

TEXT BOOK OF VETERINARY MEDICINE

VOLUME 2 (OF 5)

JAMES LAW



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VETERINARY MEDICINE.

DISEASES OF THE DIGESTIVE ORGANS.

Second in importance to pulmonary complaints in solipeds; equal in ruminants. Extent of digestive organs and character of food, in carnivora, herbivora, and omnivora. Ruminant's stomachs. Gastric fermentation. Foreign bodies. Torpor. Unwholesome fodder.

In the horse these maladies are only second in importance to those of the respiratory organs, while in ruminants they are equally frequent and important. The varying susceptibility of the digestive organs to disease in different families and the special proclivity of different parts of these organs may be, in great part, explained by the great variation in the food, by the relative extent of the gastro-intestinal surface, and by the amount of work devolving on the respective viscera.

In **carnivora** the entire gastro-intestinal surface is little more than half the area of the skin, for their rich animal food does not require a prolonged retention and an elaborate series of intricate processes to insure digestion and absorption. This system of organs is accordingly less liable to disorder in carnivora than in herbivora and omnivora. Add to this that the carnivorous stomach is very capacious relatively to the intestine, that the digestion of the great bulk of the food (nitrogenous elements) is nearly completed in this viscus, and that the contents of this organ are easily and completely discharged by vomiting whenever they prove irritating, and we have ample explanation of the comparative immunity of these animals from digestive disorders.

The **herbivora** stand at the opposite extreme, the gastro-intestinal surface being over double the area of the skin in the horse, and nearly three times that extent in the ox. The hard, fibrous and comparatively innutritious vegetable food of these animals necessitates its prolonged retention in the alimentary canal in order to the completion of digestion and the absorption of the nutritive constituents. Hence the great liability of the herbivora to diseases of the digestive organs.

Omnivora occupy a place intermediate between these two classes, as regards both the nature of the food and the extent of surface of the alimentary canal, and they are in similar ratio little liable to digestive disorders. They have besides in common with carnivora a great facility in the rejection of irritant matters by vomiting, and in thus protecting themselves against gastric and intestinal disorders.

A fair idea of the area of the intestinal surface may be given by stating the length of the canal relatively to that of the body:—in the dog : : 6 : 1, in the rabbit : : 10 : 1, in the ass and mule : : 11 : 1, in the horse : : 12 : 1, in swine : : 14 : 1, in the ox : : 20 : 1, and in the sheep : : 27 : 1. The calibre of the intestine varies however and with it the capacity. Thus in the relatively shorter intestine of the horse, the capacity is much greater in ratio with the size of the animal than is the relatively much longer intestine of the pig. The ox's intestine though twice the length of that of the horse has little more than half the capacity.

Among **herbivora** the monogastric (horse, ass, mule), and polygastric (ruminants) animals manifest varying pathological susceptibility according to the relative development of the different digestive viscera and the habitual character of their food. The horse and other large solipeds have small stomachs (16 qts.) and capacious intestines (196 qts.). Digestion is restricted in the stomach and largely carried on in the spacious bowels. The small stomach requires to be frequently replenished in moderate amount, but, if this is secured, its liability to disease is slight while that of the intestines is very considerable. In the ox the stomachs have a total capacity of 252 qts., while that of the intestines averages 103 qts. In this animal the capacious and hard working stomach is a

frequent seat of disorder, while the comparatively small intestines are to a large extent exempt. The small stomach of the horse is easily overloaded and disordered or paralyzed by an unusually full feed of grain when hungry, or one of some specially appetizing fodder, and the case is serious, as relief can rarely be obtained by vomiting. For the same reason fermentation of the gastric contents with evolution of gas and tympany usually proves fatal to the horse since relief by eructation is too often impossible. Cattle are fitted to live in damp localities where the cloven foot prevents sinking and getting bogged, and where they may draw in with the tongue a full mouthful of coarse herbage which they swallow with little mastication or admixture with saliva. This lodges in the first two stomachs, and if, from any cause, rumination is impaired, or suspended, it finds itself in conditions especially favorable to fermentation. The food too, as in the case of frosted roots, wet clover or partially ripened grain, etc., is often charged with ferments (bacteria) in a state of great vital activity, and hence the frequent tympanies of the ox. The ruminant is no less liable than the soliped to overload the stomach, and though the return of food from the first two stomachs to the mouth is a normal process, this is promptly arrested by the supervention of paresis in the overloaded and overdistended organs. This overdistension further tensely stretches and closes the lips of the œsophagean opening. The rapid swallowing of the food, with only one or two strokes of the teeth for each morsel, renders the large ruminant more liable to take in poisons, pins, nails and other injurious bodies, especially when hunger and the blunting of the sense of smell have been brought on by traveling on dusty roads. Again the large ruminants, and especially cows are wont to while away the tedious hours by chewing and unwittingly swallowing pieces of leather, cloth, bones, iron, etc. Once more the third stomach in which the food is compressed and triturated between the multiple folds, is normally comparatively dry, and is liable under dry, fibrous, heating or stimulating aliment, or in case of fever, to dry up in part or in whole, and to derange the whole process of digestion.

All herbivora are liable to disease from unwholesome fodder and the resulting affection may prove epizootic in connection with unfavorable seasons, or more local, from faulty cultivation.

The symptoms vary so much in connection with the seat and nature of the disease that it would be impolitic to attempt to generalize them.

DISEASES OF THE MOUTH.

Relative susceptibility to disease of the mouth: Food; irritants; bits; ropes; speculum; sharp metallic bodies; micro-organisms; functional; nervous.

These are met with in all domestic animals, but are above all common in horses, oxen and pigs, partly because of special susceptibilities and of the nature of the food, but largely by reason of the exposure of this part to mechanical injuries, especially in horses and cattle. Hard bits and the harder hands of cruel and ruthless drivers, nooses of rope tied over the lower jaw and tongue, iron stirrup, clevis, or balling iron used without cover to force the jaws apart, a large drenching horn employed as a lever for the same purpose, an extemporized Yankee bridle rudely applied or used in breaking a colt, the method of curing a balking or jibbing horse by tying a rope to his lower jaw and to a bar extending forward from the pole, pins, needles, thorns and other sharp bodies, and irritants in food or medicine are among the causes of such disorders. Then there are the many irritating microörganismal ferments in food, water, mucus, etc., and irritant and hot medicines and food to account for local inflammations.

FUNCTIONAL DISORDERS.

Among these are the convulsive closure of the jaws in *tetanus*, the flaccid state of the lips, cheek, and tongue in *paralysis*, and the pendent state of the lower jaw in *paralytic canine madness*. See these different subjects.

STRUCTURAL DISEASES.

INFLAMMATION OF THE LIPS, CHEILITIS.

Causes of Cheilitis: Local injuries; poisoned; envenomed; secondary disease. Symptoms: swelling; salivation; difficult prehension; cracks; blisters; ulcers; indurations. Treatment: obviate causes; astringents; antiseptics; derivatives; gravitation; for venoms antacid; antiseptic, Iodine.

Causes. Blows, pricks, wounds and bruises with bits or twitch, and other mechanical and chemical irritants, irritant vegetables, bites of leeches or snakes, stings of insects, etc. It may be a skin disease dependent on disorder of some remote organ, or a local engorgement due to a constitutional state. (See, Urticaria Surfeit, Purpura hæmorrhagica, Variola, Strangles).

Symptoms. Swelling, stiffness, heat and tenderness of the lips, with or without local abrasion, or incised or punctured wound. Food may be entirely refused from inability to take it in with the rigid tender lips, and saliva drivels from the mouth because of their imperfect apposition. Cracks, blisters and raw sores or ulcers may or may not supervene. In old standing cases the lips become indurated and comparatively immobile.

Treatment. Remove the cause whether irritants in food, or drugs, sharp pointed bodies lodged in the tissues, injuries by bit, twitch or otherwise. Local applications have comparatively little effect, being promptly removed by the tongue, yet a lotion of vinegar and honey;—of borax 10 grains and honey or glycerine 1 oz.;—or of alum in a similar medium will often prove useful. A dose of laxative medicine will favor resolution, and if there is great tumefaction, feeding thick gruels from high manger, and tying to a high rack so as to prevent drooping of the head, will favor recovery. In snake bites and stings the local application of aqua ammonia and its administration internally (horse and cow 1 oz., sheep 2 dr. in 20 times its volume of water) should be practiced; or permanganate of potash may be used.

When the heat and tenderness subside, leaving much thickening and induration it may be repeatedly painted with a lotion of one part of tincture of iodine in three parts of glycerine.

CANCROID OF THE LIPS. EPITHELIOMA.

Epithelioma: Animals susceptible; accessory causes; symptoms; lesions. Treatment: Warts and polypi. Actinomycosis: Wounds; abrasions; infection atriæ. Symptoms; treatment. Trombidiosis: infected regions; not compulsory parasite; European and American trombidia; distinct from chigoe. Symptoms. Treatment.

This has been observed in the cat and the horse, commencing at the angle of the mouth and doubtless partially determined in the latter animal by the irritation of the bit.

It is characterized by thickening of the tissues of the lips, in the form of small irregularly rounded masses, and tending to the formation of a spreading ulcer. The thickened tissues are invaded, pushed aside and infiltrated by epithelial or epithelioid cells, which, no longer confined to the surface as in the natural state, grow in the interior of the tissues and destroy them.

Treatment. The disease has little tendency to cause secondary deposits in other organs and may often be arrested by local measures. In its earliest stages it may be arrested by the thorough removal of the diseased structures with the knife, the resulting deformity being obviated by bringing the raw edges together by suture, so as to secure their adhesion, or the actual cautery may be used. The tendency to irritation from putrefaction products escaping from the mouth may be counteracted by occasional sponging with a weak lotion of carbolic acid (1 part to 50 of water) or an ointment of one part of very finely powdered boracic acid to two parts of simple ointment.

Leblanc has repeatedly succeeded in these cases by the use of chlorate of potash, locally and generally. The local application may be a solution of two drachms in four ounces of water, while the dose of the powder for the horse is 2 to 4 drachms daily.

Warts and Polypi. These are common on the outer and even the inner side of the lips, especially in dogs. They are easily removed by the scissors, after which their roots should be thoroughly cauterized with a pointed stick of lunar caustic or chloride of zinc.

ACTINOMYCOSIS OF THE LIPS.

In the rich river bottom lands of northern Germany and Russia where actinomyces abound actinomycosis is common in the form of papillæ of greater or lesser size on the lips and nose of horse and ox. The abrasion of these parts by thorns, thistles, stubble, dry fibrous fodders and other irritants, appears to produce a raw surface for the colonization of the germ, which is not slow to avail of the opportunity. The resulting lesions take the appearance of warty looking elevations, more or less indurated, which on section show the sulphur yellow actinomyces tufts of club-shaped cells converging to a central mycelial mass.

Treatment is simple as the disease is at first essentially local, and is easily checked by the local application of iodine. The wartlike elevations may be shaved off with a razor or cut off with sharp scissors and the surface painted once or twice daily with tincture of iodine. If there is suspicion of distant or deepseated actinomycosis the internal treatment with potassium iodide will be in order.

TROMBIDIOSIS OF THE NOSE AND LIPS. HARVEST ITCH.

In different parts of Europe and America, and especially in the warmer regions, or in sheltered gardens, shrubberies, and pastures, different species of the trombidium abound, and the young hexapod larvæ attack man and beast, burrowing under the cuticle and giving rise to extreme itching and persistent and irritating rubbing of the affected part. These parasites belong to the family of acari or mites, so that the condition they produce is one of acariasis or mange, only the offender is not a compulsory parasite, but appears to survive in certain soils and in the vegetation independently of animal hosts. Their parasitism is therefore accidental and non-essential to their survival.

The trombidian parasite usually found in Europe is the *Trombidium Holosericeum* or silky trombidium, so small (in its larval state) that it is just visible to the naked eye as a bright scarlet point when moving on a dark background. It was formerly called *Leptus Autumnalis* and is familiarly known as the red beast, *bete rouge*, *harvest bug*, etc. The common American species is of a dull brick red, so that it is less easily detected even on a dark background. It is familiarly known as the *jigger*, though quite distinct from the *chigoe* or burrowing flea of the West Indies.

The domestic herbivora get these parasites on the nose and lips while browsing on the pastures and contract an intolerable itching which may lead to violent rubbing, abrasions and scabby exudations. The skin becomes thickened, scabby and rigid, and as new accessions are constantly received the malady continues until cold weather sets in. The affection is not in any sense dangerous, and the attacks may be warded off by a daily application of one of the common parasitocides—decoction of tobacco, tar water, solution of creolin, naphthalin, etc. The mere seclusion of the infested animal indoors, without green food, will cure, as the larvæ pass through their parasitic stage in a few days and drop off.

GENERAL CATARRHAL STOMATITIS. BUCCAL INFLAMMATION.

Mature animals most subject: Causes in horse, mechanical, chemical, microbial irritants—alkalies, acids, caustics, hot mashes, ferments, fungi, rank grasses, excess of chlorophyll, clover, alfalfa, acrid vegetables, bacterial infection secondary, acrid insects in food; symptomatic of gastritis, pharyngitis, diseased teeth, specific fevers. Symptoms: Congestion and tumefaction of buccal mucosa, lips and salivary glands; Epithelial desquamation; foetor; salivation; froth; papules; vesicles. Prognosis. Treatment: Cool soft food; antiseptics; wet applications to skin; derivatives.

This is much more common in the adult than in suckling domestic animals. None of the domestic mammals or birds can be considered immune from it, but as its causes and manifestations differ somewhat it seems well to consider it separately in the different genera.

GENERAL CATARRHAL STOMATITIS IN SOLIPEDS.

Causes. These may be classed as mechanical, chemical, microbial and other irritants. In the horse it is often due to the reckless administration of irritant liquids as remedies. Owing to the length of the soft palate the horse can refuse to swallow any liquid as long as he chooses, and some of the worst cases of stomatitis I have seen resulted from the retention in the mouth of caustic alkaline liquids given under the name of "*weak lye*." Strong acids and caustic salts dissolved in too little water or other excipient, or suspended in liquids in which they cannot dissolve, or made into boluses which are crushed between the teeth are not infrequent conditions. Too hot mashes given to a hungry horse is another cause of this trouble. Fermented or decomposed food is often most irritating. Coachmen will sometimes induce it by attaching to the bit bags of spicy or irritant agents, to cause frothing and make the animal appear spirited.

Fungi in fodders are among the common causes. The rust of wheat (*puccinia graminis*), the caries of wheat (*tilletia caries*), the blight (*erysiphe communis*), ergot (*claviceps purpurea*), the fungus of rape (*polydesmus excitiosus*) and the moulds (*penicillium* and *puccinia*) have all been noticed to coincide with stomatitis, and charged with producing it. On the other hand, at given times, one or other of these cryptogams has been present extensively in the fodder without any visible resultant stomatitis. The apparent paradox may be explained by the fact that these fungi vary greatly in the irritant or harmless nature of their products according to the conditions under which they have grown, and the stage of their development at which they were secured and preserved. Ergot notoriously differs in strength in different years, on different soils, under various degrees of sunshine, shade, cloud, fog, etc. In different States in the Mississippi valley it is not uncommon to find stomatitis in horses in winter, fed on ergoted hay, while cattle devouring the same fodder have dry gangrene of feet, tail and ears. Yet in other seasons the ergot fails to produce these lesions. Rank grown, watery vegetation, especially if it contains an excess of chlorophyll is liable to cause stomatitis. Red and white clover, trefoil, hybrid and purple clover, and alfalfa have all acted more or less in this way, though in many cases, the food has become musty or attacked by bacterial ferments. Some of the strongly aromatic plants, and those containing acrid principles (*cicuta virosa*, *œnanthe crocata*, mustard, etc.) cause buccal inflammation and salivation.

The irritation in many such cases is not due to one agent only, the vegetable or other irritant may be the starting point, acting but as a temporary irritant, the action of which is supplemented and aggravated by the subsequent attacks of bacterial ferments on the inflamed, weakened or abraded tissues. The bacteria present in the mouth, food or water would have had no effect whatever upon the healthy mucosa, while they make serious inroads on the diseased. On the other hand the vegetable, mechanical or chemical irritant would have had but a transient effect, but for the supplementary action of the bacteria.

In horses that have the bad habit of retaining masses of half masticated food in the cheeks the growth of cryptogams is greatly enhanced and such food often becomes violently irritating.

Among other mechanical causes may be named pointed or barbed hairs or spines (barley awns, spikes, thorns, etc.) which, lodging in a gland orifice, or in a wound of the gum or mucosa, form a source of irritation or a centre for bacterial growth and abscess.

Again, irritants of animal origin must be named. These are not taken by choice, but when lodged in fodder, or in the pastures they are taken in inadvertently with the food. In this way poisonous insects, and especially hairy caterpillars, cantharides, potato bugs, etc., gain access to the mouth.

It must not be overlooked that stomatitis occurs as an extension, sympathetic affection or sequel of diseases of other organs. Gastritis is usually attended by redness and congestive tenderness of the tongue, especially of the tip and margins, and other parts of the buccal mucosa, notably the palate just back of the incisors, are often involved. In other cases it appears as a complication of pharyngitis, laryngitis, of affections of the lower air passages, of the teeth and periodontal membrane or of the salivary glands.

It appears also in a specific form in certain fevers, as in horsepox, pustulous stomatitis, aphthous fever and even in strangles. Mercurial stomatitis, rarely seen at the present time, is one of the worst forms of the disease, and like the infectious forms will be treated separately.

Lesions and Symptoms. At the outset and in the slighter forms of congestion there is merely heat and dryness of the buccal mucosa. Redness may show on the thinner and more delicate portions of the membrane, as under the tongue, on the frænum, and on the sublingual crest. But elsewhere it is hidden by the thickness of the epithelium, and the manifestations are merely those of suppressed secretion with local hyperthermia.

As the congestion is increased there is seen, even at this early stage, a slight thickening or tumefaction of the mucosa, especially on the gums, lips, the sublingual area, the orifices of the salivary glands, and the palate back of the upper incisors. On the dorsum of the tongue, the cheeks and lips, generally the lack of loose connective tissue tends to prevent the swelling.

With the advance of the inflammation the redness of the mucosa extends, at first in points and circumscribed patches, and later over the entire surface. The epithelium drying and degenerating in its surface layers forms with the mucus a sticky gummy film on the surface, which, mingling with decomposing alimentary matters gives out a heavy, offensive or even fœtid odor.

The different parts of the mouth are now tender to the touch, and this, with the fœtor and even bitterness of the bacterial products combine with the general systemic disturbance in impairing or abolishing appetite. In any case mastication becomes slow and infrequent, and morsels of food are the more likely to be retained, to aggravate the local condition by their decomposition.

The dry stage is followed by the period of hypersecretion, and in this the salivary glands take a prominent part, so that ptyalism (slobbering) becomes the most marked feature of the disease. The saliva mixed with the increasing secretion of mucus and the abundance of proliferating and shedding epithelium, escapes from the lips and falls in stringy masses in the manger and front of the stall. When there is much motion of the jaws and tongue it accumulates as a froth around the lips.

A careful examination of the mucosa will sometimes detect slight conical elevations with red areolæ, representing the tumefied orifices of the obstructed mucous follicles, and later these may show as minute erosions. Even vesicles have been noticed (Weber, Dieckerhoff, Kusters), but when these are present one should carefully exclude the specific stomatitis such as horsepox, contagious pustular stomatitis, aphthous fever, etc.

Erosions of the mucosa and desquamation of the epithelium have been noticed in horses fed on purple (hybrid) clover, buckwheat or ergot, and in some of these cases the inflammation has extended (in white faces especially) to the skin of the face, the mucosa of the nose, and the adjacent glands, and as complications icterus, constipation, colics, polyuria, albuminuria and paresis of the hind limbs have been observed. These latter are common symptoms of cryptogamic poisoning.

Prognosis. In uncomplicated cases the disease is not a grave one, lasting only during the continued application of the local irritant, and recovering more or less speedily when that has been removed. Complications are dangerous only when due to some specific disease poison (glanders, actinomycosis, strangles, etc.), and even poisoning by the usual cryptogams of leafy or musty plants is rarely persistent in its effects.

Treatment. This resolves itself into the removal of the irritant cause and the soothing of the irritation. When the cause has been definitely ascertained the first step is easy.

In the direction of soothing treatment, a careful selection of diet stands first. Fibrous hay and even hard oats, barley or corn may have to be withheld, and green food, or better still, bran mashes, gruels, pulped roots or fruits allowed. Scalded hay or oats, ensilage, sliced roots, or ground feed may often be taken readily when the same aliments in their natural condition would be rejected or eaten sparingly.

Medicinal treatment may often be given in the drinking water which should always be allowed in abundance, pure and clean. In the way of medication chlorate of potash, not to exceed one-half to one ounce per day according to the size of the animal, may be added, together with an antiseptic (carbolic acid, borax, permanganate of potash, common salt, naphthol, creolin, hyposulphite of

soda). In case of severe swelling, a cap made to fit the head with strips wet in alum and vinegar or other astringent solution maintained against the intermaxillary space may be desirable. Support for the tongue may be necessary as mentioned under glossitis.

In case of complications on the side of the bowels, liver or kidneys, laxatives, diuretics and antiseptic agents may be called for.

GENERAL CATARRHAL STOMATITIS IN CATTLE.

Dense resistant mucosa protective: Affection usually circumscribed. Action of violent irritants, and toxins of specific fevers. Mechanical irritants. Symptoms: Salivation; congestion; eruptions; erosions; ergot; acrid vegetables; caustics. Treatment: Astringents; antiseptics; refrigerants; derivatives; tonics. Removal of foreign bodies. Lesions and symptoms in sheep.

The mouth of the ox as Cadeae well says has a cuticular epithelium too thick and resistant to be easily attacked by microbes. It follows that infected inflammations are far more frequently circumscribed than in the thinner and softer buccal mucosa of the horse. The more general buccal inflammations come more particularly from the use of food that is too hot or that contains strongly irritant agents. The thickness of the buccal epithelium however, is no barrier to the local action of poisons operating from within as in rinderpest, or aphthous fever, or in malignant catarrh, nor is it an insuperable barrier to the local planting of the germs of cow pox, anthrax, actinomycosis, or cryptogamic aphtha (muguet). The wounds inflicted by fibrous food make infection atria for such germs, hence the great liability to such local inflammations, in winter when the animals are on dry feeding. For the same reason, perhaps, the prominent portions of the buccal mucosa,—the papillæ—are sometimes irritated themselves while serving as protectors for the general mucous surface, and hence they become specially involved in inflammation, which constituted the “*barbs*” of the old farriers. Utz records a buccal inflammation occurring in herds fed on green trefoil, first cutting, showing that even in cattle this agent may determine a general stomatitis.

Symptoms. These do not differ from those of the horse, and resemble, though often in a milder form, the buccal manifestations of aphthous fever. There is the difficulty of mastication and indisposition to take in fibrous aliment, the drivelling of saliva from the mouth, or its accumulation in froth around the lips, the frequent movement of the tongue and jaws, and the congestive redness, papular eruption, vesication, or even erosion of the affected mucous membrane. It is always necessary to guard against confounding the simple stomatitis, and the slighter infected inflammations, from the more violent infections above referred to. The special diagnostic symptoms must be found under the respective headings. The aphthous fever is not to be expected in American herds, but the stomatitis which is associated with ergot in the food is met more particularly in winter and spring, and must not be confounded with the specific disease, on the one hand nor with the simpler forms of buccal inflammation on the other. In the case of ergoted fodder the signs of ergotism in other situations will be found, in the affected animals, such for example as necrotic sloughs and sores around the top of the hoof, sloughing of the hoof or of one or more digits, or of the metatarsus, of the tip of the tail or ear; abortions, convulsions, delirium, lethargy or paralysis. If not seen in the same animals some of these forms may be observed in other members of the herd. Then the buccal lesions are in themselves characteristic: soft, whitish, raised patches of the epithelium (rarely blisters) are followed by desquamation and exposure of the red, vascular surface beneath, and this tends to persist if the ergoted fodder is persisted in.

Treatment. Simple stomatitis of the ox generally tends to spontaneous and early recovery. The simplest astringent and antiseptic treatment is usually sufficient to bring about a healthy action. Borax given in the drinking water, not to exceed four ounces per day, or the same amount mixed with syrup or honey and smeared occasionally on the tongue, or hyposulphite or sulphite of soda, or weak solutions of carbolic acid will usually suffice, after the irritant cause has been removed. Vinegar, or highly diluted mineral acids may be used but are somewhat hurtful to the teeth. Decoctions of blackberry bark or solutions of other vegetable astringents may be used as alternatives. When there is evidence of irritant matters in the stomach or bowels, a saline laxative will be advisable to be followed by vegetable bitters or other tonics. Thorns and other foreign bodies imbedded in the tongue or other part of the mouth must be discovered and removed.

CATARRHAL STOMATITIS IN SHEEP.

The more delicate buccal mucosa in these animals would render them more subject to inflammations, but this is more than counterbalanced by the mode of prehension of aliments, not by the tongue, but by the delicately sensitive lips, and further by the daintiness and care with which these animals select their food. The *treatment* would not differ materially from that prescribed for the ox.

GENERAL CATARRHAL STOMATITIS IN DOGS.

Causes: burns; spiced food; bones; sepsis; ferments; pin caterpillar; dental and gastric troubles. Symptoms: careful prehension and mastication; congestion; swelling; eruption; erosion; furred tongue; stringy salivation; foetor; swelling of lips, cheeks, intermaxillary space, and pharynx. Treatment: demulcent foods; antiseptics; derivatives; tonics; care of teeth and gums.

Causes. Hot food is a common cause in hungry dogs. Spiced food in house dogs fed scraps from the table tend to congestion of mouth and stomach alike. Irritation through wounds with bones, especially in old dogs with failing teeth, and in exceptional cases the impaction of a bone between the right and left upper molars are additional causes. Putrid meat must also be recognized as a factor, the septic microbes seizing upon the wounds and spreading from this as an infecting centre. Lactic acid and other irritant products developed through fermentation of particles of food retained about the gums and cheeks soften the epithelium and irritate the sub-epithelial tissue, causing congestion. Megnin draws attention to the fact that the pin caterpillar (*bombyx pinivora*) found on the stalks of couch grass (*Triticum repens*) produces buccal irritation when chewed and swallowed to induce vomiting. As in other animals more or less buccal congestion attends on gastric congestion and inflammation. Dental troubles are often sufficient causes.

Symptoms. The animal becomes dainty with regard to his food, picking up the smaller or softer pieces and rejecting the larger or harder. Mastication is painful and selection is made of moist or soft articles which can be swallowed without chewing or insalivation. The mouth is red and hot, and at times the mucous membrane eroded, or blistered, the lesions concentrating especially on the gums and around the borders of the tongue. The dorsum of the tongue is furred, whitish, yellowish or brownish. Saliva collects in the mouth and escapes in filmy strings from its commissures, and the odor of the mouth becomes increasingly foul. Swelling of the lips, cheeks or intermaxillary space marks the worst cases.

Treatment. Withdraw all irritant and offensive aliments. Give soups, mushes, scraped or pounded lean meat in small quantities, washing out the mouth after each meal with a 20 per cent. solution of permanganate of potash or borax or a two per cent. solution of carbolic acid. Cadeac advises against chlorate of potash on account of its known tendency to bring about hæmoglobinæmia in dogs. A laxative and bitters may be called for in case of gastritis or indigestion, and any morbid condition of the teeth must be attended to. Decayed teeth may be removed. Tartar especially must be cleaned off by the aid of a small wooden or even a steel spud and a hard brush with chalk will be useful. A weak solution of hydrochloric acid is usually employed to loosen the tartar, but this is injurious to the structure of the teeth and had best be avoided if possible. Tincture of myrrh is especially valuable both as a gum-tonic and as a deodorant and antiseptic. This may be rubbed on the irritated gums as often as the mouth is washed.

GENERAL CATARRHAL STOMATITIS IN SWINE.

Causes: Irritants; ferments; noose on jaw; specific poisons. Symptoms: Careful feeding; thirst; frothy lips; champ jaws; redness; swelling; foetor. Treatment: Cooling, astringent, antiseptic lotions; mushy food; derivative; tonics.

Causes. Swine suffer from simple stomatitis when exposed to thermal, mechanical or chemical irritants. Food that is too hot, or that which is hard and fibrous, or that which contains spikes and awns, capable of entering and irritating gland ducts or sores, or food which is fermented or putrid, food or medicine of an irritant character. The habit of catching and holding swine with a running noose over the upper jaw, and the forcing of the jaws apart with a piece of wood in search of the cysticercus cellulosa are further causes. In several specific infectious diseases inflammation of the mucous membrane with eruption or erosion is not uncommon. Thus aphthous fever is marked by vesicular eruption, muguet by epithelial proliferation and desquamation, hog cholera and swine plague by circumscribed spots of necrosis and erosion. Patches of false membrane are not unknown, and local anthrax, tubercle and actinomycosis are to be met with. Inflammation may start from decaying teeth.

Symptoms are like as in other animals, refusal of food, or a disposition to eat sparingly, to select soft or liquid aliments, to swallow hard materials half chewed or to drop them, to champ the jaws, and to seek cold water. Accumulation of froth around the lips is often seen, and the mouth is red, angry, dry, and hot, and exhales a bad odor.

Treatment does not differ materially from that adopted in other animals. Cooling, astringent, antiseptic lotions, honey and vinegar, and in case of spongy or eroded mucosa, tincture of myrrh daily or oftener. Soft feeding, gruels, pulped roots, or well kept ensilage may be used, and clean, cool water should be constantly within reach. In case of overloaded stomach or indigestion a laxative followed by bitter tonics will be in order.

CATARRHAL STOMATITIS OF BIRDS. PIP.

Causes: hurried breathing; local irritants; exposure; filthy roost. Symptoms: gaping; ropy cry; epithelial pellicle on tongue, larynx. or angle of the bill. Treatment: pick off pellicle; smear it often with glycerized antiseptic. Remove accessory and exciting causes.

This form of inflammation of the tongue of birds is characterized by the increased production and desiccation of the epithelium so that it takes on a horny appearance. According to Cadeac it may accompany various inflammatory affections of the air passages, which cause hurried breathing with persistently open bill, and thus entail evaporation of the moisture. More commonly it has its primary cause in local inflammation of the surface in connection with damp, cold, draughty hen-roosts, and above all, the accumulation of decomposing manure and the exhalation of impure gas. Even in such cases the abnormal breathing with the bill open is an accessory cause of the affection.

Symptoms. The breathing with open bill should lead to examination of the tongue, but above all if at intervals the bird with a sudden jerk of the head emits a loud shrill, raucous sound, which reminds one of the cough of croup. The tip and sides of the tongue are found to be the seat of a hard, dry, and closely adherent epithelial pellicle, which suggests a false membrane.

Treatment. The common recourse is to pick or scrape off the indurated epithelial mass, leaving a raw, bleeding surface exposed. This is then treated with a solution of borax, or chlorate of potash. Cadeac deprecates this treatment as useless and dangerous, and advises the disintegration of the dry epithelial mass with a needle taking care not to prick nor scratch the subjacent sensitive tissue, and to wash with a 5 per cent. solution of chlorate of potash. A still more humane and effective method is to make a solution of hyposulphite of soda in glycerine and brush over the affected surface at frequent intervals. This may be conveniently applied through the drinking water.

In case of implication of the lower air passages or lungs, the treatment must be directed to them, and soft, warm, sloppy food and the inhalation of water vapor will prove of great advantage. Secure clean, sweet, dry pens, pure air, and sunshine. (See pseudo membranous enteritis.)

LOCAL STOMATITIS.

Division of circumscribed buccal inflammations: palatitis; gnathitis gingivitis; glossitis. Causes: injuries; acrid; venomous or caustic agents; diseased teeth; foreign bodies in gland ducts; malformed jaws; infections, etc. Symptoms: salivation; difficult prehension and mastication; dropping half masticated morsels; distinctive indications of different caustics; abrasion; abscess; slough; infective disease lesions. Treatment: for *palatitis*, massage by hard corn ears, scarification, laxatives; for *gnathitis*, care for teeth and ducts, astringent washes, eliminate mercury; for *glossitis*, remove cause, use antidote to venom, or to chemical irritant, astringent, antiseptic lotions or electuaries, evacuate abscess, soft, cool diet, elevate the head, suspend the tongue.

Localized inflammations in the buccal cavity are named according to the portion of the lining membrane attacked;—*palatitis* if seated in the roof of the mouth; *gnathitis* if restricted to the cheeks; *gingivitis* if to the gums; and *glossitis* if to the tongue.

Palatitis. Lampas. Congestion of the hard palate behind the upper front teeth. This is usually seen in young horses during the period of shedding the teeth and is caused by the irritation and vascularity consequent on teething. The red and tender membrane projects beyond the level of the wearing surfaces of the upper incisors, and may materially interfere with the taking in of food. A common practice in such cases is to feed unshelled Indian corn, the nibbling of which seems to improve the circulation in the periodontal membrane and by sympathy in the adjacent palate. Superficial incisions with the lancet or knife will usually relieve, and may be followed by mild astringent lotions if necessary. If apparently associated with costiveness or gastric or intestinal irritation a dose of physic will be demanded. Nothing can excuse the inhuman and useless practice of burning the parts with a hot iron.

Gnathitis. Inflammation of the Cheeks. Usually resulting as a distinct affection from irregular or overgrown teeth, or the entrance of vegetable spikes into the gland ducts, these cause local swelling and tenderness, slow imperfect mastication, dropping of food half chewed, accumulation of food between the cheeks and teeth, thickening, induration and sloughing of the mucous membrane with excessive foetor.

Treatment. Consists in correcting the state of the teeth and ducts and using one of the washes recommended for glossitis.

Gingivitis. Inflammation of the gums. This is either connected with the eruption of the teeth in young animals and to be corrected by lancing the swollen gums and giving attention to the diet and bowels; or it is due to scissor-teeth or to the wear of the teeth down to the gums in old horses; or it is dependent on diseased teeth, or mercurial poisoning, under which subjects it will be more conveniently considered. Barley awns or other irritants must be extracted.

Glossitis, Inflammation of the Tongue. *Causes.* Mostly the result of violence with bits, ropes, etc., with the teeth, or with the hand in giving medicine; of scalding food, of acrid plants in the food: of irritant drugs (ammonia, turpentine, croton, lye, etc.), or of sharp, pointed bodies (needles, pins, thorns, barley and other barbs, etc.) which perforate the organ. In exceptional cases leech and snake bites are met with especially in cattle, owing to the tongue being exposed when taking in food. Local infections and those of the specific forms, determine and maintain glossitis.

Symptoms: Free flow of saliva, difficulty in taking in food or drinking, and red, swollen, tender state of the tongue, which in bad cases hangs from between the lips. The mucous membrane may be white, (from muriatic acid, alkalies, etc.), black, (from oil of vitriol, lunar caustic, etc.), yellow, (from nitric acid, etc.), or of other colors according to the nature of the irritant. It may be raised in blisters, may present red, angry sores where the epithelium has dropped off; may become firm and indurated from excessive exudation; may swell and fluctuate at a given point from the formation of an abscess; or may become gangrenous in part and drop off. Breathing is difficult and noisy from pressure on the soft palate. There is usually little fever and death is rare unless there is general septic infection.

Treatment will depend on the cause of injury. In all cases seek for foreign bodies imbedded in the

organ and remove them. If snake bites are observed use ammonia or potassium permanganate locally and generally, or cholesterin as a local application. If the irritation has resulted from mineral acids, wash out with calcined magnesia lime water, or bicarbonate of soda or potash. If from alkalies (lye) use weak vinegar. If from caustic salts employ white of egg, vegetable-gluten, boiled linseed, slippery elm, or other compound of albumen or sheathing agent. In ordinary cases use cold astringent lotions, such as vinegar and water; vinegar and honey; borax, boric or carbolic acid, chlorate of potash, alum or tannin and honey. Poultices applied around the throat and beneath the lower jaw are often of great value. The bowels may be relieved if necessary by injections, as it is usually difficult to give anything by the mouth. If ulcers form touch them daily with a stick of lunar caustic or with a fine brush dipped in a solution of ten grains of that agent in an ounce of distilled water. For sloughs use a lotion of permanganate of potash, one drachm to one pint of water, or one of carbolic acid, one part to fifty of water. If an abscess forms give a free exit to the pus with the lancet, and afterward support the system by soft nourishing diet, and use disinfectants locally. As in all cases of stomatitis, the food must be cold gruels or mashes, or finely sliced roots will often be relished.

The mechanical expedient of supporting the tongue in a bag is essential in all bad cases, as if allowed to hang pendulous from the mouth inflammation and swelling are dangerously aggravated.

APHTHOUS STOMATITIS. FOLLICULAR STOMATITIS.

Causes: in horse, ox, dog; rough, fibrous food, blistering ointments, bacteria. Symptoms: general stomatitis, and special; papules with grayish centres and red areolæ, vesiculation, ulceration. Treatment: Astringent, antiseptic, derivative, tonic, stimulant.

This is a rare affection in ruminants where the thickness of the epithelial covering appears to be a barrier to infection or injury, while it is common in the more delicate and sensitive buccal mucosa of the horse and dog. In the horse the ingestion of irritant plants with the food and the penetration of vegetable barbs into the mucous follicles may be charged with causing the disease, while in both horse and dog the licking of blistering ointments and the local action of fungi and bacteria are factors in different cases.

Symptoms. With the ordinary symptoms of stomatitis, there appear minute firm, whitish, circular elevations representing the openings of the inflamed mucous or salivary follicles, having a reddish areola, and grayish white vesicular centre. They may amount to a line or more in diameter, and on bursting leave red cores or ulcers. The whole mouth may be affected or the disease may be confined to the lips, gums or tongue.

Treatment. Beside the general astringent washes, this affection is greatly benefited by the local use of antiseptics, as sulphite or hyposulphite of soda, 2 drachms in a quart of water. Borax, permanganate of potash, carbolic acid or other antiseptic in suitable solution may be substituted. Saline laxatives are often useful to remove sources of irritation in stomach and intestines, and iron salts (chloride or nitrate) in full and frequently repeated doses may be given internally. Ulcers may be cauterized and soft food and pure water given from an elevated manger.

ULCERATIVE STOMATITIS. GANGRENOUS STOMATITIS.

Causes: specific disease poisons; debility; rachitis; cancer; chronic suppuration; irritation—mechanical, chemical, thermic, venomous, etc. Symptoms: difficult, imperfect prehension and mastication, salivation, bleeding, swollen, puffy epithelium, blisters, extending erosions, deep or spreading. Duration. Treatment: correct constitutional fault, tonics, soft, digestible food, antiseptics, mild caustics.

This is characterized by the formation of necrotic spots and patches of the buccal epithelium, with desquamation, and the formation of more or less rodent ulcers of the sub-epithelial mucosa. Like other ulcerative processes it is usually due to microbic invasion, and in this way it may supervene on other and simpler forms of stomatitis. It also varies in its manifestations and nature according to the genus of animal, and the specific microbe present.

ULCERATIVE STOMATITIS IN SOLIPEDS.

Causes. Apart from the ulcerations and erosions of specific diseases (glanders, horsepox, pustulous stomatitis, aphthous fever, etc.,) this condition is especially liable to appear in anæmic and debilitated subjects (Cauvet), as in rachitis (Friedberger and Fröhner), cancer (Cadeac) chronic internal abscess (Cadeac), etc. As an exciting cause and as a means of furnishing an infection atrium for the microbes of ulceration all conditions of simple lesion of the mucous membrane—mechanical, chemical, thermic, venomous, etc., are operative. Dieckerhoff has described it in connection with diphtheritic rhinitis, Friedberger with a nasal and conjunctival catarrh, Zeilinger and Kohler with aphthous fever, Mobius and Hackbarth with trefoil poisoning.

Lesions and Symptoms. There is the usual dainty feeding and disposition to masticate imperfectly or even to drop the partly insalivated morsels, working of the lips, the formation of froth on their margins, and the drivelling of saliva in long strings or filaments. As the disease advances this becomes bloody and foetid. The local lesions may be at first like white pulpy spots of softened and degenerating epithelium, which is exceptionally, raised in blisters. This is followed by desquamation and the formation of open sores which are indolent, and show a disposition to further erosion and extension. They may be rounded or irregularly indented in their borders, and contain a brownish, blackish or greenish viscid debris. They vary widely, however, in general appearance and in their disposition to speedy or sluggish healing, being apparently influenced by the nature of the pathogenic microbe and the susceptibility of the subject. In some cases the molecular degeneration extends deeply into the mucosa, and even over the edges of the lips into the adjacent skin. Recovery and complete cicatrization may take place in one week, or successive outbreaks may take place in the same animal lasting in all for months as in Cadeac's case associated with chronic abscess of the mesentery.

Treatment. The first consideration is to correct the debility on which the affection is based. Iron and bitter tonics, mineral acids, and nourishing food given in the form of soft mashes, pulped roots, or farinas, which will require little mastication, and the antiseptic cleansing of the mouth after each meal are the main features of the treatment. As antiseptics, vinegar is inimical to the microbes of the mouth, which affect alkaline media, borax, boric acid, carbolic acid, sulphurous acid, the sulphites and hyposulphites, permanganate of potash, chlorate of potash, creolin, and sulphate or chloride of iron furnish a sufficient choice of comparatively nontoxic agents. Ulcers may be touched with tincture of iodine, lunar caustic, or sulphate of copper.

ULCERATIVE STOMATITIS (DIPHTHERIA) IN CALVES.

Accessory causes. Infection. Experimental inoculation. Bacillus, grows on blood serum. Lesions in mouth, nose, air passages, intestines, digits. Symptoms: difficult sucking, fever, swollen, whitish spots on buccal mucosa, phagadenic sores, foetor, symptoms of extending disease, anorexia, debility, prostration. Duration. Diagnosis from foot and mouth disease, from actinomycosis, from tuberculosis. Prevention: cleanliness, antiseptics, segregation, diet of dam, sterilized milk. Treatment: antiseptic and eliminating: locally antiseptic.

This has been observed at frequent intervals in calves, as a serious, fatal, communicable disorder occurring in the first few weeks of life.

Causes. It has been attributed to unhygienic conditions of the dams, close, damp, impure stables, unwholesome or spoiled food, and privations of various kinds, and these, in all probability, increase the susceptibility. The congestion and traumatism connected with the cutting of the teeth is another predisposing cause. The ultimate cause is, however, the contagious element and the disease has been conveyed to healthy lambs by the introduction into their mouths of the necrotic products from the diseased subjects (Dammann). Sheep inoculated in the conjunctiva presented violent conjunctivitis in forty-eight hours. Inoculated rabbits died of septicæmia. Mice showed the same symptoms as calves, while guinea pigs showed an abscess only at the seat of inoculation (Löffler).

The identity of the germ has not been fully demonstrated. Dammann found a micrococcus, but testimony from the inoculation of its pure cultures is wanting, and the buccal mucosa of the sucking calf is full of varied germs some of which are irritating and pathogenic to an injured mucosa.

Löffler found in the epithelial concretions (false membranes) of the mouth and intestines, a bacillus of half the thickness of the bacillus of malignant œdema, five times as long as broad and usually connected with its fellows to form filaments. He failed to obtain cultures of this in nutrient gelatine, but grew it successfully in blood serum from a calf. Transferred to fresh serum the culture failed. The pure culture does not seem to have been tried on the calf.

According to Dammann the lesions occur indiscriminately in the mouth, the nose, the larynx, trachea, lungs, the intestinal canal and the interdigital space.

It has been suggested that the mouth of the calf rendered susceptible by the congestion caused by suction, is infected by licking the previously infected umbilicus.

Symptoms. There are the usual symptoms of indisposition to suck, salivation, redness of the buccal mucosa, and general indisposition. In two or three days the mucosa shows raised, pulpy, white or grayish patches about a line in diameter. These gradually soften and break down and in four or five days leave dark red angry sores one-sixth to one-third inch in diameter dotted with grayish points and surrounded by a congested areola. These exhale an offensive odor and tend to extend in superficial area and in depth, invading indiscriminately the various subjacent tissues. The lips may be perforated, the muscles, cartilages, periosteum, and periodontal membrane invaded, the teeth may be shed, and the alveoli filled with the offensive debris of ulceration. Swelling of the throat may follow from implication of the pharynx and its lymph glands, symptoms of laryngitis, bronchitis, and pneumonia may succeed, also infective gastritis and enteritis. These various parts may be infected by the direct transference of the infecting saliva, but the germ is also held to be transmitted through the blood to implicate distant organs.

Appetite is gradually lost, a blackish, foetid diarrhœa, sets in and the calf is sunk in a profound prostration and debility due partly to the enforced abstinence and colliquative diarrhœa, but much more to the absorption of toxic matters. Death may ensue from the sixth to the twelfth day. In case of recovery a month may be requisite for the completion of convalescence.

Diagnosis. This has to be distinguished especially from aphthous fever by the absence of the large, and clearly defined vesicles of that disease, by the fact that the mammary region and interdigital spaces usually escape, and especially by the immunity of the dam and of other more mature animals. From actinomycosis of the tongue it is diagnosed by its more rapid progress, by the marked constitutional depression, and prostration, and by the absence of the marked induration of the actinomycotic organ (holzzunge) and by the sulphur yellow pin head-like nodules of actinomyces. Tuberculosis is rare in the first weeks after birth in calves, and never makes the

rapid progress nor causes the profound depression of this disorder.

Prevention. The first object must be to destroy the infection, and the second to obviate the susceptibility of the young animal. The clearing away of all accumulations of litter, filth, and even fodder from the stable proper, including the stalls where the dams lie, should be followed by a thorough whitewashing or disinfection, with sulphate of copper or of iron, or even mercuric chloride, (1:500.) If the disease has already appeared in a stable the calves should be penned singly to avoid the possibility of infection through sucking each others navels. In all cases an antiseptic (tannin, carbolic acid) should be applied to the navel of the new born. The food of the dam and nurse should be nutritive and free from any suspicion of mustiness or decomposition, and when possible the calf should be allowed to draw its own milk from the teat. When this cannot be allowed, artificial feeding should be surrounded by all the safeguards, named under acute indigestion of calves.

Treatment. Cadeac strongly recommends $\frac{1}{2}$ oz. common salt daily with the food, or alcohol $\frac{3}{4}$ oz., or a strong infusion of coffee mixed with the milk. Lenglen advises quinoa in the form of tincture, $\frac{1}{2}$ to 1 oz. McGillivray sulphate of soda. Tincture of chloride of iron 30 drops in an ounce of water with each meal would be an excellent resort.

Locally antiseptics are our main reliance. Naphthol, naphthalin, salicylic acid, or salicylate of soda, may be applied directly to the diseased mucous membrane. Tincture of chloride of iron in water (1:2) is one of the best agents (James). Carbolic acid 1 drachm in 6 oz. water, and 1 oz. alcohol has been used safely and with excellent results (Lenglen). Like most other antiseptics, however, this latter must be used with caution as regards the amount. No actively poisonous antiseptic is admissible. The antiseptic should be swabbed over the whole interior of the mouth after each meal.

In case of deep gangrenous masses excision and antiseptics are demanded.

ULCERATIVE STOMATITIS IN LAMBS AND KIDS.

Causes: Accessory, locality, youth, debility, unsuitable food, impure air, parasitism, contagion. Bacteria. Symptoms: difficult sucking, frothing, salivation, buccal redness and swelling, white, softened patches, suppuration, granulation, foetor, emaciation, debility, bowel symptoms, respiratory. Duration. Treatment: Artificial feeding, antisepsis, disinfection, mild caustics, etc.

Causes. This has been noticed as an enzootic affection in young and debilitated animals, while the mature and more robust ones escape. Anæmic lambs, those that are fed on watery, innutritious materials (potatoes, grains, waste of sugar factories), those kept in close confinement, indoors, and those that suffer from distomatosis show the disease. Impure air, damp, dark places and impure water have their influence. The disease is manifestly contagious, but the infecting microbe has not been demonstrated. It was formerly supposed to be the *oidium albicans*, the fungus of *muguet*, but Neumann demonstrated its absence, and though he found *leptothrix buccalis*, *bacilli*, *spirochæte* and *micrococci* he failed to show that any one of these in pure culture would cause the disease. Rivolta charged it on *bacterium subtile agnorum* and Berdt on the *polydesmus exitiosus* which according to him the sheep contract from eating rape cake. The withdrawal of the cake led to a rapid recovery.

Symptoms. The disease may begin insidiously without at first very marked symptoms. Sucking is painful and infrequent, an acid froth collects about the mouth, and white patches appear on the gums or other part of the buccal mucosa, with at times redness and swelling, and the separation of the gums from the teeth. The white epithelial patches soften and are easily detached, leaving bright red patches, which bleed easily, and tend to extension and coalescence. These are covered by a viscid mucopurulent matter, and may become the seat of granulations, or they may involve the subjacent tissues in ulceration causing evulsion of the teeth, or necrosis of the jaw bone. The odor of the mouth is foetid. Prostration and emaciation set in, and often bear a ratio to the extension of the disease to the digestive and respiratory organs. This is manifested by uneasy movements of the hind feet, shaking of the tail, frequent lying down and rising, constipation or diarrhœa: or by cough, snuffling breathing, swelling of the submaxillary and pharyngeal glands, and hurried, oppressed breathing. The complication of vesicular and pustular eruption has been noticed. Death may occur in eight or ten days, or more commonly recovery ensues.

Treatment must proceed on the same lines as in the calf. Artificial feeding on gruels, with antiseptic washes for the mouth at each meal are indicated. Chlorate of potash, chloride of lime, borax, sulphites and hyposulphites of soda, carbolic acid, and the salts of iron afford an ample field for selection. For ulcers, a pointed stick of nitrate of silver, or a solution of muriatic acid in three times its volume of water, applied by means of a glass rod or pledget of cotton will serve a good purpose.

ULCERATIVE STOMATITIS IN SWINE.

Causes: improper food; filthy pens; debility; toxins of specific diseases; microbial infection. Symptoms: inappetence; grinding teeth; champing jaws; salivation; foetor; buccal swelling and redness; pulpy spots; desquamation; ulcers; pharyngeal, enteric and osseous complications. Treatment: Segregation; disinfection; local antiseptic washes; tonics.

This is the Scorbutus of Friedberger and Fröhner, the gloss-anthrax of Benion.

Causes. It has been attributed to insufficient or irritant food, to damp, close pens, and to chronic debilitating diseases and all these act as predisposing causes. In gastritis and in infectious fevers like hog cholera, swine plague, and rouget (hog erysipelas) the spots of congestion and petechiæ on the buccal mucous membrane may become the starting points for ulcerative inflammations. These conditions appear, however, to be supplemented by infection from bacteria present in the mouth or introduced in food and water, and as in the case of other domestic animals the most successful treatment partakes largely of disinfectant applications.

Symptoms. Loss of appetite, grinding of the teeth, champing of the jaws, the formation of froth round the lips, foetor of the breath, redness of the gums and tongue, and the formation of vesicles or white patches which fall off leaving red angry sores. These may extend forming deep unhealthy ulcers, with increasing salivation and foetor. As the disease advances the initial dullness and prostration become more profound, and debility and emaciation advance rapidly. Unless there is early improvement an infective pharyngitis, or enteritis sets in, manifestly determined by the swallowing of virulent matters from the mouth, and swelling, redness and tenderness of the throat, or colics and offensive black diarrhoea hasten a fatal issue. Rachitis may be a prominent complication, as it seems in some instances to be a predisposing cause.

Treatment. Isolate the healthy from the diseased and apply disinfection to all exposed articles and places. Employ local antiseptics as on the other animals. Sulphuric or hydrochloric acids in 50 times their volume of water, or tincture of iron, chlorate of potash, or chloride of ammonia, or borax have been used successfully. Bitters and aromatics have also been strongly recommended.

ULCERATIVE STOMATITIS IN CARNIVORA.

Causes: dietary causes; constitutional debilitating diseases; dental disorders; microbial infection; microbes. Symptoms: difficult sucking or mastication; salivation; dullness; prostration; mucosa red with gray patches, erosions, and ulcers; foetor; loose teeth; excess of tartar. Extensions to face, throat, lymphatics, nose, eyes, stomach, liver, bowels. Duration. Treatment: clean teeth; antiseptics; mild caustics; stimulants.

Causes. This affection is more common in this class of animals than in the herbivora, being apparently dependent in great part on their artificial habits of life, the sweet and stimulating diet and the derangement of the digestive organs. The lowering of the general health in connection with privation or disease and especially canine distemper, rachitism or indigestion must be recognized as predisposing causes, while the accumulation of tartar on the teeth, or the decay of the teeth themselves, constitutes a potent exciting local cause. In connection with such cretaceous deposits the decomposing elements of the food collect, and the irritant products of their fermentation lead to disease of the gums, congestion and ulceration. Superadded to this is the bacteridian infection of such diseased parts, through which the ulceration is started, maintained and extended. This infection is not that of a specific microbe, but usually of a multiplicity of germs, one or more of the bacteria that live habitually in the healthy mouth, taking the occasion of the existence of a wound, or of a reduction of vitality to colonize the mucosa which would otherwise have remained sound. The microbes actually found in the ulcers are very varied. Pasteur isolated a spirillum, Fiocca the bacillus salivarius septicus, others have found pus bacilli, and in sucking kittens the bacillus coli communis.

But the attempts made to convey the disease to healthy mouths by the transfer of the microbes have usually failed (Pasteur, Netter, Cadeac). To establish their pathogenic action therefore, it appears to be necessary to furnish a susceptible mucosa as well as an infecting microbe. This explains why the disease does not spread as an infection, the average mouth is immune and it is only when it becomes the seat of a wound, bruise or other injury, or when the general system has become so reduced that the resisting power is a minor quantity, that the hitherto harmless germ becomes actually pathogenic.

Symptoms. There is indisposition to suck or eat, the patient leaves the teat or the food, and looks dull, depressed and disposed to lie down apart. There is evident salivation and on opening the mouth we may find the offensive odor, the tartar covered teeth with red or ulcerated gums, and on the cheeks, lips and tongue dark red patches of congestion, or whitish or yellowish gray, soft, pulpy spots of disintegrating epithelium. This is followed by shedding of these epithelial patches, and the formation of rounded ulcers of a line in diameter or less. These are tender, and bleed readily. They may extend to the skin of the lips, or deeply into the mucosa, the muscles or bones, and the attendant morbid process may cause loosening and evulsion of the teeth. There may be implication of the pharynx, the lymph glands, the nose, the eyes, the stomach, the liver, or the intestines with corresponding symptoms. Death may supervene in from six to thirty days, or a more or less speedy recovery may take place.

Treatment. The first step as a rule is to remove the tartar from the teeth. This is often done with a wooden spud dipped in a weak solution of hydrochloric acid. A steel scraper will usually act well and without the solvent action of the acid.

Next will come the removal of all diseased teeth which are operating as local irritants and as centres for infectious microbes and their hurtful products.

Then antiseptics in the form of liquids applied as in the other animals with each meal, will be necessary to counteract infective action, and give the tissues an opportunity to re-establish their integrity. Cadeac recommends a 10 per cent. solution of oil of thyme, as a safe and efficient application. Boric acid, borax, salol, salicylic acid, tannic acid, sulphurous acid, or carbolic acid largely diluted may be substituted. Internally iron tonics and bitters are of great value in improving the tone of the system and securing antiseptics of the intestinal canal. The sulphites too may be given with advantage internally. In depressed conditions alcoholic stimulants may be used both as local antiseptics and general stimulants. As in other animals ulcers may be touched with a rod dipped in tincture of iodine, or a strong solution of chloride of zinc, or nitrate of silver.

MERCURIAL STOMATITIS.

Animals suffering. Causes: mercurial baths, ointments, blisters and surgical dressings; mercurial vapors; deposits on vegetation; rat poisons; malicious poisoning. Lethal dose in horse, ox, sheep and goat. Mature and old eliminate more slowly. Symptoms; Salivation; red, swollen buccal mucosa; gingivitis; loosening of teeth; foetor; ulceration; anorexia; gastro-intestinal tympany; loose, foetid stools; fever; weakness; dyspnoea; langor; blood extravasation in nose, mouth, throat, bowels, womb, skin; abortion; skin eruptions. Lesions in mouth, stomach, intestines, serosæ, kidneys, muscles, encephalon. Treatment; stop the introduction of mercury; as antidote potassium sulphide; emetic; cathartic; mucilaginous and albuminous antidotes; potassium iodide as an eliminating agent. Locally potassium sulphide or chlorate. Iron tonics.

This has been especially seen in the sheep, dog and ox, and less frequently in other domestic animals.

Causes. In sheep the use of baths containing corrosive sublimate, or of mercurial ointment for acariasis or other cutaneous parasitism. In other animals it comes mostly from licking mercurial dressings applied to the skin—calomel, red precipitate, mercurial ointment, protoiodide of mercury. The red iodide being more irritating is less frequently taken in. The modern extensive usage of mercuric chloride solutions as surgical antiseptics opens up a new channel of infection. In the injection of the uterus or of large abscesses, or in the daily irrigation of large wounds a dangerous amount may be absorbed. The application of this agent as a caustic in cases of tumors is correspondingly dangerous. Vapors from metallic mercury in confined spaces as in ships' holds, or from fires on which the mercurial compounds have been thrown, are ready means of poisoning, acting primarily on the air passages and lungs and later on the mouth. The condensation of mercury on vegetation and other food products in the vicinity of factories where mercury is handled (Idria) affects domestic animals directly. Finally the small animals are poisoned by eating the mercurial rat poisons, and all animals are subject to malicious mercurial poisoning, with sublimate especially.

Stomatitis with fatal pharyngitis and enteritis will result in the horse from 2 drs. of corrosive sublimate. About one-half of this may poison the ox, and one-fourth the sheep or goat. Ruminants are more susceptible to the toxic action of mercury than monogastric animals, one evident reason being the long delay of the successive doses in the first three stomachs, so that finally a large quantity passes over at once into the fourth stomach and duodenum for absorption. The old too are more readily poisoned than the young, as the functions of the kidneys are more impaired in age and the poison is not eliminated with the same rapidity.

Symptoms. Mercurial stomatitis is a local manifestation of a general poisoning. Salivation is one of the most prominent phenomena, the watery saliva falling in streams from the angles of the mouth. The buccal mucosa generally becomes red and swollen and the tongue becomes indented at the edges by pressure against the molars. The gums especially suffer and the teeth raised in their sockets by the swelling of the periodontal membrane, become loose, and easily detached. The mucosa of the gums becomes soft and spongy, bleeds readily under pressure and soon shows erosions and ulcers. This condition extends to the lips, cheeks and lower surface of the tongue while the upper surface of the latter organ, the fauces and pharynx commonly escape. The breath and buccal exhalations are very offensive, and the animal loathes food, and has little power of mastication or deglutition. Sometimes the ulcers extend even to the bones.

Along with these local symptoms there are usually gastro-intestinal irritation, tympany, inappetence, continuous rumbling in the belly; badly digested foetid stools, often diarrhoea, small weak pulse, hyperthermia, accelerated breathing, cough, and great langor and prostration. A tendency to blood extravasation is shown in sanguineous fæces, epistaxis, bleeding from the mouth, the throat or the womb and even into the skin. Pregnant females may abort. The eyes are dull and sunken, and the conjunctiva yellow. Eczematous or pustular eruptions may appear on the skin on the nose, lips, neck, back, loins, croup or perineum.

Lesions. In addition to the lesions described above, there are usually gastro-intestinal inflammation, œdema of the peritoneum and pleura, in the lung as well as in the serosæ, (pneumonia is not uncommon especially in sheep), intestines, kidneys and muscles, hæmorrhagic spots are not uncommon, the blood forms a loose black coagulum, and the encephalon is anæmic

and softened.

Treatment. The first consideration is to cut off the supply of mercury. Mercurial applications on the skin should be washed off with tepid water and if necessary soap. An application of sulphide of potassium will precipitate the mercury in an insoluble form. For mercurial agents in the alimentary canal an emetic may be given (if the animal is one susceptible to emesis) followed by a saline laxative. This may be combined with or followed by raw eggs, mucilage, wheat gluten or other albuminoid, sulphide of potash or sulphur, to precipitate the mercury and prevent its absorption. Later, when the bowels have been cleared, iodide of potassium in small doses will serve to dissolve and remove what mercury may be lodged in the tissues.

Locally one of the best applications is chlorate of potash as a mouth wash, 2 drs. to the quart of water. To this may be added tannic acid or other vegetable astringent and even alcohol.

Finally a course of iron and bitter tonics will serve a good purpose in restoring the general tone.

STOMATITIS FROM CAUSTICS.

Caustic Alkalies; symptoms, lesions and antidotes. Caustic Acids; symptoms, lesions and antidotes. Caustic salts; symptoms, lesions and antidotes.

Caustic Alkalies (soda, potash, ammonia and their carbonates) often cause stomatitis. What is supposed to be *weak lye*, given to counteract indigestions, colics, and tympanies often proves dangerously irritating, and some of the worst forms of stomatitis we have ever seen in the horse originated in this way. As the animal refused to swallow, the caustic liquid lay in the mouth and virtually dissolved the epithelium and surface layers of the fibrous mucosa. The surface in such a case is usually of a deep red, and where the cuticular covering remains, it is white and corrugated. The antidote is a weak, non-irritant acid, such as vinegar, boric, citric, or salicylic acid. When the caustic alkali has been thoroughly neutralized in this way the ordinary treatment for catarrhal stomatitis may be followed. The attendant gastritis must receive its special treatment.

Caustic Acids. Sulphuric, nitric and hydrochloric acids act by abstracting liquids and charring the tissues. The lesions from strong sulphuric acid turn black, those due to nitric acid, yellow, (zanthoproteic acid,) and those due to muriatic acid are white, with the characteristic odor of chlorine. The antidote in such cases is a non-irritant basic agent, such as chalk, lime water, soapsuds, calcined magnesia, and mucilaginous liquids, albumen, gluten, flax seed, with opium. The same agents are applicable to the attendant gastritis and when the acids are thoroughly neutralized the treatment is as for simple inflammation.

Caustic Salts. Among caustic salts may be named mercuric chloride, sulphates of copper and iron, chlorides of iron and zinc, tartar emetic. These may be treated by albumen, blood, white of egg, milk, gluten, mucilage and other sheathing, protecting agents which will form with the salts insoluble and harmless coagula. The subsequent treatment will follow the lines marked out for simple stomatitis. To prevent infection of the raw surface Cadeac recommends: tannic acid 1 oz., benzo-naphthol 3 drachms, powdered gentian 6 drachms, honey, sufficient to make an electuary.

MYCOTIC STOMATITIS IN FOALS, CALVES AND BIRDS. THRUSH. MUGUET.

Oidium (saccharomyces) albicans; a parasite of the young; cultures. Symptoms in foals and calves; congested buccal mucosa; curd-like concretions; erosions. Diagnosis from rinderpest. Treatment; disinfection; sunshine; open air; exercise; locally antiseptics.

This is a form of stomatitis manifested by a raised white patch on the mucous membrane and determined by the presence of the **oidium albicans (saccharomyces albicans)**, a cryptogam discovered by Berg in 1842 in thrush in children. It is closely allied to the *mucor*, and attacks only the young and feeble. The white crust consists of epithelial cells intermingled with an abundance of the white mycelium and oval spores of the fungus. Andry in his artificial cultures found that it was pearly white when grown on gelatine, dirty white on potato, and snow white on carrot.

Foals and Calves. *Symptoms.* The buccal mucosa red, congested and tender, shows here and there white curdy looking elevations, or red erosions caused by the detachment of such masses. These bear a strong resemblance to the concretions seen on this mucosa in rinderpest, but are easily recognized by the absence of the attendant fever, and by the discovery, under the microscope, of the specific microphyte. The eruption may extend to the pharynx and œsophagus and interfere fatally with deglutition, but usually it merely renders sucking painful and is not serious.

Treatment. It is always well to destroy floating germs by cleansing and whitewashing the stable, and to invigorate the young animals by sunshine, free air and exercise. Locally the most effective agent is the old favorite remedy borax which arrests the growth of the parasite whether in artificial cultures, or in the mouth. The powder may be rubbed into the sores or it may be mixed with honey or molasses and used as an electuary. As substitutes boric acid, salol, thymol, chlorate of potash, or permanganate of potash may be used.

Birds. The affection has been twice observed as occurring in the œsophagus and crop of two chickens. Martin tried in vain to inoculate it on other fowls, and Neumann failed to convey it from child to chicken by feeding. The element of individual susceptibility was manifestly lacking. From its seat in the crop the malady passed unnoticed during life. In cases that can be recognized, treatment would be the same as in young mammals.

PARALYSIS OF THE TONGUE. GLOSSOPLEGIA.

Causes: Nervous lesions—central or peripheral, parasitic, inflammatory, infectious, traumatic or degenerative. Symptoms: unilateral and bilateral. Treatment: remove cause; use nerve stimulants, embrocations, blisters, frictions, galvanism, suspension of tongue.

Paralysis of the tongue depends on a lesion of the medulla oblongata, or of the 7th or 12th cranial nerve. The central lesions may be connected with cœnurus or other parasites in the brain, hydrocephalus, meningitis, cerebro spinal meningitis, infectious pneumonia, abscess (strangles), and tumors. The distal or nerve lesions may be due to neuroma, tumors, traumas, lacerations, bruises, or violent distension of the tongue. Parotitis, abscess of the guttural pouch and tubercle may be added as occasional causes. As direct traumatic injuries those caused by wearing a poke by a habitual fence-breaker, excessive dragging on the tongue in operations on the mouth, and compression of the tongue by a loop of rope passed over it, require mention.

Symptoms. In **unilateral** paralysis the affected half of the tongue remains soft and flaccid and is liable to be crushed between the teeth, the active muscles of the opposite half pushing the organ over to the paralyzed side. In **bilateral** paralysis the tongue hangs out of the mouth, and being crushed and torn by the teeth, it swells up, and may even become gangrenous.

Treatment. Will vary according to the cause. After removal of the central or nervous lesions, the remaining functional paralysis may be treated by strychnia, internally or hypodermically, by frictions or stimulating embrocations to the intermaxillary region, or by electricity. The tongue must be suspended in a sling to prevent œdema, inflammation and wounds by the teeth. In bad cases of bilateral traumatic glossoplegia in meat producing animals it has been advised to have the subject butchered.

DISEASES OF THE SALIVARY GLANDS.

Modifications of the secretion are commonly simple excess or deficiency, with a correspondingly high or low specific gravity of the product. There may, however, be a virulent element as in the case of rabies.

SUPPRESSION OF SALIVARY SECRETION. XEROSTOMIA.

Causes; fever; vascular vacuity, after bleeding, diarrhoea, etc.; destruction of glands; Calculus. Symptoms; slow, difficult mastication; digestive disorder. Treatment; remove mechanical obstruction; correct constitutional disorder; employ stimulation to gland—pilocarpin, electricity.

Entire suppression of salivary secretion is usually the result of some other disease. It may be a manifestation of the general tendency to retain water in the febrile system, or it may be an indication of vacuity of the vascular system as after bleeding, profuse diarrhoea, diuresis, or diaphoresis, or it may be the result of the entire destruction of a salivary gland or the obstruction of its duct by some foreign body or calculus. In proportion to the completeness of the suppression, mastication and deglutition become difficult or impossible. The condition must be met by the removal of the cause which is operative in the particular case. The treatment may be surgical for the removal of obstructions, or medical with the view of overcoming anæmia, fever, profuse secretions from other emunctories, or the simple physiological inactivity. To meet the last indication small doses of pilocarpin, or the application of a gentle current of electricity will usually succeed.

EXCESSIVE SECRETION OF SALIVA. SALIVATION. PTYALISM.

Causes; a symptom of other diseases, of the mouth, teeth, throat or stomach; rank aqueous vegetation, lobelia, pilocarpin, muscaria, tobacco, mustard, and other acrid vegetables; caustic alkalies, acids, salts; compounds of mercury, gold, copper, iodine; palsy of lips; harsh bit; fungi on clover, sainfoin, etc. Symptoms; salivary escape; frequent deglutition; thirst; disordered digestion, etc. Treatment; remove cause; astringent washes; sedatives; embrocations to the glands.

This is often a symptom of some other affection such as aphthous fever, dumb rabies, epilepsy, stomatitis, pharyngitis, dentition, caries and other diseases of the teeth, wounds and ulcers of the mouth, gastric catarrh, etc. In other cases it is due to direct irritants in the food or medicine, as very rank, aqueous, rapidly grown, spring grass, lobelia, pilocarpin, muscarin, tobacco, wild mustard, colchicum, pepper, garlic, ginger, irritant and caustic alkalies, acids and salts, and the compounds of mercury, gold, copper, or iodine employed locally or internally. The application of mercurials to the skin is especially liable to salivate cattle and dogs, partly because of a special susceptibility to the action of this metal and partly from the tendency of these animals to lick the medicated surface. Paralysis of the lips causes a great flow of saliva from the mouth though no more than the normal amount is secreted. The irritation of a large or harsh bit will increase the secretion and still more the former habit of attaching to it small bags of spicy or irritant chemicals. Certain fungi determine salivation. Mathieu saw profuse salivation in horses, cattle and sheep fed on clover and sainfoin which had become brown.

Symptoms consist in the profuse flow of saliva, either in long stringy filaments, or if there is much movement of the jaws, in frothy masses; frequent deglutition; increased thirst and disordered digestion (tympany, inappetence, colics, constipation, diarrhœa). In mercurial salivation there may be loose teeth, swollen, spongy, ulcerated gums, tympany, rumbling, and the passage of foetid flatus and soft ill-digested stools.

Treatment consists in removing the cause, whether this is to be found in faulty food or drink, diseased teeth or gums, disordered stomach, or the irritant food medicine or poison ingested. If more is wanted simple astringent washes like those recommended for stomatitis and a free access to pure water will often suffice. Tartar emetic or opium has been known to succeed in obstinate cases. Friction over the parotid or submaxillary gland with camphorated spirit, tincture of iodine or soap liniment is sometimes required. In mercurial salivation chlorate of potash is especially to be commended, and when the bowels have been unloaded of the agent, iodide of potassium will hasten its elimination from the tissues and blood.

DILATED SALIVARY DUCTS. SALIVARY CALCULUS. SALIVARY FISTULA.

These are all surgical diseases and are to a large extent inter dependent. The impaction of the calculus in the duct leads to overdistension of the duct posterior to the obstruction, and the rupture or incision of the distended duct, determines the fistula. It is only necessary here to point out the seat of these lesions: the distended sublingual ducts constituting a more or less rounded swelling to one side of the frænum lingui, the Whartonian duct forming a tense rounded cord from the papilla back of the lower incisor teeth backward on the inner side of the lower jaw, and the Stenonian duct forming a similar tense cord from near the middle of the cheek down around the lower border of the jaw in company with the submaxillary artery and backward on the inner side of its curved border to the parotid gland.

For the more precise lesions, symptoms and treatment of these, see a work on surgery.

INFLAMMATION OF THE PAROTID GLAND. PAROTITIS.

Causes: traumatic; calculus; grains; barley and other beards; infecting microbes. Symptoms: fever, dullness, buccal heat, salivation, difficult mastication, swelling of gland and duct, protruded nose, stiff neck, foetor, dyspnoea, facial paralysis, induration of gland, abscess. Diagnosis from pharyngitis, abscess of guttural pouch or pharyngeal glands; from tumors. Treatment: avoidance of causes; derivation; astringent, antiseptic washes; wet antiseptic bandages to throat; cool pultaceous diet. Open abscess and disinfect. For induration deobstruents. For sloughing antiseptics.

This may be caused by traumatism, such as incised punctured or bruised wounds. Wounds inflicted by the goad, by horns, and even by the yoke in cattle must be looked on as factors. It occurs from obstruction of the salivary ducts by calculi, or by grains, seeds, or pebbles introduced from the mouth; from their irritation by the beards of barley and other plants (brome, rye, wheat, etc.); and from the localization in the gland of specific inflammations like strangles, pyæmia, canine distemper, tuberculosis and pharyngitis. In most of these cases infective microbes are prominent factors. They enter with penetrating bodies from the skin; they extend through the weakened and debilitated tissues in bruises; they penetrate the Stenonian duct with the various foreign bodies from the mouth; irrespective of foreign objects they make their way up the duct by continuous growth from the buccal orifice; in case of calculus or other obstruction their extension is favored by the local congestion and debility and by the stagnation of the saliva above the point of arrest. When present these microbes even favor the deposition of the salivary salts and formation and increase of calculi so that the affection may advance in a vicious circle, the microbes favoring calculus and the calculus favoring the increase of microbes.

Symptoms. In the horse in particular there may be premonitory symptoms of fever, dullness, heat of the mouth, ptyalism, slow and imperfect mastication, and the retention of food in the cheeks.

The Stenonian duct becomes swollen and painful. The parotid becomes hard, hot, tender, and is surrounded by a softer pitting infiltration which may extend down around the entire throat, and even along the intermaxillary region to the chin. When the canal is obstructed it may stand out as a thick rope-like resilient swelling extending around the lower border of the jaw and upward toward the cheek as far as the point of obstruction. When one parotid only is involved, the contrast with the other is quite marked. The head is extended and carried stiffly. When the nose is depressed, or when the head is turned to one side or the other, the patient gives evidence of suffering from compression or stretching of the inflamed region. The breath and mouth exhale an offensive odor, determined by the decomposition of mucus and of the retained food products.

Among remote effects may be named dyspnoea and threatened suffocation from pressure on the pharynx and laryngeal nerves, and facial paralysis from pressure on the seventh nerve.

The disease may go on to induration and remain permanently in this condition, or it may suppurate and discharge through the skin, into the pharynx or through the duct of Stenon. It may communicate with both the duct and the skin and determine a fistula. When suppuration occurs there is an access of fever, a chill may be noticed, the swelling becomes more tense, harder, more tender to the touch, and even emphysematous, and finally points internally or externally. This may take place from the fifth to the tenth day or later. When it opens into the duct it may be seen oozing from the orifice in the cheek when the mouth is opened, and in case the jaws are suddenly parted, it may escape in a jet. In such a case and especially if the microbes have come originally from the food the odor is very foetid. The abscess is not always single and when multiple the pus may escape externally by a variety of orifices. The pus is usually whitish, yellowish or grayish and creamy, but it may be grumous or bloody or serous and of a most offensive odor. In exceptional cases the gland becomes more or less gangrenous and such parts, exposed in the wound are hard, bloodless and insensible, and add very materially to the foetor. This may lead to general septic infection, or the

necrosed masses may slough off and the cavities fill up by granulations.

Diagnosis. Parotitis is distinguished from pharyngitis and abscess of the guttural pouch by the absence of cough and nasal discharge; from abscess of the pharyngeal glands it is differentiated by the limitation of the hard swelling to the parotid gland and by the superficial seat of the resulting abscess. The co-existence of active inflammation serves to distinguish it from ordinary tumors.

Treatment. By way of prevention, the avoidance of injuries by yokes, forks, pokes, and goads is important. Also the disinfection of the mouth by a liberal supply of pure water and even by antiseptic washes:—borax, boric acid, creolin, tannin, chlorate of potash. Also by the removal of foreign bodies or calculi from the canal.

When the inflammation has set in, a saline laxative is often of value. Wash the mouth with a solution of vinegar and salt, or other antiseptic, repeating this at least after every meal. The swollen, painful gland may be covered with a damp compress or anointed with vaseline to which may be added a little creolin, naphthol, carbolic acid or salicylic acid, together with lead acetate and belladonna or other anodyne. The diet must be soft, cool mash, sliced or pulped roots or any bland agent that will demand little or no mastication. Cool, fresh water should be allowed *ad libitum*. When the laxative has set, it may be followed by cooling diuretics such as nitrate or acetate of potash.

If suppuration occurs it should be opened as soon as the pus can be definitely recognized, and the cavity treated antiseptically to prevent further local or general infection by the microbes. In deep abscess there is a certain danger of wounding blood vessels and salivary ducts, but this can be to a certain extent obviated by making an incision through the skin only and then boring the way into the abscess with a grooved director or the points of closed scissors. When the cavity is penetrated the pus will ooze out through the groove or between the scissor blades. When the pus has been evacuated the cavity should be washed out two or three times a day with mercuric chloride solution (1 : 1000), or permanganate of potash solution (1 : 100).

When the gland becomes indurated and indolent seeming to merge into the chronic form it may be stimulated to a healthier action by a cantharides blister, or it may be subjected to daily massage, or to a daily current of electricity for ten or fifteen minutes. If the inflammation is slight or unrecognizable, the surface of the gland may be daily painted with tincture of iodine, and iodide of potassium may be given internally, in daily doses of ½ to 1 drachm.

Gangrene, the result of septic microbes, a weak system or too severe treatment, may be met by astringent and antiseptic agents locally, and by tonics, stimulants and a generous diet internally.

In **cattle** the disease usually responds readily to local antiseptics, and stimulating germicidal embrocations. Camphorated spirit, alone or combined with tincture of iodine; cantharides ointment with carbolic acid; and camphor and phenol may be cited as examples.

SUB MAXILLARY ADENTITIS. MAXILLITIS.

Mostly in solipeds and unilateral. Causes; traumatic; calculus; infections; ablation of papillæ. Symptoms; tardy mastication; salivation; buccal heat and fœtor; submaxillary swelling and tenderness; morsels retained under tongue; papilla and duct swollen, tender and firm; abscess. Treatment; remove causes; dislodge foreign bodies; antiseptic lotions and packing.

This is rarely seen in other animals than solipeds, is mostly unilateral, and due to the introduction of microbes along with vegetable spikes (barley awns, brome, wheat or oat spikes or glumes) or other foreign bodies. It may also be caused by calculi obstructing the duct. The orifice of each duct, to one side of the frænum lingui, is imperfectly closed by a triangular valvular projection, which in some countries is erroneously cut off as a diseased product (barbs), thus opening the way for the introduction of foreign objects. The microbes are usually pus germs and tend to abscess of the gland. As in the case of the Stenonian duct the presence of these germs tends to the precipitation of the salivary salts and the formation of calculi.

Symptoms. The animal may seem hungry, but masticates tardily and imperfectly, and may even drop morsels partly chewed. He prefers ground feed to whole, and soft mashes to ground feed, while hay and other fibrous aliments may be altogether rejected. Salivation may be excessive, the secretion drivelling from the lips, the mouth may feel hot and the submaxillary salivary gland swollen and tender. This may be detected in the intermaxillary space, but is especially noticeable along the lower and lateral aspect of the tongue. If the mouth is opened and the tongue drawn to one side a mass of food may be found to one side of the frænum lingui, and beneath this the projecting, red inflamed papilla which covers the Whartonian orifice. Extending backward from this the duct is felt as a thickened cord, and when this is compressed a purulent liquid flows from the orifice. The mouth becomes offensively fœtid.

The tendency is to suppuration, and if this is determined in the Whartonian duct only, by the presence of foreign bodies, calculi, or microbes it may recover in connection with an abundant muco purulent discharge and a free secretion of saliva. If it occurs in the gland tissue itself by reason of the penetration of the microbes into the follicles, the tendency is to circumscribed abscess, which may point and burst by the side of the root of the tongue, or externally in the intermaxillary space. In the first case the tongue is displaced upward and to the other side of the mouth by the hard, firm swelling, which is felt on one side beneath the back part of that organ, and later there is the wound, the profuse muco purulent discharge, and intense fœtor. If on the other hand the abscess forms nearer the skin, there is the firm, painful intermaxillary swelling, which finally points and bursts discharging pus of a septic odor. It may be mixed with the foreign bodies that have penetrated through the canal, with morsels of necrosed gland tissue and with blood.

Treatment. The first consideration is to extract any foreign bodies which have lodged in the duct causing irritation and infection. The finger passed along the line of the swollen duct may detect the seat of such foreign body by the extra swelling, and may extract it by manipulation from behind forward. This may sometimes be assisted by the introduction of a grooved director as far as the foreign body, or even by a catheter which can be made to distend the canal in front of the object and open the way for its easier passage. In case of failure and in all cases of the introduction of small bodies like vegetable awns or spikes pilocarpin may be given to cause an excessive secretion and thus as it were purge the canal of its offensive contents. Incision of the canal over the foreign body is the *dernier resort*.

This accomplished, the injection of antiseptic solutions (permanganate of potash, boric acid), and the liberal use of pure water and detergent lotions in the mouth (vinegar, borax, carbolic acid or salicylic acid in solution) will go far to establish a cure. In case of an abscess bursting internally the antiseptic solutions should be injected into its cavity. When the abscess bursts externally this is doubly demanded, as the introduction of aerial germs tends to produce very unhealthy action. The cavity may be stuffed with carbolized, or iodoform, or acetanilid cotton, or with boric or salicylic acid.

SURGICAL LESIONS OF THE SALIVARY GLANDS.

Among these may be named calculi of the Stenonian and Whartonian ducts, ranula, stenosis and fistulæ of these ducts, tumors, special infections like actinomycosis.

TONSILITIS IN PIGS, AND OTHER ANIMALS.

Causes; debilitating, climatic, microbial. Symptoms; fever, dullness; lies under litter; ears and tail droop; watery eyes; anorexia; vomiting; pharyngeal swelling; buccal redness and foetor; tonsils swollen with pus or caseous mass in follicles; cough dry and hard, later loose. Abscess. Calculus. Course. Treatment; antiseptic electuaries; embrocations; laxatives; diuretics; tonics.

This is seen in both the acute and chronic form. In the former it has the general causes and symptoms of pharyngitis. There is more or less fever, dullness, a disposition to lie with head extended and buried in the litter, ears drooping, eyes watering and red, carelessness of food, deglutition painful, and liable to be followed by vomiting. The mouth is red and hot, the breath foetid and the tonsils swollen, and their alveoli filled with muco purulent matter or at times with a foetid cheese-like product. The cough is at first dry and hard and later loose and gurgling.

In the chronic form there is general swelling of the tonsils with the overdistension of the follicles by the above mentioned whitish putty-like masses, which are often even calcareous. These are due to the proliferation of microbes which find in these alveoli a most favorable field for their propagation. A similar condition is found in the carnivora and to a less extent in the horse, in keeping with the restricted development of the amygdalæ in these animals. It may be attended by ulceration, or in rare cases by the formation of veritable calculi in the follicles of the tonsils.

The gravity of the disease is largely determined by the nature of the infecting microbe and the debility and susceptibility of the animal attacked. The affection usually ends in recovery, but may go on to grave local ulceration, and general infection.

Treatment consists largely in astringent and antiseptic applications to the buccal mucous membrane. In the acute forms frequent smearing of the mouth with electuaries of honey or molasses and borax, boric acid, salammoniac, chlorate or permanganate of potash, and the application of stimulating embrocations to the skin around the throat. In other cases solutions of tincture of chloride of iron, or of tincture of iodine can be used with profit. The iron can be swallowed with advantage, but it is objectionable to pour liquid rapidly into the mouth of the pig, because of the danger of its entering the lungs and setting up fatal pneumonia. A better way is to apply it to the interior of the mouth and fauces on a swab or sponge dipped in the liquid. Short of this one of these agents may be mixed with the drinking water, or muriatic acid may be used in the same way, though at some detriment to the teeth. The general health must at all times be attended to. Any costiveness may be corrected by Glauber salts or jalap, and elimination through the kidneys must be sought through the use of nitrate of potash or other diuretic.

CALCULI IN THE TONSILS.

Diagnosis and treatment of tonsillar calculi; spud; acid dressings. Trauma of soft palate by stick, probang, file, molar. Abscess of palate. Treatment; laxative; expectorant; antiseptic; lancing. Cleft palate and hare lip.

Rudimentary as these organs are in the equine race they are important enough to have become the seat of hard calculous masses. These have been found by Goubaux and Blanc in old asses, and by the author in old horses. They vary in size from a pin's head to a pea and consist of concentric layers of a granular material arranged around a central nucleus, which is usually a foreign body introduced with the food. This nucleus is usually of a vegetable nature, while the enveloping material is made up largely of the imprisoned and degenerated epithelium of the follicle. Both diagnosis and treatment are difficult in such cases. The adventitious masses should be dislodged by the aid of a smooth, blunt metallic spud, and the surface thereafter washed or swabbed with an antiseptic and astringent solution. Swabbing with a solution of hydrochloric acid will tend to dissolve and remove them.

INJURIES TO THE SOFT PALATE AND FAUCES.

The region of the fauces is sometimes injured by sharp pointed bodies swallowed in the food, by the giving of boluses on the end of a pointed stick, or by the careless use of a probang or of a file upon the posterior molars. An overgrown last molar will sometimes lacerate the velum. In other cases the inflammation of sore throat is especially concentrated on this part, giving rise to cough, difficulty of swallowing, redness, infiltration and swelling of the parts, and even abscess. In the dog it is often associated with tonsilitis.

Treatment. A laxative is usually desirable to be followed by salammoniac or chlorate of potash. In case of actual traumatic lesions, the astringent and antiseptic lotions advised for tonsilitis will be in order, and if abscess is recognized it should be opened promptly.

CLEFT PALATE.

In exceptional cases the soft palate has failed to unite in the median line, and is represented by two lateral flaps separated by a v-shaped hiatus in the middle. In a specimen in the N. Y. S. V. College, taken from a trotting colt, the fissure is continued forward for several inches between the palatine bones and the palatine processes of the superior maxillary, establishing a direct communication between the mouth and nasal chambers. In still other instances the fissure is continued forward between the maxillary and anterior maxillary bones, throwing the whole length of the buccal and nasal chambers into one irregular cavity, and forming harelip.

It would be possible to remedy some of these conditions by plastic operation, but the value of the young animal will rarely warrant any such resort.

CATARRHAL PHARYNGITIS.

Causes: traumatic; thermic; gaseous; medicinal; chemical; physiological irritants; in solipeds, cattle, swine, dogs; debility; exposure; cold baths; youth; age. Microbes in solipeds, cattle, dogs, birds; facultative microbes. Symptoms: constitutional; difficult swallowing; nasal rejection of water; pharyngeal swelling and tenderness; extended head carried stiffly; cough loose; salivation; in cattle, grinding of teeth; in dogs, rubbing of chops; buccal heat and redness; often foetor. Course. Duration. Diagnosis from parotitis, from abscess of guttural pouch, from pharyngeal tuberculosis, from actinomycosis, from adenitis and phlegmonous pharyngitis, from specific fevers affecting the pharynx. Lesions: redness and swelling of mucosa, epithelial degeneration, elevations, erosions, and ulcers; lesions of tubercle, glanders, rabies, anthrax, actinomycosis, etc. Prevention. Treatment: soothing; dietetic; laxative; expectorant; eliminating; locally antiseptic astringents in solid, liquid, or vapor; embrocations and blisters; tonics.

Causes. As in stomatitis the starting point of pharyngitis is usually in a local injury or a systemic condition which lowers the vitality of the pharyngeal mucous membrane. It may come in all animals from the hot air of burning buildings, from acrid gases inhaled, food, drink or medicines given at too high a temperature, from caustic alkalies, acids or salts, from physiological irritants like croton, euphorbium, cantharides, from barley and other spikes entangled in the follicles, from drinking freely of iced water. In *solipeds* there are the injuries caused by giving boluses on pointed sticks, and the wounds caused by tooth files in careless hands, and by coarse fibrous fodder, which has been swallowed without due mastication. In *cattle* injury comes from foreign bodies impacted, from the rough use of probang, rope or whip and even of the hands in relieving choking. *Swine* have the part scratched and injured by rough or pointed objects which they bolt carelessly with the food. *Dogs* and especially puppies are often hurt by solid and irritant bodies that they play with, and swallow accidentally or wantonly. They also suffer at times from the pressure of a tight or badly adjusted collar.

The system is debilitated and rendered more susceptible by chills consequent on exposure to cold blasts, or draughts, or rain or snow, when heated and exhausted, by cold damp beds, by pre-existing disease, by underfeeding and by overwork. In the larger animals this may come from the excessive ingestion of iced water, while in dogs the plunging in rivers, ponds or lakes may chill.

The weakness of *early age* and *old age* have a perceptible predisposing influence especially in solipeds and carnivora.

Finally as in other catarrhal inflammations the local action of disease germs on the mucous membrane must ever be borne in mind. These may be the germs of specific diseases localized in the pharynx;—in **Solipeds** the streptococcus of strangles, the bacillus of glanders, the diplococcus (streptococcus) of contagious pneumonia, the germ of influenza, and actinomyces;—in **Cattle** the bacillus tuberculosis, the bacillus of anthrax, actinomyces, the germs of aphthous fever and of pseudomembranous angina; in *dogs* canine madness and distemper;—in birds the bacillus of pseudomembranous pharyngitis.

In addition to such specific germs the micrococci, streptococci and bacilli which are normally present and harmless in the mouth and pharynx, enter, colonize and irritate the debilitated tissues in case of trauma, inflammation or constitutional disorder and serve to perpetuate and aggravate the affection.

Symptoms. Acute pharyngitis is manifested by impaired or lost appetite, dullness, weakness, by difficulty in deglutition, by the rejection through the nose of water or other liquids swallowed, by swelling over the parotid and above the larynx, and by a disposition to keep the head extended on the neck and the nose raised and protruded. Fever is more or less marked according to the severity of the attack the temperature being raised in mild cases to 100°, and, in the more violent, to 104° or 106°. The pulse and breathing may be excited, amounting sometimes to dyspnoea, the throat is tender to the touch and its manipulation rouses a cough, the nasal mucosa is congested and the buccal membrane, and especially along the margin of the tongue may be red and angry. Salivation is shown more or less, in *solipeds* the saliva accumulating especially during mastication in froth and bubbles at the commissures of the mouth, while in *ruminants* the grinding of the teeth or frequent movement of the jaws in the absence of food or actual mastication leads to a free escape of the filmy liquid at the same points. *Dogs* will rub the jaws with the foot as if to remove some

irritating object from the mouth. In the last named animals the swelling of the tonsils, fauces and pharyngeal mucous membrane, may be seen marked by patches and spots of varying redness and swelling, covered with glairy or opaque mucopurulent secretions, or particles of food, or even showing erosions.

The cough of pharyngitis is painful, paroxysmal, and softer and more gurgling (even in the early stages) than that of laryngitis or bronchitis. It is roused by handling the throat, by swallowing, by a draught of cold air or by passing out of doors, in *dogs* by opening the mouth, and in *cattle* by pulling on the tongue which causes pain and resistance. The cough is followed by the rejection, mainly through the nose in *solipeds*, but also through the mouth in other animals, of a glairy mucus or an opaque mucopurulent discharge often mixed with and discolored by the elements of food or in bad cases by blood.

The *course* of the disease is comparatively rapid, and it usually ends in recovery in seven to fifteen days, in cases that are not complicated by dangerous local infections.

Diagnosis is mainly based on the stiff carriage of the neck with the nose elevated, the swelling and tenderness of the throat, manipulation above the larynx rousing the cough, the soft or rattling nature of the cough, the ejection of liquids and foods through the nose, the movements of the jaws apart from mastication and the salivation. From *parotitis* it is distinguished by the concentration of the swelling and tenderness to the deepseated region above the larynx, by the abundance of the discharge, by the ejection of liquids through the nose, and by the readiness with which the cough is aroused. From *abscess of the guttural pouch* it is differentiated by the more continuous discharge from the nose, rather than the intermittent one. From *tuberculous pharyngeal glands* by its acute nature, by the absence of the glandular swellings in which the tuberculosis is concentrated, also by the absence of tubercles in other parts of the body. From *actinomycosis* by its more rapid progress and by the absence of the hard indurated cutaneous or subcutaneous swellings, and of the open sores with minute sulphur colored granules that mark that affection. From *adenitis* and *phlegmonous pharyngitis* it is distinguished by the absence of the glandular swelling and dyspnoea which attend on that affection. From the various fatal *febrile affections*, the germs of which may be localized in the throat, it may be diagnosed by the absence of the more profound constitutional disturbance, and of the more characteristic local symptoms of these which are seldom altogether wanting, though often greatly modified.

Lesions. Beside the thick covering of mucopurulent and alimentary matters, the pharyngeal mucosa, when washed, shows redness, ramified or reticulated, more or less swelling amounting at times to oedema, a soft friable consistency, which like the oedema may in bad cases extend into the submucous tissue, granular elevations, and raw abrasions caused by the destruction and removal of the epithelium. In some instances the ulcers may become quite extensive.

In the more specific inflammations (tubercle, glanders, rabies, aphthous fever, contagious pneumonia, anthrax, actinomycosis), the lesions will vary according to the specific nature of the disease.

Prevention. Avoid the various thermal, chemical, mechanical, and unhygienic causes already referred to, and the exposure to such infectious diseases as are liable to localize themselves in the throat.

Treatment. A piece of blanket or sheepskin placed round the throat with the wool turned inward, a moderately warm stall with pure air, and a diet composed of soft, warm or tepid mashes, (all hard or fibrous food, oats, hay, etc. being withheld) are important conditions.

If costiveness exists a dose of Glauber salts for the larger animals, and of jalap for the small, may be useful. Or pilocarpine or eserine may be given hypodermically. Following this, mild saline diuretics will serve at once to eliminate offensive products of the disease and lower the general temperature.

The most important resorts however, are the local applications of dilute acids, astringents and antiseptics to the pharyngeal mucosa, mouth and nostrils. In severe cases benefit may be derived from inhalation of water vapor, but this is rendered far more effective by the addition of vinegar, carbolic acid, creolin, camphor, tar, or sulphurous acid. The last may be obtained by the frequent

burning of a carefully graduated quantity of sulphur in the stall, the others by mixing them with hot water, saturating cloths hung in the stall, or sprinkling them on sand laid on the floor.

Chlorate of potash or borax may be dissolved in the drinking water, care being taken not to exceed the physiological dose. Mercuric chloride (1:2000) may be used to wash the lips and nostrils, but cannot be safely injected into mouth or nose. Powdered alum or tannic acid may be used by insufflation.

As a mouth wash and general medicament a saturated solution of chlorate of potash in tincture of muriate of iron, diluted by adding thirty drops to the ounce of water, may be given every hour or two. Or a solution of chlorine water diluted so as to be non-irritating, may be substituted with somewhat less effect. Even a weak solution of hydrochloric acid may be employed.

Borax may be used in a solution of 2 per cent., or carbolic acid in one of 1 per cent., or bisulphite of soda in the proportion of $\frac{1}{2}$ oz. to the pint, or salicylate of soda $\frac{1}{2}$ oz. to the pint of water. The same agents may be made into electuaries with honey or molasses and smeared every hour or two on the tongue or cheek. In such cases the addition of powdered liquorice, and, if the suffering is acute, of extract of belladonna will serve an excellent purpose.

The danger of infection of the stomach and bowels may be met by combining with the above or administering separately salol in doses of 2 to 3 drachms or naphthol in doses of 4 to 5 drachms to the larger animals. For sheep or swine one-fourth of these doses may be given, and for a shepherd's dog one-sixteenth to a twentieth.

As alternate antiseptics may be named, boric acid, permanganate of potash and salicylate of bismuth. These from their comparative absence of taste are especially useful in carnivora.

Counter-irritants to the throat are useful. For the horse, sheep, dog and cat, use equal parts of strong aqua ammonia and olive oil. For the solipeds a cantharides blister. For cattle or swine equal parts of strong aqua ammonia and oil of turpentine with a few drops of croton oil, or grains of tartar emetic.

Finally during convalescence a course of iron and bitters may be useful, especially in debilitated subjects.

PHLEGMONOUS PHARYNGITIS.

Submucous inflammation and abscess. Solipeds especially. Specific, or due to microbial pus infection. Traumatism; from foreign bodies in tonsillar and mucous follicles, from rough, fibrous food, instruments, etc.

As a sequel of catarrhal pharyngitis. Symptoms; as in catarrhal form, with more swelling and tenderness, glandular swelling, dyspnoea, and difficulty in swallowing; local induration followed by fluctuation and pointing. Complications; asphyxia, laryngeal oedema, purulent or inhalation pneumonia, pharyngeal fistula, palsy of vagus, secondary abscesses, septicæmia. Lesions, local, general. Treatment; General and local, fomentation—hot or cold and antiseptic. Embrocations. Lancing. Tracheotomy.

As distinguished from catarrhal pharyngitis this is inflammation of the submucous tissue and adjacent lymph glands, tending to abscess.

It is especially common in solipeds and rather rare in other classes of domestic animals. As a specific infectious disease it has its type in strangles (infectious adenitis), also in cattle in the complicated infection of purulent tubercle, but apart from such it is often the result of the penetration of the pus microbes from a catarrhal pharynx into the lymph plexuses and lymph glands. Traumatism may play an important part in causation as when vegetable barbs, awns, chaff or seeds, or strong hairs or bristles enter the open mouths of the mucous follicles, or the tonsillar cavities. Similarly trouble may arise from scratches by tough, fibrous fodder, from pricks by pointed or cutting instruments, by fractures of the hyoid, or by bruises by probangs, or tooth rasps. An overgrowth of the last molar, and a resulting wound and ulcer of the soft palate, and the presence of local deposits like those of glanders and actinomycosis, are other occasions of the entrance of the pus organisms. It will be recognized that this affection is not necessarily due to a difference in the infecting organism, but rather of the tissue involved, the microbes gaining the submucous tissues and expending their violence on these instead of confining their ravages to the surface layers of the mucosa. For this reason the deeper or phlegmonous affection may supervene on a catarrhal inflammation which may have already persisted for several days.

Symptoms. Beside the general phenomena of catarrhal pharyngitis, this form of the malady is characterized by a greater swelling and tenderness of the throat, extending from ear to ear, and from the trachea forward in the intermaxillary space; by nodular and painful swellings of the pharyngeal lymph glands, by the greater difficulty of deglutition, the muscular tissue being involved; by wheezing breathing amounting at times to violent roaring and threatened asphyxia. Perspiration on the throat, the ear, the side of the head or neck, of the fore arm, or of the dorsal region is not uncommon, and has been attributed to the compression of the vagus, or of the superior cervical ganglion of the sympathetic by the swelling. Fever usually runs higher than in simple catarrhal pharyngitis, which may be partly accounted for by the implication of the deeper and important structures but also in no small degree by the entrance into the circulation of the ptomaines and toxins, which in the catarrhal affection escape largely from the inflamed surface.

The resulting abscess is usually in or near a gland or group of lymph glands. The part passes through the usual succession of changes, of soft pitting swelling; firm, tense, painful condition in which the exuded lymph has coagulated; and softening and fluctuation which progresses from the centre toward the circumference. The abscess points variously according to its seat. If in the intermaxillary space it opens externally. If sub-parotidian or peripharyngeal it may burst inwardly into the pharynx or outwardly through the skin. If supra-pharyngeal (retro-pharyngeal), it may be so thickly encapsulated in unyielding walls that it may remain long indolent and inactive becoming a cold or chronic abscess. When an abscess opens into the pharynx, there is a sudden and copious flow of pus by the nose, and it may be by the mouth and a simultaneous subsidence of the inflammation.

Among the *complications* of the affection are asphyxia, oedema glottidis; abscess of the guttural pouch; rupture of an abscess into the larynx, and the descent of pus into the lungs; the entrance of saliva and alimentary matters into the lungs; gangrenous pneumonia; pharyngeal fistula; pressure on the vagus and paralysis of the pharynx or larynx; secondary abscesses; septicæmia.

Lesions. Besides the general inflammatory lesions some rather remarkable ones have been observed. Fractured hyoid, dissection of the mucous from the muscular coat, by aliments, for

nearly the whole length of the œsophagus (Brückmüller), purulent infiltration of the supra-pharyngeal muscles (Wakefield), ulceration of the pharyngeal or guttural sac mucosa, or even gangrene, purulent effusion in the tonsils, around the hypoglossal nerve, the lingual branch of the fifth, or the vagus, embolic inflammations, suppurations or gangrene of the bronchia, and implication of the lung tissue and pleura. Catarrhal enteritis and fatty liver and kidney are common.

Treatment. Beside the general measures advised for catarrhal pharyngitis, this type demands especially measures to moderate the intensity of the suffering, and when abscess appears inevitable to hasten its maturation. The first demand is met by hot fomentations persistently applied to the throat. This may be done by spongio-piline, or simply by well washed wool or cotton bound upon the throat and wet at frequent intervals with water rather hotter than the hand can bear. The addition of a little carbolic acid will secure at once some local anæsthesia and a measure of antiseptics. In warm weather the substitution of cold water has been resorted to with apparently good effect. If adopted it should be frequently removed so as to keep up the constant action of cold and moisture. These have been especially recommended in dogs injured by a tight or ill-fitting collar.

When suppuration appears imminent as shown by the dense, hard, circumscribed phlegmon, stimulating embrocations may be used to hasten its progress. Camphorated spirit is suitable for carnivora and sheep. It may be combined with tincture of cantharides for horses. For cattle and swine, oil of turpentine may be added, the three being used in equal proportions. A liniment of ammonia and oil may be used more or less frequently and energetically according to the relative thickness and insensibility of the skin of the animal affected.

When matter has formed and fluctuates, it should be at once evacuated and the cavity treated by antiseptic dressings. In this way secondary abscesses, septic infections, molecular ulcerations and other injurious sequelæ may be largely obviated.

In case of threatened asphyxia the *dernier resort* of tracheotomy is always available, and this often acts very favorably in improving the æration of the blood, in restoring the flagging vital functions which depend on hæmatosis, and in removing the friction and irritation consequent on the passage of air through the narrowed and tender passages.

SUPRA-PHARYNGEAL (RETRO-PHARYNGEAL) ABSCESS.

A sequel of phlegmonous pharyngitis. Symptoms; masked by its depth; pharyngeal wheezing or roaring with little local swelling; difficult swallowing; resisting tissues tend to chronicity. Results; pharyngeal fistula, burrowing along œsophagus, rupture into chest or blood-vessels, lymphadenitis, compression of vagus, or jugulars, permanent infected cavity with small orifice. Diagnosis from pus in guttural pouches. Treatment; external opening; antisepsis.

This is a natural result of phlegmonous pharyngitis, but it is possessed of so great importance alike in its chronicity and its results that it seems to deserve a special article. Like its initial morbid condition it is especially common in the soliped, and like that may be traceable to strangles, influenza, and local traumatism.

The *symptoms* are at first those of phlegmonous pharyngitis, and, if the local swelling, induration and tenderness are less marked than in other cases, it is due to the location of the inflammatory lesion deeply between the pharynx and the atlas and occiput. Indeed the moderate aspect of the external swelling, conjoined with the noisy wheezing or violent roaring, may be taken as important diagnostic indications. The supra-pharyngeal region is so closely confined on its lateral aspects, by the union of the fascia of the sternomaxillaris and mastoido-humeral muscles, that the swelling is confined in the early stages just as the pus is later. As this resistant fascia prevents any relief by lateral expansion, the engorged tissues press downward on the softer and less resistant upper wall of the pharynx and seriously impair both respiration and deglutition. Similarly when pus has formed, these lateral fibrous barriers, reënforced by organized lymph, stand in the way of the advance of the pus toward the skin, and lead it to dissect its way downward toward the pharynx. Even here the thickening of the tissues by the organized products of the lymph will often interpose a serious bar, and the pus remains pent up indefinitely, a source of wheezing, roaring and impaired deglutition, and a constant threat of secondary abscess or septic infection. Even the dense fibroid tissues may soften and degenerate and the pus may make its way spontaneously to the pharynx, or less frequently through the skin of the parotid, or intermaxillary region, or into the œsophagus or larynx. A fistula of the pharynx opening externally and allowing the escape of alimentary matters has been often noticed. These are especially liable to follow puncture of the abscess.

Among the less common sequelæ are fistula of the œsophagus; purulent pneumonia in connection with the purulent dissection of the œsophagean walls and rupture into the chest (Fichet, Schneider); ulceration of the blood vessels in the cow (Jonge); adenitis and lymphangitis of the neck, and the thoracic glands, followed by pericarditis and pleurisy (Cadeac); multiple embolic abscesses of internal organs (Dieckerhoff); compression and degeneration of the vagus nerve, with consequent respiratory and digestive troubles (Baudon); and compression and obstruction of the jugulars with passive congestion of the brain and vertigo. (Delamotte, Debrade). Even when the abscess opens into the pharynx the orifice is usually small, the pus escapes imperfectly, and food materials enter and the fistula may thus persist for a length of time. The same imperfect discharge is liable to take place with an external orifice and the pent up pus becomes inspissated, caseated and even calcified.

Diagnosis. Supra-pharyngeal abscess is to be distinguished from pus in the guttural pouches, by the lack of coincidence of the discharge with the dependence of the head in grazing, eating roots or drinking from a bucket; by the absence of the intermission when the head is elevated; and by the fact that the discharge is less frequently limited to the one nostril. The hearing too is less likely to be affected.

Treatment. As soon as the presence of pus can be recognized it should be evacuated. This is often attempted through the roof of the pharynx, but with such an opening there is always danger from

the entrance and decomposition of alimentary matters. If fluctuation can be felt externally, it is better to be opened through the skin. The integument may be incised with a lancet, and the tissues further penetrated by manipulations with the finger nail, a grooved sound or the point of closed scissors. In this way the vessels and nerves are pushed aside and the dangers of hemorrhage, fistula and paralysis avoided. The cavity must be irrigated with an antiseptic solution (carbolic acid 3:100; or acetate of aluminum 1:20).

PSEUDOMEMBRANOUS (CROUPOUS) PHARYNGITIS.

False membranes not due to a common microbial cause. Accessory causes in solipeds; caustics, smoke infection. Lesions: Congestion, necrosis; croupous exudate, extending to patches on bowels and bronchia; kidney infarctions; blood altered. Symptoms: fever, dyspnœa, mucous rattle in throat, swelling, painful, difficult deglutition, yellow or cyanotic mucosæ, pinched face, weakness, prostration. Duration. Diagnosis. Treatment, as for catarrhal pharyngitis with antiseptics by inhalation and electuary. Iron.

Pharyngites attended by the formation of false membranes are met with in all the domestic animals and may be grouped together as a special class. The collection of these in one group, however, must not be taken to imply that all of these, as met with in the different animals have the same pathology, and are due to one invariable cause. Above all it must not be inferred that they are identical with the malignant diphtheria of the human being. The bacillus diphtheriæ hominis isolated by Klebs in 1883, and proved pathogenic by Löffler in 1884, has not been successfully inoculated upon any of the larger domestic animals, and has not been found in any of the casual pseudomembranous pharyngitis of these animals. The common feature of the group is to be found in the formation of the false membrane, and the fact that a given disease is placed in the group must not be held to apply to any special character, of microbial origin, nor communicability by infection.

PSEUDOMEMBRANOUS PHARYNGITIS IN SOLIPEDS.

Cases of pharyngitis with false membranes have been seen in horses by Delafond, Targue, Rey, Bouley, Riss, Sonin, Robertson, Dieckerhoff and Schneidemühl.

They have been attributed to various causes, as caustic alkalies and acids, the smoke of a burning building (Bouley, Rey, Riss), to an infection which operated on dogs and horses (Robertson), to bacteria and other irritants.

Lesions. The mucous membrane of the mouth, pharynx and even the nares presents active inflammation with branching redness, petechiæ, circumscribed foci of necroses, and false membranes of a grayish, yellowish, reddish, greenish or blackish color. These are formed of a pellicle consisting mainly of fibrine and epithelium, pus globules and numerous cocci, and ovoid bacteria. The false membranes have been found on other parts of the intestinal canal (colon, cæcum); and broncho-pneumonia and pulmonary dropsy have been concomitants. The effect of the toxic products is seen in hæmorrhagic inflammation and infarctions of the kidneys, and in a black color of the somewhat diffluent blood.

Symptoms. Besides the usual phenomena of pharyngitis, there is intense hyperthermia (105° – 106°), hurried breathing threatening suffocation, painful cough roused by the slightest pressure on the swollen throat and often causing the discharge from the nose of shreds of false membrane. Auscultation of the pharynx gives a loud gurgling sound. Deglutition is very difficult and painful, liquids and even solids being rejected through the nose. The face is pinched and anxious and the mouth is often held open and the tongue pendant. Weakness and prostration are marked symptoms from the first, and the walk may be unsteady and swaying. The visible mucous membranes are congested and usually have a more or less deep tinge of yellow. The disease makes rapid progress and may prove fatal under six days. When it takes a favorable turn, recovery and convalescence may be equally prompt.

Unless the expectoration of false membrane is detected, such cases are difficult of diagnosis, though a fair inference may be deduced from the extreme severity of the symptoms, and the unusual degree of prostration which is present. When a pharyngeal speculum, passing through the nose, can be availed of, it may become possible to reach a more definite conclusion.

Treatment. Beside the measures advised for catarrhal pharyngitis (poultice, counter-irritants, laxatives, antithermics, alkalies, etc.), the main reliance must be placed on antiseptics. Persistent inhalations of warm water vapor with carbolic acid, creolin, tar, lysol, camphor or sulphurous acid are in order: also a mixture of one or other of these agents or of boric acid, bisulphite of soda, or salicylic acid in honey or molasses to be frequently smeared on the teeth. One of the best agents is the saturated solution of chlorate of potash in tincture of muriate of iron, of which a drachm may be added to three ounces of water and given every hour or two. Calomel may be injected through the nose during inspiration, by means of an insufflator, care being taken not to exceed the physiological dose.

PSEUDOMEMBRANOUS PHARYNGITIS IN CATTLE.

Most common in calves. Inoculations successful on rabbits, mice and sheep. Bacillus: its cultural characteristics. Predisposing causes. Symptoms: nasal mucosa congested; false membranes; snuffling, wheezing breathing; painful, rattling cough; agonized expression; salivation; bowel disorder. Course. Duration. Lesions; intense congestion and false membranes. Treatment: as for horse; special antiseptics; solvents; anodynes; tracheotomy.

This has appeared especially in calves, and though apparently readily transmissible among the young, it rarely attacks aged cattle. Cadeac and others inoculated it on guinea pigs and rabbits without success. Dammann, on the other hand, had his inoculated rabbits die in twenty-four hours with hemorrhages in the seat of inoculation. Löffler inoculated it hypodermically on mice and produced extensive infiltration of the entire walls of the abdomen, and often of the peritoneum including the surface of the liver, kidneys and intestines, on which was formed a thick, yellowish exudate containing the microbe. Damman claimed to have successfully inoculated the sheep as well.

Causes: Microbe. Löffler found in the deeper layers of the exudate a long delicate bacillus, five or six times as long as broad, and about half the thickness of the bacillus of malignant œdema. Several bacilli were usually joined so as to form long filaments. These failed to grow in nutrient gelatine, or sheep blood serum, but grew readily in the blood serum of the calf.

Beside the specific microbe Cadeac enumerates as predisposing causes: sudden chills, rapid changes of temperature, suppression of perspiration, inhalation of irritant gases, swallowing of irritant liquids, and traumatic injuries.

Symptoms. The nasal mucosa is violently congested, reddened, thickened, and covered at intervals by false membranes which block the normally narrow passages and produce snuffling, wheezing, and difficult breathing. The throat is swollen, and tender, the slightest touch producing a painful gurgling cough which leads to the discharge of mucopurulent matter, shreds of false membrane and even blood. These membranes may be seen on the nose or mouth. There is high fever, rapid, small pulse, cyanosed mucous membranes, pinched countenance, and usually open mouth, pendant tongue and drivelling saliva. There may be either constipation or diarrhœa.

The course of the malady is rapid, death sometimes supervening in 24 to 48 hours. Recovery and convalescence may be prompt, or the disease may last for weeks.

Lesions. The congestion is intense and may invade the mouth, nose, pharynx, larynx and bronchia, with at intervals the patches of yellowish white false membranes. These may be soft and diffuent when recent, and tough and resistant when of longer standing. The deeper layers are often bloodstained. Preitsch has seen them extend to the gullet, paunch and manifolds, and attended with considerable ulceration of the subjacent mucous membrane.

Treatment. This does not differ materially from that recommended for the horse. Among the additional antiseptics employed have been, iodoform, oil of turpentine, sulphide of calcium, silver nitrate and coal tar. To loosen and detach the false membranes ipecacuan and sulphates of soda and magnesia have been largely resorted to. Papayin and pepsin might be tried. Also as anodynes digitalis, morphia, aconite and belladonna. Finally tracheotomy has been employed when asphyxia seemed imminent.

PSEUDOMEMBRANOUS PHARYNGITIS IN SHEEP.

Cause; infected dust on susceptible subject; inoculation. Symptoms; movement of jaws; frothy lips; salivation; viscid nasal discharge; difficult swallowing and breathing; swollen tender throat; extended head; anorexia; cyanosis; open mouth; cough expels shreds of false membrane; asphyxia. Lesions. Treatment; Glauber salts or muriatic acid in water; antiseptic fumigation and drinking water; antiseptics of the pharynx.

Roche-Lubin speaks of this disease as common in flocks, as the result of moving them around for twenty-four hours in a narrow enclosure covered with dust which is raised in a cloud and settles in the fleece so as to increase its weight. The fever and excitement caused by the constant driving and the local action of the infected dust on the respiratory mucous membrane is said to bring about the intense exudative inflammation. It has been seen especially in the spring in young lambs shortly after weaning. Damman claims that he transmitted the disease to sheep by inoculating the diphtheritic exudate of the calf.

Symptoms. There were constant movements of the jaws, with the accumulation of frothy saliva round the lips or the drivelling of this secretion from the mouth, the discharge of a viscid white material from the nose, difficulty of deglutition, hurried, panting, snuffing breathing, swelling and tenderness of the throat, and the occurrence of cough and the discharge of mucopurulent matter whenever it was pressed. The head and neck are held rigidly extended, the eyes are dull or glazed, the appetite is completely lost, the mucosæ red and cyanotic and the animal weak and unsteady upon its limbs. By the third or fourth day respiration has become so difficult that the mouth is held constantly open, the tongue protruded and the painful convulsive cough leads to the expulsion by the nose and mouth of shreds of false membrane. Careful examination of the nose or of the fauces may detect the grayish or yellowish patches of false membrane at an earlier stage. Death by asphyxia is common.

Lesions do not differ materially from those seen in the calf. The inflammation and pseudomembranous exudate may extend as far as the trachea and bronchia in which case the indications of death by asphyxia are clearly marked.

Treatment is the same as for the calf. Tepid drinks slightly acidified with muriatic acid, or the addition to the drinking water of one pound of sulphate of soda to every fifty sheep has been especially recommended. Fumigation with sulphurous acid or chlorine is easy of application in flocks. As alternatives for addition to the water may be named hyposulphite or bisulphite of soda, borax, carbolic acid, or spirits of turpentine. For treatment individually swabbing the throat with antiseptics and dilute caustics, electuaries, and hot poultices to the throat may be tried.

PSEUDOMEMBRANOUS PHARYNGITIS IN SWINE.

Prevalence in herds, in close pens, and in young. Relation to swine plague and hog cholera. Symptoms: sore throat, prostration, hoarse cough, yellowish discharge with shreds of false membrane, pellicles on mouth, fauces, tonsils. Diagnosis from swine plague. Treatment: Isolation, disinfection, antiseptics to throat, febrifuges.

This has long been recognized as a contagious affection, occurring especially where the animals were kept in herds and too often in close and filthy pens. These are more liable in youth than in maturity, partly no doubt because the older animals have already suffered and attained to an immunity. Modern observation has shown that pharyngitis with formation of false membranes is especially common in swine plague, and the present tendency is to refer all such cases to that category. It is however altogether probable that the occurrence of local irritation with the addition of an irritant or septic microbe altogether distinct from those of swine plague or hog cholera, gives rise at times to this exudative angina. Certain it is that septic poisoning with the food is not at all uncommon in the hog, in the absence of these infectious diseases.

The *symptoms* are those of severe sore throat with profound prostration, a hoarse, painful cough, a yellowish discharge from nose and mouth, and great muscular weakness. The base of the tongue, tonsils and soft palate are red and tumid with here and there grayish or yellowish patches of false membranes. The identification of swine plague may be made by the history of the outbreak, the number of animals affected, the tendency to pulmonary inflammation, the enlarged lymph glands, the presence of the non-motile bacillus which does not generate gas in saccharine media, and which readily kills rabbits and Guinea-pigs with pure cultures of the germ.

Treatment will depend largely on the nature of the attack—swine plague or simple pseudomembranous infection. Isolation, cleansing and disinfection will be demanded in both cases. In swine plague all additional precautions to prevent its spread must be resorted to. In the simpler exudative inflammations the antiseptic local treatment and general febrifuge measures will be demanded.

PSEUDOMEMBRANOUS PHARYNGITIS IN DOGS.

Relation to diphtheria in man and horse. Symptoms; fever, prostration; swollen throat; cough; vomiting; false membranes on fauces and tonsils; cyanosis. Treatment: local antiseptics; febrifuges.

Rossi and Nicholski claim that dogs contract diphtheria by swallowing the excrements of diphtheritic infants, but these observations lack confirmation, and the infrequency of such an occurrence argues against it. Robertson records cases of canine angina with false membranes occurring at the same time as a similar affection in the horse. The victims were puppies and the mortality was high. Exact observations are, however, lacking.

The *symptoms* were dullness, prostration, anorexia, a hard cough, swollen throat, vomiting, diarrhoea, and the presence of grayish or yellowish false membranes on the fauces and tonsils. The breathing was difficult and painful and the mucosæ cyanotic.

Treatment has been essentially local, consisting of swabbing with solution of boric acid (1:200), chlorate of potassa, perchloride of iron, or nitrate of silver.

PSEUDOMEMBRANOUS STOMATITIS OF PIGEONS AND CHICKENS.

Contagious and destructive nature of the disease. Mode of extension from the mouth and pharynx. Causes: bacillus diphtheriæ columbarum; its characters: pathogenesis to birds, mice, rabbits, Guinea pigs; dogs, rats, and cattle immune; diagnosis from bacillus diphtheriæ. American disease. Incubation. Symptoms: prostration; wheezing breathing; sneezing; difficult deglutition; false membrane on fauces; necrotic changes in mucosa; perforations; lesions of internal organs; blood infection; nostrils stuffed; bill gapes; lesions on eye, tongue, gullet, crop, intestine; diarrhœa; vomiting. Skin lesions. Course, acute, chronic. Paralysis. Mortality. Prognosis. Diagnosis from coccidiosis, from croupous angina of Rivolta, from aspergillus disease. Treatment: isolation; destruction of carcasses; hatching; destruction of dead wild birds and rabbits; exclusion of living; quarantine of new birds; disinfection; locally, antiseptics by inhalation, swabbing, and internally, iron in water.

This affection prevails in certain countries and causes heavy losses among young pigeons, so that it might with great propriety be included among animal plagues, which should be dealt with by the State. The malady is a local inflammation leading to the formation of false membranes and its usual course is to progress from the mouth and pharynx, to the nasal passages, lachrymal ducts and sacs, the larynx, trachea, bronchia, intestines and skin.

Causes. The essential cause of the disease is held by Lœffler to be the *bacillus diphtheriæ columbarum*, which is a short bacillus with rounded ends, a little longer than the bacillus of fowl cholera and not quite so broad. It is usually found in irregular clusters, especially in the interior of the hepatic capillaries. It is ærobic, non-motile, non-liquifying, and grows on nutrient gelatine, blood serum or potato. In gelatine it forms a white surface layer, and spherical colonies along the line of puncture, which show a yellowish brown tint under the microscope. On blood serum and potato it forms a grayish white layer.

Pathogenesis. The bacillus is inoculable on other **pigeons** and as it usually appears in the young birds in the nest, still fed by the parent bird, it is probable that no inflammation nor abrasion is necessary to make it take. Pure cultures inoculated in the mouth gave rise to the usual local type of the disease. When inoculated subcutem it caused a local necrotic inflammation.

In **mice** subcutaneous injections proved fatal in five days with general dissemination of the bacillus. There are congested and hemorrhagic spots on the lungs, enlarged spleen, and the liver is marbled by numerous necrotic white masses, in the centre of which the capillaries are found to be blocked with the bacilli. This is so pathognomonic that Löffler looks on the inoculation of mice as the best means of diagnosis.

Inoculated **rabbits** showed inflammation in the seat of inoculation and sometimes fibrinous peritonitis and enlarged spleen. Inoculation on the cornea produced a false membrane.

In **Guinea pigs** induration and ulceration occurred in the seat of inoculation but recovery followed in 14 days.

Sparrows inoculated in the pectoral muscles died in three days with yellowish necrotic tissue highly charged with bacilli.

Inoculation of the **chicken** by Löffler and Megnin produced a circumscribed redness which soon disappeared. On the other hand Krajewski, Colin, Loir and Ducloux seem to have inoculated chickens successfully, and Cadeac says that the cultures are infecting for **sparrows, pigeons, turkeys, chickens** and **ducks**. It rests uncertain therefore whether the pseudomembranous pharyngitis of hens is a distinct disease as alleged by Löffler and Megnin or if the chickens used by these observers were not already immune by reason of a prior attack.

Löffler's experiments showed that *dogs* and *rats* were immune. Loir and Ducloux failed to infect cattle.

In infected dove-cots a comparative immunity is attained by the older pigeons, which continue to

harbor the germ, but do not suffer materially from its presence. They however communicate it to the susceptible young in the milky secretion produced in the crop and with which they feed them, and these accordingly perish in large numbers. Thus pigeons that are themselves in fine condition become the propagators of the bacillus to the more impressible.

Sparrows and other *small birds* are also held to be common propagators of the germ, and if they too can secure an individual immunity and yet harbor the bacillus, their passage from yard to yard may be attended with great danger. The grains soiled by their bills and not swallowed are common media of transmission.

Loir and Ducloux found the affection transmissible between man and pigeon. The identity of the bacillus with that of genuine diphtheria in man appears to have been thoroughly disproved by the observations of Roux and Yersin.

The following differential characters have been noted:

<i>Bacillus Diphtheriæ (Klebs-Löffler).</i>	<i>Bacillus Diphtheriæ Columbarum</i>
1. In gelatine cultures grows only above 23°C.	1. In gelatine cultures grows at 15–17°C.
2. Kills Guinea-pig and dog.	2. Guinea-pig and dog nearly immune.
3. Mice immune.	3. Mice usually die with hepatic necrosis.
4. Does not grow on potatoe.	4. Grows luxuriantly on potatoe.

It may be accepted as demonstrated that the common diphtheria of birds is essentially distinct from the genuine diphtheria of man, and that when such diphtheria of the bird is conveyed to man as has been often alleged (Richter, Gips, Bonig, Gerhart, etc.), it is one of the forms of pseudo diphtheria that is produced, and not that which is caused by the Klebs-Löffler bacillus. Dr. V. A. Moore, who has cultivated specimens of the bacillus diphtheriæ Columbarum obtained from Germany, considers the germ as belonging to the group of the bacillus coli communis, and as not the cause of the chicken diphtheria in America. Further investigation must settle whether the bacillus diphtheriæ Columbarum is the one cause of this affection in Europe, and what is the microbial cause or causes of the disease in America.

Incubation. This is very variable. False membranes may form in twenty-four hours in some cases; in other cases they may be delayed from four to fourteen days (Colin, Babes, Puscarin, Marinescu).

Symptoms. There is dullness, prostration, sunken head, ruffled feathers, altered hoarse voice, drooping wings, wheezing breathing, difficult deglutition, sneezing, and patches of dark red congestion in the fauces covered with a thin film, at first translucent, but soon becoming dense, adherent, opaque, whitish or yellowish. As it becomes older this deposit becomes granular, wrinkled, dry and friable. It is more adherent in chickens than in pigeons and causes bleeding when detached. Necrotic changes may take place in the mucosa leading to considerable loss of tissue, and even to perforations of the soft palate, pharynx or œsophagus. It may remain circumscribed by the region of the mouth and end in an early recovery, or it may extend to the organs of the chest and abdomen, or the germs may proliferate largely in the blood and induce fatal results. On the other hand it may become subacute or even chronic, and, as already noted in the case of the parent pigeons, it may persist as an infecting disease without materially injuring the general health of a comparatively immune animal.

The affected *nasal passages* become filled by frothy liquid and blocked by false membranes, so that the bird is driven to breath through the open mouth. The skin around the nares, and eyelids and the cavity beneath the eye may be covered with the false membrane, by the increase of this product the bones may be driven out of place, so that the palatines press downward, the eyeball is pressed outward and the root of the beak may seem swollen. The false membranes that form on the skin or reach the surface are soft, creamy, cheesy, or dry, granular and friable.

When the *eye* is specially affected there are swelling of the lids, profuse lachrymation, closure of the lids by adhesion, and formation around their borders or on their inner surface and on the

membrana nictitans of false membranes which press the lids outward more or less unevenly, and may be easily recognized when the lid is everted. The cornea and even the interior of the eye may suffer, leading to perforation, internal tension, and in some cases atrophy, with permanent blindness.

The *tongue* may suffer on the tip as in *pip*, or on its dorsum, from which the disease extends to the larynx, trachea and even the air sacks, which become filled with false membranes, that are coughed up, and decomposing in the mouth, add to the infection and foetor. Dyspnœa and cyanosis of comb and wattles are marked features.

The extension may take place downward along the alimentary track, the false membranes forming on the *gullet* or *crop* and interfering with swallowing or digestion, or on the intestine and determining a foetid, often greenish or bloody diarrhœa with indications of false membranes. Vomiting may be a marked symptom.

The *skin* is usually attacked secondarily around the margin of the beak, the eyelids, the nares, the ears, the comb, the wattles, the anus, but it may develop at any point where the infecting material has touched an abraded surface.

Trinchera found that in acute cases the acme was reached in fifteen days after which improvement might be looked for. A chronic form affecting the gullet might however persist indefinitely in pigeons without proving incompatible with good health.

Paralysis of the wings or limbs may remain after the healing of the local lesions.

Mortality. Prognosis. The disease is very fatal to both pigeons and chickens, 50, 70 or even 100 per cent. perishing when a flock is attacked for the first time. In flocks that have previously suffered, on the other hand, a large number are practically immune, and even if they contract the disease it assumes a mild form, and they survive but may retain the germ and continue to communicate it to others. Even the young of such immune flocks suffer less severely, coming as they probably do from less susceptible and therefore surviving birds, or having already perhaps contracted a mild (non-fatal) type of the disease from their parents.

Differential Diagnosis. From *psorospermiosis (coccidiosis)* it is distinguished by its origin on the mucous membranes, and not on the skin, the skin lesion being a secondary one. In psorospermiosis the primary lesion is usually on the skin, from which it extends to the mouth and especially along its floor. In psorospermiosis the morbid deposit assumes the form of rounded warty-like masses, on comb or wattles; is easily propagated by inoculation, is promptly checked by antiseptics, does not tend to produce internal extension nor generalization, and on microscopic examination shows numerous spheroidal coccidia intermingled with the epidermic cells and possessing amoeboid movement. By virtue of this automatic movement they make their way between and into the epidermic cells in which they multiply.

From the *croupous angina* of Rivolta it is distinguished by the absence of the infusoria (*monocercomonas gallinæ*) to which he, Delprato and Pfeiffer attributed that affection. The *monocercomonas* is a flagellate organism 14μ to 25μ in length and 5μ to 7μ in breadth. Its rounded end bears one flagellum as long as the body, and its acute end three flagella which give it active motions. These are found in the yellowish white swellings of the mucosa, which vary in size from a millet seed to a pea, covering a hyperæmic spot and composed of epithelial cells, blood globules—white and red,—leucocytes, granules and the infusoria. The false membrane is remarkable for its lack of consistency and its tendency to invade the mouth and gullet rather than the air passages. These infusoria are not colored by picrocarminate of ammonia, but stain by methyl-violet and then appear as round or slightly irregular hyaline bodies.

From *aspergillus disease of pigeons*, by the absence of the characteristic, miliary, white nodule of that disease showing caseated contents intermixed with an abundant mycelium of *aspergillus fumigatus*. The aspergillus disease attacks especially the mouth but may also implicate the gullet, lungs, liver, intestine and kidneys. The microscopic examination of the exudate is conclusive, by reason of the presence of the bacillus diphtheriæ columbarum, and the comparative absence of the filamentous mycelium.

Treatment. This is mainly prophylactic. The first step must be to separate the sick and healthy,

destroying the former, or shutting them up in a special enclosure apart from all other birds. In the case of valuable chickens, their eggs may be set under other hens and the young raised apart from the suspected flock. This may even be attempted in pigeons, the common eggs being removed and the valuable ones put in their place under a healthy sitting dove. In the case of pigeons that have been recently through the disease they should be kept strictly by themselves, even though they may appear to have regained perfect health. The dead bodies must be burned or deeply buried. Sparrows and even rabbits dying in the vicinity must be similarly disposed of, and where the disease prevails sparrows and small birds may be exterminated as probable bearers of infection.

The purchase of strange birds must be carefully guarded, none being taken that show weeping eyes, nasal discharge, labored or wheezing breathing, and all new birds should be placed by themselves in quarantine for ten to fifteen days. Finally a thorough disinfection of the place where the sick have been is of first importance. Thorough cleaning of the poultry house, followed by a coat of white-wash, every gallon of which contains four ounces of chloride of lime, or one drachm of mercuric chloride will usually prove effective. The poultry runs should be liberally sprinkled with a solution of sulphuric or hydrochloric acid, one part to 1000. The same may be used on the building, which may further be fumigated by burning sulphur.

Poultry shows should be kept under the most rigorous sanitary supervision.

Curative treatment is only profitable in the case of specially valuable birds, and even then only, as a rule, when the disease is confined to the nose, mouth, larynx and pharynx. The affected parts may be brushed with a solution of chloride of iron (1 dr. of the tincture to 1 oz. water), nitrate of silver (2 grs. to 1 oz. water), sulphide of calcium ($\frac{1}{2}$ dr. to 1 oz. water), tannin (10 grs. to 1 oz.). Tincture of iodine may be applied direct, or a solution of carbolic acid or of creosote or creolin (1 part to 50) will often succeed. Thomassen recommends the removal of the false membranes and the application of boric acid followed by dry sulphur. Benoist says the majority recover when made to inhale the fumes of oil of turpentine evaporated at a gentle heat twice a day.

As internal medication, or to correct the intestinal affection, sulphate of iron may be dissolved in the drinking water, or salicylic acid may be given in pill form with molasses.

CHRONIC PHARYNGITIS.

Sequel of acute: or subacute from the first. Due to œstrus, in cattle to summer catarrh, tubercle or actinomycosis. Lymphatic horses predisposed; attends chronic indigestion; in swine tonsillitis. Symptoms: chronic cough, easily roused, wheezy or mucous; nasal discharge; low condition; lack of spirit. Lesions: congestion; softenings; erosions; cicatrices; tonsillitis; abscesses; specific deposits. Treatment: hygienic; antiparasitic; astringent; antiseptic; derivative; counter-irritant; tonic inhalations and electuaries. Bitters. Iron.

Causes and Nature. Chronic pharyngitis in animals may be a simple continuation of the acute, in a milder form, or it may assume a subacute or chronic type from the first and never rise to the intensity that would characterize the acute. It may be a simple catarrhal affection or it may become more or less follicular or glandular. Again in horses it is not infrequently a result of the hibernation form of the œstrus (bots) attached to the delicate pharyngeal mucosa, and in cattle from the extension of the chronic summer catarrh, or from the local development of tubercle or actinomycosis in the walls of the pharynx or in the adjacent lymph glands. Horses of a soft, lymphatic constitution, with a heavy coat, confined in close warm stalls, and which perspire abundantly are especially liable to the affection. It may also be an accompaniment and result of chronic gastric indigestion. In swine the affection is commonly associated with tonsillitis.

Symptoms. In many cases the main symptom is a chronic cough which is aroused by any cause of irritation, feed, especially dry or fibrous fodder, cold drinking water, sudden passing from the hot stable to the cold outer air, reining in, pressure on the throat, or sudden active exertion. If the cartilages are calcified it may be impossible to rouse the cough by pressure. The cough is often dry and wheezy, rather than soft and gurgling as in the second stage of acute pharyngitis, and is repeated several times paroxysmally. In the intervals there is more or less stertor or wheezing, or a distinct rattle especially when the neck is curved by drawing the nose inward. Deglutition may be interfered with but this shows most with the first swallow, which in the case of liquids may be returned through the nose, whereas those that follow go down without difficulty. A lateral swelling of the parts above the larynx or a bulging of the parotids is not uncommon. Discharge from the nose of a mucopurulent character is usually present, but often so scanty as to be overlooked. There is usually loss of flesh and lack of vigor even if the subject is well fed.

Lesions. In the simple catarrhal form the mucous membrane of the lateral pharyngeal walls, the posterior pillars of the palate and the back of the soft palate, is red, congested, with arborescent vessels, thickening, and puckering into rugæ. The epithelium has lost its translucency, become opaque and granular, and its desquamation in spots and patches may leave erosions, ulcers more or less deep, and white drawn cicatrices. When the follicles and mucous crypts are involved (follicular) they stand out like millet seed, peas or beans, and may show ulceration or minute abscess. In pigs especially, tonsillitis is liable to be present, and the tonsillar follicles are filled and distended with tenacious mucous, a caseous granular *debris*, or even a cretaceous material. In the vicinity of the tonsils, minute abscesses may exist in or beneath the mucosa.

Ulceration may be the result of tubercle, glanders, actinomycosis, aspergillus, sarcoma, or some local infection, and attendant symptoms of one or other of these diseases will guide the diagnosis. Thus in tubercle there will be the implication of the adjacent lymph glands and usually of distant ones; in glanders the deposits in the nose, submaxillary lymph glands and lungs will enable one to diagnosticate; in actinomycosis the hardness of the neoplasm and the presence of the yellowish tufts which present under the microscope the concentrically arranged club-shaped elements, will show its nature; and in sarcoma or carcinoma the structure of the new tissue will decide its character. The pharyngeal muscles are the seat of granular or fatty degeneration or of fibroid change. Friedenreich speaks of a fold from the vault of the pharynx which had nearly closed the passage and had killed the horse by inability to swallow.

Treatment. Chronic pharyngitis is usually a very obstinate affection and demands careful hygienic as well as medicinal treatment. Hot, foul stables, unduly thick coats, unwholesome food, irregular feeding, excessive meals at long intervals, overwork, undue exposure to cold and wet, lack of sunshine or of grooming are to be corrected. Next, the removal of mechanical irritants such as pharyngeal bots, actinomycosis growths, etc., will be in order. Then the use of astringents and

antiseptics internally and of derivatives externally will be demanded. An occasional embrocation of mustard, or the application of ammonia and oil, will often serve a good purpose, and in obstinate cases the hot iron in points will sometimes prove effective.

Internally the inhalation of the fumes of tar, carbolic acid, creolin, oil of turpentine, or of burning sulphur kept up continually or frequently repeated. Giving all drink in the form of tar water will often have a good effect. Electuaries made with boric acid, salicylate of soda, ammonium chloride or iodide, borax, with honey, molasses, liquorice, Iceland moss, or gum arabic will often prove beneficial. Agents that stimulate the mucosa may follow, such as balsams of Peru or Tolu, copaiba, cubebs, pilocarpin, wild cherry bark, or these may be combined with the former. Finally a course of tonics are usually of the first importance; iron sulphate, copper sulphate, arsenious acid, arsenite of strychnia may furnish examples.

DEPRAVED APPETITE. STUMP SUCKING. PICA. LICKING DISEASE.

Common features of group. Ruminants; depraved appetite; objects swallowed: hair balls. Sheep eating wool in winter. Pigs eat bristles. Puppies swallow marbles, etc., wantonly. Solipeds swallow hair, plaster, earth, sand, and lick manger or rack. Fowls eat their feathers. Causes: soil exhaustion, lack of lime, soda, potash, phosphorous; relation to osteo malacia; granitic or sandy soils, peat, muck, causative; digestive disorder; faulty food; yearly breeding and heavy milking; constant stabling; dry seasons. Course: chronic. Lesions; emaciation; anæmia; serous exudate; catarrh of the bowels. Treatment: soil; good fodder; salts of soda, potash and lime, phosphates; tonics; apomorphine. Wool eating; example: digestive disorders; emaciation. Treatment: open air; good fodder; salts of the bones and soft tissues; clip nurses; apomorphine.

Definition. We have here a class of morbid habits, which cannot be referred to any constant lesion or group of lesions, and which appear in certain cases to result from example and to constitute nothing more than a bad habit.

Symptoms. *Ruminants* without any appreciable cause, lick the clothes of their care-takers, chew and swallow articles of clothing of all kinds, bones, old shoes, gloves, socks, cuffs, collars, small forks, pocket-knives, nails, wires, needles, coins, stones, lumps of clay, hair, which may give rise to secondary troubles of a more or less serious kind. Pregnant cows are especially subject to this infirmity. The small pointed objects like pins, needles, ends of wires, etc., which are mostly taken by accident with the food are especially apt to be entangled in the alveoli of the reticulum and make their way to the heart, with fatal effect, or through the abdominal walls creating a fistula. Hair aggregates with saliva, mucus and phosphates, to form balls in the first two or three stomachs. Other indigestible objects may also become encrusted and prove sources of irritation. Licking the skin of another animal is doubtless at times encouraged by the taste of the salts of perspiration, but in other cases it has all the appearance of a mutual kind service as the cow with itching head will walk up and present it to its fellow which rarely fails to respond to the invitation. Stump licking is not uncommon.

Sheep shut up in the winter get in the habit of chewing each other's wool, thus virtually depilating their fellows and accumulating wool balls in their stomachs.

Pigs when running at large eat human fæces often infecting themselves with the cysticercus cellulosa, and devour their own or their fellows' bristles, which form ovoid and irritating aggregations in the stomach.

Puppies are proverbial for swallowing every small object that comes in their way, coal, pebbles, marbles, leather, hair, etc., with the result of inducing nausea and vomiting, or more seriously, wounds of the stomach, gastritis and enteritis. In older dogs the habit is more likely to imply rabies.

Solipeds will lick and swallow each others hair, eat off the hair from each other's tails and manes, eat their clothing, lick the wall plaster, earth or sand, and even the manger or rack. The last named habits are usually connected with disease.

Fowls can digest almost anything they swallow, but if they take to picking their feathers, they create serious injury to the skin and indirectly to the general health.

Causes and Nature. In general terms it may be said that the causes of depraved appetite are very numerous, so that the trouble must be looked upon as a symptom of many morbid conditions in place of a disease *sui generis*.

Heredity has been invoked as a cause, mainly, it would appear, because the disease appears enzootically on certain exhausted soils, or in herds kept in the same unhygienic conditions. In such cases the real cause is usually to be found in faulty conditions of soil, water, buildings, food, etc., on the correction of which the trouble disappears. When, however, from a long continuance of unhygienic conditions, a weakness of constitution is transmitted from parent to offspring, such hereditary debility may be accepted as a predisposing factor.

An exhausted soil, lacking especially the elements of lime and phosphorus, is a common cause, though by no means the only one. Nessler who analyzed the hay and water, furnished to cattle suffering from this disease in the Black Forest found a notable absence of the soda salts. In others in which osteo malacia was the prominent symptom the lack was in phosphate of lime as well. In the nature of things the soil that has been continuously cropped to exhaustion is robbed of both

earthy and alkaline salts, and the animals fed on its exclusive products suffer not only as regards the nutrition of the bone, but also of the soft parts. Hence Trasbot says that in osteomalacia, pica is never absent. Roloff and Röhl hold that it is the first symptom of osteomalacia. In South Africa where the land has been cropped with oats year after year without manure and as long as it will bear, the disease became prevalent in the street car horses fed on the oats, and was corrected by the addition of phosphates, or phosphate bearing food, to the ration. In the older dairying farms of New York which have been kept under grass for a great length of time, and all the milk products sold off, depraved appetite in all its forms is quite frequent. Where the land is originally light and sandy and naturally deficient in lime, osteomalacia is often a concurrent disorder. The two conditions may however occur independently of each other, and especially may pica appear alone, in keeping with the greater solubility of the soda and potash salts and the readiness with which these can be washed out of the soil, while the less soluble lime salts in part remain.

Lemcke, Haubner and Siedamgrotzky attribute the disease to a nervous disorder. Lemcke indeed traces the disorder to a lack of phosphorus, and claims that osteomalacia only supervenes where the rheumatic diathesis is also present.

It may be shortly stated that the disease prevails especially on granitic or sandy soils, or on those which are mainly composed of organic debris (peat, muck). Limestone soils and those which contain any considerable proportion of potash or soda are usually exempt.

Digestive disorder though starting from a different point may tend to the same end. A hyperacidity of the stomach has been observed to coincide with the malady, and by interfering with easy and normal digestion, it may stand in the way of such assimilation as is necessary to vigorous health.

Faulty food operates in a similar manner. The exhausted soils, and their products deficient in alkaline and earthy salts have been already referred to; we must also note the evil effect of fibrous fodders, the main nutritive elements of which have been washed out by intemperate weather after they were cut, the rank aqueous products of wet or swampy soils, the fibrous and siliceous plants (rushes, carex, equisetums, etc.) which grow on poor, wet or soured soils, the innutritious and fermented products of beet sugar factories, and generally the spoilt food which has undergone fermentation.

Yearly breeding and constant milking, by undermining the general health, predisposes so strongly that in many cases the affection is seen in dairy cows, while oxen and young cattle escape. The last period of gestation when the demands for the growing calf are greatest, is the period of especial danger.

Permanent stabling which denies the invigorating influence of sun, exercise and pure air contributes toward the general debility and therefore, in animals that are closely stabled for the winter the spring is especially to be feared, when compulsory inactivity, poor feeding, gestation and milking have combined to reduce the system.

Dry seasons have been noticed to increase the affection manifestly by reducing the supply of food.

Course. The affection is chronic and unless arrested by the supervision of more favorable conditions, may last for a year or more. Spontaneous recovery may set in when turned out to pasturage and open air life, and especially if a rich grain feeding is added. Without change in the conditions however, the tendency is to a fatal result.

Lesions. The victims of the disorder are emaciated, the fatty tissue contains a yellow serum, there is little blood, and that is thin and watery and coagulates loosely, the muscles are pale and flabby, and the gastro-intestinal mucous membrane is the seat of catarrh.

Treatment. To treat rationally and successfully we must adapt the measures to the obvious causes. When the soil has been scourged and exhausted, a change of pasture, and of land used for hay or soiling crops is the first consideration. If these cannot be secured then grain and seeds rich in protein, and alkaline and earthy salts should be added to the ration. Wheat bran, middlings, peas, beans, cotton seed meal, linseed meal, rapeseed may be named among available resorts, or in their absence, daily doses of phosphate of lime, and sodium chloride or bicarbonate, or potash salts

may be allowed, or even bone dust.

If imperfect digestion is a manifest factor, sodium chloride, or potassium chloride, calcium phosphate, iron and bitters will serve a good end. In hyperacidity, limewater, chalk, or magnesia may be given. If the digestion is torpid, hydrochloric acid with bitters may be resorted to.

Feser and especially Lemcke strongly recommend apomorphia. It is used hypodermically in doses of 2 grains for horse or cow repeated daily for three days.

Secondary Symptoms in Wool-eating Lambs. Lambs from two to six weeks old especially such as suck ewes with woolly udders (merino, Cotswold) first swallow the wool inadvertently, and then acquire a liking for the saline matters in the abundant yolk (merino), till finally the accumulating wool balls produce digestive and nervous disorder and a craving for the indulgence. Thus the breed must be considered in estimating the symptoms. For the same reason the wool about the hips or elsewhere soiled with salts of the urine or liquid fæces prove attractive to the victim. The proximity of other wool eaters is another cause which starts others to follow the bad example. The general conditions of debility, exhausted soil, and the absence of alkaline and earthy salts must be borne in mind. So too with prolonged confinement indoors in winter, the absence of invigorating exercise and the restriction of the animals (dams) to food which is deficient in saline matters.

Beyond the mere eating of the wool and the destruction of fleeces, the lambs do not usually suffer seriously. But if the consumption of wool is excessive the accumulating balls of the size of marbles in the stomach, and the blocking of the pylorus and small intestine, may give rise to intermittent constipations and diarrhœas, deranged digestion, muco-enteritis, mucous covered stools, loss of condition, emaciation and retarded development.

Treatment consists first in the securing of a more healthy regimen. This is but one of the evils of the close winter confinement of an animal preëminently adapted to freedom and exercise. Turning out in a wide range, especially if pasture is available, is a prime consideration. The separation from the flock, of the first wool eaters, will check the propagation of the vice by imitation. Food that is defective in one or more constituents must be supplemented by that which will correct the deficiency. Salt, potassic salts and above all phosphate of lime or bone meal will sometimes benefit. May recommends the separation of the lambs from the ewes except when nursing, three times a day. Finally Lemcke claims for apomorphia the same curative effect as in other animals. The dose is 2 grains, subcutem, as in the cow and may be repeated three days in succession.

LACERATION OF THE PHARYNX.

Trauma of pharynx from objects swallowed; from whip or other instrument in choking; lesions. Symptoms: swelling; rapidly extending; dysphagia; salivation; retching; dyspnoea; roaring; asphyxia. Treatment: as for pharyngitis; open pouch, suture laceration; use antiseptics; liquid diet.

Laceration of the velum palati has been already referred to, and the remaining walls of the pharynx sometimes suffer in the same way and from identical causes. Pins, needles, and other sharp pointed bodies taken with the food sometimes perforate the walls and determine an advancing ulceration which furnishes a way for their escape externally in the region of the throat. In other cases a rigid staff, a whip, or even a probang introduced to overcome choking, is forced through the walls of the pharynx forming a pouch for the accumulation and septic fermentation of ingesta, and extensive ulcerative and gangrenous lesions.

Lesions and Symptoms. These depend mainly on the extent of the laceration. If there is a mere abrasion, superficial laceration or prick of the mucosa, it determines a prompt inflammation, with exudation which covers or closes the wound and a speedy healing may ensue. When, however, the whole thickness of the mucosa has been extensively lacerated and a pouch has been formed beneath it, it becomes filled with decomposing mucus and ingesta, and the resulting septic products determine ulceration, abscess, or gangrene. The result is too often a general and fatal septic infection.

In the milder forms there are only the common indications of a moderate pharyngitis. In the more severe form, the throat swells at first on the lacerated side and later all around. This swelling soon fills the intermaxillary space and extends over the face and the entire head. From the first, deglutition is extremely difficult or impossible, liquids are returned through the nose and saliva flows abundantly from the mouth. Retching is not uncommon and saliva mixed with alimentary matters is discharged by the nose (solipeds) or mouth (other animals). The swelling of throat and head has a doughy, oedematous feeling, it is very tender, and soon causes rattling, wheezing breathing, roaring, dyspnoea and asphyxia.

Necropsy shows the general oedematous exudate, the laceration of the pharyngeal walls, and the collection of debris and pus in the lacerated cavity. The pus may have extended between the muscles following the course of the gullet and trachea as far as the chest. Extensive patches of necrosis may also be shown. *Treatment.* In the slighter cases the ordinary treatment for catarrhal pharyngitis is demanded. In the more severe the lesions are so redoubtable and their progress so rapid that a fatal result is virtually inevitable. As a desperate resort the septic pouch may be opened from without, its contents removed, the pharyngeal wound sutured if possible, and a thorough irrigation with antiseptics (acetate of aluminium solution) employed at frequent intervals to check if possible the septic process. The animal should be fed with well boiled milk or other liquid which will not add to the fermentation, and this may be given through a stomach tube, or by the rectum when deglutition is impossible.

PARALYSIS OF THE PHARYNX

From nervous bulbar lesion, or toxic from the pathogenic bacteria. Transient or permanent and fatal. Symptoms: dysphagia: liquids expelled by nose or enter lungs. Inhalation pneumonia. Facial palsy. Roaring, laryngeal thrill. Atrophy. Gangrene. Treatment: remove cause: combat bulbar hyperæmia and cephalic congestion: cold: derivatives, electricity: blisters: antiseptics.

This has been described as a rare affection, yet it is often a marked symptom of cerebro spinal meningitis, and has been observed in infectious pneumonia, and influenza of the horse (Cadeac, Palat) as well as in rabies, and traumatic injuries of the brain.

The existence of the condition usually implies disease of the bulb at the roots of the vagus and glossopharyngeal nerves, or swellings affecting these nerves or the sympathetic along its course. The morbid condition may be transient in which case a speedy recovery may follow, or it may be permanent and end fatally.

Symptoms. Swallowing is impossible and the animal refuses food and drink or if the latter is forced on him it is rejected by the nose or mouth when the head is lowered or still worse, it enters the larynx and descends into the lungs. The larynx innervated by the same trunks is usually involved and the alimentary solids and liquids determine gangrenous bronchitis and pneumonia, with labored breathing, foetid breath and violent dyspnœa. In other cases the facial nerve is involved, the nostrils and lips are flaccid on one or both sides, and the eyelids and ears may droop. There may be snuffling breathing from the closure of the alæ nasi, or roaring from the approximation of the arytenoids and vocal cords. If the affection is unilateral the difficulty of breathing is greatly diminished and even deglutition may be effected with some effort. There is usually, however, the obvious unilateral paralysis of the face, and especially of the larynx, with the distinct thrill, during inspiration, conveyed to the finger placed on the larynx.

Complications in the form of gangrene and atrophy of parts supplied by the same nerves, or those adjoining the pharynx, have been recognized in different cases.

Treatment. This must depend on the obvious cause of the affection. If due to an infectious disease the first attention must be given to that. If due to tumors or abscesses pressing on the nerves they may be removed. If there is bulbar hyperæmia or effusion attention must be devoted to derivation and other means of combating that. Cephalic congestion and heat may be met by cold applications. Derivation toward the bowels may be secured by eserine, pilocarpin or physostigmine administered subcutem. Reabsorption of exudate may be sought by pilocarpin, or diuretics—the latter administered by the rectum. Electricity in weak current may be tried when the acute febrile symptoms have moderated, accompanied by hypodermic injection of strychnia (2 grs.). Frictions around the throat with essential oils or even a cantharides blister may be used to advantage. Antiseptic washes may be injected into the mouth,—vinegar, boric acid, borax, sulphite of soda. Finally the animal must be nourished by rich gruels and soups given by the rectum, or in the smaller animals by the stomach tube.

TUMORS OF THE PHARYNX.

Varieties of neoplasms. Malignant invade adjacent and distant parts. Symptoms: sore throat; stertor; dyspnœea; dysphagia. In cattle, lymphadenoma, tubercle, actinomycosis. Cause cough, ptyalism, discharge, foetor, dysphagia. Dogs and pigs vomit. Treatment: medical; surgical.

Tumors of the pharynx are not common in the *horse* yet they occasionally appear as either primary or secondary neoplasms. They are of various kinds, as, epithelioma (Labat, Mathis), carcinoma (Casper, Dupuy, Mathis), sarcoma (Siedamgrotzky, John), lipoma (Fricker), cystoma (Degive) and melicerous (Lesbre). The malignant forms tend to invade the surrounding tissues and spread widely into the nose, palate, tongue, pharyngeal glands, and, secondarily, into the small intestines. The simple tumors like the lipomata and fibromata tend to detach themselves and hang by a pedicle (see pharyngeal polypi). The same is true of the melicerous cyst which originating in an obstructed mucous or salivary duct projects as a mass as large as a hen's or pigeon's egg into the fauces or pharynx.

Symptoms. There are symptoms of intense sore throat with stertor continuous or intermittent, increasing to dyspnœa at intervals or on exertion. In case of pediculated tumors these attacks correspond to the displacement of the tumor into the glottis. Deglutition is difficult or impossible, liquids or even solids returning through the nose, mixed with mucus and at times tinged with blood. Nasal discharge and ptyalism are present.

In **cattle** pharyngeal tumors may be of the same nature as mentioned for the horse, but they are far more frequently lymphadenoma, and above all tubercle or actinomycosis. Zimmer found that of seventy-three such tumors fifty-four were actinomycosis.

The *symptoms* are wheezing breathing, cough, nasal discharge, ptyalism, bleeding from the nose, foetid breath, difficult deglutition attended by cough and rejection of the ingesta through the nose, and the presence of a solid body in or on the pharynx which may be manipulated from without or within and tends to increase in size.

In **dogs** there are the same general symptoms with vomiting. The vomited material is usually remasticated and swallowed. The swelling in the pharynx can be felt from without, or seen through the open mouth. The tonsils are usually enlarged. Pressure on the pharynx or gullet produces instant regurgitation.

Treatment consists in the removal of the tumor when possible. Malignant growths and multiple tumors are not favorable for treatment. Actinomycosis can be treated throughout by iodides, or these may supplement the surgical measures. In the short-faced animals an ecraseur, or a wire-snare passed through a tube may be employed. (See pharyngeal polypi).

ESOPHAGITIS. INFLAMMATION OF THE GULLET.

Causes: Alimentary and therapeutic; parasitic and accidental traumatisms; mechanical irritants; acrids; caustics; parasites—gongylonema, coccidia, spiroptera. Extension inflammations. Lesions: hyperæmia; epithelial degeneration and desquamation; erosion; petechiæ; suppuration; fibroid contraction; sacculation; polypi. Symptoms: dysphagia, difficult deglutition; eructation; cough; upward wave motion in jugular furrow; colicky pains; probang arrested; fever. Treatment: liquid or semi-liquid food; for caustics, antidotes; cold water; ice; antiseptics; derivatives; open abscess; potassium iodide.

Causes. This usually arises from injury to the mucous membrane and in the milder forms remains confined to this structure. In the more severe, it extends to the muscular coat and even to the periesophagean tissues. The causes may be divided into alimentary and therapeutic irritants; parasitic or accidental traumatisms; and extension of inflammation from the pharynx or other adjacent part.

Among *irritants* taken as food, may be named hot mash, bolted by a hungry and gluttonous horse, and temporarily arrested in the gullet by reason of the resulting irritation of the mucous membrane. In other cases, coarse fibrous fodder is bolted without previous mastication, and scratches and abrades the œsophagean mucosa leading to transient or progressive inflammation. In other instances diseases of the teeth, jaws, temporo-maxillary joint, or salivary glands prevent the necessary trituration of the food, and it is swallowed in a rough, fibrous, or even a dry condition. Again the impaction of a solid body (turnip, apple, potatoe, egg) or of a quantity of finely divided grain or fodder so as to obstruct the lumen of the gullet, is an occasional cause. The density of the epithelium reduces these dangers to the minimum, yet a too rough morsel, or an undue detention of the less irritating material will determine hyperæmia and even inflammation and infective invasion. Acrid and irritant vegetables in the food are less injurious when thoroughly insalivated, as their contact with the œsophagean walls is then very slight and transient.

Irritant and caustic chemical agents given for therapeutic purposes, attack the mouth, pharynx and stomach, more severely than the gullet through which they are passed with great rapidity. In some cases, however, the agent will adhere by reason of its powdery, gummy or balsamic character and will then act as a direct irritant. Solutions of caustic alkalies (weak lye) given to correct acid gastric indigestion in the horse, and ammonia to remedy tympany in cattle, when insufficiently diluted, will dangerously attack the œsophagean mucosa.

Parasitic irritation is not so common here as in other parts of the intestinal canal where the contents are longer delayed and are passed with less friction, yet certain parasites are found in this region and may even produce considerable irritation. The gongylonema of the thoracic œsophagean mucosa of ruminants and swine are apparently harmless. The psorospermia of the œsophagean muscles of the same animals are alleged to cause œdema of the glottis, asphyxia and epilepsy. The spiroptera microstoma of the horse has in one instance known to us caused extensive denudation of the muscular coat within a foot of the cardiac end of the gullet. Finally we have found bots hooked on to the œsophagean mucosa close to the cardia, causing much irritation and spasm. The spiroptera sanguinolenta is often present in chambers hollowed in the œsophagean mucosa of the dog.

Traumatic causes appear in the form of contusions and bruises from without, but much more frequently from foreign bodies, and probangs operating from within. The use of a whip or of a rope without a cup-shaped end for the relief of a choked animal. Short of the occurrence of laceration this often produces contusion and abrasion which results in local inflammation. Even the too forcible dislodgment of a solid body by a probang of approved pattern, may bruise and scratch the gullet when the seat of violent spasm. Pins, needles, wire, thorns and other sharp bodies are liable to do serious damage during their passage in an ordinary bolus and when they transfix the mucosa violent infective inflammation may ensue.

Extension inflammations from the throat, and from phlegmous, abscesses, tumors, etc., in the

jugular furrow need only be mentioned in this connection, as the primary disease will be clearly in evidence.

Lesions. These are usually circumscribed when due to a traumatic injury and extended when caused by caustics or irritants. The affected section is swollen, and surrounded by some serous effusion. When the muscular coat is involved it is often paler than normal, and microscopically shows extensive granular and fatty degeneration. The mucosa usually sloughs off its epithelial layer, sometimes over an extensive area (thoracic portion, Renault; whole gullet, Bertheol), and the exposed raw surface is of a deep red or violet. When the epithelium is not shed, it is infiltrated, swollen and friable breaking down under the slightest manipulation. Petechiæ and slight blood extravasations are abundant, and diffuse suppuration is not uncommon. In traumatic injuries necrosed areas are found in the muscular and mucous coats. Strictures, dilatations, and polypoid growths are liable to follow as sequelæ.

Symptoms. These usually manifest themselves from two to four days after the operation of the cause. There is much difficulty in deglutition, the effort to swallow either solids or liquids causing acute suffering, with extension of the head on the neck and strained contraction of the facial muscles. If the liquid succeeds in passing the pharynx, it is arrested at the seat of inflammation and regurgitated through the nose and mouth, or in solipeds through the nose only. This takes the appearance of emesis even if nothing actually comes from the stomach. The animal shakes the head violently, breathes hurriedly, and has fits of paroxysmal coughing. A wave extending from below upward along the jugular furrow and followed by nasal discharge is a marked symptom, as the violence of the inflammation increases. Uneasy movements of the limbs, pawing and lying down and rising, indicate the existence of colic, and this is aggravated by the administration of anodynes or antispasmodics by the mouth. In cattle, rumination is arrested, froth accumulates around the lips, the rumen becomes tympanitic, and colicky movements appear. Oftentimes a swelling extends upward in the jugular furrow, and even in its absence, pressure with the fingers along the furrow will often detect an area of tenderness with or without local swelling. Fever with more or less elevation of temperature, is a general symptom. There may be wheezing breathing or loud stertor. The passage of a probang is arrested by the swelling or spasm at the diseased part and when withdrawn may be covered with pus or foetid *debris*. In the horse a small probang may be passed through the nose.

Treatment. In a slight congestion at the seat of a recent obstruction and which tends to renewed obstruction, little more is necessary than to restrict the feed for a few days to soft mashes so that irritation of the sensitive surface, spasm and the arrest of the morsel may be obviated. Plenty of pure water or of well boiled linseed or other gruel should be allowed.

In cases in which the obstruction is still present in the gullet, its removal by probang or looped wire is the first consideration, to be followed by the measures mentioned above.

In case of the swallowing of a caustic agent, no time should be lost in giving an antidote. For the mineral or caustic organic acids, lime water, magnesia, or other bland basic agent is demanded. For caustic alkalies or basic agents, bland acids, such as vinegar, citric acid, or even a mineral acid very largely diluted will be in order. In both these cases and in that of caustic salts, albuminous and mucilaginous agents, eggs, linseed tea, slippery elm, gums, and well boiled gruels are indicated. To these may be added small doses of laudanum when the irritation is great. Iced drinking water, iced milk, or iced gruels are often soothing to the suffering animal, and cold compresses, snow or ice applied along the jugular furrow is often valuable. To counteract the septic developments on the affected mucous membrane, chlorate of potash, boric acid, salol, naphthalin, naphthol, pyoktannin, or even weak solutions of phenic acid or creolin may be used. In the slighter forms of inflammation or when the acute form threatens to persist, an active counter-irritant of mustard or cantharides may be applied along the jugular furrow.

In case of abscess, as manifested by fluctuation following a hard, indurated, painful swelling, a free incision should be followed by frequent injections of antiseptic lotions or by the packing of the cavity with such bland antiseptics as salol, boric acid, or iodoform on cotton.

As inflammation subsides, potassium iodide may be given, both as an antiseptic and a resolvent, to counteract the tendency to fibroid contraction and stricture of the gullet.

SPASM OF THE ŒSOPHAGUS. ŒSOPHAGISMUS.

Causes: nervous disorders or lesions, pharyngeal, œsophagean, or gastric disease, œsophagean parasites, choking, tumors, ulcers, cold drinks. Symptoms: extended drooping head, working jaws, frothing, pawing, attempts at swallowing, alkaline regurgitation, cries, rigid gullet, tenderness. May be paroxysmal with intervening dullness. Treatment: by sound; by removal of obstruction; by antispasmodics. Embrocations. Tonics.

Causes. This has been noticed as a concomitant of certain diseases of the nervous centres, such as rabies, tetanus, or epilepsy, and those of the pharynx or stomach. Cadeac has seen it in connection with stricture, and the present writer has observed it as a result of larvæ of œstri hooked on to the mucosa above the cardia. It is an important factor in most cases of choking, and may depend on tumors, ulcers, or even cold beverages. Animals with a specially nervous organization are particularly subject to it and it may thus be an hereditary family trait. It has been especially noticed in solipeds and calves.

Symptoms. A feeding animal suddenly ceases to eat, extends the head on the neck, drops the nose toward the ground, moves the jaws constantly, froths at the mouth or lets the saliva drivel to the ground, moves the fore feet uneasily pushing the litter under the belly, makes efforts at deglutition during which, waves may be seen to descend along the jugular furrow, followed by regurgitation and discharge of the liquid as by emesis. The act is often followed by a slight cry. Manipulations of the left jugular furrow detects the gullet as a firm, rigid cord, unless when liquids are passing as above, and auscultation reveals a rattling or gurgling noise as if in jerks. Pressure on the gullet is often very painful, increasing the spasm and rigidity, and causing the animal to cry out. Wheezing breathing may attend the discharge of saliva through the nose, and violent paroxysms of coughing may be caused by the entrance of this liquid into the larynx.

In the majority of cases no food is swallowed and nothing but saliva is disgorged, which together with the absence of an acid odor distinguishes this from true vomiting. In an exceptional case of the author's, occurring in a colt, the animal continued to masticate and swallow green food which gradually filled the whole length of the gullet, practically paralyzing it. In ordinary cases a small sound can usually be passed into the stomach. In cases of obstruction, however, by a solid morsel, or by an accumulation of soft solids, the probang will enable one to detect the condition. The acute symptoms may occur in paroxysms of a few minutes in length, between which, the animal remains dull and dissipated until the new attack supervenes. Recovery is at times as sudden as the onset, though there remains, for a length of time, liability to a relapse. Cadeac has seen a succession of such attacks which extended over a year and a half.

Treatment. In many cases the passage of a probang or sound, will, by the mere distension of the gullet, overcome the local spasm, though it may be necessary to repeat the operation several times. In case the sound causes much pain the end of the instrument may be well smeared with solid extract of belladonna, and after passing this as far as the obstruction a short time may be allowed, before its passage is again attempted. In case obstruction by soft solids has taken place, the passage of the wire loop will serve to break up the mass and even to draw it up toward the mouth.

The administration of antispasmodics is the next indication. Chloroform or ether by inhalation or in solution in water, chloral hydrate as an enema, morphia or atropia hypodermically may be used according to convenience. Bromide of potassium and other antispasmodics given by the mouth, too often fail to pass the obstruction and thus prove useless, except in the intervals of the spasms.

Fomentations of the lower border of the neck with warm water, and frictions over the region of the gullet with camphorated spirit, essential oils, ammonia, or in calves with oil of turpentine, often contribute to relieve the spasm.

Finally after the severity of the attack has passed, a course of bitter tonics and above all of nuxvomica will fortify the system against a relapse.

PARALYSIS OF THE ŒSOPHAGUS.

Causes: nervous lesions and disorders; arytenectomy; over distension; stricture; parasites. Symptoms: dysphagia; regurgitation; cough; dyspnœa; hard packed gullet. Inhalation pneumonia. Lesions. Treatment: remove cause; liquid food; dilatation; nerve sedatives and stimulants; electricity; counter-irritants.

Causes. This has been noticed in a number of cases in solipeds, and attributed to central nervous lesions, cerebral concussion (Straub), encephalitis (Hering, Bornhauser), paralysis of the fore extremities (Meier), pharyngeal paralysis (Puschmann). Möller has seen it several times consequent on arytenectomy, while Dieckerhoff and Graf have seen it occur without any clearly defined cause. In a case referred to above, the present writer found it connected with the attachment of larvæ of œstri in the lower end of the gullet. Stricture and impaction may be a further cause.

Symptoms and lesions. There is more or less interference with deglutition, culminating in complete inability to swallow, and the rejection of morsels of masticated food by the nose. Cough may also occur from the descent of food toward the lungs, with more or less dyspnœa and oppression of the breathing. Manipulation along the left jugular furrow, detects the œsophagus as a prominent hard, rope-like mass which fills up the groove unduly. When death occurs rapidly the gullet is found gorged with masticated food throughout its entire length. In certain instances gangrenous pneumonia is found, the result of the penetration of food into the bronchia. In other cases there are lesions of the medulla oblongata, or of the vagus or glossopharyngeal nerves or their œsophagean branches.

Death usually results from obstruction, inanition, or, in case the paralysis is partial, from pneumonia or exhaustion.

Treatment. First remove or correct the existing cause of the disease. Impaction may be broken up by the use of the wire loop, or pincer probang; parasites may be expelled by passing a cupped probang; the impactions following arytenectomy can be obviated by feeding gruels, milk and other liquid foods only, and from a bucket set on the ground; stricture may be dilated by the use of graduated sounds; and nervous diseases may be dealt with according to their specific nature in each several case. When any definite cause of this kind has been overcome the persistent use of strychnia, subcutem, or by the mouth, may be effectual in overcoming the paresis of the gullet. Hypodermic injections are best made along the left jugular groove, and frictions, stimulating embrocations, and galvanic currents may be employed with excellent effect.

ŒSOPHAGEAN TUMORS.

Forms of neoplasm in gullet of horse, ox, sheep, pig, dog. Symptoms: dysphagia; eructation; vomiting; bloating; cough; dyspnœa; stertor; fœtor; palpitation. Treatment.

These have been often noticed in the lower animals. In the *horse* have been noticed *melanoma* (Olivier, Röhl, Kopp, Besnard, Pouleau), *fibroma* (Dandrieu, Dieckerhoff), Carcinoma (Chouard, Lorenz, Cadeac, Laurent), epithelioma (Blanc, Lorenz), Leiomyoma (Lucet, Lothes), cystoma (Caillau, Legrand), mucous cysts (Lucet).

In **cattle papilloma** is especially common, having been noted by *Johne*, Mons, Fessler, Schütz, Lusckar, Gratia, Beck, Cadeac and Kitt. **Tubercles**, and **fibroid masses** with cystic purulent centres are not uncommon. **Actinomycosis** is also frequent, sometimes hard and warty and at others soft and vascular.

In the **Sheep**, Dandrieu found between the muscular and mucous coats a hard tumor as large as a hen's egg, the removal of which put a stop to a persistent choking. In both **cattle** and **sheep**, swellings from **coccidiosis** are common; in **cattle** and **swine** from **gongylonema**, and in **sheep** from **filaria** (Harms) or **spiroptera** (Zurn).

In **pigs**, **fibroma** is met with in the walls of the gullet (Raveski) and in **dogs fibroma, papilloma**, and the tumors of spiroptera.

Symptoms. The coccidia and spiroptera usually cause few symptoms or none, but neoplasms usually develop symptoms of obstruction, dysphagia, eructation, vomiting, and all the indications of choking according to their seat. These do not come on suddenly and recover as in simple choking, but even though there may be periodic obstructions, spasms and paroxysms, there is a slow, progressive advance as the neoplasms increase. Stertorous or mucous breathing, cough, dyspnœa and fœtid exhalations are common, the symptoms may be aggravated when the head is bent, and the tumor may even be felt on palpation of the throat or left jugular furrow. In ruminants tympany occurs after feeding.

Treatment is surgical and consists in the removal of the tumors by incision and ecraseur or otherwise. Thoracic œsophagean tumors are usually inoperable.

IMPACTION OF THE CROP. INGLUVIAL INDIGESTION.

Gallinaceæ and Palmipeds. Causes; Overfeeding after privation; fermentation; lack of water; green food in geese and chickens; food containing paralyzing element. Symptoms; dull; motionless; erect plumes; drooping wings and head; gapes; ejects liquid from bill; firm cervical swelling. Treatment; manipulation; incision; surgical precautions. Convalescent feeding.

The cervical dilatation of the œsophagus known as the crop is well developed in all granivorous birds, (Gallinaceæ, etc.) and like the macerating cavities of the ox (first two stomachs) is subject to overdistension and paralysis. In the palmipeds (ducks, geese) there is no distinct crop but in its place the cervical portion of the gullet has a fusiform dilatation, and under given conditions this may be also the seat of impaction.

Causes. The impaction may result from overfeeding when the bird has been starved, or when it suddenly gains access to food of a specially appetizing kind and to which it has been unaccustomed. The crop like every other hollow viscus is rendered paretic by overdistension. Then the food undergoes fermentation still further distending the cavity, affecting the brain by reflex action, and paralyzing the vagus and its peripheral branches in the lungs, heart, stomach, liver, intestines, etc. When the food is dry as in the case of beans, peas, bran, farinas, it may be a simple firm impaction which the muscular walls of the crop are unable to break up or move onward. When green food is taken there is often superadded the additional evil of active fermentation from the great number and activity of the bacterial ferments contained in it and the soft aqueous fermentescible nature of the food (See *tympany in ruminants*). Dupont states that young geese led out to fresh spring grass may lose two-thirds of their number in a few hours from such overloading and that some species of *Carex* and *cynodon dactylon* are particularly injurious. Chickens also gorge the crop with clover, etc. In all such cases, plants that contain a paralyzing principle like *lolium temulentum*, ripening *lolium perenne*, chick vetch, etc., are to be specially dreaded. (See *Trichosoma Contortum*).

Symptoms. There are first dullness and sluggish movements, followed by indisposition to move, the bird standing in one place with ruffled feathers and drooping wings, and at intervals, projecting the head forward with open beak and in some cases a little liquid is rejected. If the bird is now caught and examined the crop is found to be firmly distended, and more or less compressible or indentable according to the nature of the food impacted. In most cases and especially if the food has been green or aqueous, there is a certain resiliency from the presence of gas outside the solid impacted mass.

Treatment. This must be in the line of seconding the physiological efforts of regurgitation which is a normal and common act in birds. The duck which has gulped a mouse half-way down the cervical part of the œsophagus will readily disgorge it when he finds it impossible to pass it further. The carnivorous birds often reject by vomiting the indigestible debris such as feathers and bones, after all the more soluble parts have been disposed of in the stomach. The pigeon even feeds its young by disgorging into their open bills, the semi-digested food and milk from its crop. Following these indications we must break up the contents of the crop by manipulation and force them in small masses upward into the bill and downward to the proventriculus. The rejection by the bill may be further stimulated by introducing the finger into the fauces to rouse the reflex active emesis. Usually the crop can be quickly and satisfactorily emptied in this way.

When this proves impossible there remains the operation of direct incision through the walls of the crop and the evacuation of its contents. This can be done by a pocket knife or even a pair of scissors. The crop is punctured in its lower part and the incision is continued upward as far as may be necessary to allow the escape of the contents. Usually half an inch will suffice. Then the crop is squeezed so as to press the contents through this opening and it is emptied by a process of enucleation. If the contents are fibrous it may be necessary to employ forceps to dislodge the material. The empty crop may be washed out with tepid water, any food attached to the raw edges of the wound must be removed and the skin stitched accurately together. The wound rarely fails to heal by first intention. To avoid stretching it, the food for a day or two should be restricted to milk, gruels, or a little soft mash.

Lerein notices jaundice as a sequel of impacted crop, and recommends treatment by sulphate of

soda in the water.

TYMPANITIC INDIGESTION IN THE RUMEN. BLOATING.

Definition. Susceptible Genera. Causes; gastric paresis, overloading, cold, fear, exhaustion, poisons, fermentescible food,—new grain, leguminosæ, frosted vegetables, ruminitis, foreign bodies in rumen, microbial ferments. Symptoms, abdominal, general. Gases formed under different aliments—carbon dioxide, marsh gas, hydrogen sulphide, nitrogen, oxygen. Lesions, rupture of rumen or diaphragm, compression or rupture of liver or spleen, petechiæ, congestion of lungs and right heart, of cutaneous and cerebral vessels. Prevention, avoid indigestible and fermentescible aliments, correct adynamic conditions, tonics, avoid injurious ferments, make alimentary transitions slowly. Treatment, exercise, bath or douche of cold water, rubbing and kneading, rope round abdomen spirally, gag in mouth, dragging on tongue, movement of a rope in fauces, probang, stimulants, antiseptics, alkalies, ammonia, oil of turpentine, oil of peppermint, alcohol, ether, pepper, ginger, soda, potash, lime, muriatic acid, carbolic acid, creosote, creoline, sulphites, kerosene, chloride of lime, chlorine, tar, common salt, hypochlorite of soda, magnesia, eserine, pilocarpin, barium chloride, colchicum, lard, trochar, Epsom salts, rumenotomy. Treatment of diseased gullet, mediastinal glands, stomach or intestines.

Definition. The condition is a combination of paresis of the rumen and gaseous fermentation of its contents. The initial step may be the paresis or in the more acute forms the fermentation.

Genera susceptible. While all ruminating animals are subject to this disorder, it is much more frequent in cattle and sheep than in goats.

Causes. It commences in *paresis of the rumen* in the weak, debilitated, convalescent or starved animals which are suddenly put on rich, and appetizing food. Hence it is common in animals that break into a cornbin, a store of potatoes, a field of growing corn or small grain, or that are turned out on green food in early spring. Cadeac maintains that paresis of the rumen is the essential cause in all cases, while the nature of the aliments ingested fills a secondary and comparatively insignificant rôle. According to this view the torpid stomach can neither relieve itself through regurgitation for rumination, nor expel through the œsophagus the constantly evolving gas which therefore distends the viscus to excess. In support of this view may be adduced the occurrence of tympany through fatigue, fear, cold, enlarged (tubercular) mediastinal glands pressing on the gullet and vagus, obstruction of the œsophagus by a solid body (choking), impaction of a morsel of solid food in the demicanal of the calf as noticed by Schaubert, and the cessation of the normal vermicular movements of the rumen in connection with inflammation of its coats, or extensive inflammation elsewhere or finally of fever. Even in paralysis of the stomach by poisons like lead, tympany may be a result. Cadeac attributes tympany following the ingestion of green food wet with a shower, or drenched with dew, of frosted potatoes or turnips, or of iced water, to the paralyzing action of the cold on the rumen. This view is manifestly too extreme, as the bloating occurs often after a warm summer shower, or after the consumption of potatoes and other roots and tubers which have been spoiled by frost but which are no longer at a low temperature when consumed.

Tympany may also start from the ingestion of certain kinds of food which are in a very fermentescible condition. Green food, especially if the animal has been unaccustomed to it, is liable to act in this way. Clover and especially the white and red varieties, lucern (alfalfa), sainfoin, cowpea and other specially leafy plants, which harbor an unusual number of microbial ferments, and which contain in their substance a large amount of nitrogenous material favorable to the nourishment of such ferments are particularly dangerous in this respect. All of these are most dangerous when wet with dew or when drying after a slight shower, partly no doubt at times by reason of the chilling of the stomach, but mainly because the ferments have been stimulated into activity by the presence of abundance of moisture. Drenching and long continued rains are less dangerous in this respect than the slight showers and heavy dews, manifestly because the former wash off a large portion of the microbes, which under a slight wetting multiply more abundantly.

Frosted articles act in a similar way, partly when still cold by the chilling and paralyzing of the stomach, but cold or warm, by reason of the special tendency of all frozen vegetables to undergo rapid fermentation when thawed out. This is true of green food of all kinds when covered by hoarfrost, of turnips, beets, potatoes, carrots, apples, cabbage, etc., which have once been frozen, and of frosted turnips and potato tops, though, in the case of the latter agent, a narcotic principle is added.

In the case of Indian corn, the smaller cereal grains, and certain leguminous plants (vetches, tares, peas, beans) which have the seed fully formed but not yet quite hardened nor ripened, there is the double action of a paralyzing constituent and an aliment that is specially susceptible of fermentation.

Inflammation of the rumen, already quoted as a cause, may be determined by hot as well as cold food, by irritant drugs and poisons, and by narcotico-irritant and other acrid plants in fodder or pasture. In the same way the inflammation caused by the introduction of foreign bodies into the rumen, such as nails, tacks, needles, pins, wires, knife blades, and masses of hair or wool may at times cause tympany.

The two main causative factors, of paresis of the rumen on the one side and of specially fermentescible food and a multiplicity of microbial ferments on the other, must be recognized as more or less operative in different cases, and in many instances their combined action must be admitted. The tympany is the symptom and culmination of a great variety of morbid causes and conditions, and its prevention and treatment must correspondingly vary.

Symptoms. The whole left side of the abdomen being occupied by the rumen, its distension leads to an uniform swelling of that side, differing from that caused by simple excess of solid ingesta in being more prominent high up between the last rib and the outer angle of the ilium, and in giving out in this region a clear tympanitic or drumlike resonance on percussion. It has also a tense resiliency, like that of a distended bladder, easily pressed inward by the finger but starting out to its rotundity the moment the pressure of the finger is withdrawn. The distension caused by overloading with solids bulges out lower down, is not resonant but dull or flat when percussed, and yields like a mass of dough when pressed retaining the indentation of the finger for some time. The swelling of tympany, when extreme, rises above the level of the outer angle of the ilium and even of the lumbar spines on the left side, and if no relief is obtained the right side may undergo a similar distension.

Auscultation detects an active crepitation over the whole region of the rumen, finer in some cases and coarser in others, according to the activity of evolution and the size of the bubbles of gas. The crepitation is especially coarse and loud in fermentation of green food, and of spoiled potatoes or other tubers or roots.

In all acute or severe cases, there is anorexia, suspension of rumination, and the normal movements of the compressed bowels seem to be largely impaired, though the anus is protruded and a little semi-liquid fæces or urine may be expelled at intervals. The breathing is accelerated, short, and labored. The nostrils are dilated, the nose extended, the face anxious, the eyes bloodshot and the back arched. Froth may accumulate around the lips, or the mouth may be held open with the tongue pendent. Sometimes a quantity of gas may suddenly escape with a loud noise, but without securing permanent relief. The heart beats are violent and accelerated, the pulse increasingly small and finally imperceptible, and the visible mucous membranes are congested and cyanotic. Pregnant females are very liable to abort.

When the right flank as well as the left rises to the level of the lumbar spines death is imminent, and this may take place as early as fifteen or thirty minutes after the apparent onset of the attack. Death may result from nervous shock, from suffocation, or from the absorption of deleterious gases, or from all of these combined.

In the less acute cases the animal may live several hours before the affection terminates in death or recovery. As a rule he stands as long as he can and finally drops suddenly, the fall often leading to rupture of the diaphragm or stomach, to protrusion of the rectum, or the discharge of ingesta by the mouth and nose.

In still slighter cases relief comes through vomiting or more commonly through frequent and abundant belching of gas, the swelling of the flanks subsides, rumbling of the bowels may again be heard, and usually there is a period of diarrhœa.

Gases present. When the rumen is punctured before or after death so as to give exit to the gas in a fine stream it proves usually more or less inflammable, the lighted jet burning with a bluish flame. The usual inflammable ingredients are carbon monoxide, hydrogen carbide (marsh gas) and

hydrogen sulphide, yet the relative proportion of the gases varies greatly with the nature of the food and the amount of gas evolved, carbon dioxide being usually largely in excess. The following table serves to illustrate the variability:

Gas.	Pflüger.	Gmelin, from clover	Lassaigue.	Lameyran and Fremy, from clover.	Vogel.	Raiset.
CO	60–80	5	29	5	27	74.23
CO	28–40					
CH		15	6	15	48	23.46
HS		80		80		
O			14.7			
N			50.3		25	2.22

The most elaborate observations on this subject are those made by Lungwitz on the different aliments kept in closed vessels at the body temperature, and on similar agents fed for days as an exclusive aliment to oxen provided with a fistula of the rumen for purposes of collection. He found carbon dioxide to be the predominating gas in all cases, but that it was especially so in extreme tympanies and varied much with the nature of the food. The following table gives results:

Percentage of CO ₂ .		
Buckwheat (Polygonum fagopyrum)		80
Alfalfa (Mendicago Sativa)		70–80
Clover (Trifolium pratense)		70–80
Meadow grass		70–80
Indian corn (Zea Maïs)		70–80
Spurry (Spergula arvensis)		70–80
Hay of alfalfa or clover		70–80
Oats with cut straw		70–80
Yellow Lupin (Lupinus luteus)		60–70
Vetch (Vicia sativa)		60–70
Oats cut green		60–70
Potato tops		60–70
Potatoes		60–70
Meadow hay		60–70
Leaves of beet		50–60
Leaves of radish		50–60
Cabbage		40–50

The marsh gas varied from 16 to 39 per cent., being especially abundant in cases of abstinence. It should, therefore, be in large amount in the tympanies which accompany febrile and other chronic affections. Hydrogen sulphide was found only in traces, recognizable by blackening paper saturated with acetate of lead. Oxygen and nitrogen were in small amount and were attributed to air swallowed with the food. In the work of fermentation the oxygen may be entirely used up.

Lesions. These are in the main the result of compression of the different organs, by the overdistended rumen. Rupture of the rumen is frequent. The abdominal organs are generally bloodless, the liver and spleen shrunken and pale, though sometimes the seat of congestion or even hemorrhage. Ecchymoses are common on the peritoneum. The right heart and lungs are gorged with black blood, clotted loosely, and reddening on exposure. The right auricle has been found ruptured. Pleura, pericardium and endocardium are ecchymotic. The capillary system of the skin, and of the brain and its membranes, is engorged, with, in some instances, serous extravasations.

Prevention. This would demand the avoidance or correction of all those conditions which contribute to tympany. In fevers and extensive inflammations, when rumination is suspended, the diet should be restricted in quantity and of materials that are easily digested (well boiled gruels,

bran mashes, pulped roots, etc.) and all bulky, fibrous and fermentescible articles must be proscribed. In weak conditions in which tympany supervenes on every meal, a careful diet may be supplemented by a course of tonics, carminatives and antiseptics such as fœungrec oxide of iron, hyposulphite of soda and common salt, equal parts, nux vomica 2 drs. to every 1 lb. of the mixture. Dose 1 oz. daily in the food, or ½ oz. may be given with each meal.

Musty grain and fodder should be carefully avoided, also mowburnt hay, an excess of green food to which the stock is unaccustomed, clover after a moderate shower, or covered with dew or hoarfrost, frosted beet, turnip, or potato tops, frosted potatoes, turnips or apples, also ryegrass, millet, corn, vetches, peas with the seeds fairly matured but not yet fully hardened. When these conditions cannot be altogether avoided, the objectionable ration should be allowed only in small amount at one time and in the case of pasturage the stock should have a fair allowance of grain or other dry feed just before they are turned out. Another precaution is to keep the stock constantly in motion so that they can only take in slowly and in small quantity the wet or otherwise dangerous aliment.

When it becomes necessary to make an extreme transition from one ration to another, and especially from dry to green food, measures should be taken to make the change slowly, by giving the new food in small quantities at intervals, while the major portion of the diet remains as before, until the fæces indicate that the superadded aliment has passed through the alimentary canal. Another method is to mix the dry and green aliments with a daily increasing allowance of the latter. Some have avoided the morning dew and danger of fermentation by cutting the ration for each succeeding day the previous afternoon and keeping it in the interval under cover.

Treatment. Various simple mechanical resorts are often effective in dispelling the tympany. Walking the animal around will sometimes lead to relaxation of the tension of the walls of the demicanal and even to some restoration of the movements of the rumen with more or less free eructation of gas. The dashing of a bucket of cold water on the left side of the abdomen sometimes produces a similar result. Active rubbing or even kneading of the left flank will sometimes lead to free belching of gas. The same may be at times secured by winding a rope several times spirally round the belly and then twisting it tighter by the aid of a stick in one of its median turns.

A very simple and efficient resort is to place in the mouth a block of wood 2½ to 3 inches in diameter and secured by a rope carried from each end and tied behind the horns or ears. This expedient which is so effective in preventing or relieving dangerous tympany in choking appears to act by inducing movements of mastication, and sympathetic motions of the œsophagus, demicanal and rumen. It not only determines free discharge of gas by the mouth, but it absolutely prevents any accession of saliva or air to the stomach by rendering deglutition difficult or impossible. A similar effect can be obtained from forcible dragging on the tongue but it is difficult to keep this up so as to have the requisite lasting effect. Still another resort is to rouse eructation by the motions of a rope introduced into the fauces.

The passing of a hollow probang into the rumen is very effective as it not only secures a channel for the immediate escape of the gas, but it also stimulates the demicanal and rumen to a continuous eructation and consequent relief. Friedberger and Fröhner advise driving the animals into a bath of cold water.

Of medicinal agents applicable to gastric tympany the best are stimulants, antiseptics and chemical antidotes. Among stimulants the alkaline preparations of ammonia hold a very high place. These, however, act not as stimulants alone, but also as antacids and indirectly as antidotes since the alkaline reaction checks the acid fermentation which determines the evolution of the gas. They also unite with and condense the carbon dioxide. Three ounces of the aromatic spirits of ammonia, one ounce of the crystalline sesquicarbonate, or half an ounce of the strong aqua ammonia may be given to an ox, in not less than a quart of cold water. Next to this is the oil of turpentine 2 oz., to be given in oil, milk, or yolk of egg. But this too is an antiferment. The same remark applies to oil of peppermint (½ oz.), the carminative seeds and their oils, and the stronger alcoholic drinks (1 quart). Sulphuric or nitrous ether (2 oz.) may be given in place. Pepper and ginger are more purely stimulant and less antiseptic. Other alkalies—carbonate of potash or soda, or lime water may be given freely.

Among agents that act more exclusively as antiseptics may be named: muriatic acid 1 to 1½ drs. largely diluted in water; carbolic acid, creosote or creolin, 4 drs. largely diluted; sulphite, hyposulphite or bisulphite of soda 1 oz.; kerosene oil ½ pint; chloride of lime 4 drs.; chlorine water 1 pint; wood tar 2 oz. The latter agent is a common domestic remedy in some places being given wrapped in a cabbage leaf, and causing the flank to flatten down in a very few minutes as if by magic. The extraordinarily rapid action of various antiseptics is the most conclusive answer to the claim that the disorder is a pure paresis of the walls of the rumen. The affection is far more commonly and fundamentally an active fermentation, and is best checked by a powerful antiferment. Even chloride of sodium (½ lb.) and above all hypochlorite of soda or lime (½ oz.) may be given with advantage in many cases.

Among agents which condense the gasses may be named ammonia, calcined magnesia, and milk of lime for carbon dioxide, and chlorine water for hydrogen.

Among agents used to rouse the torpid rumen and alimentary canal are eserine (ox 3 grs., sheep ½ gr. subcutem), pilocarpin (ox 2 grs., sheep ⅓ gr.), barium chloride (ox 15 grs., sheep 3 to 4 grs.), tincture of colchicum (ox 3 to 4 drs.). Trasbot mentions lard or butter (ox 4 oz., sheep ½ oz.), as in common use in France.

In the most urgent cases, however, relief must be obtained by *puncture of the rumen*, as a moment's delay may mean death. The seat for such puncture is on the left side, at a point equidistant from the outer angle of the ilium, the last rib and the transverse processes of the lumbar vertebræ. Any part of the left flank might be adopted to enter the rumen, but, if too low down, the instrument might plunge into solid ingesta, which would hinder the exit of gas, and would endanger the escape of irritant liquids into the peritoneal cavity. In an extra high puncture there is less danger, though a traumatism of the spleen is possible under certain conditions. The best instrument for the purpose is a trochar and cannula of six inches long and ⅓ to ½ inch in diameter. (For sheep ¼ inch is ample.) This instrument, held like a dagger, may be plunged at one blow through the walls of the abdomen and rumen until stopped by the shield on the cannula. The trochar is now withdrawn and the gas escapes with a prolonged hiss. If the urgency of the case will permit, the skin may be first incised with a lancet or pen knife, and the point of the instrument having been placed on the abdominal muscles, it is driven home by a blow of the opposite palm. In the absence of the trochar the puncture may be successfully made with a pocket knife or a pair of scissors, which should be kept in the wound to maintain the orifice in the rumen in apposition with that in the abdominal wall, until a metal tube or quill can be introduced and held in the orifices.

When the gas has escaped by this channel its further formation can be checked by pouring one of the antiferments through the cannula into the rumen.

When the formation of an excess of gas has ceased, and the resumption of easy eructation bespeaks the absence of further danger, the cannula may be withdrawn and the wound covered with tar or collodion.

When the persistent formation of gas indicates the need of expulsion of offensive fermentescible matters, a full dose of salts may be administered. If the presence of firmly impacted masses can be detected, they may sometimes be broken up by a stout steel rod passed through the cannula. If the solid masses prove to be hair or woolen balls, rumenotomy is the only feasible means of getting rid of them.

In *chronic tympany* caused by structural diseases of the œsophagus, mediastinal glands, stomach or intestines, permanent relief can only be obtained by measures which will remove these respective causes.

CHRONIC TYMPANY OF THE RUMEN.

Causes: catarrh of rumen, impaction of manifolds, debility, paresis, peritoneal adhesions, neoplasms, concretions, sudden change in diet, gastric congestion, lesions of gullet, or of mediastinal glands. Symptoms are usually after feeding only, inappetence, rumbling, costiveness, rumen indentable. Treatment: obviate causes, give salines, acids, bitters, and water, laxative food, carminatives, antiseptics, electricity, emetic tartar, eserine, pilocarpin, barium chloride, apomorphin.

Causes. The persistence of causes of acute tympany may lead to the appearance of the condition after each meal, or even in the intervals between meals. Among the more specific causes may be named catarrhal inflammation of the rumen, impaction of the third stomach, paresis of the rumen, general debility, peritoneal adhesions affecting the viscus, tuberculosis, actinomycosis or other morbid productions in its walls, hernia of the reticulum into the chest, hard stercoral, hair or wool balls, or masses or foreign bodies in the rumen, and the ingestion of a very fermentescible quality of food. When the rumen is affected by catarrh or paresis or debility, even ordinary food will lead to tympany, but much more so any food to which the animal has been unaccustomed (green for dry, or dry for green, grain for grass or hay, or beans or peas for grain). Also food in process of fermentation, or the seat of fungoid growth.

Again, so intimately related are the different stomachs that derangement of one instantly impairs the functions of the other, and thus a slowly progressive impaction of the third stomach leads to torpor of the first, and the aggregation of more or less of its contents into solid, fermenting masses. In the same way congestion of either the third or fourth stomach impairs the functions of the rumen and induces tympany.

Morbid conditions affecting the functions of the œsophagus and interfering with rumination and eructation of gas are familiar causes. For example, strictures and saccular dilations of the tube, and enlargements—tubercular, sarcomatous, actinomycotic,—of the mediastinal glands.

The *symptoms* do not differ from those of acute tympany excepting that they are less severe; and are continuous or remittent, suffering a material aggravation after feeding. Rumination may be suppressed or tardy, the bowels also are torpid, the fæces glazed, and the ordinary intestinal rumbling little marked. When the tympany has temporarily subsided, the knuckles, pressed into the left side, can often be made to strike against the hard, solid impacted mass of ingesta. Symptoms of impacted manifolds may also be patent and the patient steadily loses condition.

Treatment must be directed toward the removal of the special cause of the trouble, and if this cannot be secured, as in tuberculosis, the case is hopeless. In cases of solid masses in the rumen the free use of common salt with a drachm of hydrochloric acid, and one grain strychnine with each meal, and a free access to water may succeed. The food had best be restricted to gruels and sloppy mash. The daily use of electricity through the region of the paunch is an important accessory. The common salt may be increased as required, so as to keep up a very relaxed condition of the bowels.

In obstinate cases of this kind puncture may be resorted to and an attempt made to break down the impacted masses with a steel rod introduced through the cannula. Should this also fail the solid masses or foreign bodies may be extracted by rumenotomy.

In simple catarrh of the rumen the continued use of strychnine with gentian, and sulphate of iron, may prove successful under a carefully regulated diet. Oil of turpentine, balsam of copaiba, or balsam of tolu may also prove useful, or in other cases extract of hamamelis, or of wild cherry bark. While strychnine and electricity are to be preferred to rouse the muscular activity of the viscus, such agents as tartar emetic, emetine, apomorphin, eserine, pilocarpin and barium chloride are recommended and may be resorted to in case of necessity.

OVERLOADED (IMPACTED) RUMEN.

Definition. Causes, excess of rich unwonted food, gastric torpor, paresis, starvation, debility, partially ripened, poisonous seeds, paralyzing fungi or bacteria, lead, cyanides, congestion of rumen, chlorophyll, acrids, dry, fibrous innutritious food, lack of water, enforced rest on dry food, over-exertion, salivary fistula or calculus, diseased teeth or jaws, senility. Symptoms, suspended rumination, inappetence, anxious expression, arched back, bulging pendent left flank, impressible, no friction sounds, excessive crepitation, hurried breathing, colics, grunting when moved, diarrhœa, stupor, cyanosis. Signs of improvement. Phrenic rupture. Diagnosis from tympany, pneumonia, or gastro-intestinal catarrh. Treatment, hygienic, antiseptic, stimulants, puncturing, purgation, rumenotomy.

Definition. The overdistension of the rumen with solid food is characterized by two things, the excess of ingesta which produces the torpor or paresis which is common to all over-filled hollow viscera, and the comparative absence of fermentation and evolution of gas. If the ingesta is of a more fermentescible nature the rapid evolution of gas occurs before this degree of repletion with solid matters can be reached, and the case becomes one of tympany, but if the contents are comparatively lacking in fermentability they may be devoured in such quantity as to cause solid impaction.

Causes. Overloading of the rumen is especially common as the result of a sudden access to rich or tempting food to which the animal has been unaccustomed. Accidental admittance to the cornbin, breaking into a field of rich grass, clover, alfalfa, corn, sorghum, vetches, tares, beans, peas, or grain, or into a barrel of potatoes or apples will illustrate the common run of causes. A pre-existing or accompanying torpor or paresis of the stomach is a most efficient concurrent cause, hence the affection is especially common in animals debilitated by disease or starvation, but which have become convalescent or have been suddenly exposed to the temptation of rich food. For the same reason it is most likely to occur with food which contains a paralyzing element, as in the case of the following when they have gone to seed but are not yet fully ripened: Rye grass, intoxicating rye grass, millet, Hungarian grass, vetches, tares and other leguminosæ, and to a less extent, wheat, barley, oats and Indian corn. The same may come from the paralyzing products of fungi or bacteria in musty fodder or of such chemical poisons as lead, and the cyanides.

A catarrhal affection of the rumen, and the congestion produced by irritant plants, green food with an excess of chlorophyll, and the whole list of irritants and narcotico-acrids, will weaken the first stomach and predispose to overdistension.

Anything which lessens the normal vermicular movements of the rumen and hinders regurgitation and rumination tends to impaction, and hence an aliment which is to a large extent fibrous, innutritious, and unfermentable, such as hay from grass that has run to seed and been threshed, the stems of grasses that have matured and withered in the pastures, fodder that has been thoroughly washed out by heavy rains, sedges, reedgrass, rushes, chaff, finely cut straw, and in the case of European sheep, the fibrous tops of heather contribute to this affection. Lack of water is one of the most potent factors, as an abundance of water to float the ingesta is an essential condition of rumination. Hence pasturage on dry hillsides, prairies or plains, apart from streams, wells or ponds is especially dangerous unless water is supplied artificially, and the winter season in our Northern states, when the sources of drinking water are frozen over, and when the chill of the liquid forbids its free consumption, is often hurtful.

Gerard attributes the affection to constant stabulation. This, however, has a beneficial as well as a deleterious side. It undermines the health and vigor, and through lack of tone favors gastric torpor and impaction, but it also secures ample leisure for rumination, which is so essential to the integrity of the rumen and favors the onward passage of its contents. With dry feeding and a restricted water supply it cannot be too much condemned, but with succulent food and abundance of water the alleged danger is reduced to the minimum.

Active work and over exertion of all kinds must be admitted as a factor. At slow work the ox can

still ruminate, but in rapid work or under heavy draft this is impossible, and the contained liquids may pass over from rumen to manifolds conducing to impaction of the former, or fermentations may take place, swelling up the mass of ingesta and distending the walls of the first stomach. Similarly, cattle and sheep that are hurried off on a rapid march with full stomachs are greatly exposed to both tympany and impaction.

In speaking of dry, fibrous food and lack of water as factors, we must avoid the error of supposing that succulent or aqueous food is a sure preventive. In a catarrhal condition of the rumen or in a state of debility, impaction may readily occur from the excessive ingestion of luscious grass, wheat bran, potatoes, apples, turnips, beets, or cabbage.

Finally defects in the anterior part of the alimentary tract may tend to impaction. Salivary fistula or calculus cutting off the normal supply of liquid necessary for rumination, tends to retention and engorgement. Diseased teeth and jaws interfering with both the primary and secondary mastication has the same vicious tendency. Old cows, oxen and sheep in which the molar teeth are largely worn out, suffer in the same way, especially when put up to fatten or otherwise heavily fed. In this case there is the gastric debility of old age as an additional inimical feature.

Symptoms. These vary with the quantity and kind of ingesta also to some extent with the previous condition of the rumen, sound or diseased. They usually set in more slowly than in tympany. On the whole the disease appears to be more common in the stable than at pasture. The animal neither feeds nor ruminates, stands back from the manger, becomes dull, with anxious expression of the face, arching of the back and occasional moaning especially if made to move. The abdomen is distended but especially on the left side, which however hangs more downward and outward and tends less to rise above the level of the hip bone than in tympany. If it does rise above the ilium this is due to gas and it is then elastic, resilient and resonant on percussion at that point. The great mass, and usually the whole of the paunch is nonresonant when percussed, retains the imprint of the fingers when pressed, and gives the sensation of a mass of dough. The hand applied on the region of the paunch fails to detect the indication of movements which characterize the healthy organ. The ear applied misses the normal friction sound, but detects a crepitant sound due to the evolution of bubbles of gas from the fermenting mass. This is especially loud if the impaction is one of green food or potatoes, even though the gas remains as bubbles throughout the entire fermenting mass, instead of separating to form a gaseous area beneath the lumbar transverse processes.

The respiration is hurried, labored and accompanied with a moan, the visible mucosæ are congested, the eyes are protruded and glassy from dilatation of the pupils, the feet are propped outward, and the head extended on the neck. There may be signs of dull colicky pains, movements of the tail and shifting of the hind feet, in some cases the patient may even lie down but never remains long recumbent. There may be occasional passages of semi-liquid manure, though usually the bowels are torpid and neither passages nor rumbling sounds on the right side can be detected. When moved the animal usually grunts or moans at each step, and especially when going down hill, owing to the concussion of the stomach on the diaphragm. In cases due to green food the irritation may extend to the fourth stomach and intestines and a crapulous diarrhœa may ensue. The temperature remains normal as a rule. The disease is more protracted than tympany, yet after several hours of suffering and continual aggravation the dullness may merge into stupor, the mucosæ become cyanotic and death ensues from shock, asphyxia, or apoplexy.

Course. Termination. Many cases recover in connection with a restoration of the contractions of the rumen, the eructation of gas, in some rare cases vomiting or spasmodic rejection of quantities of the ingesta, and the passage of gas by the bowels. This may be associated with a watery diarrhœa, and loud rumbling of the right side, which may continue for twenty-four hours or longer. With the subsidence of the diarrhœa there comes a return of health, or there may remain slight fever, inappetence, suspended or impaired rumination, dullness, listlessness, and a mucous film on the fæces. This indicates some remaining gastro-enteritis.

In some instances there is rupture of the diaphragm with marked increase in the abdominal pain and the difficulty of breathing. In others there is a laceration of the inner and middle coats of the rumen so that the gas diffuses under the peritoneum and may even be betrayed by an

emphysematous extravasation under the skin.

Diagnosis. From tympany this is easily distinguished by the general dullness on percussion, the persistence of the indentation caused by pressure, the outward and downward rather than the upward extension of the swelling, and the slower development of the affection.

It is far more likely to be confounded with pneumonia, which it resembles in the hurried, labored breathing, the moans emitted in expiration, in the dullness on percussion over the posterior part of the chest, it may be even forward to the shoulders, and in the cyanotic state of the mucosæ. The distinction is easily made by the absence of hyperthermia, and of crepitation along the margins of the nonresonant areas in the lungs, by the fact that the area of chest dullness covers the whole posterior part of the thorax to a given oblique line, and by the history of the case and the manifest symptoms of overloaded stomach, not with gas but with solids. From gastro-intestinal catarrh it may be distinguished by the more rapid advance of the symptoms and by the absence of the slight fever which characterizes the latter.

Treatment. Slight cases may be treated by hygienic measures only. Walking the animal uphill, injections of cold water, friction on the left side of the abdomen to rouse the rumen to activity, antiseptics as in tympany to check further fermentation, and stimulants to overcome the nervous and muscular torpor, may be employed separately or conjointly. When it can be availed of, a rubber hose may be wound round the abdomen and a current of cold water forced through it.

When further measures are demanded we should evacuate any gas through the probang or a cannula, as in tympany, and thus relieve tension and then resort to stimulants and purgatives. Common salt 1 lb. is of value in checking fermentation, and may be added to 1 lb. Glauber salts in four or five quarts of warm water. A drachm of strong aqua ammonia or 2 oz. oil of turpentine and ½ drachm of nux vomica may be added. Bouley advocated tartar emetic (2 to 3 drachms), and Lafosse ipecacuan (1 oz. of the wine) to rouse the walls of the rumen, and more recently pilocarpin (ox 3 grs.), eserine (ox 2 grs.) and barium chloride (ox 15 grs.), have offered themselves for this purpose. The three last have the advantage of adaptability to hypodermic use, and prompt action. The repetition of stimulants and nux vomica may be continued while there appears any prospect of restoring the normal functions of the paunch, and when all other measures fail the only hope lies in rumenotomy.

Rumenotomy. The warrant for this operation is found in the entire lack of movement in the rumen, the absence of eructation, the cessation of rumbling and motion of the bowels, and the deepening of the stupor in which the patient is plunged. The longer the delay and the deeper the stupor and prostration the less the likelihood of a successful issue from the operation. The animal is made to stand with its right side against a wall, and its nose held by the fingers or bulldog forceps. If judged necessary a rope may be passed from a ring in the wall in front of the shoulder around the animal to another ring behind the thigh and held tight. Or a strong bar with a fulcrum in front, may be pressed against the left side of the body, and well down so as to keep the right side fast against the wall. A line may be clipped from the point of election for puncture in tympany down for a distance of six inches. A sharp pointed knife is now plunged through the walls of the abdomen and rumen in the upper part of this line, and is slowly withdrawn, cutting downward and outward until the opening is large enough to admit the hand. The lips of the wound in the overdistended stomach will now bulge out through to the wound in the abdominal walls, and three stitches on each side may be taken through these structures to prevent displacement as the stomach is emptied and rendered more flaccid. A cloth wrung out of a mercuric chloride solution may be laid in the lower part of the wound to guard against any escape of liquid into the peritoneal cavity. The contents may now be removed with the hand, until the organ has been left but moderately full. Two or three stable bucketfuls are usually taken, but it is by no means necessary nor desirable that the rumen be left empty, as a moderate amount of food is requisite to ensure its functional activity. As a rule at least fifty pounds should be left. Before closing the wound and especially in cases due to dry feeding, it is well in a tolerably large animal to introduce the hand through the demicanal to ascertain if impactions exist in the third stomach and to break up these so far as they can be reached. This done, the edges of the wound in the stomach are to be carefully cleansed, washed with the mercuric chloride solution and sewed together with carbolated catgut, care being taken to

turn the mucosa inward and to retain the muscular and peritoneal layers in close contact with each other. It will usually be convenient to cut first the two lower stitches through the abdominal walls, and suture from below upward. When finished the peritoneal surface of the gastric wound may be again sponged with the mercuric chloride solution, together with the edges of the wound in the abdominal walls. Finally the abdominal wound is sutured, the stitches including the skin only or the muscular tissues as well. The smooth surface of the paunch acts as an internal pad and support, and with due care as to cleanliness, antisepsis and accuracy of stitching, it is rare to find any drawback to continuous and perfect healing. It is well to restrict the animal for three days to well boiled gruels, and for ten days to soft mashes in very moderate amount lest the wound in the paunch should be fatally burst open before a solid union has been effected.

RUMINITIS. INFLAMMATION OF THE RUMEN.

Prevalence in different genera. Causes, as in tympany and impaction, irritants, specific fevers. Symptoms: impaired rumination, tympanies, impactions, depraved appetite, fever, nervous disorders. Lesions: hyperæmia, petechiæ, exudates, ulcers, desquamation, swollen or shrunken papillæ. Treatment: remove cause, mucilaginous food, or gruels, sodium sulphate, or chloride, bismuth, bitters, mustard cataplasm, electricity.

This is not a prevalent disease but affects animals at all periods of life and is a cause of tardy and difficult digestion and rumination. It usually shows itself as a catarrhal inflammation and by favoring fermentation in the food, and torpor of the muscular walls of the organ contributes to tympany and impaction. It is more common in the ox than in the sheep owing, perhaps, to the more habitual overloading of the stomach and to the hurried, careless manner of feeding. In the goat it is rare.

Causes. Among the causes may be named tympany and overloading, so that all the dietary faults that lead to these may be set down as causes of inflammation. Irritants taken with the food, whether in the form of acrid plants (ranunculaceæ, euphorbiaceæ, etc.), musty fodder, irritant products in spoiled fodder, aliments which are swallowed while very hot or in a frozen state, and foreign bodies of an irritating kind are especially liable to induce it. Congestions of the paunch are not uncommon in specific infectious diseases like Rinderpest, malignant catarrh, anthrax, and Texas fever, and specific eruptions sometimes appear in aphthous fever and sheepox.

Symptoms. Rumination is slow and irregular, appetite capricious, tympanies appear after each feed, and there is a marked tendency to aggregation of the ingesta in solid masses, which resist the disintegration and floating which is necessary to rumination, and favor the occurrence of putrid fermentation. There is usually a tendency to lick earth, lime from the walls, and the manger, and a depraved appetite shown in a desire to chew and swallow foreign bodies of many kinds. Vomiting or convulsive rejection of the contents of the rumen is not unknown (Vives, Pattaes). There is slight fever with heat of the horns and ears, dry muzzle, and tenderness to pressure on the left flank. The bowels may be alternately relaxed and confined, and bad cases may end in a fatal diarrhœa. In other cases the disease may become acute and develop nervous symptoms, as in tympany and impaction. When the disease takes a favorable turn, under a careful ration, recovery may be complete in eight or ten days.

Lesions. These are violet or brownish patches of hyperæmia on the mucosa of the rumen, circumscribed ecchymoses, exudates in the sense of false membranes and even pin's head ulcerations. On the affected portions the mucosa is swollen, puffy, dull and covered with mucus, and epithelium may desquamate. The papillæ are often red, and thickened or shrunken and shortened. In the specific affections like aphthous fever and sheepox the lesions are rounded vesicles containing liquid. The ingesta is more or less packed in masses.

Treatment. If irritant foreign bodies have been taken rumenotomy is demanded. If caustic alkalies, acetic or other mild acid. If acids, lime water or magnesia. Feed well boiled flax seed, or farina gruels, and wheat bran or middlings in limited quantity. Solids may be at first withheld, coarse or indigestible food must be. It may be necessary to rouse the organ by 10 or 12 ozs. of sulphate of soda with a little common salt and abundance of thin gruels as drink. As a tonic the animal may take nitrate of bismuth $\frac{1}{2}$ oz., powdered gentian $\frac{1}{2}$ oz., and nux vomica 20 grains, twice a day. The application of a mustard pulp or of oil of turpentine on the left side of the abdomen may also be resorted to. A weak current of electricity through the region of the paunch for twenty minutes daily is often of great service.

HAIR BALLS IN THE RUMEN AND RETICULUM. EGAGROPILES.

Balls of hair, wool, clover hairs, bristles, paper, oat-hair, feathers, chitin, mucus, and phosphates. Causes: Sucking and licking pilous parts, eating hairy or fibrous products. Composition. Symptoms: Slight, absent, or, gulping eructation, vomiting, tympany, in young putrid diarrhoea, foetid exhalations, emaciation. Diagnosis. Treatment.

Definition. The term egagropile, literally goat-hair, has been given to the felted balls of wool or hair found in the digestive organs of animals. The term has been applied very widely, however, to designate all sorts of concretions of extraneous matters which are found in the intestinal canal. In cattle the hair licked from their skin and that of their fellows rolled into a ball by the action of the stomach and matted firmly together with mucus and at times traces of phosphates, are the forms commonly met with. In sheep two forms are seen, one consisting of wool matted as above and one made up of the fine hairs from the clover leaf similarly matted and rolled into a ball.

In pigs the felted mass is usually composed of bristles, (exceptionally of paper or other vegetable fibre), and in horses felted balls of the fine hairs from the surface of the oat, mingled with more or less mucus and phosphate of lime make up the concretion. These are found in the stomach, and intestines. In predatory birds the feathers and in insectivorous birds chitinous masses are formed in the gizzard and rejected by vomiting.

Causes. Suckling animals obtain the hair from the surface of the mammary glands hence an abundance of hair or wool on these parts favors their production. The vicious habit of calves of sucking the scrotum and navel of others is another cause. In the young and adult alike the habit of licking themselves and others especially at the period of moulting is a common factor.

Composition. Hair, wool, and the fine hairs of clover are the common predominant constituents, but these are matted together more or less firmly by mucus and phosphates, the ammoniamagnesian phosphate uniting with the mucus and other matters in forming a smooth external crust in the old standing balls of adult animals. The centre of such balls is made up of the most densely felted hair. In balls of more recent formation the external crust is lacking and the mass is manifestly hairy on the surface, and the density uniform throughout. These have a somewhat aromatic odor, contain very little moisture, and have a specific gravity approximating .716 (sheep) to .725 (ox). Ellagic, and lithofellic acids, derivatives of tannin, are usually present, and are abundant in the egagropiles of antilopes.

In the balls of recent formation, as seen especially in sucking calves, the hair is only loosely matted together, and often intermixed with straw and hay, and is saturated with liquid and heavier than the old masses. These are usually the seat of active putrefactive fermentation, and being occasionally lodged in the third or even the fourth stomach, the septic products act as local irritants, and general poisons. They are therefore far more injurious than the consolidated hairballs of the adult animal, and often lay the foundation of septic diarrhoeas and gastro-enteritis.

The balls may be spherical, elliptical, ovoid, or, when flattened by mutual compression, discoid.

Symptoms. Generally these balls cause no appreciable disturbance of the functions of the stomach. This is especially true of the large, old and smoothly encrusted masses. The museum of the N. Y. S. V. College contains specimens of 5½ inches in diameter, found after death in a fat heifer, which had always had good health and which was killed for beef. This is the usual history of such formations, they are not suspected during life, and are only found accidentally when the rumen is opened in the abattoir.

The smaller specimens, the size of a hen's or goose's egg, or a billiard ball, have produced severe suffering, with gulping, eructation, vomiting and tympany from obstruction of the demicanal or gullet, and such symptoms continued until the offending agents were rejected by the mouth. (Caillau, Leblanc, Prevost, Giron). Again they may block the passage from the first to the third stomach (Schauber, Feldmann, Adamovitz, Tyvaert, Mathieu).

In calves on milk they are especially injurious as beside the dangers of blocking the passages already referred to, the unencrusted hairs and straws irritate the mucous membranes and still worse, the putrid fermentations going on in their interstices, produce irritant and poisonous products, and disseminate the germs of similar fermentations in the fourth stomach and intestine.

Here the symptoms are bloating, colics, impaired or irregular appetite, foetid diarrhoea, foetor of the breath and cutaneous exhalations, and rapidly progressive emaciation.

Diagnosis is too often impossible. Tympanies, diarrhoea, colics, etc., may lead to suspicion, but unless specimens of the smaller hair balls are rejected by the mouth or anus there can be no certainty of their presence. If arrested in the cervical portion of the gullet they may be pressed upward into the mouth by manipulations applied from without. The looped wire extractor may be used on any portion of the oesophagus. If lodged in the demicanal the passage of a probang will give prompt relief. If retained in the rumen and manifestly hurtful, rumenotomy is called for as soon as a diagnosis can be made.

FOREIGN BODIES IN THE RUMEN AND RETICULUM.

Common. Harmless or injurious. Perforating objects. Traumatism of contiguous organs. Causes; hurried primary mastication, morbid appetite. Bodies found. Lesions; catarrh, perforations, congestions, ulcerations of mucosa, abscess, trauma of liver, spleen, diaphragm, abdominal and thoracic walls, lung, pleura, pericardium, heart. Symptoms; absent, or, indigestion, tympany, eructations, hepatic, respiratory or circulatory disorder, colics, local tenderness, crepitation, substernal exudate, costiveness, difficult urination or defecation, bloody faeces, nervous disorder. Treatment; Prevention; avoidance of causes, gravitation methods, incision.

These are so frequent that they can hardly be looked on as abnormal, but they must be accepted as pathological when they cause serious irritation or digestive disorder. This result is seen especially in the case of cutting or sharp pointed bodies, which beside wounding the walls of the rumen, show a marked tendency to advance to the heart and penetrate it, or to perforate the liver, diaphragm or abdominal walls and even to cause a fistula through which the ingesta escapes.

Causes. The common cause in cattle is the habit of swallowing, after one or two strokes of the teeth, any small object that is mixed with the provender. Next to this comes the habit of stabled cows, and of such as suffer from a lack of phosphates or other important element in the food, of licking, chewing, and swallowing articles that can in no sense be considered as alimentary.

Among the rounded or smooth bodies found in the rumen and reticulum may be named coins, rivets, fragments of wood, cords, pieces of rope, leather, gloves, cloth, small garments like vests or caps, ribbons, bones, pieces of lead, dried paints, cotton waste used as packing for machinery, shot, and even small animals such as frogs, toads, and snakes; also sand and pebbles.

Among sharp or pointed bodies the most common are nails, pins, needles, baling wire, pieces of iron or other metals, knives, scissors, forks, fragments of glass, thorns, etc.

Lesions. These are as varied as the nature of the traumatic agent, the seat and nature of the trauma. The rounded bodies, if non-poisonous, act merely by attrition of the walls and tend to induce a local catarrhal inflammation. Yet even sharp pointed bodies may prove comparatively harmless. The museum of the N. Y. S. V. College contains a pocket knife which had remained open in the rumen for a length of time without producing any visible injury.

Sharp and pointed bodies are especially liable to be entangled in the cells of the reticulum; so that this viscus is the most common seat of the resulting trauma. Around this there occur hyperæmia, exudation, thickening and centrally ulceration, which may lead into a fistula or abscess, confined it may be to the wall of the viscus, or continued into the surrounding organs. In this way may be implicated, the liver, the spleen, the diaphragm, the abdominal or thoracic walls, the lung, the pericardium or the heart. The pus is always foetid and usually mixed with alimentary matter. If it approaches the surface it may burst and allow exit to the offending body. If it encroaches on the liver, symptoms of hepatic disorder supervene. Its progress through the lung or pleura is marked by objective symptoms of pulmonary or pleural inflammation (crepitation, flatness or percussion, creaking or friction sounds), but without the customary amount of hyperthermia, and with some evidence of gastric disorder. When the pericardium is reached there are the usual signs of pericarditis, attended by comparatively little fever, and a doughy swelling beneath the sternum is added to the objective signs of exudation in the pericardium.

Among the peculiar routes followed by such bodies may be named the following: to the side of the ensiform cartilage; through an intercostal space; into a chondro-costal articulation; through the muscles of the flank; and even in the region of the croup. If the attendant abscess or fistula bursts into a serous cavity it determines septic peritonitis or pleurisy, while in the lung it may cause septic pneumonia. As a rule, however, this is prevented by the excessive quantity of exudation.

Symptoms. These are extremely variable according to the seat and nature of the lesion. So long as the foreign body is confined in the rumen there is usually no symptom. Even when it has penetrated surrounding organs the symptoms are usually for a time very obscure. A few years ago a cow entered the prize ring, at the New York State Fair, was awarded first prize, and died a few minutes later from a piece of baling wire penetrating the pericardium. When symptoms are patent there are usually early indications of indigestion in the rumen, capricious appetite, sluggish and

imperfect rumination, dullness, tardy movements, frequency and foetor of eructations, colic pains, grunting when moved, and wincing under pressure in the left hypochondrium. Pressure below, to the left of the ensiform cartilage is sometimes particularly painful.

If the object is advancing toward the heart a broad area or line of dullness may often be detected by percussion on the left side of the chest and under the acts of respiration or walking, gurgling sounds may be heard along this line. The movements of the ribs on the same side are limited as compared with the other side, and straining in defecation or urination may be manifestly painful and accompanied by groaning. For the same reason costiveness is liable to set in.

When the body approaches the skin there is formed a large, hot, phlegmonous swelling similar to that which marks the advance of an intercostal abscess.

In special cases there are symptoms of disease of the particular organ penetrated. Hepatitis, splenitis, and peritonitis are occasionally seen. Eggeling notes a fatal hemorrhage from the wounding of the œsophagus by a nail, and Brauer bloody fæces from penetration of the pylorus by a piece of glass. In other cases fatal results have followed on trauma, thickening and obliteration of the pylorus. (Olivier).

In cases of the ingestion of shot, the spray of bullets, white or red paint or other form of lead, the special symptoms of lead poisoning supervene. (See lead poisoning).

Treatment. As a rule this is unsatisfactory and especially in cases implicating the pericardium, as the symptoms may be entirely overlooked until sudden death occurs. Hence the great value of preventive measures, and above all the careful removal of all nails from the vicinity of fodders. Bailed hay is always dangerous, and when used, each bale should be carefully freed from its wires and any short pieces removed. Pointed metallic bodies of all kinds should be removed from the pastures and stables.

In case the migrating foreign body leads to the formation of a superficial phlegmon at any point, this should be freely opened and the offensive agent extracted.

If the lesion in the reticulum has been diagnosed, the combined methods of Kolb and Schobert should be tried. Turn the animal on its back with the head and shoulders up hill, and employ strong pressure, with the foot, in jerks, over the ensiform cartilage. The object is to slide the foreign body back into the viscus, and success is claimed in seven cases out of nine.

Failing in such methods there remains only the operation of rumenotomy and the removal of the offending bodies so far as they can be reached.

TUMORS OF THE RUMEN AND RETICULUM.

Tumors of different kinds have been found in the walls of these organs, though by no means frequently. Epithelial hypertrophy and papilloma have been found in the ox the former undergoing necrotic changes. Chondroma is reported by Kitt, Sarcoma by Cadeac and Beylot. There seems to have been a special tendency to invade the demicanal, and to interfere with deglutition, rumination, and the passage of food into the third stomach. The impairment and loss of appetite and of rumination, the presence of tympany, and the general loss of condition are suggestive. If the disease of the demicanal leads to antiperistaltic movements of the œsophagus which can be felt by the hands pressed on the jugular furrows the diagnosis may possibly be made.

Treatment is manifestly hopeless. To be effective it must be surgical and would too often entail excision of the affected part of the viscus and careful suture of its walls. This would be even more hopeless when the demicanal was the seat of disease.

Temporary palliation might be secured by a sloppy diet, the withholding of all rough food which would demand rumination, and the use of common salt, saline laxatives and abundance of water.

ANIMAL PARASITES OF THE RUMEN AND RETICULUM.

Infusoria. *Amphistomum Conicum*. Actinomycosis. Tumors: Papilloma, Chondroma, Sarcoma. Treatment—palliation or surgical.

Colin describes and figures as many as eight varieties of infusoria found habitually in the first two stomachs. All appear to be introduced with the food, in the infusions of which they also appear, and they find in the fermenting mass of ingesta in the first two stomachs a favorable medium in which to grow and multiply. It cannot be shown that they are in any way detrimental and they have even been supposed to be beneficial to digestion as glycogen has been demonstrated in their protoplasm. Like the bacterial ferments they doubtless assist in the disintegration of the mass of food.

Amphistomum Conicum. This is a trematode worm about the size of an apple-seed (10 millimetres long by 2 millimetres thick), rounded at both ends, slightly curved on itself, and, as usually found, of a bright red color. It attaches itself by its sucker (on thick end), usually in the vicinity of the demicanal. Its life history is closely allied to that of the distomata, but as it is not known to prove at all injurious to its host, it possesses no pathological importance.

Actinomycosis of the Rumen and Reticulum. Tumors of this fungus are sometimes found in the walls of the two first stomachs projecting in the form of polypi, or imbedded in the thickness of the coats. Where they are completely covered by the mucous or serous membrane their true nature is not readily recognized. When incised they show the characteristic yellow granules made up of club-shaped cells, though the usual stellate arrangement may be somewhat imperfect. From the serous surface the growth may invade the different adjacent organs. It is impossible to diagnose a primary actinomycosis of the rumen, if unaccompanied by more superficial lesions, but, if the disease is recognized elsewhere, the same general treatment with iodide of potassium will dispose of these formations as well.

IMPACTION OF THE OMASUM (THIRD STOMACH).

Definition. Synonyms. Causes, torpid action, defective insalivation, inactive rumen, fever, inflammation, spinal paresis, dry, fibrous, innutritious food, fungi, ergot, smut, privation of water, or of succulent food, microbial ferments and their products, chronic heart disease, dry farinas, extreme changes of diet, brain disease. Symptoms: slight or violent; ill health, impaired appetite and rumination, grunting, tympany, diarrhoea, constipation, baked coated faeces, percussion signs, separation from herd, red eyes, stiffness, agalactia, foetid eructations, paralysis, drowsiness, stupor, delirium, nervous symptoms mostly in acute cases. Course in chronic and acute cases. Diagnosis; from pneumonia, and overloaded rumen. Lesions; solid impacted omasum, baked contents, shedding of epithelium, congestion, petechiae, ulceration, empty, and congested abomasum and small intestine. Treatment: laxative food, purgatives, stimulants, antiseptics, enemata, stimulants of peristalsis, counter-irritants, mucilages, laxative diet, tonics.

Definition. This may be defined as a form of indigestion of which the prominent feature is the drying and impaction of the ingesta between the folds of the third stomach. It may seem to be a primary disease, but in very many cases it occurs as a result of some acute febrile or inflammatory affection.

Synonyms. As the disease has been long popularly known it has received a variety of popular names which are more or less characteristic. Dry murrain, Clewbound, Fardelbound, Stomach staggers, Grass staggers, Vertigo, Chronic dyspepsia, and Chronic indigestion may serve to illustrate these.

Causes. Torpor of the manifolds and the suppression of secretion of saliva, together with the absence of a continuous access of waves of liquid floating the finely divided food from the mouth or rumen to the third stomach are prime conditions of dessication of the contents. The third stomach, like the two first, has no provision for liquid secretion, but is dependent for its supply on constant flushing from in front. If therefore feeding and rumination are interrupted as the result of a febrile disease, if the secretion of saliva is in great part suppressed, if the vermicular movements of the rumen and resulting overflow into the third stomach are checked, and if in addition the omasum itself is rendered torpid, the ingesta compressed between its folds becomes drained of its liquid, and in no great length of time, to such an extent that it may be rubbed up into a dry powder. All this is a necessary result of an acute febrile condition, and therefore all febrile and inflammatory affections tend to drying and impaction of the contents of the omasum. If therefore the observer were to go no further than this he would have a very simple pathology, for all or nearly all fevers and inflammations would be to him simply impacted omasum. In the great majority of cases this condition is to be looked on as a secondary and subsidiary affection, while the real primary disease has still to be sought for.

Some explanation of the special susceptibility of the third stomach in such constitutional troubles, is found in the source of innervation of the viscus. Colin and Ellenberger could rouse the movements of the first two stomachs but not of the third by electric stimulation of the vagus, while the third stomach was excited to action by excitation of the spinal cord and of the sympathetic twigs proceeding from this to the manifolds. Ellenberger indeed avers that the walls of this viscus are abundantly furnished with ganglionic cells which are called into action by this sympathetic stimulus. The innervation being derived from an independent source, derangement of the third stomach may be quite independent of any primary disorder of the first, and the omasum deriving its motor supply from sources so closely related to the vaso-motor ones, may give an additional explanation of the intimate connection of its disorders with febrile and inflammatory diseases.

But while acknowledging the controlling influence of torpor or paresis of the omasum, it would be an error to follow Cadeac in denying the influence of food as a cause of impaction. It has long been notorious that impaction of the omasum is preëminently a disease of winter, or of the period of dry feeding. It occurs in cattle fed on dry, fibrous, innutritious fodder, and especially when there is a scarcity of water, or when in connection with severe frost the usual water supply has been frozen up. It prevails in stock turned in spring or autumn on pastures in which the fresh green grass grows up among the dead, dried and withered stems of a previous growth and tempts them to eat them. It appears when the stock consumes corn or cornstalks affected with smut or certain other fungi, or the cereals or grasses suffering from ergot or smut, but this is especially the case when

there is also a privation of water, whereas, with an abundant water supply or a partial ration of roots, potatoes or ensilage the danger is greatly reduced. Sometimes a change from soft to very hard water appears to act as a cause but whether from a special astringent action or a disinclination to consume the usual amount has not been made clear.

It must be allowed that the sheep and goat which habitually drink little, suffer far less from this affection than the ox which drinks freely, yet allowance must be made for the constitution and long settled habits of the genus, and we must not forget that it is usually under privation of water or a restricted supply that the ox suffers.

Among other causes must be named fermented food, the microbial ferments and their products, serving to render the organ torpid, but also to produce fever, lessened secretion and an arrest or retardation of liquid supplies from the mouth or rumen.

Chronic heart disease, causing blood stasis in the omasum, appears to induce torpor and favor impaction.

The ingestion of lead has a very direct action in producing paralysis and consequent impaction.

Finally, finely divided dry food like meal or bran, swallowed hastily, tends to pass in large amount directly into the omasum, and, before the animal has become accustomed to the ration, is liable to clog the viscus and induce impaction.

In nearly all cases, the commencing impaction entails a certain rise of temperature and suppression of secretions, so that the malady tends to move in a vicious circle, each new step tending to aggravate the already existing condition. In chronic cases, which are very common, a careful record of bodily temperature shows oscillations, above and to the normal, at irregular intervals, each rise tending to add to the impaction.

The most acute and fatal forms of the affection occur in connection with a sudden change from dry to rich, luscious, green food in spring, the unwonted stimulus giving rise to general irritation of the whole gastric mucosa, with disordered and impaired function of all four stomachs, but especially of the third. Such cases are usually congestive and inflammatory and the suspension of the gastric movements is a grand cause of impaction. In such cases too the brain or spinal cord, or both, are seriously involved, and the early death is preceded by torpor, paralysis, violent delirium or convulsions, following largely the type of acute lead poisoning.

Symptoms. These vary according to the degree of impaction or gastric torpor, from simple, irregular, or suspended rumination (loss of cud) to the most severe gastric and nervous disorder.

The slighter or less acute cases are marked by a failure to re-establish regular rumination on partial convalescence from a fever or inflammation. The hyperthermia subsides, but the appetite remains poor and capricious, the muzzle dry, the eyes dull, the spirits low, breathing quickened and occasionally accompanied by a moan, especially when moving down hill, slight tympanies of the rumen may appear and the contents of that organ seem consolidated and may be felt as solid masses when pressure is made by the hand. The mouth is hot, clammy and foetid, and the bowels costive, the faeces being passed in small amount and in the form of hard, black pellets, covered by a film of mucus, or streaks of blood, and containing particles of undigested food. This not unfrequently merges into a transient diarrhoea to be followed in turn by renewed constipation, and such alternations may repeat themselves again and again. The omasum is so deeply seated under the ribs on the right side that exploration is unsatisfactory, especially in the milder cases, yet pressure of the closed fist upward and forward below the middle of the chest will give the impression of a specially solid resistance and the patient may indicate suffering by a moan. Percussion with the closed fist has the same effect. There may be slight tremors of the body, the horns, ears and limbs are cold, and the hair erect in patches, dry and lustreless.

In cases occurring independently of previous disease, diarrhoea may be the first symptom noted, the malady being preceded by local irritation and congestion, but this soon gives place to constipation with alternating diarrhoea and the general train of symptoms above mentioned. The animal leaves the herd and is found lying apart on its left side with the nose in the right flank, the pulse and breathing quickened, the eyes congested, and a moan emitted occasionally in expiration. This is increased if the patient is raised and driven, especially down hill. He walks with stiff, arched

back, unsteady gait and dragging limbs. Appetite may not be entirely lost at first, but only impaired and irregular, and as rumination ceases, grinding of the teeth becomes common. The secretion of milk is diminished or altogether arrested, and emaciation advances day by day. Foetor of the eructations, the result of prolonged and septic fermentation in the rumen, is often a marked symptom.

This form may last from ten to fourteen days and merge finally into paralysis of the hind limbs, drowsiness and stupor, or delirium and convulsions.

In the more acute cases resulting from a sudden access of green food, a change of water, or the ingestion of irritant plants, the affection partakes more or less of the nature of congestion or inflammation of the viscus, and may run a rapidly fatal course. The animal may be seen apart from the herd in the characteristic recumbent position, with eyes red and glassy, eyelids semi-closed, and much drowsiness and stupor, but when raised he may still feed in a sleepy, listless manner. The bowels may be loose or confined, the pulse and breathing accelerated, the right hypochondrium firm and tender, and as in the other forms the crepitating sound of fermentation is slight or absent over the region of the manifolds. Soon nervous disorder appears, the eyes glare wildly, the animal seeks relief in motion, it may be in a straight line, or to one side, and being blind and unconscious of obstacles he may fall into pits, or ditches, dash against trees, fences or buildings, and if they offer sufficient resistance he will continue pushing, breaking teeth or horns, and subject to violent muscular contractions, causing even the grubs to start from the back. The nervous disorder is often further shown in loud and terrified bellowing as if chased by a dog or gored by one of its fellows.

Course. Chronic cases may continue almost indefinitely the victims showing merely poor health, impaired digestion, and steady loss of condition. After death the omasum is sometimes found to contain dried materials, such as the animal has not had access to for from three to five months.

In those which end in an early recovery there occurs a free and abundant diarrhoea, the faeces containing solid flattened masses with black baked or polished surface, the result of the detachment of the impacted layers from between the folds of the third stomach. The tympany of the rumen subsides, crepitation is renewed in the rumen and omasum, there is free rumbling in the bowels, and the appetite gradually improves. The softening and removal of the dessicated contents are slow and it may be weeks before there is a complete restoration to normal conditions.

Diagnosis. The hurried pulse and breathing and the grunting with expiration may be mistaken for pneumonia or pleurisy, but the distinction can be made as in impacted rumen. There is at first no fever, the tenderness is confined to the right side, the percussion dullness of the chest is in the posterior part and distinctly referrible to the loaded abdominal viscera, it is attended by no pulmonary crepitation, indeed crepitation in rumen and omasum is lessened or abolished, there is no pleural effusion, but there are the unquestionable signs of gastric and intestinal disorder.

It may be confounded with overloading of the rumen, but in the latter case the distension occurs rapidly, there is little or no indication of movement of the viscus, appetite and rumination are usually early suspended and the gaseous eructations are not putrid.

Postmortem Appearances. These are essentially connected with the impaction of the omasum. This organ is gorged to twice its normal size or larger, firm, solid and resistant, not easily taking an impression of the finger, and having at times an almost stony hardness. When incised the intervals between the folds are sure to be packed with dessicated food, often so dry in the upper part that it may be rubbed down into a grayish powder, and it has been compared to the cakes of linseed as they come from the press. The surface of such cakes is smooth and dark, and usually covered by a layer of epithelium which has detached itself from the surface of the fold. This is usually quoted as a morbid desquamation, but inasmuch as we frequently see it in perfectly healthy conditions in animals killed in abattoirs, it must be admitted to occur also as a normal physiological exfoliation. The exposed mucosa shows spots and patches of congestion, extravasation, and even at times ulceration, or slight areas of necrosis.

The rumen shows the result of torpor and inactivity. The ingesta is largely packed into solid masses, which have advanced from the simple acid fermentation, to evident putrefaction with offensive emanations. The abomasum is empty or nearly so of ingesta, but contains abundance of

mucus and shows patches of congestion as in prolonged abstinence.

The small intestine is also empty and collapsed, with considerable redness and congestion. The larger intestine contains a small quantity of feculent matter, dry, massed in small pellets and with smooth glistening surface. Mucus is abundant and dense.

Treatment. This must follow the same lines as in impaction of the rumen with the understanding that the response is less certain and the result somewhat more tardy in reaching complete convalescence. In mild and chronic cases a liberal allowance of flaxseed tea, several bucketfuls per day, will often succeed.

In using purgatives those are usually the best which lead to drinking abundantly. For the ox a pound each of Epsom and common table salt, with an antiseptic stimulant like aqua ammonia (3 to 4 drachms) or oil of turpentine (2 oz.) will often act favorably. The sodium chloride is antiseptic, and induces ardent thirst and if there is free access to water, tepid or not too cold, purgation is early secured and the impacted cakes in the manifolds are slowly softened, detached and removed. But unless water is given freely the salt will prove irritating and even injurious.

In obstinate cases, and in the absence of indications of gastric or cerebral congestion the addition of 20 croton beans or 20 drops of croton oil will be excellent. Nux vomica (1/2 drachm) is also of value in rousing the torpid nervous action. Injections are always in order, and it is recommended to use these cold so as to rouse the muscular action of the intestine and stomach.

Some of the newer remedies which rouse the contractility of the digestive organs and at the same time stimulate secretion serve an excellent purpose in these cases. Eserine 1 1/2 grain, veratrine 1 grain, barium chloride 10 to 15 grains, or pilocarpin 3 grains may be given hypodermically in addition to the usual purgative. The pilocarpin is theoretically the best as its tendency is to cause free secretion from all mucous surfaces, and even a slight secretion from the omasal folds will greatly favor detachment and discharge of the impacted plates. These as well as the stimulants may be repeated as the effects pass off. The purgatives on the other hand should be given at first in a large dose, and not repeated except under the stress of necessity as their constant repetition in small doses seems to nauseate the animal and even to retard action. In the case of profuse secretion from the kidneys however it may be supposed that the saline agents have passed off in that way and a purgative may be safely repeated. It may be well however to use one which is less likely to stimulate the kidney, such as castor, olive, or raw linseed oil or senna.

The patient may be several days or even a week without alvine discharge and yet do well. If there are fever and other indications of gastric congestion a blister to the right hypochondrium may be of value. Rub well with oil of turpentine and then with a pulp of the best ground mustard and tepid or cold water and cover with sheets of thick paper to prevent evaporation.

If nervous, symptoms are manifested by dilated pupils, blindness, congested conjunctiva, hot horns and ears, and drowsiness, or excitability apply cold water or an icebag to the head and continue as long as may be needful. If the patient should become violently delirious he may be fastened to a beam overhead in the centre of the stall so as to prevent him from injuring himself or others.

In these cases the more violent and irritant purgatives are to be avoided, and decoctions of slippery elm, linseed or gum may be given to sheathe and protect the irritated membrane.

Even though a free action of the bowels has been secured it is not to be assumed that all impacted material has been removed. A specially laxative diet of roots, ensilage, or succulent green food, with a liberal supply of salt, and free access to water should be kept up for some weeks to secure a complete softening and expulsion of the impacted material. Repeated small doses of laxative medicine may be requisite to bring this about. As a rule a course of tonics, and above all of nux vomica is valuable in re-establishing the normal tone of the stomachs and intestines.

INFLAMMATION OF THE OMASUM.

Involved in rinderpest, Texas fever, malignant catarrh, etc. Diphtheritis, Tuberculosis, Irritant poisons, Traumatism, Impactions, change to green food, etc. Lesions: Congestion, ramified redness, petechiæ, desquamation, softening, necrosis, false membranes, ulcerations, pigmentation, papillary growths, impaction. Symptoms: those of impaction with fever. Course, Treatment: demulcents, laxatives, blisters, bismuth, eserine, veratrine, pilocarpin, electricity, careful diet.

Like the rumen the omasum is the seat of local inflammatory lesions in certain specific fevers. Thus in Rinderpest, and Texas fever it is almost always the seat of patches of congestion and blood extravasation, and in the latter of necrosis and perforation of the folds. Similar lesions sometimes appear in malignant catarrh and anthrax. Dieckerhoff describes exudates, ulcerations and even perforations in pseudo-diphtheritis, and Brückmüller, congestions and ecchymoses in connection with a cutaneous rash. Tuberculosis of the organ is somewhat rare and is held to be due to the swallowing of bronchial secretions in cases of pulmonary tuberculosis.

Cases of primary inflammation are rare, in keeping with the soft finely divided condition in which the food reaches the organ. It may, however, occur in case of the ingestion of arsenic and other irritant poisons, or of goring, kicks and other injuries on the right hypochondrium, or from the irritation attendant on impaction, or again from the stimulus of a sudden change to rich green food.

The *lesions* in such a case are congestion of the folds with patches of ramified redness, blood extravasations, desquamation, softening or even gangrene. False membranes, perforating ulcers, and erosions are sometimes present. In the chronic forms grayish or slate colored pigmentation of the mucosa, congestions and papillary growths are common.

In both acute and chronic forms the congestion entails loss of contractility and thus impaction and drying of the ingesta between the folds of the organ are constant.

Symptoms. These are the symptoms of impaction of the manifolds, impaired appetite and rumination, formation of solid masses in the rumen, tympany, tenderness or pressure on the right hypochondrium, irregularity of the bowels, arching of the back and grunting when made to walk. The addition of fever, as evidenced by rectal hyperthermia, hot horns, ears, legs and muzzle, serves to diagnose it from simple impaction.

The *course* of the malady is the same as in impaction, but with an even greater tendency to aggravation and a fatal result as the inflammation entails a paresis of the walls of the viscus which favors a constant accumulation and dessication of the interlamina material.

Treatment. This must be largely on the same line as in impaction, laxatives of sulphate of soda, a diet of flaxseed or barley gruel, and drinking water rendered demulcent with slippery elm. These must be supplemented by a mustard or other blister to the right hypochondrium, by soothing doses of nitrate of bismuth ($\frac{1}{2}$ ounce), and hypodermic injections of eserine ($1\frac{1}{2}$ grain), veratrine (1 grain), or pilocarpin (3 grains). A current of electricity sent through the right hypochondrium once or twice a day, will further be desirable. When convalescence has set in, mashes of wheat bran and middlings may be allowed, to keep up the flagging vigor, and the patient should be returned to solid, fibrous food by slow degrees only.

TUMORS OF THE OMASUM.

Papilloma. Sarcoma. Actinomycosis.

Tumors of the omasum have been seen only as papilloma, and sarcoma.

The **papillomata** result from hypertrophy of the normal papillæ, and their general appearance resembles those of the pharynx, gullet and paunch. They sometimes grow to the size of the fist or larger, with a cauliflower appearance, their increase and the formation of pedicles being favored by the active contractions of the muscular coat of the manifolds. They may be red and vascular if recent, are usually white if older, and may become somewhat horny on the surface, but soft and friable within. When they attain a large size they may obstruct the passage to the fourth stomach, tending to impaction of the manifolds and arresting digestion and nutrition.

Sarcoma of the third stomach has been recorded by Paule, Kitt and Schütz as developing in the subserous tissue and forming a layer on the surface of the organ. This bulges out in rounded swellings of irregular sizes, and may show various degenerations—caseous, calcic or necrotic. The structure shows fusiform and rounded cells more or less numerous imbedded in a fibrous stroma.

ACTINOMYCOSIS OF THE ABOMASUM.

This has been seen once by Professor Axe. Its true nature is unrecognizable during life, but if other formations of the same kind betray the nature of the lesion, the treatment by iodide of potassium may be resorted to with good hope of success.

INDIGESTION OF THE ABOMASUM.

Causes: excess of water—ice cold, or after privation. Symptoms: colicky pains, local perspirations, right flank gurgling, diarrhoea, arched back, anorexia, or nervous symptoms. Prevention: Treatment: stimulants, carminatives, exercise, electricity, friction, stimulants of peristalsis.

This has been observed as the result of ingestion of an excess of water, and especially ice cold water, by work oxen, or overdriven animals which have been long exposed to the heat of the sun and subjected to violent exertions without drink. The habit of allowing water only at long intervals, though it is being abundantly eliminated not only by kidneys and bowels, but also by the accelerated breathing and the sudation, causes consuming thirst, and when brought to the drinking place, the subject drinks inordinately before eating. Much of this liquid is passed at once into the abomasum, which with a capacity of 20 to 25 quarts, becomes overdistended and irritated. Much of the water passes speedily into the bowels, rousing these also into unwonted action. The sudden distension appears to cause spasmodic contractions of the abomasum, which are aggravated if the liquid is cold, and a violent though transient suffering is induced.

Symptoms. These appear suddenly after the drinking of the cold water and consist in the most violent colicky pains, twisting of the tail, kicking at the belly, lying down and rising at short intervals, moaning, looking at the flanks, anxious countenance, and the breaking out of perspirations around the ears, on the neck or belly. There is no tympany of the rumen but there are some fullness and active gurgling on the right side of the abdomen. The attack does not usually exceed one or two hours in length, and a profuse diarrhoea brings relief, the alvine discharges being very watery with considerable mucus and some undigested food principles.

In exceptional cases it has lasted for six hours and even in the more transient cases, there is liable to remain for a time dullness and prostration, advancing of the hind legs under the body, anorexia and suspended rumination. Cruzel records two cases in which the small intestine was ruptured as the result of too vigorous driving of the patients. Other cases have perished from the coexistent diarrhoea. Nervous symptoms also may appear as in other gastric disorders. The usual result is recovery after a very transient illness. This short and favorable course, and the evidence of cause and symptoms sufficiently identify the disease.

Treatment. Prevention should be sought by avoidance of cold water in excess, when the animal is heated, fatigued and thirsty. A little food, an occasional mouthful of water, or a drink of warm water and meal will act prophylactically. When the animal is attacked alcoholic drinks, ammonia, carminatives (pepper, ginger, fennel, caraway, peppermint, chamomile) or even strong tea or coffee may be used to advantage. Careful walking exercise is also useful with friction to the abdomen, or the use of electricity. Cadeac advises stimulants of the peristalsis—eserine, veratrine or pilocarpin subcutem; senna, podophyllin, or castor oil by the mouth.

INDIGESTION OF THE FOURTH STOMACH IN THE YOUNG.

Causes: Symptoms: Dullness, recumbency, inappetence, colic, acid eructations, abdominal tension, costiveness, diarrhoea, emaciation. Lesions: Sour coagula in stomach, puffy or congested mucosa, undigested casein in bowels and faeces. Prevention as in infector gastro-enteritis. Treatment: Elimination, antacid, antiseptic, carminative, stimulant, rennet.

While the fourth stomach in the mature animal is protected against danger by the preparatory work of the first three, and by their action in retarding the food in its progress, and allowing it to pass into the fourth only when thoroughly comminuted and then only in small quantity at a time; in the suckling on the other hand the milk passes at once into the abomasum, which is thus rendered as susceptible as in the monogastric animal.

Causes. The causes are almost identical with those set forth under *infective gastro-enteritis* of the suckling, acting however with less force, or on a less susceptible system. Overloading and the resulting paresis; unsuitable milk from another genus, or from unhealthy, overworked, or over-fed specimens of the same genus; the ingestion of hard, insoluble, indigestible or toxic aliments, and exposure to cold and wet are among the most common direct causes. As secondary causes are overfeeding of the cows, and bringing up the calves on the pail with all kinds of substitutes for the milk of the mother.

Symptoms. Dullness, lack of sportiveness and of appetite. The patient lies down a good deal, but is nervous or restless, and when up shows colicky pains by movements of the tail and hind limbs. He may moan gently or bellow frequently. The muzzle is dry, the mouth clammy, hot and sour, the abdominal muscles rigid and the belly often somewhat swollen, and resonant on percussion. Acid eructations are common. At first there is costiveness, but in a few hours diarrhoea sets in and usually proves critical, clearing away the offensive and irritant materials and paving the way for recovery. The tension of the belly lessens, by degrees, the appetite returns, the bowels resume their normal tone and in twenty-four or forty-eight hours health may be fully restored.

There is, however, always danger of the supervention of gastro-enteritis of which in many instances the above-named symptoms indicate the first stage. In all cases it interferes with the growth and fattening of the subject.

Lesions. We observe the presence in the stomach of masses of coagulated milk, undigested, mixed with an excess of mucus, and exhaling a sour or even a septic odor. The mucosa is more or less red and congested with swelling and opacity of the epithelium. The bowels also contain the undigested flocculi of casein, more or less fermented and which have escaped the action of the peptic liquids.

Prevention. This consists in the avoidance of the causes, and as these are in the main the accessory causes of infective gastro-enteritis in the suckling, it will save repetition to refer to the article on that subject.

Treatment. In an attack which is caused and maintained by undigested and irritant materials in the stomach, the first consideration must be the elimination of these offensive matters. An ounce of castor oil with a teaspoonful of laudanum for calf or foal will usually effect this purpose. Or $\frac{1}{2}$ oz. calcined magnesia or carbonate of magnesia, or of manna $2\frac{1}{2}$ drs. or cream of tartar may be substituted for the oil. The addition of a carminative or stimulant (1 dr. syrup of anise, or tincture of cinnamon, 1 oz. whisky or brandy, or $\frac{1}{2}$ oz. oil of turpentine for calf or foal) will often check the diarrhoea and fermentation.

In weak subjects the stimulant may be used with a drachm of chalk or of bicarbonate of soda, and 5 grains powdered nux vomica.

In all cases alike the use of rennet is very advantageous. One-eighth of a calf's rennet being steeped in a bottle of sherry wine and the liquid given to the amount of a tablespoonful ($\frac{1}{2}$ oz.) with

each drink. This secures proteid digestion and checks fermentation thereby hindering the formation of the offensive products which maintain the irritation and disorder.

INFECTIVE GASTRO-ENTERITIS IN CALVES, LAMBS AND FOALS. WHITE SCOUR.

Causes: early life, exclusive activity of fourth stomach, faulty milk, absence of colostrum, milk from advanced lactation, milk of other genus, or altered by excitement, or unwholesome food, excess on hungry stomach, soured, fermented, feverish milk, putrid milk, leucomaines, overdistension of stomach, farinaceous food, hair balls, morning and evening milk, milk after first calf, composition of milk by genus, ruminant's milk to monogastric animal, infectious microbes—bacilli, micrococci. Symptoms: costiveness, inappetence, listlessness, tense abdomen, acid eructations, foetid diarrhoea, becoming yellow or white, general foetor, staring coat, pallid mucosæ, tucked up tender abdomen, weakness, emaciation, fever, bloating, frothy dejections, arthritis, peritonitis, pneumonitis, hepatitis, ophthalmia, laminitis, etc. Mortality: in foals, calves, lambs. Lesions: gastric and intestinal congestions, exudations, necrosis, incoagulable blood in foals, anæmia. Prevention: normal feeding, expulsion of meconium, care of nurse, adapt composition of cow's milk to genus of nursing, warmth, lime water, rubber teat, Pasteurizing, disinfection, separation from infected animals and places, breed from robust parents. Treatment: elimination, antiseptics, boiling milk, rennet, ipecacuan, carminatives, astringents, tar, calomel and chalk, gum, flaxseed, elm bark.

Causes. The abomasum in the adult is protected against disorder, by the normal activity of the first three stomachs, macerating the food, presiding over the second and more perfect mastication, grinding it between the omasal folds into a firmly attenuated pulp and delaying its progress so that it arrives at the fourth stomach at short intervals and in small quantities only at a time. It follows that this organ is rarely involved in serious disorder unless as the result of the ingestion of poisons, or of excess of water, or from the presence of parasites. In the very young ruminant, however, the condition is reversed, the first three stomachs are as yet undeveloped, and incapable of receiving more than the smallest quantity of food or of retaining the same, and the abomasum alone is functionally active and receives at once practically everything that may be swallowed. In the first few weeks of life therefore the ruminant is exposed to almost the same dangers, from overloading, indigestion, inflammation and poisoning as is the monogastric animal. For the time, indeed, the undeveloped ruminant is in its physiological and pathological relations, a monogastric animal. For this early life therefore whatever applies to the soliped applies equally well to the ruminant.

When allowed to suck at will from a healthy nurse, which completed its gestation about the time the young animal was born, indigestion is rare. But whatever interferes with the normal supply is liable to cause derangement. The withholding of the first milk—colostrum—the laxative properties of which are essential to clear away the intestinal accumulations of foetal life—meconium; the placing of new-born offspring on the milk of nurses that bore their young many months before; bringing up of foals on cow's milk; working, overdriving, hunting, shipping by rail, or otherwise exciting the dams; allowing too long intervals between the meals—feeding morning and night only, or morning, noon and night, the nurse being kept at work or pasture in the interval; feeding unwholesome food to the nurse; bringing up by hand, on cold and even soured milk, or that which has become contaminated by putrid leavings in the unscaled buckets. Some of these causes should be emphasized, for example the milk of excitement and fever, milk that is soured or putrid, and milk suddenly swallowed in excess. The nurse which is fevered or subjected to over-exertion has produced an excess of tissue waste and leucomaines which largely escape from the system in the milk. This milk is therefore at times unwholesome and even poisonous. Mares subjected to severe work or that fret much under lighter work, cows carried by car or boat, or driven violently, and any nursing animal which has been thrown into a fever from any cause whatever, is liable to yield toxic milk. This would include the milk of all severe diseases, as being liable to become charged with toxins and ptomaines and thus poison the young animal, which subsists upon it as an exclusive diet, even though the actual pathogenic microbe may not be present in the secretion.

With regard to fermented milk, that which has been simply soured, relaxes the bowels and the attendant congestion contributes to further derangement and even infection by any pathogenic germ which may be present, or by microbes which are habitually saprophytic, but take occasion to

dangerously attack the weakened mucosa. If the milk has undergone putrefaction in the feeding bucket, the co-existence of the septic germ and the septic ptomaines and toxins, often determines indigestion and irritation of the mucosa. These poisons may further be absorbed and produce general constitutional disorder which reacts most injuriously on the stomach and digestion.

Milk swallowed rapidly and in excess by a hungry calf or foal, overdistends the stomach, which, like other hollow viscera in such conditions, is rendered paretic or paralytic, and suffers from suspension of both the vermicular contractions and the peptic secretions. Under these conditions the milk, which is one of the most admirable culture media for bacterial ferments, undergoes rapid decomposition, with the production of a series of toxins and ptomaines varying according to the different kinds of microbes that may be present. Under such conditions microbes which are normally harmless, vigorously and destructively attack the mucous membrane and determine some of the worst types of juvenile diarrhoea.

In artificial feeding there is another serious danger. Calves in particular are brought up largely on gruels made from farinaceous material. These contain a large quantity of starch which requires the action of the saliva (ptyaline) to resolve it into glucose, and fit it for absorption. But in the early days of life the salivary glands are almost entirely inactive, and it is only as the first three stomachs develop that this secretion becomes normally abundant. This is sought to be met by fixing in the feeding bucket a rubber teat, which the young animal is made to suck so as to solicit the secretion of saliva. The benefit obtained is however more from the slower ingestion of the milk than from any material increase of saliva from the as yet functionally inactive glands.

The presence of hair balls in the stomach, derived from the skin of themselves or others is one of the most injurious of the causes of juvenile indigestion. Lying as these do at this early age in the one well developed stomach they interfere with its normal secretions, and being at first open in texture they become saturated with putrefying ingesta, which gives out the most poisonous products.

The milk is materially affected by the food eaten by the nursing animal and such variations in the milk tend at times to derange a weak stomach. The following table from Becquerel and Vernois gives the results of dry and succulent food on the amount of the different proximate principles in the milk.

Nature of Food.	Water. Parts in 1,000.	Casein and extractive matter. Parts in 1,000.	Milk, Sugar. Parts in 1,000.	Butter. Parts in 1,000.	Salts. Parts in 1,000.
Cows on winter feed: Trefoil or lucerne 12–13 lbs.; oat straw, 9– 10 lbs.; beets, 7 lbs.; water, 2 buckets	871.26	47.81	33.47	42.07	5.34
Cows on summer feed: Green trefoil, lucerne, maize, barley, grass and 2 buckets water	859.56	54.7	36.38	42.76	6.30
Goats milk on different rations:					
Straw and trefoil	858.68	47.38	35.47	52.54	5.92
Beets	888.77	33.81	38.02	33.68	5.72

The decrease of the solids but especially of the casein, sugar, and salts is very marked in the cow on poor winter feeding. In the goat fed on beets alone the increase of sugar and decrease of other solids is striking.

To the same effect speaks the following table giving the results of an experiment with a ration of corn and cob meal, in contrast with one of sugar meal. Each cow had a common ration of 12 lbs. corn fodder and 4 lbs. clover hay, in addition to the test diet which was 12½ lbs. corn and cobmeal in the one case, and 10 lbs. sugar meal in the other. To avoid the misleading effects of a sudden transition from one food to the other, each special ration was fed for seven days before the commencement of each test period.

Animal and Ration.	Milk. Lbs.	Fat. Per Cent.	Solids	Fat. Lbs.	Solids Lbs.	Ratio of fat to solids not fat.
Grade Shorthorn Cow:						
1st 21 days: Corn and cob meal	631.25	3.43	11.57	21.67	73.02	422.:1000
2d 21 days: Sugar meal	641.50	4.04	12.53	25.93	83.38	476.2:1000
3d 21 days: Corn and cob meal	559.00	3.22	11.86	17.97	66.32	371.7:1000
Grade Shorthorn Cow:						
1st 21 days: Corn and Cob meal	604.75	3.57	11.95	21.56	72.28	425.1:1000
2d 21 days: Sugar meal	582.00	3.91	12.37	22.74	72.57	456.3:1000
3d 21 days: Corn and cob meal	527.00	3.37	12.05	17.78	63.48	389.1:1000
Grade Shorthorn Cow:						
1st 21 days: Sugar meal	753.50	3.97	12.43	29.94	93.67	469.8:1000
2d 21 days: Corn and cob meal	601.50	3.15	11.45	18.97	68.89	380.0:1000
3d 21 days: Sugar meal	560.50	3.85	12.16	21.58	68.16	463.3:1000
Grade Shorthorn Cow:						
1st 21 days: Sugar meal	487.50	4.15	13.27	20.25	64.69	455.6:1000
2d 21 days: Corn and cob meal	379.00	3.51	12.00	13.30	48.09	382.3:1000
3d 21 days: Sugar meal	374.50	3.72	13.01	13.95	48.74	401.0:1000

Here we find a material increase of the solids and particularly of the fat whenever the sugar (gluten) meal, rich in fat and albuminoids was furnished. It is interesting to note the relative amount of fat and albuminoids in the corn and cobmeal mixture as compared with the sugar meal.

Constituents.	Corn and cob meal. Per Cent.	Sugar meal. Per Cent.
Water	13.37	6.10
Salts	1.43	1.17
Fat	2.81	11.16
Carb-hydrates	65.99	52.66
Woody fibre	8.03	8.64
Proteids	8.37	20.27

—*Bulletin: Iowa Agricultural Experiment Station.*

Such variations in the quality of the milk under different rations, occasionally affect the weak stomach of the new born, and as the same constitution is likely to predominate in the same herd, a number may be attacked together as a result of some change in feeding.

Other conditions, however, lead to variation in quality. Hassall found that the morning milk of the cow furnished 7.5 per cent. of cream, while the evening milk gave 9.5 per cent. Boedecker found that morning milk had 10 per cent. of solids and evening milk 13 per cent. The first drawn at any milking is poorer in cream than that which is drawn last. The first may have only one-half or in extreme cases one-fourth of the cream that the strippings have. When the cow is in heat the milk not only contains more of the solids, but has granular and white blood cells like colostrum and often disagrees with the young animal. The milk of the young cow with her first calf is usually more watery than that of the adult, and that of the old one has a greater tendency to become acid. The longer the period which has elapsed since calving the greater the tendency to an excess of salts. Certain breeds like the Channel Island cattle produce an excess of butter fat (4–5 per cent.), whereas others like Holsteins, Ayrshires and Short Horns have less on an average (3–4 percent.), the casein and, it may be, the water predominating. Hence Jersey and Guernsey milk will scour

calves which do well on that of one of these other breeds.

Overkept, fermented and soured food tends to produce acidity and other changes in the milk. Old brewers' grains, swill, and spoiled gluten meal, or ensilage, especially such as has been put up too green, are especially injurious to the milk. The milk of cows fed on raw Swedish turnips or cabbage acquires a bitter taste and odor.

The milk of different genera of animals offer such strong contrasts that it is always dangerous to attempt to bring up the young of one genus upon the milk of another. The following table giving the composition of the milk in woman and each of the domestic mammals serves to illustrate this and to furnish a basis for adjustment:

	Woman.	Cow.	Goat.	Ewe.	Camel.	Mare.	Ass.	Sow.	Bitch.
Density	1032.67	1033.38	1033.53	1040.98		1033.74	1034.57		1041.62
Water	889.08	864.06	844.90	832.32		904.30	890.12	854.90	772.08
Solids	110.92	135.94	155.10	167.68	134.00	95.70	109.88	145.10	227.92
Butter	26.66	36.12	56.87	51.31	36.00	24.36	18.53	19.50	87.95
Casein and extractive matters	39.24	55.15	55.14	69.78	40.00	33.35	35.65	84.50	116.88
Sugar	43.64	38.03	36.91	39.43	58.00	32.76	50.46	30.30	15.29
Salts(by incineration)	1.38	6.64	6.18	7.16		5.23	5.24	10.90	7.80

Becquerel and Vernois.

Not only does the milk vary so widely with the genera, but that of the ruminating animal with its great excess of casein coagulates in the stomach into large dense clots which are not easily penetrated and digested by the peptic juices while that of woman or soliped forms loose clots, easily permeable by the gastric fluids and therefore much more readily digestible. Indeed the milk of these monogastric animals often form loose floating flocculi only, instead of solid clots. As cows are usually selected for foster-mothers to the orphaned animals of other genera this becomes a source of danger to the young and must be obviated by modifying the milk more in keeping with that of man or soliped.

As predisposing causes, must be named a weak constitution and damp, dark, filthy, or otherwise unwholesome buildings. Buildings with no drainage nor ventilation beneath the floors, standing on filth-saturated soil, and those with double walls holding dead rats and chickens are especially to be dreaded. In breeds of inconstant color the lighter colored calves (light yellow, light brown) are more subject to such attacks than the darker shades (dark browns, reds, blacks). The weak constitution may be a result of close breeding, without due consideration of the strength and vigor of the parents. Then young animals kept indoors in impure air, damp and darkness are more susceptible than those that are kept in pasture and are invigorated by exercise, pure air and sunshine.

Aside from the general run of causes, predisposing and exciting, we must recognize the contagious element. Jensen has sought to identify the microbe as a small ovoid bacillus united in pairs, or in long chains. This was present not only in the ingesta, but in the lesions of the mucosa, and in the lymph glands. Its cultures ingested in milk, or injected into the rectum sometimes produced the affection. Microscopically it resembled the bacillus foetidus lactis, but the latter failed to produce the disease. He looks upon it as a sport of the bacillus coli communis.

Perroncito found micrococci, usually arranged in pairs and comparable to the cultures of those obtained from the blood in the pneumonia of calves. The injection of the cultures into the thorax of a Guinea-pig caused pleuro-pneumonia with or without dysentery. The rabbit proved immune. At the necropsy the Guinea-pig like the calf showed the diplococci in the blood. Nikolski who studied the affection in lambs seeks to incriminate both micrococci and bacilli.

It is premature to specify any particular microbe as the sole cause of the affection. It seems not improbable that bacterial ferments of one or more specific kinds, which in a healthy animal have no injurious effect, may by special combination, or by growth in a mucosa in a given morbid condition, acquire properties which render them not only violently irritating, but may retain such properties

so as to render them actively contagious. In this condition they may overcome the resistance of the most healthy stomach and bowels and attack all young animals into which they may secure an entrance. Certain it is that the infection may persist in the same stable for years, will enter a new herd with a newly purchased cow or calf bought out of a previously infected lot, and will follow the watershed and affect in succession the different herds drinking from a stream as it flows downward.

The similarity of the germ found by Jensen to the *bacillus coli communis*, suggests that in this as in a number of other contagious affections a pathogenic sport from this common saprophyte is at least one of the microbial factors in this disease.

Symptoms. These may set in just after birth but usually the disease occurs within the two first weeks of life. When delayed for a few days after birth it may be preceded by some constipation, the fæces appearing hard, moulded, and covered with mucus. This is especially the case when the meconium has been retained and has proved a cause of irritation. The young animal is careless of the teat or refuses it (or the pail if brought up by hand), yawns and seems weary. The abdominal muscles are tense and the belly may be swollen if fermentation has already set in but this is rarely excessive. Straining to defecate usually causes eructations of an acid odor, and sometimes vomiting of solid soursmelling clots. Abdominal pain may be manifested by uneasy movements of the tail and hind limbs, by looking toward the flank and even by plaintive cries. This is followed within six hours by liquid dejections, at first merely soft, slimy and sour but soon complicated by a peculiar odor of rotten cheese which becomes increasingly offensive as the malady advances. The tail and hips become soaked with the discharges and as the putrid fermentation goes on after their discharge of the fæcal matters, the air becomes more and more repulsive. The same odor pervades the mouth and the breath and the tongue is coated with a whitish, grayish or yellowish fur.

The fæces become more watery and slimy, with much casein in course of putrefaction, and the patient is rapidly run down by the profuse discharge and the general poisoning by absorbed putrid products. In the worst cases this may prove fatal in one or two days.

When the illness is more prolonged the alvine passages which at first number five or six per day, increase to fifteen or twenty and are passed with more effort, usually leaving the anus in a liquid stream. The color of the stools changes from a yellow to a grayish yellow or dirty white, hence the common name of *white scour*, and the foetor is intensified.

Appetite may be in part preserved for a time but is gradually lost, and the subject becomes dull, listless and weak, indisposed to rise and walking unsteadily when raised. A general appearance of unthriftiness, staring coat, scurfy, unhealthy skin, pallor of the mucous membranes, arching of the back, tucking up and tenderness of the abdomen, excoriation of the margins of the anus, and congestion of the rectum as seen everted during defecation, mark the advance of the disease. Emaciation becomes very marked, and weakness and prostration extreme.

Fever usually sets in as the disease advances, as marked by hyperthermia, hot dry muzzle, hot ears, accelerated pulse and breathing.

When the intestinal fermentation is extreme there may be distinct bloating, more acute colicky pains, rumbling of the bowels and a frothy and even bloody condition of the dejections. The prostration may become extreme and the temperature reduced to the normal or below.

Death may result from inanition and exhaustion, or from nervous prostration and poisoning.

The affection may be complicated by purulent arthritis, peritonitis, pneumonia, hepatitis, keratitis or laminitis. It may prove fatal in from three to ten days.

Mortality. This is always high. For foals it has been set down at 80 per cent. of the numbers attacked, for calves at 54 to 90 per cent., and for lambs at 66 per cent. In 500 lambs Beresow found 300 attacked and 200 died. Kuleschow sets the losses at 30 to 40 per cent. of the lambs, and Cadeac advises that even the survivors should be fitted for the butcher as they are unfit for reproduction.

Lesions. In foals these are mainly confined to the intestines which show a more or less extended area of redness and congestion with catarrhal or pseudomembranous exudate on the mucosa, and the submucous connective tissue is infiltrated softened and marked by intense punctiform redness.

The epithelium is swollen, opaque and easily detached, and Peyers patches infiltrated and prominent. The blood in the intestinal vessels is incoagulable. Exudation into the peritoneum and softening of the liver are not infrequent.

In calves the lesions are very similar, but the 4th stomach is usually implicated, the congestion and epithelial desquamation being most marked in the pyloric region. The contents of the intestines are mucopurulent, grayish, yellowish or red, and intensely foetid. The follicles of the small intestine are red and projecting with an areola of congestion. Softening and even necrotic centers are found in the liver and kidneys and the mesenteric glands are swollen, red and softened.

In lambs the lesions are nearly the same in the 4th stomach, intestine, lymph glands, liver and kidneys. There is usually marked emaciation and the spleen and nerve centres are anæmic.

Prevention. The first consideration is to avoid the various causes which have been enumerated. Give the young the warm milk of its dam or of a nurse of the same species and at the same time after parturition. If it is necessary to give older milk to the new born don't fail to clear out the bowels by a tablespoonful or two of castor oil (foal, calf,) or two teaspoonfuls (lamb), or to add manna or linseed decoction to the milk. Protect both nurse and nursling against cold storms, overheating, overwork, excitement, and sudden changes of diet, (dry to green, etc.). If the nurse has been overheated or overexcited draw off the first milk by hand and let the nursling have only that which is secreted later. Avoid the milk of diseased and especially fevered animals. If the milk of one nurse disagrees, correct any obvious cause in the food or general management, and if none can be found get another nurse. If fungi appear in the milk (inducing ropiness or not) withhold the food or water from which they have been probably derived and give bisulphite of soda (cow or mare 2 to 4 drs., ewe $\frac{1}{2}$ dr. daily). When an animal of one genus has to be brought up on the milk of another, let the milk be so modified by the addition of water, sugar, cream, etc., as will approximate it somewhat to the normal food. The milk of the cow may be given unchanged to lambs or kids, while for the foal it should be diluted by adding $\frac{2}{5}$ of water, and sugar enough to render it perceptibly sweet. Even more sugar is wanted for the young ass. In place of simple water, barley water may be used, as this not only loosens the coagulum formed in the stomach, but renders it especially open and permeable to the digestive fluids. Another method of special value for puppies is to let the cow's milk stand for several hours and then take only the upper half (containing most of the cream) for feeding. This must be watched lest it should unduly relax the bowels. In all cases the milk artificially prepared should be given milk warm. To retard the acid fermentation which is liable to occur early and injuriously in adapted milk, the addition of an ounce of lime water to each quart of milk is of great advantage. Pigs and puppies can usually adapt themselves readily to the milk of the cow. In all cases in which a young animal is raised by hand and especially if on the milk of another species, it is desirable to provide against sudden overloading of the stomach. The artificial rubber teat fixed in the feeding pail serves a good purpose in this respect. Pasteurizing is admissible but boiling of the milk is objectionable, as rendering the milk constipating and thereby favoring irritation. In condensed milk this tendency is largely reduced by reason of the excess of sugar and consequent looseness of the clot, only care should be taken to dilute it sufficiently with boiled water.

Among the most important measures of precaution, is the separation of the sick from the healthy, and to disinfect thoroughly the buildings in which the infected have been. Straw, and when possible dung should be burned; if not, they should be buried together with the urine. The stalls should be thoroughly cleansed and then saturated with mercuric chloride (1 : 1000), or sulphuric acid (3 : 100), or a saturated solution of sulphate of copper. Here as elsewhere chloride of lime (4 oz. to the gallon) with as much quick lime as will make a good white wash, does admirably, as it is at once seen if any part has been missed.

Esser remarked that the calves of cows that had been removed to another stable some time before parturition, usually remained healthy, provided they were kept from the other and sick calves.

Lastly, it is important to use for breeding purposes such animals only as have a strong, vigorous constitution, and to furnish a healthful, abundant aliment and to allow a sufficient amount of exercise during gestation. Vigor and stamina are the great desiderata, but these are usually found

with the darker colors.

Treatment. The old treatment of eliminating offensive matter by a laxative is still good, and thus castor oil (2 ozs. for a foal or calf, 2 drs. for lamb), or rhubarb (1 dr. foal or calf, 1 scr. for lamb), or manna ($\frac{1}{2}$ oz, foal or calf, 1 dr., lamb), may be given with laudanum (1 dr., foal or calf, 10 drops, lamb), and salicylate of soda (16 grs., foal or calf, 5 grs., lamb). The milk should be given boiled. An old and excellent remedy to follow the laxative is solution of rennet made by adding $\frac{1}{8}$ of a calf's abomasum to a quart of 20 per cent, alcohol (or sherry). A tablespoonful may be given with each meal. The value of this as an antiferment is liable to be overlooked, yet both the hydrochloric acid and pepsin are strongly antiseptic, and neither of these is produced to any extent in the diseased stomach. In addition to this ipecacuan has been used and by its stimulant action on both stomach and liver it furnishes the two most important natural disinfectants of the alimentary canal (foal or calf 1 dr., lamb 10 drops ipecacuan wine, thrice a day).

In addition to these or separately, antiseptics, carminatives and astringents may be employed. An excellent preparation is prepared chalk 1 oz., white bismuth 1 oz., tincture of cinnamon 8 ozs., gum arabic $\frac{1}{2}$ oz. A tablespoonful thrice a day will often check the disorder.

Cadeac advises, subnitrate of bismuth 5 grains, salicylic acid 5 grains, naphthol 20 grains, syrup 150 grains, distilled water 100 grains. One or two tablespoonfuls in the mouth after each drink (foal or calf).

Filliatre obtained excellent results in calves, with a solution of tar 150 grams in 6 litres boiling water, given in the dose of $\frac{1}{3}$ litre every half hour. It may also be used as an enema. The diarrhoea is promptly checked, and the tar water may then be restricted to $\frac{1}{4}$ litre mixed with the milk of the next two days. One-tenth of the dose may be given to lambs.

Among other antiseptics in use may be named salicylic acid and tannin, salol, boric acid, betol, diaphthol, bruzonaphthol, salicylate of bismuth, creolin, naphthalin, and lactic acid.

When icteric membranes, white discharges and extreme foetor indicate hepatic disorder, calomel 1 part and chalk 12 parts may be resorted to three or four times a day (foal or calf 6 grs., lamb 1 gr.).

Among the carminatives may be named anise, fennel, dill, cinnamon, and chamomile. Beside their stimulant action these are all more or less antiseptic.

In addition to the boiling of the milk, and in certain cases as a temporary substitute, may be used sterilized mucilaginous agents, gum arabic, flax seed, and slippery elm.

ACUTE GASTRIC INDIGESTION IN SOLIPEDS. TYMPANITIC STOMACH.

Definition. Causes: overloading, lessened secretion, mastication or insalivation, frosted food, fermenting food, appetizing food, cooked food, debility, disease, starvation, overwork, fatigue, violent exertion after a meal, anæmia, parasitism, injury to vagi or their centres, iced water, wading or swimming in iced water, food with excess of proteids, drink after grain, alkaline waters. Symptoms: in slight cases, in severe, violent colic, weariness, pinched countenance, acid mouth, bloating of abdomen, anorexia, dysphagia, no rumbling of bowels, no fæces, tries to eructate, dullness, stupor, coma, vomiting. Rupture of stomach. Recovery. Lesions: overdistended stomach, ruptured peritoneal hemorrhage, ruptured diaphragm, small bloodless liver and spleen. Diagnosis. Treatment: Aromatics, stimulants, antiferments, laxatives, stimulants of peristalsis, exercise, friction, electricity, chloral hydrate, puncture of stomach and colon, probang, stomach pump, dieting, tonics.

Definition. Suspension of the normal functions—motor and secretory—of the stomach and the supervention of fermentation in its contents.

Causes. The small size of the stomach in the soliped (26 quarts) and the rapidity with which the alimentary matters normally traverse it, render this organ much less subject to disorder than the complex stomachs of ruminants. In his native state the horse eats at frequent intervals and digestion is constantly going on, so that the viscus is never distended to paresis, nor the secretions, nor vermicular movements retarded by excess of ingesta. But the limited capacity of the viscus becomes in its turn a cause of indigestion whenever the animal is tempted by hunger to hurriedly swallow too great a quantity of food; when the decreased secretions fail to act with sufficient promptness on the contents and leave them to undergo fermentation; when from imperfect mastication and insalivation the food is left in large masses comparatively impermeable to the gastric juice, and accumulates in firmly packed masses; when frosted food (roots, potatoes, apples, turnip tops, etc.), taken in quantity temporarily chills and paralyzes the stomach, and starts a speedy and gaseous fermentation; when the food swallowed is already in process of fermentation (musty or covered by cryptogams) and full of toxic fermentation products which tend to paralyze the stomach. Old animals are especially liable because not only are the teeth and salivary glands ineffective, but the functions of the stomach are habitually below par.

The paralysis of the stomach by overloading is seen especially in animals that have been fasting for too great a length of time, and are then furnished with a food, rich, appetizing and abundant. Few horses are proof against the temptation to overeating when they get to the cornbin, the ripening grain or maize, or the field of rich red clover. Some are natural gluttons and on gaining access to grain or green food, will suddenly overload the stomach beyond its power of active contraction on its contents, and without sufficient mastication or insalivation. The food is literally bolted whole, with no admixture of saliva, and no facility for admixture of gastric juice, even if the overloading had left the stomach capable of secreting the latter. Cooked food is especially dangerous by reason of its bulk, the facility with which it is swallowed, and the rapid and excessive dilatation of the stomach caused by it, rather than from lack of trituration and saliva. If fed judiciously, cooked food is more fattening for both horse and ox, any lack of ptyaline being counterbalanced by the presence of amylase in the intestines.

Such paresis and indigestion, however, are more common as the result of a general debility or a special gastric atony caused by disease, starvation, overwork or fatigue. In all acute febrile and inflammatory diseases the gastric functions are weak or suspended, and if the animal is tempted to eat, the ingesta is unaffected by the digestive fluids and forms a suitable fluid for injurious fermentations. In convalescence especially, when the starved system once more craves support, tempting food is liable to be taken to excess, unless the attendant is especially judicious and careful in grading the feed as the stomach can dispose of it. The horse that has been starved must be fed little and often, of easily digested material until the gastric functions are restored. Long continued severe work, exhausts the motor and secretory power of the stomach, as it debilitates the system at large, and the animal may be at first unable to digest a feed of grain even if he will take it. In such a case as in that of the very hungry glutton a drink of gruel or a handful of hay which he must masticate will often obviate the danger.

Violent exertion immediately after a meal arrests digestion, and tends to a fatal indigestion. An

animal fed grain and immediately put to severe work, or subjected to confinement for a painful operation, may die in two hours from tympanitic indigestion.

This weakness of the digestion may come from profuse bleeding, from the anæmia caused by parasites (sclerostomata), or from injuries to the pneumogastric nerves or their centres. It can be produced experimentally by cutting both vagi; the gastric contents then remain packed and solid, without peptic juices and without digestion.

Iced water, like frozen food, may temporarily arrest the gastric functions and entail fermentation. It acts most dangerously on the overheated and exhausted horse, and though the indigestion may not prove fatal, it may induce a sympathetic skin eruption or laminitis. The mere exposure to external cold is less to be dreaded as there is a compensating stimulus which drives the blood to internal organs, the stomach included. Standing in cold water or wading or swimming a cold river, is commonly less injurious than a full drink of iced water, when heated and fatigued.

Certain kinds of food are far more dangerous than others, and especially such as should be digested in the stomach. Thus the different grains—barley, rye, buckwheat, wheat, oats, Indian corn, and even bran, have been especially objected to. The amount of proteids in oats, for example, is 11.9 per cent., while those of hay are but 7 per cent. The same bulk of oats, therefore, demands nearly double the work of the stomach to reduce its nitrogenous constituents to peptones than does hay. But when fully insalivated the difference is even greater, for oats take but the equivalent of their own weight of saliva, whereas hay takes four times its own weight. There is 1 part of proteids in 16.7 parts of insalivated oats, and but 1 in 71.4 of insalivated hay. If the oats are bolted without mastication, which can never be the case with hay, the discrepancy becomes greater still. Grain is best fed often, in moderate amount, and without further loading of the stomach immediately with either solids or fluids. Above all never feed grain to a thirsty horse and then lead him direct to the watering trough. Even should he fail to have the stomach paralyzed by the cold water, and indigestion developed, yet much of the proteids will be washed out into the small intestine to threaten indigestion there.

Selenitic waters may induce indigestion by neutralizing the hydrochloric acid of the stomach and interrupting digestion.

Finally all forms of gastritis—catarrhal, toxic, and phlegmonous—induce atony, fermentation and indigestion.

Symptoms. There may be simply tardy digestion or grave disorder with impaction or tympany.

In the former case there is impairment or perversion of appetite, refusal of food, irregular feeding, licking earth or lime, or eating filth, even fæces, with some dullness, apathy, or signs of pain such as pawing with the fore feet, or looking round at the flanks. There is rumbling of the bowels, followed in favorable cases by the passage of flatus, of softened fæces containing imperfectly digested food, and distinct diarrhoea which proves curative.

The more violent attacks set in suddenly, usually within one or two hours after feeding. There are usually colicky pains, pawing, looking back at the flank, kicking of the abdomen with the hind feet, lying down, rolling, rising again quickly, yawning, anxious pinching of the countenance, rigid loins insensible to pinching, and heat and dryness with an acid odor in the mouth. There is soon observed some swelling and tension of the belly with tympanitic resonance on percussion in the left hypochondrium. There is no elevation of temperature as in gastritis, and no complete intermissions of pain as in spasmodic colic, but pain is continuous, though worse at one time than another. There is an utter indisposition to eat or drink and if liquids are given by force there is manifest aggravation of the sufferings. As a rule there is no rumbling of the bowels, and though the animal may strain violently, little or nothing is passed, except at the first a few moulded balls of dung. The bowels like the stomach are paralyzed. In some cases there are attempts at regurgitation, the fore feet are placed apart, the neck arched, the lower cervical muscles are contracted and the nose drawn in toward the breast. In some instances relief is obtained by belching gas or by actual vomiting of solid matters. Vomiting in the horse is always ground for suspicion, since it usually occurs when the muscular coat of the stomach is ruptured. An important hindrance to vomiting lies in the loose folds of the mucosa covering the cardia, in the flaccid condition, and as these folds may

be entirely effaced in hernia of the mucosa through the muscular coat as well as in the overdistended condition, vomiting may be either a fatal or a favorable indication. If vomiting takes place, without attendant prostration and sinking, and if on the contrary there is manifest improvement after it, it may be looked on as a beneficent outcome.

If no such relief is obtained the patient becomes increasingly dull and stupid; the breathing is accelerated, short, moaning or wheezing; the nostrils dilated; the nasal mucosa dark red; the superficial veins, especially those of the face, are distended and prominent.

The nervous symptoms may vary. Usually the dullness increases to stupor, the animal rests his head on the manger or against the wall, or if at liberty he may move forward or around blindly until some obstacle is met and he stumbles over it or pushes against it. In some instances there is champing of the jaws, or irregular motions of the limbs, but more commonly the dullness goes on to stupor and coma, the animal falls helpless and dies in a state of profound insensibility.

If the *stomach should become ruptured* there is often vomiting, the ingesta escaping by the nose, without any relief of the general symptoms, but with an increasingly haggard expression of countenance, sunken eye, and accelerated, weak, and finally imperceptible pulse. Cold sweats, which may have been already present, become more marked and the prostration becomes more extreme and the abdominal tenderness more marked. There are muscular tremblings of the shoulders and thighs, dilatation of the pupils, rapid breathing and stupor which presages death.

Recovery may be hoped for if rumbling in the bowels commences anew, if defecations continue and become soft and liquid, if urine is passed abundantly and if the general symptoms are improved. Complete relief may be had in five or six hours, and even in protracted cases in two days.

Lesions. The body is swollen, tense and resonant; the rectum usually projects somewhat and is dark red; the intestines, small and large, are tympanitic; the stomach is double or triple its usual size, tense and resistant, and with its contents may weigh as much as 40 pounds. When cut open its contents are seen to be disposed in the order in which they were eaten, in stratified layers, the motions of the stomach have not operated to mix them. There is no sign of digestion, unless it be in a thin surface layer or film which may be white, pulpy and chymified. The cuticular mucosa is usually unchanged further than its attenuation by stretching, the alveolated mucosa also attenuated is congested, opaque or slightly inflamed. The great curvature may be the seat of a rupture the edges of which are slightly swollen, congested and covered with small blood clots. The escaping ingesta usually remains enclosed in the omentum, which thus looks like a larger stomach with extremely thin gauze-like walls. If this is ruptured then the food floats in masses among the convolutions of the intestines. The peritoneum is red, hemorrhagic and covered with more or less exudation.

Another occasional lesion is rupture of the diaphragm. The liver and spleen are usually small and comparatively bloodless, owing to the compression.

Diagnosis. This is largely based on the speedy supervention of the attack on a feed, the animal having been apparently well before, on the onset by slight colics, rapidly passing into great and continuous suffering and stupor, with tympanitic tension of the abdomen, and suppression of the intestinal movements, in the absence of any distinct or marked hyperthermia. The rapid progress to death or recovery is equally characteristic.

Treatment. In mild cases the prompt use of aromatics will sometimes succeed; tincture of pimento, anise or coriander 2 to 3 ounces, oil of peppermint, 20 to 30 drops. Stimulants, aqua ammonia, 1 to 2 drachms, largely diluted, ether 1 ounce, brandy or whisky 1 pint, will sometimes succeed. A good combination is dilute hydrochloric acid, 1 drachm, oil of turpentine 1 ounce, olive oil $\frac{1}{2}$ pint.

Still more effective in the rousing of the torpid vermicular movements are eserine sulphate 1.5 grain or pilocarpin 2 grains, or barium chloride 7 grains subcutem.

These largely replace the old plan of giving a dose of aloes in bolus, yet in case of need aloes may still be given in ounce doses in cold water injections. The cold serves to rouse the vermicular movements of the bowels and sympathetically of the stomach.

Walking exercise, friction over the abdomen, and even electric currents through the epigastrium

and left hypochondrium may be helpful.

In very urgent cases, 1 ounce to 2 ounces chloral hydrate is often effective. It acts as a powerful antiferment, checking further extrication of gas, and counteracts spasms of the bowels, so that gas passes more freely per anum, vermicular movements are resumed and recovery may be hoped for.

Puncture of the stomach through the external abdominal wall can only be effected by transfixing the transverse colon above which it lies and few have the hardihood to undertake this. It may, however, be punctured with comparative safety through the fourteenth to the seventeenth intercostal space in its upper half (Scammel). The overdistended stomach pressing forward on the left half of the diaphragm, applies that against the inner surface of the ribs, the lung being driven forward out of the way, and the liver and colon are also displaced, so that the trochar transfixes the skin, intercostal muscles, costal and phrenic pleura, diaphragm, peritoneum and stomach. The marked drumlike resonance on percussion indicates the best point for the puncture, and the trochar should be directed inward and slightly backward and pushed until solid resistance at its point ceases. As the intestines are usually tympanitic as well it may be requisite to puncture also the cæcum and colon, to restore the peristalsis of the alimentary tract generally. Antiseptics such as sulphurous acid, the sulphites, or hyposulphites, calcium chloride, bleaching powder, potassium permanganate or chloral hydrate, may be introduced through the cannula or by the mouth.

As far as the stomach is concerned, an effective relief can be had through the probang or stomach pump. A small one-half inch hollow probang may be safely passed through the nose and gullet into the stomach, and any gas or liquid allowed to escape. With proper attachments this may be fixed to a stomach pump and the viscus exhausted of all available liquid, after which an equal amount is pumped in and again withdrawn, until the contents are reduced to a normal amount. The water pumped in may be rendered antiseptic by sodium chloride, sodium bisulphite, or other antiferment, so that further extrication of gas will be prevented. If it is necessary to use the ordinary probang or stomach tube introduced through the mouth, great care must be taken in introducing it to see that the soft palate does not deflect it downward into the larynx. Its presence in the gullet above and beyond the larynx can be felt by manipulation from without, and until this is ascertained it should on no account be pushed onward.

The importance of a measure of mechanical relief such as this, is the greater that the stomach of the soliped is non-absorbing, and relief from undue pressure of contents can only be had by their passage upward or downward. Then again, the horse cannot vomit like the carnivora and omnivora, nor regurgitate like ruminants, and if left to himself with engorged stomach, his case is hopeless indeed.

The contingent weakness in cases of recovery may demand careful feeding and a course of bitter tonics.

GASTRIC INDIGESTION IN CARNIVORA.

Causes: too frequent feeding, indigestible food, epidermic products, bones, tendons, rubber, cords, marbles, pebbles, catarrh, inflammation, intestinal lesions, fever. Symptoms: dullness, restlessness, eating grass, vomiting, seeks seclusion, colics, giddiness, tense, tender abdomen. Treatment: emetic, diluents, dieting, tonics.

Causes. Unintermittent work of the organ wears out its tone and indigestion follows. The more indigestible the food, and the admixture with the food of indigestible materials the greater the tendency to this fatigue and atony. Thus epidermic materials, hair, horn, wool, bristles, feathers are often injurious; also bones, tendons, and above all pieces of rubber, caouchouc, cords, marbles, pebbles and other small objects. Fortunately such agents are usually rejected by vomiting, but if retained, the movements of the viscus become tardy and its secretions defective, and fermentation, indigestion and irritation ensue. The evil effects however mostly come of catarrhal and other inflammations of the stomach, and serious lesions of the intestines, (inflammation, obstruction, volvulus, intussusception, tumors, strangulated hernia,) or acute fever. In any such case the stomach ceases to act, and its contents become a center of active fermentation with more or less irritation.

Symptoms. In transient cases the dog will appear dull and restless, moving about, and, if opportunity affords, perhaps eating grass so as to hasten emesis and relief. With this relief the subject may remasticate and swallow the very materials he has vomited.

If relief is not secured by vomiting, the animal remains dull, anxious, retiring, seeking perhaps seclusion and darkness, is taciturn, moves continually, lying down first on one side and then on the other, or successively in different places, looks round at the belly, starts up suddenly with a piercing cry, and may appear giddy and uncertain in his gait. The abdomen is usually tense or even full and tender to touch.

Treatment. The first resort is the evacuation of the stomach by vomiting. Give tepid water and tickle the fauces with a feather or the finger. Or $\frac{1}{2}$ to 1 gr. tartar emetic in a tablespoonful tepid water, or a teaspoonful of ipecacuan wine. Or $\frac{1}{2}$ dr. of a $\frac{1}{100}$ solution of apomorphia hypodermically. Beyond this, little is wanted except careful feeding as regards quantity and quality. Nux vomica $\frac{1}{2}$ gr. twice a day will serve to restore tone. Further treatment will come naturally under catarrhal gastritis.

ACUTE GASTRIC INDIGESTION IN SWINE.

Causes: fermented or putrid swill, spoilt vegetables, frozen aliments, caustic alkalies (powdered soaps) from kitchen, indigestible materials, poisons. Symptoms: dullness, grunting, restlessness, seeking seclusion, colics, vomiting, rumbling, tense, tucked up abdomen, diarrhoea. Treatment: emetic, bland acids, laxative, dieting, bitters, iron.

Causes. Swine have such a varied dietary, are so constantly fed swill containing all manner of ingredients and often kept in barrels, etc., that are never emptied and cleansed, and therefore so often the seat of septic fermentation, that both gastritis and enteritis are often produced. Spoilt turnips, potatoes, apples and other succulent vegetables, or those that have been exposed to frost, or which are devoured while frozen are additional causes of irritation. The various caustic alkaline powders used in washing the table dishes and the product added to the swill is another cause of such outbreaks which, attacking a whole herd at once, is attributed to hog cholera. Then indigestible materials (hoofs, hair, bristles, tree bark, etc.) when they fail to be rejected by vomiting cause gastritis and indigestion. Finally a long list of medicinal and toxic substances act in this way.

Symptoms are like those seen in dogs, dullness, arching of the back, drawing the feet together, erection of the bristles, hiding under the litter, grunting, restlessness, frequent movement from place to place, lifting of the hind feet, grubbing in the litter with the snout, tension of the abdomen, and often abdominal rumbling followed by diarrhoea and recovery. More commonly, however, relief comes from early rejection of the irritant matters by vomiting.

Treatment. Induce emesis as in the dog. Give vinegar in case of alkaline poisoning. Follow this by a laxative if the irritants have gained the intestines, and finally a course of iron or bitters. Careful dieting is absolutely essential.

ACUTE CATARRHAL GASTRITIS IN THE HORSE.

Causes: wet fermented food, cryptogams, bacteria, sprouted green potatoes, sand, irritants, hot food, frosted food, ill health, starvation, anæmia, siliceous plants, diseased teeth or salivary apparatus, parasites, calculi, specific inflammations. Symptoms: depraved appetite, licking soils, walls, filth, etc., clammy mouth, red bordered tongue, eructations, colic, rumbling, tympany, icterus, costive, coated fæces, tender hypochondrium, anorexia, emaciation. Lesions: stomach full or nearly empty, pyloric sac congested, mucosa thickened, petechiated, with excess of mucus and desquamated epithelium, cells swollen opaque, congested duodenum, pale yellow liver, with excess of pigment in cells, also in urine, ruptured stomach, hemorrhagic infiltrations. Treatment: careful dieting, laxative, stomachics, pepsin, antiferments, bitters, ipecacuan.

Causes. This is a much less common affection in horses than dogs and is usually charged on some fault in diet. Fodders that have been badly harvested or wet from any other cause and are musty, dusty, rusty, or covered with any irritant or poisonous cryptogams or bacterial ferment, sprouted oats, or potatoes that have become green by exposure to the sun, sand and gravel in the grain, irritant plants like ranunculus, euphorbium, veratrum, etc.; cooked food given too hot, or vegetable food given frosted; putrid water, and indeed all the causes of indigestion tend to produce gastric catarrh. Weakness of the stomach and gastric functions from any cause is a concurrent factor. Thus extensive inflammations and violent fevers, prolonged abstinence, starvation, anæmic and parasitic affections, the action of frozen food on the viscus, are to be feared. A horse which has been for a week or more without food is extremely subject to such attacks unless fed with the greatest caution at first. Irritant plants like carex, equisetum, etc., which act mechanically by reason of the contained silica, food imperfectly masticated on account of disease of the jaws, teeth or salivary glands, parasites like spiroptera and bots and phosphatic and oat-hair calculi, act mechanically. Finally certain affections like petechial fever, influenza, pneumonia, pustulous stomatitis and horse pox may develop local foci of inflammation in the stomach. When once started, microbial infection tends to maintain and aggravate it.

Symptoms. These are very indefinite, depending very much on the complications. Some loss or perversion of appetite, a licking of the soil or walls, eating litter, filth and even manure, a clammy mouth, a redness along the margin of the tongue, eructations or attempts to eructate, or actual vomiting, colicky pains which are usually dull until the bowels are implicated, more or less rumbling in the bowels, sometimes icterus, in other cases tympany, and nearly always tardy passage of hard and scanty mucus-covered fæces. The colics may be intermittent, appearing only just after food is taken, or they may be continuous, the animal pawing incessantly hour after hour. A slight hyperthermia and a distinct tenderness of the epigastrium and left hypochondrium to pressure are valuable symptoms. Percussion causes even keener suffering.

If the gastric contents are abundant and fermentation active, death may ensue from gastric tympany. In other cases, the persistence of colics at the time of feeding, of impaired appetite, constipation and loss of condition are the main symptoms. In the last named cases the patient may die of marasmus.

Lesions. In cases terminating in fermentation and fatal tympany the stomach is full; in other types it is empty of all but water, mucus, and perhaps some irritant contents, or decomposed food. The alveolar mucosa of the right sac and above all of the pylorus is red, congested, petechiated, macculated, thickened to double its normal thickness or more, thrown into rugæ, and covered with tenacious mucus. This mucus is highly charged with detached epithelial cells, and at different points the mucosa is abraded by their desquamation. The epithelium generally shows swollen, opaque cells. The red congested spots show active engorgement of the capillaries, and this is especially marked around the glandular follicles, with more or less formation of embryonic cells. The duodenum is often implicated with similar lesions of the mucosa and its epithelial layers, which may block the orifices of the pancreatic and especially of the biliary duct. In this case there is a yellowish discoloration of the liver, excess of pigment in the hepatic cells, and hemorrhagic spots in the liver and even in the kidneys. The urine may be yellow or reddish brown from the presence of bile or blood pigment. In ruptured stomach, spiroptera, bots, and other irritants, we find their characteristic lesions, and in petechial fever there is excessive and partly hemorrhagic infiltration of the mucosa and submucosa. In protracted cases ulcers may be present on both stomach and

intestine. When it is a localization of some specific fever the characteristic lesions of that affection will be found.

Treatment. If appetite continues, diet should be restricted to a very moderate allowance of green food, pulped roots, bran mash, boiled flaxseed, boiled middlings, with pure water or whey. If there are irritants in the stomach they may be got rid of by a laxative (aloes 4 drachms, or sulphate of soda $\frac{1}{2}$ pound). Sodium bicarbonate ($\frac{1}{2}$ drachm 2 or 3 times daily) is desirable to stimulate peptic secretion and check acid fermentations. Pepsin (2 drachms) should be given at equal intervals. Fermentations should be checked by the use of salol (1 to 2 drachms), naphthalin (1 to 2 drachms), benzo-naphthol (1 to 3 drachms), or calcium salicylate (2 drachms).

In this connection bitters are of value to improve the tone of the gastric mucosa, nux vomica, gentian, quinia and quassia in combination with ipecacuan giving good results.

PHLEGMONOUS (PURULENT) GASTRITIS IN THE HORSE.

Definition: deep inflammation tending to abscess. Causes: invasion by pus microbes, infectious diseases, parasitism, traumas. Symptoms: hyperthermia, colic, tenderness, icterus, coincident disease, hæmatemesis. Lesions: submucous or subperitoneal abscess, parasites, peritonitis, exudation, thickening, neoplasm of mucosa, catarrhal complications. Treatment: careful diet, antiseptics, bitters, laxatives.

Definition. This is a gastric inflammation affecting the membranous layers, and tending to submucous or subperitoneal abscess. It is much less frequent than the catarrhal form.

Causes. It may be attributed to invasion of the gastric walls by pus microbes, and appears as secondary abscess in pyæmia and above all in strangles. The microbes are introduced more directly through the wounds inflicted by the larvæ of œstrus, or by the burrowing of these (Argus, Schlieppe, Schortmann), or of spiroptera (Argus). Wounds by sharp pointed bodies taken in with the food, furnish other infection—atria, and in their turn ulcers connected with catarrhal or toxic inflammation may furnish a means of entrance.

Symptoms. These resemble those of catarrhal inflammation, but are usually attended by greater hyperthermia, and the colicky symptoms are more marked. There is also greater tenderness in the epigastrium and left hypochondrium, and icterus is more marked. When it occurs as an extension of strangles or pyæmia the symptoms of these affections elsewhere are pathognomonic. When the abscess bursts into the stomach there may be vomiting of bloody mucus (hæmatemesis) which is not necessarily followed by a fatal result.

Lesions. As these are seen only in fatal cases, the presence of an abscess is the characteristic feature. This is usually submucous, or less frequently subperitoneal, and may vary in size from a hazelnut upward. The tendency appears to be to open into the stomach, though it may burst into the peritoneum and cause general infection of that membrane. In case of parasites, the spiroptera or œstrus larva may be found in the abscess cavity having a narrow opening into the stomach. In certain cases the abscess on the pyloric sac has been found opening into the duodenum. Congestion, thickening, puckering into rugæ and laceration of the adjacent mucosæ may be a marked feature, a circumscribed catarrhal gastritis complicating the local phlegmon.

Treatment. This is less hopeful than in catarrhal gastritis, but should be conducted along the same lines. The same careful diet, with daily antiseptics and bitters may prove valuable in limiting the inevitable suppuration, and, if the pus should escape into the stomach, in healing the lesion. Sulphites of soda, sulphide of calcium, chamomile, and quinia, are to be commended and pepsin may be added to secure at once proteid digestion and antisepsis. Laxatives may be required to counteract constipation or expel irritants, and these may be combined with the antiseptics already named or with salol, eucalyptol, sodium salicylate or other non-poisonous agent of this class.

TOXIC GASTRITIS IN SOLIPEDS.

Causes: caustics or irritants acting on mucosa, accidentally, or maliciously. Symptoms: colics, pinched face, small rapid pulse, hurried breathing, hyperthermia, sometimes salivation, color of buccal mucosa, odor, congestion of tongue, thirst, urination, icterus, albuminuria, analysis of urine or vomited matter. Lesions: congestion, corrosion, necrosis or ulceration of gastric mucosa, discoloration. Treatment: antidote, stomach pump, demulcents, coagulants.

Causes. Toxic gastritis in solipeds is peculiar in this that it must be due to one or other of the more caustic or irritant agents, which act chemically on the tissues, while those agents that require to be absorbed to establish a physiological irritation are comparatively harmless. This depends on the fact that few or none of the poisonous agents are absorbed by the gastric mucosa of the soliped, and if ingested they must pass on into the duodenum before they can be absorbed into the tissues and blood-vessels. Hence the horse is injured mainly by actual caustics like mercuric chloride, zinc chloride, ferric or cupric sulphate, the caustic alkalies or earths, or alkaline carbonates, and the mineral acids. These may be taken accidentally or administered maliciously, or as medicines.

Symptoms. Morbid symptoms vary according to the agent swallowed. There are colics, anxious countenance, small accelerated pulse, rapid breathing, hyperthermia, and salivation, especially marked with mercuric chloride. The buccal mucosa may give valuable indications, becoming white with muriatic acid, or zinc, or antimony, or mercuric chloride, yellow with nitric acid, and white changing to black with sulphuric acid or silver nitrate. Ferric or cupric sulphate may give their respective colors to the saliva, and the former will darken the fæces.

The mouth is usually dry, hot, and clammy, and the edges of the tongue red. Temperature is usually elevated, yet with tartar emetic it may be distinctly reduced. Thirst is usually marked, and urination frequent. Icterus and albuminuria attend on phosphorus poisoning. When vomiting takes place the appearance or analysis of the matters rejected, or otherwise of the urine, will often indicate the nature of the poison.

Lesions. The gastric mucosa is congested and discolored, but the corrosion and even the ulceration are especially characteristic. Patches of necrotic mucous membrane may be more or less detached exposing a deep red submucosa. The coloration otherwise varies;—white or black with sulphuric acid or silver nitrate; white with muriatic acid, the caustic alkalies, or zinc chloride; yellow with nitric acid; or green with salts of copper.

Similar lesions are found on the buccal, œsophagean and intestinal mucosæ, and even at times on the respiratory.

Treatment. In the treatment of this form of gastritis the first consideration is to expel, or use an antidote to, the poison. In the soliped, emetics are useless. The stomach pump or tube may, however, be applied with good effect in nearly all cases, alternately throwing in water and drawing it off. Demulcents and coagulants are also universally applicable. Milk, eggs beaten up in milk, blood albumen, flaxseed tea, well boiled gruels, or slippery elm bark, may be used as may be most convenient. Next come the other chemical antidotes the use of which however demands a previous knowledge of the poison present. For the mineral acids one can make use of calcined magnesia, lime water, chalk, or carbonate of soda in weak solution. For alkalies the appropriate antidote is vinegar. For carbolic acid, vinegar, alcohol, or failing these a weak solution of soda or oil. For tartar emetic, gallic or tannic acid. For bichromate of potash or chromic acid, calcined magnesia, magnesia carbonate, or lime carbonate. For phosphorus, old oil of turpentine and demulcents—no oil. For ammonia, vinegar followed by almond, olive or sweet oil. In case of œdema glottidis, tracheotomy. For copper salts yellow prussiate of potash, which precipitates the copper in an insoluble form, and demulcents. For mercuric chloride, demulcent drinks can be resorted to, there is no other reliable antidote. In all cases after the evacuation of the stomach and the use of the antidote, mucilaginous agents must be given freely with morphia or other anodynes.

CATARRHAL INFLAMMATION OF THE FOURTH STOMACH.

Usually a complication. Causes, predisposing, exciting, changes of food or water, spoiled, frosted or fermented food, green potatoes, caterpillars, nitrogenous food, irritants. Symptoms: Separation from herd, grinding teeth, eructation, depraved appetite, fever, tender epigastrium, coated dung, red eyes, fixed or retracted, dilated, blind eyes, drooping ears, nervous symptoms, reckless unconscious movements, bellowing, tender skin, tremors. Lesions: Congestion and exudate in gastric mucosa, hemorrhagic discoloration, desquamation, excess of mucus, resemblance to rinderpest, Texas fever and malignant catarrh. Treatment: Empty stomach by bland laxatives, stimulants of peristalsis, calmatives, cold to head, counter-irritants, enemata, bitters.

This affection is by no means rare in cattle, though it is usually complicated with inflammation of the first three stomachs or of the intestines. Nevertheless, when the disease appears to be concentrated on the fourth stomach mainly, it may well take its name accordingly.

Causes. A predisposition to the affection occurs in the weak and debilitated, the overworked oxen, underfed cattle, in those that are just recovering from a severe illness and in which the gastric secretions and functions are still poor. The usual exciting cause is some fault in the food, it may be a sudden change from one kind to another, and especially from dry to green, or from one kind of green food to another and more tempting one, as when the animal breaks into a field of grain which is advancing to maturity. Even a sudden change of water, as from soft to hard has seemed in our experience to contribute to its development. Next come spoiled aliments, frosted or frozen turnips, beets, carrots, potatoes, apples, turnip tops, fermented grasses, musty hay, sun-exposed potatoes, putrid vegetables, and caterpillars on cabbages, tree leaves and other vegetables. Next come products that are highly nitrogenous, like vetches, alfalfa, sainfoin, clover, and the cakes of linseed, rape and cotton seed. Irritant plants such as colchicum, digitalis, yew, radish, etc., have been charged as causative agents.

Symptoms. These are often difficult to distinguish from those of indigestion or acute lead poisoning, and they vary in different cases according to the severity of the attack. In the milder cases there may be loss of appetite and rumination, some tympany, arching of the back, uneasy movements of the hind limbs and tail, a disposition to leave the herd, grinding of the teeth, and frequent gaseous eructations. Some show a depraved appetite, picking up and chewing various non-alimentary substances. Somewhat more characteristic are the dry, hot muzzle, the hyperthermia of the body, the tenderness to pressure of the epigastrium, and the baked appearance and glistening surface of the fæces. In the more severe forms the suffering is increased and the nervous system participates in the disorder. The eyes are congested, fixed or rolled back, the pupils dilated, the vision appears to be abolished, the ears are pendant, if tied the subject attempts to get loose, if at liberty he moves steadily in some one direction regardless of obstacles or dangers, he bellows, pushes against walls or other obstructions and may seriously injure himself or others. There is, however, no mischievous purpose, he is simply impelled to blind motion, and no regard is had to anything which may be in his way. In some instances the nervous disorder is manifested by a sensitiveness of the skin, so that the animal shrinks when handled or pinched along the chine or back. Tremors is another marked symptom.

Lesions. These consist mainly in congestion and swelling of the gastric mucosa, which is further covered by a thick layer of mucus. The folds are especially thickened and discolored, and the seat of hemorrhagic extravasations (petechiæ) and exudations in spots and patches. Desquamation of the epithelium may be met with at points and even distinct ulcers. Exudation in the submucous tissue, and petechiæ in the peritoneum are common. The condition may bear a close resemblance to what is seen in rinderpest or malignant catarrh.

Treatment. The first desideratum is the elimination of the irritant ingesta from the stomach. But for this purpose emetics are useless and we must fall back on laxatives. Again we are met by the

consideration that the inflamed stomach will neither readily absorb nor respond to a stimulus. Yet as a rule the viscus is not equally inflamed throughout, and even the affected parts do not necessarily have the whole muscular coat involved and paralyzed. We can therefore hope for a measure of response which once started will deplete and improve the adjacent and more violently affected parts. But irritant and drastic purgatives like croton, podophyllin or gamboge are proscribed as very liable to aggravate the inflammation. Pilocarpin 3 grs. hypodermically may be given or in default of this, 1 lb. each of Glauber and common salt in not less than six quarts of water, free access being allowed to pure water until it shall have operated. Bismuth may also be given as a calmative. Active rubbing of the abdomen will assist in rousing the stomachs to action, and hasten the action of the purgative. If there should be any sign of cerebral disorder, cold water or ice may be applied to the head, and oil of turpentine, followed by a pulp of the best ground mustard may be applied to the epigastrium and right hypochondrium. This may be accompanied and followed by copious enemata, and doses of quinia, gentian or still better nux vomica three times a day.

CATARRHAL GASTRITIS IN SWINE.

Definition. Causes, irritants, fermented, putrid swill, spoiled vegetables, irritant molluscs or larvæ, hot or cold food, alkalies, indigestible food, specific germs and toxins, parasites. Symptoms: inappetence, restlessness, vomiting, colic, constipation, diarrhœa, fever, stiffness, tender abdomen, arched back, chill, plaintive grunting, drooping tail. Lesions. Treatment: change diet, mucilaginous, milk, protection from saprophytes, change pen, emetic, laxative, calomel, bismuth, cleanliness, washing.

Definition. Inflammation of the gastric mucosa with mucopurulent discharge.

Causes. Irritants of various kinds, fermented or putrid swill, spoiled vegetables, irritant molluscs or larvæ, too hot or too cold aliment, excess of brine, excess of alkalies, in swill (dishwashings), indigestible foods of all kinds. The stomach may also be the seat of catarrhal inflammation in hog cholera, swine plague, rouget, diphtheritic affections and in the case of gastric parasites, so that it is very important to distinguish the affections due to simple irritants, from those dependent on plagues and parasitism.

Symptoms. There is inappetence, vomiting, restlessness and constant movement, colics, vomiting, constipation or diarrhœa, hyperthermia, stiffness, tenderness of the abdomen to handling, arched back, a disposition to hide under the straw, plaintive grunting when roused, drooping of the tail. The tendency is to a rapid recovery after the evacuation of the stomach by vomiting, though it may persist under a continuance of the irritating ingesta.

In these last cases the *lesions* may closely resemble those of the contagious affections of the abdomen, but the disease may be distinguished by its indisposition to spread beyond the herd which is subjected to the unhealthy dietary.

Treatment. Change the diet to one of pure and easily digestible materials, soups, mush, fresh whey or buttermilk, boiled farinas or flax seed, and even fresh grass. If there is violent diarrhœa boiled milk is often of great value.

Whatever food is given should be furnished in a vessel into which the animal can't get his feet, as these are usually charged with septic germs which are pathogenic to the diseased stomach, though they may have started from ordinary saprophytes.

For the same reason it is usually desirable to change the pen, as the animal grubbing in the ground charges the snout with the same offensive microbes.

If vomiting is not already established, 30 grains of ipecacuan may be given, or tepid water may be used to assist the process. If constipation is present 10 to 30 grains of calomel (according to size) may be given. In case of diarrhœa a combination of calomel 1 part and chalk 12 parts, may be given in 3 grain doses, two or three times a day. Or $\frac{1}{2}$ to 1 drachm nitrate of bismuth may be substituted.

Cleanliness in food and surroundings is among the most important measures, and if the skin has been filthy, repeated washing with soap and warm water may be resorted to with great benefit.

CHRONIC GASTRIC CATARRH IN SOLIPEDS.

Causes: Debility, age, anæmia, leucæmia, lymph gland, kidney, heart or lung disease, parasitism, dental or salivary disease, coarse, fibrous food, spoiled food, putrid water, gastric neoplasms. **Symptoms:** Impaired appetite, eating lime or earth, weariness, costiveness, coated dung, tympanies, diarrhœas, fatigue, sweating, unthrifty hide, pallid mucosæ, emaciation, colics. **Lesions:** Thickened right gastric mucosa, discoloration, mucus, petechiæ, opaque granular epithelium, gastric dilatation. **Treatment:** Remove causes, diet, watering, exercise, sunshine, bismuth, pepsin, acids, bitters, electricity, antiseptics, stomachics.

Causes. These are in the main the causes which operate in producing the acute affection. In most chronic cases they act continuously on a system rendered susceptible by debility or otherwise. Among predisposing causes may be named: The debility of old age, anæmia, leucæmia, chronic diseases of the lymph glands, of the liver, kidney, heart, or lung, parasitic diseases, diseases of the jaws, teeth, or salivary glands which interfere with proper mastication and insalivation. Among exciting causes may be named: A coarse, fibrous, innutritious diet, a too bulky diet, spoiled fodders of all kinds, putrid drinking water, and stomach parasites (spiroptera, œstrus larva). Actual disease of the stomach—papilloma, cancer, actinomycosis, tumors, and oat-hair or other concretions are further causes.

Symptoms. Impaired or capricious appetite, a disposition to lick the walls or earth, or to drink impure water, yawning, constipation with glossy mucus-covered fæces, and slight tympany, alternating with diarrhœa, small, accelerated pulse, susceptibility to perspiration and fatigue on slight exertion, unthrifty skin and hair, hide-bound, dry, hot mouth, coated tongue, pallor of the mucous membranes, loss of condition, and increasing weakness. Slight colics may occur at intervals, and the sluggishness may deepen into stupor or vertigo.

Lesions. The right sac is usually the seat of more or less hypertrophy of the mucosa, which is thickened, rugose, with patches of dark red, gray and slate color, and covered with a layer of tenacious mucus. The surface may show warty-like elevations, or papillary projections, with here and there patches of blood extravasation. The epithelial cells are increased, opaque and contain many fatty granules.

Dilatation of the stomach is not uncommon especially in old horses, and then the mucosa may be attenuated and smooth.

Hyperthermia may be present, but is so slight that inflammation cannot be predicated from it and it is difficult to establish a diagnosis from chronic dyspepsia.

Treatment. It is important to first correct any curable predisposing disorder, in teeth, jaws, salivary glands, blood, or internal organs, to carefully regulate feeding, watering and work, to secure as far as possible an outdoor life, and to employ bitter and other tonics. All over-exertion or fatigue must be carefully avoided. The food may be as advised for the more acute affection. Costiveness may be best met by boiled flaxseed, or in case of necessity by bran mashes, or green food. The irritation of the stomach may be benefited by nitrate of bismuth (3 to 4 drs.) and pepsin, and dilute muriatic acid with each meal are often of value. Nux Vomica (10 to 25 grs. twice daily) will help to restore the lost tone, and a current of electricity may be sent through the epigastrium daily. As alternatives, sulphate of quinia or gentian may replace the nux, and salol or salicylate of bismuth may take the place of the nitrate. Bicarbonate of soda in ½ dr. doses, common salt in ½ oz. doses, and fennel in ½ oz. doses are sometimes useful in re-establishing gastric functions.

CHRONIC GASTRITIS IN RUMINANTS.

Causes: As in acute form, parasites, gravid womb, insufficient ration, overwork, exhaustive milking, chronic diseases. **Symptoms:** deranged appetite, rumination, pica, eructations, regurgitations, tympanies, colics after feeding, coated dung, diarrhoea, fever, hot clammy mouth, sunken eyes, small weak pulse, palpitations, emaciation, weakness, tender hypochondrium. **Lesions:** hypertrophy of gastric mucosa, granular epithelium. **Treatment:** tonic regimen, diet, green food, roots, sunshine, bismuth, salol, strychnia, pepsin, muriatic acid, common salt, counter-irritants.

Causes. These are in the main the causes of the acute affection. There may, however, be special persistent factors like parasites (*strongylus contortus* and *filicollis*, *spiroptera*) in the stomach, the pressure of a gravid womb, an alimentation deficient in lime or phosphorus, overwork, exhausting milking, or chronic disease of important organs (heart, liver, lung, kidney).

Symptoms. These are indefinite and not easily distinguished from those of disorders of the third stomach. There is impaired or capricious appetite, a disposition to eat lime, earth, and all sorts of non-alimentary objects, rumination is rare or altogether suspended, efforts to regurgitate are ineffectual, or result in gaseous eructations only, there are tympanies and abdominal pains especially after feeding, and constipation with a firm glazed appearance of any faeces passed, may alternate for a short time with diarrhoea. The mouth is hot and clammy, the eyes sunken and semi-closed, the pulse small and weak, though the heart may palpitate, and there is a constantly progressive emaciation and prostration. Among the more characteristic symptoms are tenderness of the right hypochondrium to manipulation and percussion, and the presence of slight hyperthermia.

Lesions. The changes consist mainly in hypertrophy of the gastric mucosa, with changes in the epithelium and submucosa such as are already described in the horse. The pyloric region suffers most, and here ulcers are not at all uncommon.

Treatment. The main aim must be to remove the causes, and to build up the general health, so that the patient may rise above the debilitating conditions. More is to be expected from the change of diet to green food, roots, mash, etc., and an outdoor life than from the action of medicines, which are liable to disappear by absorption in the first three stomachs, so that they can only act on the fourth through the system at large. Yet benefit may be expected from the use of nitrate of bismuth, and salol, as calmatives and antiferments, *nux vomica* as a tonic and even from pepsin and muriatic acid as digestive agents. The two last are not dependent on the fourth stomach for their activity but will digest the contents more or less in the first three, and the finely disintegrated and partly peptonized ingesta coming to the fourth stomach in a less irritating, and less fermentescible condition, lessens the work demanded of that organ and gives a better opportunity for recuperation. Small doses of common salt and one or other of the carminative seeds may be added. The application of mustard or oil of turpentine to the right hypochondrium will sometimes assist in giving a better tone to the organ.

CHRONIC GASTRITIS IN SWINE.

Causes. Symptoms: inappetence, dullness, arched back, colic, irregular bowels, fever, emaciation. Treatment: diet, green food, milk, mash, cleanliness, bismuth, salol, sodium bicarbonate, strychnia, pepsin, muriatic acid, sunshine, washing.

Causes. These are like those producing the acute affection which may easily merge into this by a continuation of such causes.

The *symptoms* too are alike. Inappetence, dullness, prostration, arched back, vomiting, colic, constipation, with alternating diarrhoea. There is hyperthermia with hot dry snout, thirst, increasing emaciation, and anæmia.

Treatment. An entire change of diet, to green food, roots, fresh milk, and soft mash in limited quantity. Allow pure water freely. Adopt all precautions against contamination of the food by the feet or snout. The stomach may be quieted by oxide of bismuth (20 grs.) or salol (10 grs.) two or three times daily, and the tone and secretions of the stomach may be stimulated by bicarbonate of soda (1 dr.) and nux vomica (1 to 2 grs.) thrice daily. In addition pepsin and muriatic acid may be given with each meal in proportion adapted to its amount. A life in the open air, and an occasional soapy wash will do much to restore healthy gastric functions.

CHRONIC GASTRITIS IN THE DOG.

Causes: faults in diet, musty food, foreign bodies, poisons, lack of sunshine, retained fæces, parasites, ill health, chronic diseases, icy bath, septic drink. Symptoms: irregular appetite and bowels, fever, foul breath, red tongue, tartar on teeth, dullness, prostration, vomiting of mucus or bile, tender epigastrium, arched back, foetid stools, emaciation. Treatment: regulate diet, sunshine, pure water, scraped muscle, soups without fat, antiseptics, calomel, pepsin, muriatic acid, strychnia.

Causes. The irregularity and variability of the food, overfeeding, highly spiced foods, putrid or spoiled food, musty food, the swallowing of pieces of bone, and of indigestible bodies, the consumption of poisons, the absence of open air exercise, the compulsory suspension of defecation in house dogs, and the presence in the stomach of worms (spiroptera, strongylus), are among the common causes of the affection. As in other animals, ill health, debility, lack of general tone, and chronic diseases of important organs (liver, kidney, heart, lungs) must be taken into account. The plunging into cold water when heated and the licking of septic water must also be named.

Symptoms. Appetite is poor or irregular, the nose dry and hot, the mouth foetid, the tongue reddened around the borders and furred on its dorsum, the teeth coated with tartar, the animal dull and prostrate, vomits frequently a glairy mucus mixed with alimentary matters or yellow with bile, and there is constipation alternating with diarrhœa. The epigastrium is tender to the touch, the back arched, the fæces glazed with mucus or streaked with blood, and offensive in odor. Emaciation advances rapidly and death may occur from marasmus.

Treatment. Adopt the same general plan of treatment. Stop all offensive and irritating food, give regular outdoor exercise, free access to pure water, and every facility to attend to the calls of nature. Give plain easily digestible food in small amount. In the worst cases pulped or scraped raw meat, in the less severe mush, or well-prepared soups with the fat skimmed off, and bread added. Check the irritant fermentations in the stomach by salol, bismuth, salicylate of bismuth, or naphthol. In case of constipation give 8 to 10 grs. calomel. Then assist digestion by pepsin (5 grs.) and hydrochloric acid (10 drops) in water with each meal. If the bitterness is not an objection 1 gr. nux vomica may also be added.

ULCERATION OF THE STOMACH.

Causes: peptic digestion, paresis, caustics, irritants, acids, alkalies, salts, mechanical irritants, hot food, parasites, thrombosis, embolism, specific disease poisons, aneurism, tumors, infective growths, nervous disorder, debility, toxins of diphtheria, staphylococcus, etc. Symptoms: slight colics, tympany, emaciation, vomiting blood, tender epigastrium, dark or bloody stools, irregular bowels; in carnivora abdominal decubitus, arched back, bloody, mucous, acid vomit, colics after meals. Lesions: in horse erosions, ulcers, parasites, neoplasms, discolorations, extravasations; in cattle and dogs on folds, nature of ulcer. Treatment: restricted, digestible diet, lavage, anodynes, bismuth, antacids, antiseptics, salol, naphthol, chloral, pure water.

Causes. Gastric ulcers may arise from quite a variety of causes which determine necrotic conditions of the mucosa and the gradual invasion of the resulting lesion by destructive microbes. One of the simplest factors is the peptic juice, the stomach, being struck with paresis (in inflammation, fever, nervous disorder), while containing a quantity of its secretion, undergoes an autodigestion which affects particularly the lowest (pyloric) portion, toward which the liquid gravitates, and the free edges of the folds which are the most exposed to its action.

The swallowing of irritant and caustic agents (the mineral acids or alkalies, mercuric chloride, tartar emetic, antimony chloride, Paris green, arsenious acid, etc.) by corroding or causing destructive inflammation of the exposed mucous membrane may similarly operate. This is especially the case with monogastric animals, (horse, pig, dog, cat), as in the ruminants such agents tend to be diluted in the first three stomachs and rendered more harmless.

Mechanical irritants may cause the lesion and infection atrium in any of the domestic animals, pins, needles, nails, pieces of wire and other sharp pointed bodies being swallowed by horse and ox, and small stones, pieces of bone, and all sorts of irritant objects picked up by the puppy or rabid dog.

Cooked food swallowed hurriedly at too high a temperature is especially liable to start necrotic changes in the single stomach of horse, pig or dog, the ruminant being in a measure protected by the food passing first into the rumen.

The wounds caused by gastric parasites may become the starting points of molecular degeneration and ulceration. In the horse the spiroptera megastoma, s. microstoma, and the larvæ of the various œstri; in cattle and sheep the strongylus contortus, s. convolutus, s. filicollis and s. vicarius; in swine the spiroptera strongylina, Simondsia paradoxa, and gnathostoma hispida; in dogs spiroptera sanguinolenta, and in cats the ollulanus tricuspidatus act in this way.

Gastric catarrh debilitates the affected mucosa and lays it open to necrotic microbial infection especially in the pyloric sac and on the summit of the folds.

Interruption of the local circulation in the deeper parts of the mucosa as in inflammation and capillary thrombosis, arterial embolism, venous thrombosis, may lead to local sloughing and ulcerous infection. This may be seen in the petechial fever of the horse, malignant catarrh, rinderpest, and anthrax in cattle and sheep, and in canine distemper in dogs. Vogel found ulcers resulting from a gastric aneurism in the dog.

Tumors and infective growths in the walls of the stomach may prove an occasion of ulceration. Thus sarcoma, epithelioma, actinomycosis and tubercle may be the primary morbid lesion in different cases.

Gastric ulcers have also been attributed to morbid nervous influences as in dogs they have been found associated with lesions of the dorsal myelon, and the corpora quadrigemina, and faradisation of the vagus has apparently led to their production.

General constitutional debility has been alleged as a factor, and experimentally in dogs, the hypodermic or intravenous injection of various microbes or their toxins (diphtheritic toxin, Enriquez and Hallion, staphylococci, Panum, Lebert, Letulle, and a bacillus of dysentery in man, Chantemesse and Widal), have produced gastric ulcers.

Symptoms. In *horses* and *cattle* these are very obscure, being mainly in the nature of chronic gastritis. In both there are recurrent attacks of slight colicky pains, with tympany in cattle, and gradual emaciation. Vomiting has been exceptionally seen in both class of animal and if the rejected matters are very acid and above all if mixed with blood it is more suggestive of ulcer. In the horse the attacks of colic are mostly in connection with eating, or (in case the ulcer is duodenal) an hour or two after a meal. In this animal it is possible to withdraw liquids from the viscus by the stomach pump, and any hyperacidity or blood may be almost diagnostic. Tenderness to pressure on the epigastrium or hypochondrium is often present, yet the colics of ulceration are often relieved by pressure and friction. Blood is sometimes present as such in the excrements, but more commonly these are simply blackened by the exuded blood as acted on by the gastric acid and intestinal liquids. The bowels may be alternately constipated and relaxed. A gradually increasing feebleness is a characteristic feature and in cattle paraplegia may precede death.

In the *carnivora* the symptoms are less obscure. The animal is dull, prostrate, weak, lies on its belly, but rarely long in one place, and when up has arched back, stiff movements, and tucked up abdomen. The epigastrium is painful to touch, which tends to arouse vomiting of food or bloody mucus. As in the horse the rejected matters are very acid. Constipation may alternate with diarrhoea, the faeces being blackened (melæna) or even streaked with blood. The occurrence of suffering after meals, the constancy and persistency of the symptoms and the steadily advancing emaciation and weakness are very characteristic. If the tenderness is referable to a given point, it is even more distinctive.

Lesions. In the *horse* ulcers and erosions occur in the cardiac sack in connection with œstrus larva and spiroptera which destroy and remove the cuticular covering, or with sarcoma or epithelioma growing in the gastric walls. In the right sac there may also be round ulcers from the hooklets of the œstrus, or irregular excavations on the summits of the folds in connection with catarrhal inflammation. Ulcers from autodigestion are usually in the right sac, in the most dependent part of the viscus, between the folds, and of a more or less circular outline. The raw surface is black, brown, slaty gray or white. The ulcers which result from petechial fever are irregularly notched and marked by a mass of dark blood coagulated in their depth.

In *cattle* and *dogs* the ulcers are most frequent near the pylorus, and when of catarrhal origin may be round or irregular, and on the summit of the fold, or if peptic, may be round and between the folds. In malignant catarrh and rinderpest, they are mostly formed on the summits of the folds. They may vary in size from a pea to a quarter of a dollar. The surrounding mucosa is usually congested, swollen, and projecting, and the surface of the ulcer itself of a dark red, black, yellowish, slaty or gray.

The round ulcer is usually marked by surrounding infiltration and by a tendency to become deeper and to perforate the gastric walls, with the result of inducing an infective peritonitis. This is more common in cattle and carnivora than in solipeds.

Treatment. If a reasonably certain diagnosis can be made the patient should be put on a restricted diet of easily digested materials, given at regular intervals. For the carnivora scraped or pulped raw meat, and milk, and for the herbivora milk and well boiled flax seed or other farina are appropriate.

Violent emesis in carnivora may demand washing out of the stomach with tepid water with or without the aid of a stomach tube. This may be seconded by anodynes, chloral, cyanide of potassium, or even morphia.

Bismuth trisintrate or oxide is appropriate in all animals, also sodium bicarbonate, chalk or magnesia to neutralize the muriatic acid.

As antiseptics calculated to obviate the formation of irritant products from the gastric contents and to check the progress of the microbial infection in the wound such agents as the following may be used: Salol (horse or ox 1 dr., dog 5 grs.), naphthol or naphthalin (same doses), chloral (horse 2 drs., dog 5 grs.).

Sometimes it is well to relax the bowels by small doses of Glauber salts, and in all cases an abundance of fresh water, butter milk, or other bland drink.

Cases of the kind are slow in their progress and unless the animal is specially valuable, treatment may be a source of loss.

PERFORATING ULCER OF THE STOMACH

Causes: round ulcer, foreign bodies, parasites. Symptoms: those of ulcer, followed by infective peritonitis, fistula, pleuritis, pericarditis. Treatment: of fistula.

This may be the result of the gradual deepening of the round ulcer, yet in the domestic animals it mostly comes from the presence of sharp pointed bodies. These may be enumerated as needles, pins, nails, wires, sharp bones (dog), whalebone (horse), forks, knives (cattle), and even gravel. The burrowing of the spiroptera has seemed to cause perforation in the horse. All causes of ulceration may, however, lead to perforation.

The *symptoms* are those of gastric ulcer, already given, followed by the more specific ones of perforation. These in their turn differ according to the parts involved. In the horse and dog the perforating ulcer usually opens into the peritoneum, inducing a fatal infective peritonitis. In cattle the foreign body sometimes passes toward the heart, enveloped in a protecting mass of new formed tissue and proves fatal by heart disease. In other cases it has been found to proceed downward toward the sternum and to escape by a fistula formed beside the ensiform cartilage. In other cases it has taken a direction toward the right wall of the abdomen where it formed a fistula, discharging alimentary matters. In still other cases it has opened into the peritoneal cavity with fatal effects.

Treatment in the case of external fistula, without implication of the peritoneum, consists in the removal of the foreign body, and the stimulation of granulations along the tract of the fistula by the application of an ointment of tartar emetic to the interior. Should this fail the fistulous tract may be scraped to make it raw, and the edges may then be drawn together with sutures taking a deep hold of the skin.

DILATATION OF THE STOMACH.

Adaptability to bulk of food. Dilatation with atony. Eructation. Cribbiting. Vomiting. Age. Rare in cattle. Catarrh, overloading, nervous lesions, intestinal obstructions, tumors, calculi, volvulus, invagination, hepatitis. Symptoms: overfeeding, pot-belly, unthrifty hide, emaciation, eructations, cribbiting, fatigue, perspiration, indigestion, colic after meals, tympanic resonance. Lesions: varying distension, contents, action of calculus or pebbles, cardiac dilatation. Treatment: nutritive, digestible, concentrated food, lavage, strychnia, iron, faradisation, antiseptics.

The stomach has a great power of accommodation to the amount of food habitually taken. In the horse fed mainly on grain with only a little hay, it is habitually small, while in one fed on cut straw with a little grain, on hay alone, or on green food, it is very much more capacious though within the physiological limits of health. The cow wintered on grain alone, has all four stomachs lessened in capacity, and though she maintains good condition she is ill fitted to change at once to the bulky grass diet of spring. The heavily fed swine, and the farina fed dog and cat, have both stomach and intestines increased in capacity over those of the wild boar, or the purely carnivorous wolf or wild cat.

The condition becomes pathological when associated with atony, and this may occur directly from over distension. It is especially common in the horse by reason of the difficulty of relieving the over distension by eructation or vomiting, and also by reason of the habit of swallowing air (cribbiting). The dog, which has great facility in vomiting, should be correspondingly protected from the condition, yet it is very common in old dogs, doubtless from their common vice of gourmandizing and lack of exercise. Cattle are rarely attacked, the fourth stomach being protected by the others which stand guardian over it and prevent the sudden access of excess of food even if that is rapidly swallowed.

Other *causes* are: chronic catarrh which renders the stomach atonic, lessens its peptic secretion and determines indigestions and over distensions: habitual overfeeding which results in chronic indigestions and fermentations; lesions of the brain, and tumors of the jugular furrow or mediastinum which interfere with the functions of the vagus nerve; obstructions of the intestines which force the contents back into the stomach or hinder their exit. Thus tumors on the duodenum, calculi in stomach or intestines, volvulus and invagination have been charged with producing overdistension. Chronic hepatic disorder has also been quoted as a cause.

Symptoms. The subject may eat naturally or excessively yet is unthrifty, the belly is habitually distended, the hair dry and rough, there is loss of flesh, there may be eructations or (in the horse) swallowing of air, lack of endurance, a disposition to perspire easily, a tendency to indigestion and colics after meals, and hurried breathing sometimes marked by a double lifting of the flank in expiration. In the *dog* which has the stomach more accessible to examination its outline may be followed by percussion, a tympanitic resonance being produced from the eighth rib back to the umbilicus or further. If there is any difficulty the organ may be emptied of water by a stomach tube and then pumped full of air by means of a Davidson's syringe, and percussed in each condition. Or a half a teaspoonful of bicarbonate of soda may be given in a little water followed by an equal amount of tartaric acid, and the stomach percussed.

Lesions. The distension of the stomach may reach ten times its normal size in the horse (Leisering). Kitt found a stomach with a capacity of 84 quarts. Fitzroy Philipot took from a dilated equine stomach 140 lbs. of contents. The contents of the viscus are usually largely of solids which the weakened and attenuated walls failed to pass into the duodenum. On the contrary and as if by compensation, the pylorus and duodenum are constricted and the latter has liquid contents which pass from the stomach with very little of the solids.

Special dilatations are sometimes met with, thus an equine stomach has been found largely dilated at the greater curvature where concretions formed in the viscus or pebbles introduced with the food had habitually lodged. In other cases the cardia has been dilated like a funnel, so that the

animal could eructate or vomit with great facility. This last dilatation is especially common in cribbiters.

Treatment. This must necessarily be prolonged as time must be allowed for a tonic contraction of the viscus. Food must be given often in small quantity, of easy digestion, and of aqueous composition. For dogs, milk, eggs and soups, or pulped raw meat furnish examples. For horses milk gruels, boiled flax seed, pulped roots may suffice. If the stomach is loaded as is usually the case, it should be washed out with the stomach tube, which when passed into the stomach should be raised at its free end and filled with tepid water; it is then suddenly lowered so as to act as a syphon in evacuating the liquid contents of the stomach. This may be repeated again and again, the stomach in the case of the dog being manipulated so as to mix and float the solids and favor their exit through the tube. Daily washing out of the stomach by the tube is of the greatest possible value.

Meanwhile we should seek to improve the tone of the stomach by strychnia (horse 2 grs., dog $\frac{1}{60}$ gr. daily), by salts of iron, and by faradisation.

To counteract fermentation, antiseptics (salol, naphthol, freshly burned charcoal) may be given with each meal, along with pepsin and hydrochloric acid.

RUPTURE OF THE STOMACH IN SOLIPEDS.

Mainly in solipeds. Causes: overloading, fermentation, impossibility of eructation, violent concussions, falls, galop, concretions, dilatation, catarrh, ulcers, cicatrices, abscesses. Symptoms: Anamnesis, colic relieved, followed by prostration, sinking, complete anorexia, tender abdomen, vomiting, no abdominal rumbling. Lesions: tear in greater curvature, most extensive in outer coats, shreddy, bloody edges with clots, contents in omentum, other seats, partial ruptures. Treatment: in partial ruptures, stomach pump, diet. Prevention.

This is pre-eminently a disease of solipeds for the reason that they alone of domestic animals are especially liable to overload the comparatively small stomach and are mostly unable to relieve the overloaded viscus by eructation or vomiting.

Causes. These are in the main overloading of the stomach and overdistension, by the gases of indigestion. To this are usually added violent concussions when the animal throws itself down violently. The stomach distended to the fullest possible capacity, and lodged in a cavity which is not all equally tense, is comparable to a very tense bladder which is liable to burst when forcibly struck, or suddenly compressed.

Apart from such indigestion, cases are recorded in which the full stomach has been burst by a sudden fall in the shafts or elsewhere. Miles even records a case which occurred during a rapid galop after a full drink of water.

The presence of solid bodies (calculi, gravel) in the stomach or even in the intestines has appeared to cause rupture by blocking the outlet of ingesta and determining indigestion.

Certain conditions predispose to rupture, notably dilatation of the stomach with attenuation of its walls, cribbiting, old standing catarrh of the viscus, pre-existing ulcerations, cicatrices and abscesses.

Symptoms. There is usually the history of a full feed of grain, followed by violent colic, and indications of gastric overdistension, tense abdomen, dullness, then the rejection of the gastric contents by vomiting, the matters escaping by the nose, and then collapse. The violence of the colics may cease, but the pulse becomes rapid, small, and finally imperceptible, the breathing hurried, the head depressed, eyelids, ears and often the lower lips drooping, the face becomes heavy and expressionless, the belly distended and tender, the skin covered with cold sweat, and the temperature exalted above or depressed below the normal. There is never any disposition to eat nor drink. Death follows in a few hours.

In the vomiting which is independent of rupture, the symptoms are usually at once relieved, when the emesis occurs, since not only liquid and solid matters escape but also gaseous material. The pulse retains its fullness, the facial expression is that of intelligence and comfort, rumbling may be resumed in the bowels, fæces and urine may be passed, and colics are less acute. In favorable cases the animal may even desire to eat or drink.

Lesions. The usual seat of rupture is on the great curvature and may extend longitudinally for from six to ten inches. The laceration is usually most extensive in the outer coats, and the mucosa is carried outward with the escaping ingesta, which helps to efface the normal mucous folds at the cardia, and to render vomiting possible. The edges of the wound are more or less shreddy, and of a dark violet color from blood extravasation and clots. The escaping contents are rarely diffused in the cavity of the abdomen, but remain enclosed in the omentum through the thin meshes of which they can be easily seen, and which has sometimes been mistaken for the walls of the stomach reduced to this attenuated condition by disease. When the omentum gives way the contents are at once diffused through the abdominal cavity between the convolutions of the intestines. In exceptional cases the rupture has its seat in the lesser curvature, or even at the cardia. In still others the laceration implicates the muscular and peritoneal coats only, and the looser mucosa, filled with ingesta bulges outward as a hernia. In such a case a recovery seems possible if the viscus could be relieved of its contents.

Treatment is virtually hopeless. Yet a moderate laceration of the two outer coats only might be followed by recovery through the formation of a cicatrix. The first consideration would be the unloading of the stomach spontaneously or by the aid of the stomach pump, and thereafter the adoption of a rigidly restricted diet of easily digestible food (such as gruels) in small quantities at a

time.

Prevention is much more available. In violent colics with overloading or tympany of the stomach, employ anodynes to keep the animal from throwing himself down violently, give a soft bed of litter where the shock on lying down will be lessened, employ antiferments to prevent gaseous distension, and whenever possible relieve the plenitude of the viscus by the stomach pump or tube.

TORSION OF THE STOMACH IN THE DOG.

Causes: mobility of dog's stomach when empty, leaping, running down stairs. **Lesions:** viscus doubled forward, pylorus in front of cardia, duodenum compresses cardia, liver, spleen and omentum displaced, stomach tympanitic, lungs and heart compressed, latter gorged with dark blood. **Symptoms:** tympanitic abdomen, and half thorax, no rumbling, murmur in front of thorax, abdomen tender, patient stands, dyspnoea, emesis impossible. **Course:** violent symptoms in twelve hours, death in thirty-six. **Diagnosis:** sudden, severe seizure, complete anorexia, tympany, tenderness, dyspnoea, no vomiting, arrest of peristalsis. **Obstruction.** Peritonitis. Choking. **Treatment:** tapping, laparotomy, replacing the viscus.

This has been demonstrated by Kitt and Cadeac who believe that it is quite a common occurrence.

Causes. The predisposing cause is the extreme mobility of the canine stomach which hangs from the oesophagus like a pear from its stalk, the remainder of the viscus being only attached to the loose omentum, spleen, and commencement of the duodenum all of which it can carry with it easily when it rolls on itself. Its mobility is, however, very restricted when full, the liver on the one side and the spleen and intestines on the other proving almost insuperable obstacles to rotation. But when empty it moves with great freedom and by a sudden shock in leaping, gamboling or running rapidly down stairs the pylorus is carried forward and to the left until it and the commencement of the duodenum are jammed in front of the cardia. The result is the obstruction of the cardia and duodenum by their mutual pressure in crossing each other, and the interruption of the gastric circulation and functions.

Lesions. As just stated the stomach which would normally extend from the cardia downward and to the right is bent forward and doubled upon itself, the pylorus lying in front of the cardia, the duodenum extending from before backward above the cardia and tightly compressing it, the liver drawn to the left by the hepato-duodenal peritoneum, and the spleen displaced to the right by the traction on the omentum. The stomach enveloped in its omentum is distended by gas to perhaps ten times its normal dimensions and appears to fill the entire abdominal cavity while the intestines are pushed aside and concealed. The chest is compressed by the strong pressure on the diaphragm, and the lungs are congested of a deep blue and the right heart distended with dark blood. The animal appears to have perished of apnoea.

Symptoms. In fully developed cases the abdomen is greatly distended and tympanitic. The drumlike resonance is met with in the anterior part of the abdomen including the umbilical region. It extends forward over one-half of the thorax, excepting only a space of 5 or 6 inches square in the right hypochondrium, which represents the situation of the liver, and spleen. Auscultation furnishes no sound in the abdomen, and only in the anterior portion of the thorax is there a distinct respiratory murmur. The heart may beat strongly and rapidly, or weakly and slow, and the pulse is small and thready. The abdomen is tender. The animal stands, dull, and breathes with great effort. If made to walk it is done slowly, stiffly and with head extended, mouth open and tongue protruding. There is no sign of vomiting and this cannot be brought about by tickling the fauces, or even by giving apomorphine subcutem, though retching may be induced.

Course. The disease may develop into dullness and anorexia in two hours after boisterous health; in twelve hours there may be considerable tympany and dyspnoea; and a fatal result is reached in about thirty-six hours.

Diagnosis. This is based on the transition from vigorous health to sudden illness, with complete anorexia, inability to swallow or to vomit, tympany of the stomach as shown by percussion, tenderness of the abdomen, dyspnoea, disturbed heart-functions, and inactivity of the bowels. With intestinal obstruction on the other hand there is free vomiting of bilious and feculent matters. With peritonitis there is much greater and more uniform abdominal tenderness, vomiting and higher fever, but less tympany in the anterior abdominal region, and no such complete suspension of defecation. With choking there is no such progressive tympany, appetite and defecation are not so completely suspended, and liquids may often pass the obstruction in small quantities in both deglutition and vomiting. Choking is by no means so speedily fatal.

Treatment is essentially surgical. When tympany is already established the gas must be

evacuated by a small cannula and trochar. Then resort is had to laparotomy, the incision is made on the right side large enough to introduce the fingers, which must follow the great curvature of the stomach as far as the pylorus which is pulled back into its normal position on the right. The incision is now closed by an ordinary continuous suture.

FOREIGN BODIES IN THE STOMACH. HAIR, WOOL, BRISTLE, CLOVER AND COTTON BALLS.

Hair balls, wool balls, bristle balls, cotton balls, clover-hair balls, oat-hair balls, paper balls, phosphatic calculi, sand and gravel, nails, wires, needles, pins, etc., cloth, leather, whalebone, playthings, etc. Symptoms: of catarrh or colic, dullness, restlessness, arched back, in dog vomiting of blood, fistula. Diagnosis. Treatment: emetic, feed potatoes, laparotomy.

Hair Balls. These are common in the rumen of cattle and have been found in the fourth stomach. They are especially injurious to young animals by reason of their irritating the gastric mucosa, but they also occasionally block the pylorus, producing indigestion, gastric dilatation, gradually advancing emaciation and even a fatal result.

Wool Balls. These are found in sheep and are especially injurious in young lambs.

Bristle Balls. These are found in swine as round, or ovoid balls or long ellipses bent upon themselves. The sharp projecting ends of the bristles render them very irritating, especially to young pigs.

All of these are caused by licking themselves or their fellows, and particularly during the period of moulting or as the result of some skin affection. Lambs which are nursed by ewes with an excess of wool on and around the mammæ, and old sheep with a disposition to eat wool are frequent victims.

Cotton Balls. These have been found in lambs fed on cotton seed cake. A certain amount of the cotton fiber is incorporated in the cake, and this is rolled together and felted by the movements of the stomach and agglutinated by mucus.

Clover-hair Balls. The fine hairs from the clover leaf have been found rolled into balls in the abomasum of lambs producing all the evil effects of the other pilous masses.

Oat-hair Balls. The fine hairs which cover the seed of the oat are found matted together and cemented by mucus in the stomach of horses fed on the *dust* of oatmeal mills. They are especially common in Scotland, where oatmeal has been so extensively used.

Paper-ball. In the museum of the N. Y. State Veterinary College is a conglomerate ball of paper taken from the stomach of a hog by Dr. Johnson, Sioux City.

Phosphatic Calculi have been described as found in the stomach, but this is evidently an error, as the acid secretion would have speedily dissolved them. The error doubtless came from mistaking the transverse colon for the stomach.

Sand and Gravel arrive in the stomach of the *horse* from pasturing on loose sandy land, the plants being pulled up by the roots and swallowed together with the sand adherent. Also from drinking water from shallow streams with sandy bottoms. Feeding of grain from the ground is a cause of swallowing sand, earth and pebbles. Licking the soil in acidity of the stomach is another cause. Fodder that has been packed down and mixed with earth, and that which has been blown full of sand or dust, and roots eaten from the ground in wet weather lead to the ingestion of much sand or earth. Shetland ponies taken from the islands pass sand for some weeks. Dogs taught to fetch and carry, swallow stones, pebbles, marbles, etc., accidentally.

Nails, Wires, Needles, Pins, etc. More or less pointed metallic objects are often taken in with the food by gluttonous horses and though usually arrested in the intestines they sometimes irritate or wound the stomach.

Fragments of cloth, leather, or whalebone are similarly taken with the food, or in case of depraved appetite are deliberately chewed and swallowed.

Playthings and small household articles are especially taken by puppies through mere wantonness. Rubber balls, pieces of metal, thread, cord, cloth, bits of leather, sponge, horse hair, human hair, corks, bits of wood and everything obtainable of small size may be swallowed and

found in the stomach.

Pigs swallow pieces of wood and other objects.

Birds habitually swallow pebbles and ordinary objects are ground down in the gizzard. They also readily vomit feathers, bones and other offensive matters that have proven indigestible.

Symptoms. In **horses** there are no especial symptoms, though the foreign bodies sometimes cause gastric catarrh, and in other cases produce wounds and ulcers or block the pylorus causing violent colic. Most commonly the foreign bodies pass on into the intestines, where they may directly wound the walls, form nuclei for the deposition of earthy salts in the form of calculi, or in case of fibrous materials (cords) roll into firm balls.

In **dogs** the foreign bodies may cause gastric catarrh, or puncture or abrasion of the mucosa, and they may be rejected by vomiting. The more rounded, smooth bodies may lie for a length of time in the stomach without doing any manifest injury, as in the case mentioned by Nichoux in which a dog carried in its stomach for twelve years a four franc piece and a large sou. Sometimes the objects block the pylorus. Then the subject is dull, depressed, inclined to lie on the right side but continually changing his position, gives a stifled yelp when he lies down or occasionally when he stops walking. He carries the back arched, and the abdomen tucked up, and drags his hind limbs. Vomiting, is frequent and accompanied by violent and painful retching. The vomited matters may be mixed with blood. The epigastrium is tender to pressure. Death may ensue in twenty-four hours or not until after weeks or even months.

In other cases there is gastro-enteritis with vomiting, colic, anorexia, trembling, hyperthermia, constipation or diarrhoea, and finally the passage of the offending agent per anum, when recovery ensues.

In other cases sharp pointed bodies perforate the walls of the stomach, and determine the formation of abscess or fistula opening at any point around the abdominal cavity. This may be followed by recovery, by gastric or intestinal fistula, or by chronic disease of some important organ like the liver.

In **dogs**, *diagnosis* is often possible by manipulation of the stomach through the walls of the abdomen. If the belly is very lax it may be compressed between finger and thumb, or between the two hands; if more tense, pressure with both hands just behind the sternum may detect the resistance of a solid body.

Treatment. In the **horse** this is hopeless.

In the **dog** much may be expected from the use of emetics, (ipecacuan, tartar emetic, apomorphine, tepid water, tickling the fauces). In some cases of sharp pointed bodies an exclusive and abundant diet of well boiled potatoes proves successful. The object is to pass much of the starchy matter through the small intestines undigested, so that it may envelop the sharp body and protect the mucosa. When it reaches the colon, the ingesta as a whole becomes more solid and invested by this, the body is often passed without danger. Other methods failing laparotomy remains. The dog is stretched on his back on a table with the forelimbs held well apart. The skin of the epigastrium is denuded of hair and washed with antiseptics (mercuric chloride solution 1:500). Hands and instruments are also made aseptic. Then an incision is made in the epigastrium or in the situation where the offending body has been felt, and the finger is introduced to locate the body. At this point a thread is passed through the walls of the stomach, and these are drawn well out through the abdominal wound and incised to the extent of an inch or more. Through this orifice the foreign body can be easily felt and extracted. Then in case the stomach is over-filled it may be emptied, and the edges washed with the antiseptic and carefully sutured with sterilized catgut. The usual care must be taken to turn the mucosa inward and bring the muscular and serous coats in accurate opposition. Finally the abdominal wound is closed by a continued suture of silk or catgut.

The greatest care must be taken to prevent the escape of any of the gastric contents into the abdominal cavity, to render both wounds aseptic and to protect the external wound especially against infection. A wash of carbolic acid (1:100) with a little of some intense bitter (quassia) will often succeed in preventing licking or gnawing.

Even greater care must be given in the matter of diet. At first a few teaspoonfuls of cold water

only need be given. After twenty-four hours a little well strained beef tea; later milk or gruel may be added, and by degrees more solid food. In three weeks the ordinary food may usually be resumed.

In case the foreign body has escaped into the peritoneal cavity, the same method may be pursued, the edges of the gastric or intestinal wound being made raw, treated antiseptically and carefully sutured, and the abdomen washed out with an antiseptic solution (aluminum acetate solution) and closed.

TUMORS OF THE STOMACH.

In horse—sarcoma, papilloma, lipoma, adenosarcoma, epithelioma, in cattle—scirrhus, in dog—sarcoma, lipoma, epithelioma. Symptoms: chronic gastritis, periodic indigestions, colics, vertigo, salivation, impacted gullet, blackened fæces, eructations, vomiting, rumbling, stiffness, emaciation. Treatment: laparotomy in dog.

The peptic stomach in the different animals is subject to a great variety of tumors. In many of the recorded cases, however, the true nature of the tumor has been left uncertain.

Sarcoma. In the **horse** this is the common tumor of the pylorus, and less frequently it is found on the cardia and body of the stomach, especially on the greater curvature. These are usually firm and resistant, though sometimes soft and friable; they tend to swell out in lobules, and show areas of ulceration, or even suppurating excavations opening through the mucosa. In some instances, however, they start under the serous coat, and the ulcerous surface may open into the peritoneum. At other times they are but a local manifestation of a general affection.

In the **dog** multiple sarcomata have been found on the stomach varying in size and easily mistaken for recent tubercles. In these cases the small round cells were especially numerous in the centre of the tumor rendering it soft and predisposing to degeneration.

Papilloma. In the horse these are found as branching or filamentous dependent projections from the mucosa of the left sac having evidently started from the sores formed by the attachment of the œstrus larvæ. They are also found around the pylorus and of such size as to seriously obstruct that orifice (Stadler).

Lipoma. Fatty tumors have been seen on the stomach of the **dog** and **horse** in the submucosa.

Adenosarcoma. This formation in the **horse** leads to a thickening of large patches of the mucosa. It also grows out in mushroom like masses, or is irregularly lobulated.

Epithelioma. In the **horse** epithelioma has been found at the pylorus and on the great curvature of the stomach. It usually grows out as a rounded mass varying in size from an egg to an infant's head, and may be even a diffuse thickening of the mucosa. Microscopically the individual lobules, are composed of cylindroid cells surrounding a central mass of epidermoid cells. The stomach may be greatly contracted, and the surface of the neoplasm, ulcerated or even excavated. In the **dog** similar formations are found.

Carcinoma. In **cattle** *Scirrhus* of the abomasum is described. Small tumors rise to a height of $\frac{1}{2}$ to 3 inches, and are closely packed together so as to assume polygonal forms. The surface is smooth, or perforated by orifices leading into ulcerous or suppurating cavities. On section the mass shows a fibrous or a lardaceous consistency. They are most common in the pyloric region, and may partially obstruct this orifice.

Symptoms. These are necessarily obscure. In the **horse** periodic gastric indigestions and colics may be the sole indications, which are certainly not pathognomonic. In other cases, have been noticed: vertigo, salivation, impacted gullet, and blackish, sanguinolent fæces due to ulceration and hemorrhage from the tumors.

In **cattle** have been observed variable and capricious appetite, imperfect rumination, tympany, eructations, vomiting, rumbling of the bowels, constipation, slow painful walk, progressive emaciation and debility. When blood is discharged by emesis or defecation the suspicion of gastric tumor may be strengthened.

In the **dog** there are the usual signs of chronic gastritis, thirst, anorexia, stiffness, a disposition to lie, sunken eye, dyspnœa, vomiting, often of blood. The discharge of blood by mouth and anus, the distended abdomen, the tumor usually easily detected by manipulation, and the progressive loss of condition are strongly suggestive.

Treatment of these cases is hopeless. In the dog alone for a circumscribed tumor, laparotomy, the removal of the tumor and closure of the wound may be tried.

ACUTE INTESTINAL INDIGESTION IN THE HORSE.

INTESTINAL TYMPANY.

Definition. Causes: Debility—general and local, and its causes, fermentescible food, legumes, new grain, paralyzing seeds, musty fodder, defective teeth, jaws and salivary glands, iced water after grain, verminous embolism, chill. Symptoms: Anamnesis, colic, gaseous distension, stupor, death, diagnosis from spasmodic colic. Course: Fatal in two hours, or more. Recovery. Lesions: Distension of bowels with carburetted hydrogen, carbon dioxide, and nitrogen, redness of intestinal mucosa, anæmia of abdominal organs, congestion of cutaneous and surface vessels. Treatment: Stimulants, antiseptics, enemata, chloral hydrate, puncture, eserine, pilocarpin, friction, massage, exercise, dieting, bitters.

Definition. A gaseous overdistension of the intestines, from fermentations in the ingesta, but also in part from air that has been swallowed, and from carbon dioxide exhaled from the blood circulating in the intestinal mucosa.

Causes. These are to a large extent the same as those of gastric tympany. General and digestive debility resulting from former disease, from spare diet, from unsuitable or indigestible food, from anæmia, from parasites, from hemorrhages, is a potent predisposing cause.

Weakness of the alimentary canal from catarrh, or other persistent disease, from impaired innervation, from embolism of the vessels and imperfect circulation also predisposes, or again the lack of vermicular movement and of the mingling of the digestive fluids with the food, leaving the latter in a specially fermentescible condition. As direct *exciting causes* may be named:

Very fermentescible food in excess, such as the leguminous products (beans, peas, vetches, cowpea, alfalfa, sainfoin, clover) in their green condition. These contain an excess of protein compounds, which should be mainly digested in the stomach, and if passed rapidly in large quantity into the intestines, they fail to be sufficiently acted on by the trypsin, and are specially liable to fermentation. Very rapidly grown and aqueous grasses are similarly liable to decomposition.

New grain is specially liable to fermentation the more so that it sometimes contains a paralyzing agent, which acts like intoxicating ryegrass. The ripening seeds of many forage plants often act in this way, (annual and perennial ryegrass, millet, Hungarian grass, chick vetch, vetches generally). The same has been observed of the leaves of growing maize, grapevine leaves, and potato plants.

Musty and spoiled fodders of all kinds are very dangerous, the toxic principles of the fungi and bacterial ferments paralyzing the sympathetic nervous filaments.

Fodders that are *imperfectly masticated and insalivated* owing to defective teeth or diseased jaws or glands are liable to prove hurtful in a similar way.

A full drink of water and especially of ice cold water *after a feed of grain* is one of the most potent factors. The stomach and intestines are both roused to violent peristaltic action, the undigested food is washed on into the bowels, and too often the action of the cold induces congestion and partial paresis, and exposes the undigested mass to the uncontrolled action of ferments.

Circulatory troubles caused by verminous embolism (see intestinal congestion) is another very prolific factor. *A sudden chill* in an animal that is perspiring and fatigued may precipitate an attack, by causing a retrocession of blood from the skin to the intestines, with resulting paresis of their coats.

Symptoms. The condition is usually complicated with gastric tympany, so that we have a complication of symptoms. The history of the case is often diagnostic, showing one of the above mentioned causes, and above all a full drink after a feed of grain, speedily followed by abdominal pain, gaseous distension of the abdomen, causing death in two hours or upward. The distension of the abdomen usually shows more on the right than the left, and the resonance on percussion is greater. Colics are usually less violent than in intestinal congestion, and the actions of the animal

are less precipitate or disorderly. He may lie down, roll and rise, but the constant restless movement, the sitting on the haunches, and the frequent agonized turning of the nose toward the flank are rarely shown. The animal is rather dull and prostrate, passing finally into a stupor, the face is pinched and anxious, the back arched, the head pendent, the walk slow and unsteady, and respiration and pulse accelerated. There is no complete intermission of pain, though it is more acute at one time than another, for some time there is rumbling in the bowels followed by complete silence, as they are fully paralyzed, fæces may be passed at first, but this ceases as the floating colon is emptied and the gaseous distension becomes extreme, and urination which may take place in the early stages is no longer effected in the advanced ones. The compression of the thorax causes severe dyspnoea, accompanied, when the patient is down, by a slight groan.

Course. In the advanced stages the animal may sink to the ground oppressed by the shock, poisoned by the carbon dioxide which can no longer be exhaled through the lungs, and by hydrogen sulphide and other toxic products of intestinal fermentation. Death may follow two hours after a hearty meal and is rarely long delayed in fatal cases.

Improvement may be recognized by the termination of the paresis, the lessening of the abdominal tension, the return of the rumbling in the abdomen, the passage of fæces and flatus, and of urine and by general relief.

Lesions. In case of death the overdistension of the intestines and abdomen is the most marked lesion, the composition of the gas varying with the nature of the ingesta and the duration of the illness. Carburetted hydrogen compounds abound as a rule in the early stages, while carbon dioxide predominates later. Pinner found 49 per cent. of carburetted hydrogen, 8 per cent. of carbon dioxide, and 42 per cent. of nitrogen.

The contents of the large intestines are usually in considerable amount and in an undigested condition. The walls of the distended bowels are greatly attenuated and may show congestion, petechiæ, or rupture. Rupture of the diaphragm is not uncommon. Congestion of the lungs, but especially of the skin and superficial structures of the body, and of the brain are natural results of the expulsion of blood from the abdominal cavity.

Treatment. The desiderata are: relief from existing gaseous tension; arrest of further fermentation; and the restoration of the vermicular movement of the intestine.

The two first indications may sometimes be successfully met by stimulants and antiseptics. Formerly, mild cases were successfully treated by oil of peppermint and oil of turpentine in oil, and a free use of enemata. A more modern resort is a large (virtually soporific) dose of chloral hydrate (1 oz.) given in solution. This is at once a powerful antiferment and an antispasmodic. It is moreover highly volatile and in the heat of the stomach is readily passed on into the duodenum and absorbed. Employed early it not only checks the production of gas, but it relaxes the whole intestinal tract and allows the free passage of accumulated gas which passes off rapidly per anum.

But in severe cases the gaseous distension is too great to hope for relief by such measures and puncture of the cæcum or double colon is the only hopeful resort. This is made with a small trochar and cannula not more than $\frac{1}{4}$ inch in bore, which is inserted at the point of greatest resonance. The point usually advised, as in the ox, is the centre of the space circumscribed by the last rib, the ilium and the transverse processes of the lumbar vertebræ. A better plan is to percuss and puncture the point where the drumlike resonance is greatest. The higher the puncture the more promising as the cannula is less likely to be blocked by the ingesta which accumulates in the lower part of each viscus. The cannula may be left in place for some time to keep the bowel flaccid and allow time for the restoration of its contractile functions. The cannula may be utilized to inject antiferments (chloral) and peristaltic stimulants (eserine, pilocarpin, barium chloride). In cases in which puncture is not imperative these agents may be used hypodermically, eserine $1\frac{1}{2}$ gr., pilocarpin 2 grs. or barium chloride 7 grs.

Enemata of soapsuds, with or without stimulants prove effective in emptying the floating colon and soliciting the action of the large intestines generally and the passage of flatus. Friction of the abdomen and walking exercise are desirable. After recovery a restricted diet, laxatives and bitters serve to restore the lost tone of the alimentary canal.

ACUTE INTESTINAL INDIGESTION WITH IMPACTION OF THE LARGE INTESTINES IN THE HORSE.

Definition. Causes: dessication of ingesta in colon, sacculaton, constriction at pelvic flexure, debility, ill health, local peristalsis, diseased teeth, jaws or salivary apparatus, excess of food, heating grain, hard fibrous indigestible fodder, green leguminosæ, privation of water, inactivity, verminous aneurisms, tumors, strictures, obstructions. Symptoms: colics after meals, becoming more severe, tension and firmness of right side, sitting on haunches, stretching, small, hard, dry coated stools, obstruction felt on rectal exploration, frequent attempts to urinate, tympany. Course: six hours to six days or more, signs of aggravation and improvement. Lesions: large intestines tympanitic, impaction often at pelvic flexure, or other constriction, adherent mucosa has thick mucus or blood, is discolored, friable, necrosis, perforation, liquid contents of distended bowel in front, rupture, invagination, volvulus. Treatment: laxative diet, injections, aloes, pilocarpin, eserine, barium chloride, chloral hydrate, morphia, henbane, belladonna, puncture, cold or oleaginous enemata, empty or knead rectum and colon, cold compress, electricity, friction, laparotomy.

Definition. This is an impaction and obstruction of the colon, and usually of the pelvic flexure with dried and badly digested alimentary matters.

Causes. Certain anatomical and physiological conditions contribute to this disease in the horse. The ingesta as it leaves the stomach is liquid or pultaceous and throughout the small intestines it remains so, so that they are little liable to impaction. But by the time the cæcum is reached much of the liquid has been absorbed, and as the contents pass into the double colon they are usually a soft solid, which gradually becomes dried as it advances through the double and floating colon. The sacculaton of cæcum and colon tends to delay the masses and favors absorption. The pelvic flexure, the narrowest part of the double colon, is formed by an acute bend of the viscus on itself so that the dried masses advancing from in front are especially liable to become arrested and impacted at this point. Impaction may, however, occur at any part of the large intestine.

Any debility or atony of the intestines predisposes to the condition. The ingesta accumulates in the portion which does not contract sufficiently to pass it onward, and this soon becomes distended to a state of absolute paresis. All conditions of debility, and all prolonged ill health tend to operate in this way by lessening vermicular movement.

Again in cases of nausea or intestinal disorder the supervention of antiperistaltic movements, will tend to accumulate the ingesta at one point and favor impaction (Ernst).

As in the case of other indigestions the imperfect preparation of the food is an active factor. Diseased teeth, jaws or salivary glands, act in this way and a functionally weak stomach contributes to this as to other intestinal disorders.

An excess of food and especially indigestible food will contribute to impaction. Heating grain, like corn, wheat, buckwheat, passed rapidly through the stomach in an imperfectly digested condition, tends to accumulate in the larger intestines. Hard, fibrous fodders like hay and straw that have run to seed, or which have been washed out by rains, bleached or heated, rye-straw, the stalks of beans, peas, vetches, which have been similarly spoiled, and clover hay affected with cryptogams or other ferments act in the same way. Even clover eaten green, produces in foals impactions to which the hairs of the leaves and chalice materially contribute (Verrier). The allied plants alfalfa and sainfoin when passed rapidly through the stomach tend to impaction of the large intestines. But any fibrous, indigestible and innutritious fodder, taken in excess to make up the deficiency of nutriment is liable to act in this way.

Other conditions that contribute to impaction are lack of water, especially at night when much hay is consumed, and lack of exercise which tends to torpor of both liver and bowels.

Finally verminous aneurisms and embolism of the intestinal arteries induce congestion, paresis, spasms, and other disorders which tend to aggregation and impaction. Also tumors, strictures and obstructions of all kinds tend to impaction.

Symptoms. A certain amount of impaction is not incompatible with ordinary health, but as this increases all grades of colic may be met with from the most simple and transient to the most persistent and severe.

In the milder forms slight and transient colics come on after meals, for days in succession, before any serious attack is sustained. These are especially marked under dry bulky fodder (hay), less so on grain, and less on green food or roots. The animal paws, moves the hind limbs uneasily, looks at the flanks, he may even kick at the abdomen, lie down and roll, rise, pass a little manure or flatus, and seeming relieved may resume feeding, until the next attack. The intermissions may last a few minutes, a quarter of an hour or longer, and they gradually become more prolonged until they disappear for the time. Sooner or later, however, the obstruction becomes more complete and the colic more severe and persistent. To the ordinary symptoms of violent abdominal pain there are added symptoms which point to bowel impaction or obstruction. There is a special tension of the right side of the abdomen, with flatness on percussion. When down there is a tendency to sit on the haunches to relieve pressure on the diaphragm and lungs. When standing there is a disposition to stretch the fore limbs out forward and the hind ones backward. Fæces may be passed at first in a few small round balls at a time, but this soon ceases, and very little or none can be obtained even by the use of enemas. The straining is usually so violent as to expel the enemata as soon as introduced. The hand introduced into the rectum can easily detect the solid impacted pelvic flexure of the colon pressing backward into the pelvis or impinging on the right pubis. Another common symptom is the frequent passage of urine in dribblets, due to the irritation of the bladder by the pressure upon it of the impacted colon during straining. In cases of this kind the colon and cæcum become tympanitic as first shown by a resonant distension of the right flank obliterating the hollow in front of the ilium, and later by a similar condition of the left flank.

The abdominal pain is usually less acute than in simple spasmodic colic or intestinal congestion. The face is less pinched and anxious, the eye less frightened, the kicking at the belly less violent, and the lying down more deliberate and careful. Very commonly the patient merely rests on his belly or side without attempting to roll.

Course. The disease may last six to twelve hours, or even as many days before it ends in recovery or death. The colicky symptoms usually increase, with the complication of dyspnoea when tympany becomes well marked, hyperthermia in case of the supervention of enteritis, and signs of general peritonitis and collapse in case of rupture of the bowel. A sudden increase of the pain may otherwise indicate the occurrence of invagination.

As indicating a favorable termination there may be restoration of the rumbling, the passage of fæces at first perhaps in the form of solid cylindroid masses, and later as a mixture of broken up ingesta, liquid and gas, the tension of the abdomen disappears, the pains lessen and cease, and there is a gradual restoration to health.

Lesions. The abdominal walls are tense and more or less drumlike, and when these are cut through the large intestines protrude strongly. When punctured there is a free discharge of gas. The most common seat of obstruction is the pelvic flexure, but it may occur in the floating colon, or rectum, in the double colon even at other parts than its pelvic flexure, in the cæcum or in the ilio-cæcal opening. The impacted mass is firm, rather dry, covered with mucus and sometimes blood, and manifestly only partially digested. Its size and form vary greatly as it is moulded into the affected viscus. The mucosa in contact with the impacted mass is covered with a thick layer of viscid mucus sometimes streaked with blood. The mucosa itself is congested, thickened, friable, and marked with spots or patches of various colors (white, gray, green,) indicating commencing necrosis. In old standing cases this may extend to the other coats of the bowel determining perforation or laceration.

The portion of the bowel immediately in front of the obstruction is filled with liquid which has been forced down upon the barrier by the active peristaltic movements, and the distension by liquid and gas may have increased until rupture has ensued with the escape of the contents into the peritoneal cavity. Invagination, volvulus and peritonitis are common.

Treatment. This will vary according to the stage and degree of the illness. In slight cases with transient colics only after meals, a more laxative diet may suffice. Boiled flaxseed, roots, potatoes,

apples, green cornstalks, silage, or even sloppy bran mashes, with an abundance of good water and active exercise may prove efficient. Copious injections of warm water, soapsuds, or linseed oil emulsion may be added.

In the more violent cases we must resort to more active measures and yet drastic purgatives are full of danger. The free secretion from the vascular small intestines and the active vermicular movements, lead to the speedy overdilatation of the bowel just in front of the obstruction, the current being strong and active all around the contracting gut in contact with the mucosa, while a weaker return current sets in in the centre, but is effectually checked and arrested at no great distance in front of the impaction by the strong backward peripheral stream. If therefore the impaction is not broken up, it is inevitable that the gut above must be more and more distended until a rupture ensues.

Yet in a certain number of cases a moderate dose of aloes or castor oil supplemented by frequent enemata and other measures, succeeds in safely overcoming the obstruction. The solid impacted mass is gradually softened and removed, and finally after perhaps three or four days of complete obstruction the fæces begin to pass and recovery ensues.

With or without the aloes, the hypodermic use of pilocarpin or eserine or both will often succeed in obtaining successful peristalsis. Barium chloride while inducing more active peristalsis is, on that account, somewhat more dangerous.

Pain may be moderated and fermentation checked by chloral hydrate ($\frac{1}{2}$ oz.), or, an anodyne, morphia (2–4 grs.), may be given hypodermically. In the absence of these, extract of hyoscyamus or belladonna (2 drs.) may be given by the mouth, and repeated as may be necessary. If tympany is dangerous use the trochar and cannula. Enemata and other accessory measures must not be neglected.

W. Williams has resorted to rectal injections of 2 oz. aloes forced into the rectum by a syringe furnished with a long elastic tube, and repeated when expelled. Brusasco has used copious liquid injections poured into a rectal tube the end of which is raised at least ten feet above the croup, so as to gain the requisite force. Schadrin uses injections of cold water to stimulate the bowels to contractions. Injections of oils or mucilaginous matters when they can be carried far enough lubricate the walls and favor the passage of solid matters. Castor oil which acts to a large extent locally is especially applicable.

Mechanical applications are often valuable. If the obstruction is lodged in the rectum or floating colon it can usually be reached by the oiled hand and carefully extracted. If this is not successful, impactions in the floating colon or pelvic flexure may still be to a large extent broken up and loosened by the knuckles of the oiled hand in the rectum. I have often resorted to this with excellent effect. Impactions in the cæcum or elsewhere in the double colon are however inaccessible for such treatment. For these, external measures are available. Wilhelm wraps the abdomen in cold compresses. Causse and Lafosse recommend the electric current. Friction to the skin of the abdomen is a common resort. Rudofsky turns the animal on his side or his back, to remove the weight of the small intestines from the impacted cæcum or colon, and favor the exit by gravitation of the contents from the cæcum into the colon. Kneading of the abdomen with the fists or knee when in this recumbent position may also be resorted to.

As a *dernier resort*, Gaullet performed laparotomy but with no success as the animal died the following day. The horse was, however, in extremis at the time of the operation and a portion of the intestinal wall was blackish and gangrenous. To be successful such an operation should be practiced before there is any probability of gangrene, and while the patient is still in good condition for recuperation. But these are just the cases in which success is to be hoped for from less dangerous measures. Again the conditions for its success are best in case of obstruction of the pelvic flexure, as that could easily be drawn out through a spacious abdominal wound, incised, emptied and sutured with careful antiseptic precautions, and with little risk of infection of the peritoneum. But this is just the point where an obstruction can be efficiently dealt with in a less dangerous way, by kneading through the rectum for example. The operation, however, is not one to be utterly condemned, but in any case in which it is certain that the obstruction is otherwise irremediable, it should be adopted at as early a stage as possible, under anæsthesia, and with antiseptic

precautions. The after treatment would consist in a restricted diet of milk or gruel with antiseptics to prevent fermentation and bloating.

IMPACTION OF THE COLON IN RUMINANTS.

Causes: Debility, hard, fibrous food, dry winter feeding, privation of water, astringents, smut, ergot. Symptoms: Hard, moulded, coated dung, blood-streaked, and in small quantity, tympany, dullness, debility, splashing sound when right flank is pressed, rectal exploration. Treatment: Laxative food, water, salt, strychnia, eserine, barium chloride, enemata, oils.

This is not a common affection in cattle, yet it does occur in weak and debilitated conditions, and in animals fed on fibrous and innutritious aliments. The ingesta are delayed in the gut, their liquid portion absorbed and the remainder accumulates in a hard mass, which distends and weakens the bowel. Dry winter feeding, with a scarcity of water strongly contributes to its production. Astringent plants in the hay, or smut or ergot may add to the tendency.

Symptoms. The fæces are hard and firm, glazed on the surface, coated with mucus and sometimes stained with blood. They are passed in small quantity and with much effort and straining, and finally the bowels become completely blocked, nothing whatever being passed. Tympany of the rumen now appears, especially after feeding, appetite and rumination fail, there is much dullness, debility, and loss of flesh and unless relieved, the animal dies in marasmus. Pressure on the right side of the abdomen made suddenly and forcibly produces a sound of liquid splashing in an air space, derived apparently from the accumulation above the obstruction. The oiled hand introduced into the rectum may feel the solid impaction, but in any case causes pain and moaning when the seat of the impaction is pressed upon.

Treatment. In the milder cases and earlier stages a change to sloppy food, green food, or boiled flaxseed, with plenty of salt, free access to water, and scruple doses of nux vomica may prove successful.

In the more advanced conditions with complete obstruction, give $\frac{1}{2}$ lb. to 1 lb. each of sodium chloride, and sodic sulphate, and $\frac{1}{2}$ dr. nux vomica, inject hypodermically 3 grs. eserine or 7 grains barium chloride, give water *ad libitum*, and frequent and large injections of soapsuds. If these latter are given cold they will still further stimulate the missing peristalsis. In obstinate cases a second dose of the salts, or 1 quart of castor oil may be given with the addition of 20 drops of croton oil.

When relief has been obtained, a laxative and nutritive diet and a course of bitters should follow.

INDIGESTION WITH OBSTRUCTION OF THE COLON IN SWINE.

Causes: green leguminosæ, dew, rain, dry indigestible food, lack of water or exercise, debility, torpid liver. Symptoms: firm, small, coated stools, obstruction, straining, tympany, rumbling, vomiting, anorexia, lies on belly, secludes himself, restless, grinding teeth, diarrhœa. Diagnosis from hog cholera. Treatment: laxatives, enemata, antiferments, rubbing, massage, mechanical unloading of rectum, puncture, dieting, bitters.

Causes. The leguminosæ in their green state are liable to produce indigestion and flatulence in the pig. If covered by dew or rain this tendency is increased. Dry, fibrous or indigestible food with privation of water and of exercise tends to intestinal impaction. Debility from any cause, by weakening the contractility and secretory power of the bowel strongly predisposes to this condition. Torpid liver with diminished secretion of bile is another common factor.

Symptoms. The defecations are infrequent, and small, and covered by a mucus film on a glazed surface. This increases steadily until they cease altogether, when straining, tympany, rumbling and vomiting follow. The animal refuses food, and lies on its belly, hiding under the straw when that is available. Restlessness with frequent change of place and grinding of the teeth are noticed. A spontaneous cure may take place by a free secretion of liquid in which the impacted mass is loosened, disintegrated and floated off, the costiveness being succeeded by diarrhœa. Once established this diarrhœa may become persistent, causing serious loss of condition, and simulating hog cholera. It may be distinguished by the fact that it occurred without the introduction of a *contagium*, is easily accounted for by the nature of the food and is not communicated to adjacent herds treated in a different way. There is also the absence of the petechiæ on the skin, and, on post mortem, of the specific round necrotic intestinal ulcers of hog cholera.

Treatment. The first object is to rid the intestines of the irritating impacted masses, and this may be secured by giving 1 oz. castor oil, 2 drs. jalap, or 3 grs. croton farina to a 150 lb. pig. This may be seconded by frequent and copious injections of soapsuds. If fermentation and tympany are troublesome 30 grs. chloral hydrate may be given and repeated as circumstances demand it. Active rubbing of the abdomen or kneading of the same will prove useful. If tympany becomes dangerous the gas may be safely evacuated by trochar and cannula, the point of puncture being selected by the clearness of the resonance. When the impaction has reached the rectum and prevents the use of enemata, it may be extracted with the oiled fore finger. An injection of sweet oil may then be given and the finger may be used again and again as the impacted fæces come within reach. When diarrhœa has set in it may be checked by doses of 30 to 60 drops of laudanum, and a diet of boiled milk, well boiled flaxseed or other starchy gruel or mush. A course of bitters with chalk, bismuth or antiseptics will prove serviceable during convalescence.

INTESTINAL INDIGESTION IN THE DOG WITH CONSTIPATION.

Usual seat. Causes: house life, neglect of call to defecate, lack of exercise, overdistension, atony, watch dogs, over feeding, obesity, ill health, debility, loss of teeth, paraplegia, spiced and sweet food, matting of hair over anus, tumors round anus. Symptoms: small, hard, white, glazed stools, straining, no stools, hot, tender, swollen, bulging anus, abdominal manipulation, dullness, laziness, seeking seclusion, colics, tender abdomen, stiffness, arched back, drooping head and tail, vomiting—sometimes feculent, fever. Lesions: impacted mass of hard, gritty particles, catarrhal congested or necrotic mucosa, and outer coats, perforating ulcers. Treatment: air, exercise, laxative diet, mechanical extraction, purgatives, enemata, demulcents, laparotomy, enterrectomy.

In the dog, atony and impaction are common especially in the rectum, where the fæces are unduly retained in connection with house life until accumulated and dried. The impaction tends to extension forward, the new material adding continually to the old, and the overdistended rectum becoming more and more atonic in proportion to the increase of the distension.

Causes. The most prominent factor is denial of nature's call to defecate, on the part of house dogs trained to habits of cleanliness. The accumulated mass distends and weakens the rectum, enabling it to hold more without suffering, making the call of nature less imperious, and diminishing the power of expulsion. Lack of exercise usually operates in the same animals, as it also does in watch dogs, the movements of which are limited by the length of their chains.

Overfeeding contributes, in various ways, by increasing the amount of feculent matter passed on into the rectum, by hastening the food through stomach and small intestine imperfectly digested and therefore in a more irritating condition, and by contributing to obesity and lack of tone.

In mastiffs, hounds, bull-dogs, etc., which are naturally gluttonous and swallow animal food in large masses without tearing apart, or mastication, portions pass into the intestine undigested and tend to disturb and block the terminal bowel.

Dogs that are out of health, and which lack tone in general have usually torpid bowels and suffer from delay and impaction of contents. Hence all chronic and debilitating diseases are liable to become aggravated by this troublesome complication.

Old dogs with the teeth worn out and the general tone of the stomach and intestines low are habitual sufferers.

Paralysis of the posterior limbs is usually associated with paresis of the rectum and accumulation, and various other atonic nervous disorders act in the same way.

The sympathy between the skin and alimentary tract shows itself in common disorders, indigestions and catarrh becoming complicated by skin eruptions acute and chronic, by indigestions and impactions. To both conditions the nature of the food of house dogs largely contributes, the habit of eating abundantly at each meal time of the master, the consumption of highly spiced meats, of sweet cakes, pastry and puddings, and even the exclusive diet of white bread or farinaceous and starchy aliment.

In long haired dogs, the matting of these hairs together across the anus proves a factor by rendering defecation difficult and painful.

The formation of tumors around the anus, or inflammation and swelling of the anal glands are additional causes.

Symptoms. As in other animals this condition may be chronic, lasting for a long time without leading to complete obstruction. The fæces are passed in hard foetid, whitish masses, often partly divided, in pellets, dry, and polished on the surface and covered with a film of mucus, sometimes blood. They are passed at considerable intervals, slowly and with painful effort and straining.

In the more advanced and violent cases defecation becomes absolutely impossible, though the animal strains violently and frequently. The anus and rectum bulge as a rounded swelling and the congested and bleeding mucous membrane may be exposed, but nothing is passed. The anus is hot and tender to the touch and the anal glands swollen. Manipulation of the abdomen between the two hands can detect the impacted gut extended forward for a considerable distance, and even implicating the colon. The oiled finger in the rectum may detect the impacted matter as a

conglomerate mass of gritty materials. If the appetite continues this becomes all the more extensive. The affected animal is dull, prostrate and indisposed to exertion, seeking a dark retired place where he can rest undisturbed curled up into a ball. Sometimes he starts with a sharp yelp. Especially does he shrink and complain when the belly is handled. If made to walk, he does so stiffly, hangs head and tail, arches the back and tucks up the belly. The face and eyes express severe suffering. Vomiting is a frequent complication, the rejected matters being often feculent. Colicky pains are indicated by yelping or moaning, enteritis and peritonitis by hyperthermia and extreme abdominal tenderness. The acute disease may last from one to two weeks, and death may be preceded by auto-infection, by nervous symptoms of various kinds or by enteritis or peritonitis.

Lesions. These may be stated shortly as impaction, catarrh or necrotic changes of the mucosa, more or less extensive inflammations of the bowels and peritoneum, perforations, and congestions of the liver and kidneys.

Treatment. In the milder cases plenty of open air exercise and a laxative diet may succeed. In the more severe cases it is usually requisite to unload the rectum mechanically. The dog is laid on a table, and the oiled finger introduced through the anus, lubricates the mucosa as far as it can be reached. Then little by little the firm mass may be disintegrated and removed being steadied by the other hand applied on the abdomen. The handle of a teaspoon or a special spoonshaped curette may at times replace the finger to advantage, but must be used with due judgment, in view of the thinness and friability of the walls of the gut.

When the gut has been emptied in this way, or in the less severe cases without this preliminary, purgatives and frequent injections can be used to advantage. Jalap $\frac{1}{2}$ dr. and calomel 5 grains, or castor oil $\frac{1}{2}$ oz., or syrup of buckthorn have been usually employed. The impaction is usually too firm for the transient action of eserine or pilocarpin. As injections, castor oil, soapsuds, decoctions of flaxseed, mallow or elm bark may be employed being repeated as often as they are expelled and supplemented by the mechanical removal of all solid matters that come within reach.

In cases so extensive as to resist the above measures we can resort to laparotomy. The incision can be made close and parallel to the linea alba, the rectum, or floating colon drawn out through the wound, the other intestines being carefully held back by an assistant, the gut is then incised longitudinally and its solid contents removed. The wound is thoroughly cleansed, washed with an antiseptic (mercuric chloride 1:2000), and sutured with catgut, the mucosa being carefully turned in and the muscular and peritoneal coats kept in accurate contact. Finally the abdominal wound is closed by silk sutures. The patient must be placed for a week or ten days on well boiled gruels and the rectum frequently emptied by injections of tepid water.

In case the bowel is found to be necrotic, the gangrenous section may be excised and the ends brought together by Murphy's button, or simply sutured with catgut over a hollow tube of raw potato.

INTESTINAL INDIGESTION AND OBSTRUCTION IN BIRDS.

Causes: Age, debility, atony, matting of feathers, dry or indigestible food, lack of water, diseased oviducts, sand or gravel, lack of pebbles or power in gizzard. Lesions: masses of egg, uric acid, or fæces in cloaca, implicating colon and cæca. Symptoms: dullness, stupor, vertigo, staggering, erect plumage, trailing wings and tail, bulging anus, covered with matted feathers, impaction felt by finger. Treatment: extract mass, castor oil, laudanum, chalk, bismuth, pepper, demulcents, phenol, exercise, silage, green food, pebbles.

Causes. These resemble those already noted for the dog. Old age, debility, and atony of the bowel, the matting together of feathers across the anus, dry feeding, indigestible food, scarcity of water, and lack of exercise are especially to be noted. Malformations or other changes lead to obstruction of the cloaca, and of defecation. Sand and gravel passing from an atonic gizzard accumulate in the small intestine or in the cæca distending them to great excess. Imperfect trituration in the gizzard, from lack of pebbles, may prove a factor in stoneless prairies.

Lesions. The most common seat of obstruction is at the cloaca, and the impacted matter may be yellow partaking of the nature of yolk of egg, or it may consist of feculent matters and uric acid in various proportions, white, hard and fœtid. As in the dog this distension may be continued forward blocking the colon and cæca as well. Lucet mentions a case in which the impacted mass measured seven inches long, and eight in circumference at its posterior and larger end.

Symptoms. The bird is dull, sluggish, stupid, giddy or unsteady on its limbs, with feathers erect, wings, tail and head pendent and loses flesh rapidly. Often a felted mass of feathers and fæces cover the anus. In its absence there appears the rounded swelling or on manipulation the impacted cloaca or rectum can be felt firm and resistant.

Treatment. Soften and remove the external mass of fæces by the aid of tepid water, clip off the feathers, which would tend to restore it, then by the oiled finger and warm water injections break up and extract the contents of cloaca and rectum. If impaction remains farther forward give a teaspoonful of castor oil. If diarrhœa has already set in, give 5 drops laudanum, and mix chalk or bismuth and pepper in a mush to be fed to the patient. Injections of slippery elm containing a teaspoonful of carbolic acid in the pint will prove useful.

The bird should be allowed plenty of exercise, its grain being fed on a floor covered lightly with straw to encourage scratching, and silage or green food should be allowed. On the prairies where pebbles cannot be secured, imported gravel or vitrified brick broken into small pieces should be allowed.

COLIC IN SOLIPEDS FROM VERMINOUS EMBOLISM. INTESTINAL CONGESTION.

Definition. Causes: presence of sclerostoma in arteries, form, habit, nature, immature, biology, life in bowel, in submucosa, in arteries, outside the mammal, pathogenesis, blood-sucking, verminous cysts, verminous aneurisms, seats of latter, coagula, embolism, stagnation of blood, œdema and thickening of intestine, mesentery, fermentations, tympany, infective inflammations, blood extravasations, infection of liver and spleen. Symptoms: sudden attack, violent colics, reckless movements, frequent defecation followed by its arrest, palsy of peristaltic movement, of pain, prostration. Course: two to twenty-four hours, death from indigestion, tympany, obstruction, hemorrhage, poisoning, recovery, sequelæ, laminitis, intestinal catarrh or atony, debility. Treatment: aneurism worms beyond reach, treat lesions, venesection, anodynes, stimulants of peristalsis, antiseptics, compresses, sinapisms. Prevention: expel intestinal worms, exclude embryos, tartar emetic, iron sulphate, arsenic, phenol, pure water, occasional vermifuges.

Definition. Congestion and spasms of the intestines in connection with blocking (thrombus or embolism) of the mesenteric arteries, and verminous aneurism.

Causes. The essential cause is the migration of the sclerostoma equinum (*strongylus armatus*, Rud.) into the mesenteric arteries in its agamous condition. It seems appropriate therefore to here notice the life history of this parasite.

The **sclerostoma equinum** (*strongylus armatus*) is one of the common pin worms of the horse. It is distinguished by its dull gray or reddish brown body, thickest at the cephalic end and tapering off toward the caudal, but ending in a blunt point; by the round, open mouth furnished with several firm chitinous rings, of which the *outer* bears six short symmetrically arranged papillæ, an *intermediate* row of rounded blunt tooth-like projections, and the *innermost* a row of fine, closely aggregated and very sharply pointed teeth for penetration of the mucosa. Male $\frac{3}{4}$ to $1\frac{1}{2}$ inches long, with caudal membranous alæ in two lateral lobes, joined by a rudimentary central lobe: two delicate spicula. Female $\frac{3}{4}$ to 2 inches long, blunt pointed tail, vulva in posterior half of the body. Eggs ovoid with slightly raised ring around the centre: oviparous.

Habitats. They are found in solipeds in two stages of existence, the *mature worms* in the cæcum and colon, and the *immature* in the same organs encapsuled in little pellets of manure, and in cysts in the mucosa but also apart in the arterial system especially in the anterior mesenteric artery and other gastric or intestinal trunks.

The **mature sclerostomata** are found attached to the mucosa of the large intestine into which the head is sunk for the purpose of sucking the blood, and they may be gray, brown or red according to the quantity of blood which they have imbibed. The author has found them in little hernial sacs of the mucosa hanging from the peritoneal surface.

The **sexually immature sclerostomata** are found in little pill-like masses of ingesta in the large intestines and from which they project part of the body through a narrow opening. Another habitat is in cysts of the mucosa of the cæcum and colon and less frequently of the small intestine, individual cysts varying in size from a pin's head to a hazel nut, and containing the young worm rolled upon itself, and varying in size but always less than the intestinal worm and always asexual. In some cases the cyst is found empty but with a small opening toward the lumen of the bowel showing the means of escape of the parasite. A third habitat of the immature worm is in the blood-vessels, especially the posterior aorta and its divisions, and still more constantly the anterior and other mesenteric arteries.

Biology. The ova of the sclerostoma are segmented in the oviduct but are hatched out after they have been laid. The hatching may be effected in the intestine or in manure or water external to the body. When hatched out in the intestine they may pass out at once with the manure or they may envelop themselves in pellets of the finer ingesta and remain for a time in the bowel and finally pass out in this condition. Baillet has traced their development out of the body. In a watery or damp medium they are hatched out in a few days as a cylindroid worm $\frac{1}{4}$ to $\frac{1}{3}$ mm. long, thick in front and with a filiform tail. In moist environment but especially in damp manure they grow to 1 mm. or 1.5 mm. and continue for months in this condition, but remain small and asexual, until taken in, in the drink or green food of the soliped. Reaching the intestine and especially the cæcum and colon they bore their way into the mucosa and encyst themselves, or if they happen to perforate a blood-

vessel they make a habitat of that. In the cyst, development proceeds and when it has reached a certain stage the worm once more bores its way through the mucosa and reaching the intestine becomes sexually mature.

In this last migration the young worm is liable to perforate a blood-vessel in which case it is destined to a period of existence in the blood. It may, however, have blundered upon a blood-vessel at an earlier stage when seeking a temporary home in the mucous membrane, so that the sclerostomata of aneurisms may be derived from two separate sources. In the blood-vessels the parasite attains a length of 1 to 8 lines, whereas in the mucous cysts it does not exceed $3\frac{1}{2}$ lines. Yet Neumann holds that after leaving the blood-vessels they may again encyst themselves in the mucosa before escaping into the intestine.

Several moultings take place in the asexual condition.

Other views have been advanced as to the development of the sclerostomata. Colin believed that the ova deposited in the ducts of the mucous glands and in the perforations made by the parasite in blood-sucking, hatched in this situation and the embryo at once encysted itself in the mucosa.

Leuckart imagines that the embryo found in the fæces or in water outside the body of the soliped, should pass through an intermediate host before it can return to gain sexual maturity in the horse. But no evidence of the existence of such intermediate host is furnished, and the encysted intestinal worms show no indication of a special development which would have been accomplished in such host.

Willach holds to a hermaphrodite stage passed in the intestine of the soliped. He found in the bowel small worms apparently related to the sclerostomata by the appearance of the head and the caudal membrane, but not exceeding three to five lines in length. Some were evidently females and contained not only eggs with soft shells, but in one case embryos. Others had the caudal membrane of the male, yet contained also a few eggs. There is no vulva and the embryos escape by rupture of the oviducts. These embryos he supposes are developed in the same host into the familiar mature sclerostomata.

Whatever may be said of those alleged modes, the first described series of changes and migrations may be taken as the usual and regular method of development.

Pathogenesis. *Lesions.* These embrace perforations of the mucosa, cysts, aneurisms, embolisms and congestions.

Irritation of the mucosa. The adult worms, like so many leeches are continually biting and sucking blood from the mucosa and when present in large numbers, hundreds, thousands, or a million create an aggregate of irritation which may determine violent indigestions and congestions.

Verminous Cysts. These are like a pin's head, a pea or hazel nut, containing the asexual worm in a mass of purulent debris, or if empty, presenting a small orifice where it made its exit.

Verminous Aneurisms. These are perhaps the most important lesions caused by the sclerostome as they are the steppingstone to the dangerous embolisms, and too often fatal colics and congestions of the intestines. They are very common in some localities, and rare in others following the distribution of the sclerostomata. Bollinger found them in 90 to 94 per cent. of adult horses, and Ellenberger in 84 out of 85 horses dissected. They are found in all ages from six months up, and are nearly always in the short, stubby trunk of the anterior mesenteric artery. Often two or three exist in the same animal, the whole length of the posterior aorta showing patches of disease, exudations, neoplastic elevations alternating with depressions, and aneurisms and thrombosis in its different branches. In 100 horses Bollinger found 168 aneurisms, 153 in the anterior mesenteric, and its divisions, 4 in the cœliac axis and its divisions, 3 in the hepatic artery, 3 in the posterior mesenteric artery, 3 in the renal arteries and 2 in the posterior aorta.

The special predisposition of the anterior mesenteric artery is variously accounted for: 1st. There is the obvious fact that its branches are distributed to the cæcum and double colon, the home of the mature parasite, and to the small intestines which are first reached by the young parasites that are taken in with the water and the food. These are therefore most likely to get into the branches of this vessel and to follow them up toward its origin. 2nd. The anterior mesenteric artery distributes its branches to the small intestines the most motile portion of the intestinal tract, and the cæcum and

colon the most heavily loaded with solid ingesta, it is therefore the most subject to traction, and distensions, and the more so that the parent trunk is extremely short and the divisions pass in all directions and to a large extent at right angles, so that there is a dragging of the walls apart as well as an obstruction to the blood flow and an increase of internal tension. The distension, laceration, inflammation and softening of the internal coat have accordingly been regarded as the starting point of an endarteritis upon which the parasites have been implanted as a further cause of trouble. We must not forget, however, that the sharp circle of teeth of the parasite, by which it fixes itself on the intima of the vessel are quite enough to produce initial endarteritis, without any assistance from distension, traction or laceration.

The irritation of the intima from whatever cause determines here as elsewhere exudation, and coagulation, and the inflamed walls losing their tone yield more and more readily to the internal tension. Sometimes the coagulum lines the aneurism or vessel all round, leaving a narrow central passage through which the blood still flows; in other cases the clot extends into the adjacent smaller vessels, completely blocking them and disturbing circulation and innervation in the parts which they supply. As a rule the parasites are found in galleries hollowed out in the clot, and heads or tails may be seen to project into the circulating blood. Sometimes they are found imbedded in the arterial coat, or in an adjacent small abscess. The formation of aneurisms in the other arterial trunks may follow the same method.

Embolisms. These come very naturally from the formation of thrombi in the various arteries. The coagulum determined by the presence of the worms, tends to undergo retrogressive changes notably fatty degeneration, to which germs brought on the worms or in their alimentary canals contribute. This together with the movements of the parasites tends to break up the mass, and minute portions are washed on into the different smaller vessels. Soon these reach divisions which are too small to admit them, which are accordingly occluded and the circulation through them abolished. The presence of microbes as well as fibrine contributes to cause further coagulation, more absolute embolism and arrest of the circulation.

It is further alleged that the sexual instinct in the summer months (May to August) leads the worms to leave the aneurisms, to pass through the smaller divisions to the cæcum or colon where alone full sexual evolution is possible. In these migrations they cause the thrombosis of the smaller trunks and determine the verminous congestions of the bowels which are especially common in these months.

Disturbances of the Intestinal Circulation. As these usually occur in the lines of distribution of the anterior mesenteric artery a knowledge of its divisions and their destination and anastomosis, is essential to an intelligent understanding of the pathogenesis and lesions. As first pointed out by Lecoq the anterior mesenteric artery is divided into three primary bundles: (a) a *left* of 15 to 20 trunks which are destined to the small intestine; (b) a *right* which gives off cæcal branches, one to the double colon, and one to the ilium to anastomose with the last trunk of the left bundle; and (c) an *anterior* which gives one branch to the second division of the double colon and anastomosis with the colic branch of the right bundle at the pelvic flexure; and a second branch to the floating colon to anastomose with the posterior mesenteric artery.

The divisions of the left bundle anastomose so freely with each other in the mesentery and immediately above the intestine that the blocking of any one branch cannot entirely arrest the circulation in the corresponding part of the intestine. It may however produce a partial local stagnation in the vessels of a short loop of intestine, resulting in œdematous infiltration and thickening with resulting induration and stricture of the gut. Chronic and permanent lesions are produced by such blocking, but only rarely acutely fatal ones. Acute and fatal congestive lesions of the small intestine from verminous embolism, occur only when several adjacent divisions of the artery are blocked at once, and this is a rare occurrence.

The right bundle of branches furnishes the only two arteries which are supplied to the cæcum and the only artery furnished to the first half of the double colon. The ileo-cæcal branch is less involved, first, because being less dependent and smaller, it is less likely to receive an embolus, and, second, because any lack of blood supply is counterbalanced by the free anastomosis with the last iliac division of the left bundle. When the embolus blocks the undivided trunk of the right

bundle this same principle comes into play, the free supply of blood from the posterior branch of the left bundle supplying blood through its anastomosis with the iliac and cæcal branches of the right.

But when the emboli are lower down, in the cæcal branches of the right bundle, or in these and the colic branch, arrest of the circulation in the intestinal walls ensues, followed by paresis, passive congestion and hemorrhage. The cæcum and double colon thus become the seats of the grave and fatal lesions of verminous embolism.

The resulting lesions are to be variously accounted for. The stagnation of blood in the vessels below the embolus, determines a speedy exhaustion of its oxygen and increase of its carbon dioxide, so that it is rendered unfit to maintain the normal nutrition and functions of the part, and the capillary and intestinal walls are alike struck with atony or paresis. The blood filters into the stagnant vessels slowly from adjacent anastomosing trunks, and the liquor sauguinis exudes into the substance of the tissues and lumen of the intestine, leaving behind the greater part of the blood globules so that the stagnant blood is rendered more and more abnormal in composition. The walls of the capillaries soon lose their cohesion as well as their contractility, and giving way at different points, allow the escape of blood into the tissues, bowels and peritoneal cavity. It has been further claimed that the emboli already infected and in process of degeneration communicate this to the walls of the vessels and to the stagnant blood, hastening the process of degeneration and rupture.

Another series of circulatory disorders are liable to take place. The blocking of the vessels of the right bundle, tends to increase the blood pressure in the left bundle and the anterior one, and thus to determine congestions, paresis and inflammations in the small intestines, the second division of the double colon and the floating colon. The resulting inflammation and increased vascular tension may lead indirectly to implications of the brain and lung.

Extravasations so extensive as to appear like blood clots may be present between the layers of the mesentery or in the mucosa and submucosa, and blood, liquid or coagulated, may have accumulated in the abdominal cavity. Blood effusion into the intestine gives a dark red coloration to the contents which are further mixed with distinct clots.

The atonic bowels are always the seat of extensive fermentations and tympany. The microbes engaged in these fermentations and their toxins, are accountable for toxic changes occurring in the locally diseased parts and in distant organs. To this may be attributed the congestion and softening of the liver and the engorgements and hemorrhagic centres in the spleen.

Symptoms. An animal, perhaps known to harbor the sclerostoma equinum, is suddenly attacked with violent and persistent colic. He trembles, paws, moves his hind feet uneasily, kicks the abdomen, throws anxious looks at the flanks, crouches, lies down, rolls, gets up, and at once gets down again. The intensity of the suffering rapidly increases, the face is drawn and pinched, the eye is extremely anxious, the patient no longer lies down, but throws himself down reckless of consequences, when down he is not quiet for an instant, but now on his breast, then on his side, then on his back, the limbs struggling and jerked violently, the head turned first to one side and then to the other, he is a picture of extreme agony. If made to walk the same indications continue; he walks with head down and limbs semiflexed ready to drop at any moment, and often he will drop suddenly in spite of every effort to keep him on his feet. The pulse is at first strong and full, but as extensive effusion takes place into the bowels or abdomen, or as the animal is poisoned by toxins, it becomes small, weak, and it may be imperceptible. Breathing is quick and catching, and the mucous membranes are dark red. Sweating which shows first about the elbows or flanks or back of the ears finally becomes general, the surface cold and the limbs especially so. Fæces may be passed at first, a few dry balls at a time from the floating colon or rectum, but soon they are suppressed entirely. Some patients strain frequently to micturate but pass little at a time.

In some instances the acute pain seems to suddenly cease, but there is no general improvement, the patient stands with head depressed, eyes sunken and expressionless, ears lopped, cold perspiration, chilly limbs, unsteady gait and imperceptible pulse. It implies merely a paralysis of the affected bowels in connection with the extensive congestion and extravasation.

Course. Duration. The more acute cases reach their acme with great rapidity, death may occur

after two hours illness, and in other cases it may be delayed ten or even twenty-four hours. It may be caused by indigestion and tympany, by volvulus or invagination, by excessive hemorrhage, or by poisoning with toxic matters.

Recovery occurs when the vessel blocked is an unimportant one as a branch of the left bundle so that circulation may be reestablished from collateral trunks; or when a more important trunk has been but partially blocked, and after a time it either clears itself, or collateral circulation comes in with sufficient compensation. There is a more or less rapid disappearance of the colics and other symptoms, a free passage of urine, the rejection of fæces, it may be in a liquid, semi-liquid or sanguineous condition, yet enough to indicate the restoration of intestinal tone. The patient begins to pick morsels of food and soon acquires his former appetite.

In some instances, however, the recovery is not complete. Trasbot has noted a case of laminitis occurring within fifteen hours after the improvement, and in other cases there remain chronic debility and catarrh of the intestines. The appetite remains poor, there are occasional colics, the bowels are irregular, loose or costive, and the fæces are dry, glossy and covered with mucus. The back is arched, the belly tucked up, strength and vigor are both lacking, and the patient spends much time in the recumbent position.

Complications of various kinds may follow as in other diseases of the intestines. After even the best recoveries, a relapse is always to be apprehended as the original cause remains and the animal is liable to be cut off at any time.

Treatment. This is very unsatisfactory as the original source of trouble, the worms, being in the blood-vessels, cannot be reached by vermifuges that would be harmless to the host, and clots blocking the smaller intestinal vessels, cannot be dissolved and removed. Moreover, although we could compass the death of the worms in the aneurisms, we would leave their dead bodies as sources of septic change, blood coagulation and embolism.

A certain number of cases, however, are not necessarily fatal, and the worms of the blood-vessels have not an indefinite period of life, so that there is some encouragement for both therapeutic and preventive treatment. During the attack we must be content to treat symptoms. French veterinarians still trust largely to general bleeding, adopted at the very outset and to the extent of 6 to 10 quarts. It will temporarily lessen the vascular tension, more permanently dilute the blood, and calm nervous excitement, and in the most violent cases, as a kind of forlorn hope, it might be tried with the view of tiding over the acute stage until a freer collateral circulation could be established.

The use of anodynes will be more generally acceptable to American practitioners. Two to four grains of sulphate of morphia or codeine may be given hypodermically in combination with 1½ gr. eserine, 7 grs. barium chloride, or 2 grs. pilocarpin, to secure a speedy movement of the bowels.

To counteract intestinal fermentation perhaps no better agent can be got than chloral hydrate, ½ oz. of which may be given by the mouth in water, and ½ oz. more by the rectum.

Wet compresses to the abdomen, or fomentations with water rather hotter than the hand can bear or even the application of mustard is sometimes useful as a soothing or derivative agent.

In the absence of morphia or chloral, laudanum, ether, chloroform, camphor or assafoetida have been recommended.

It is important to keep the patient on a soft, littered floor to prevent injury from his throwing himself down, and walking him around may be resorted to for the same purpose.

Prevention. After a non-fatal attack and in every case in which a horse is found to harbor the sclerostoma equinum in quantity, measures should be taken to expel those present in the bowels and to prevent the entry of embryos. The infested horse may be purged and put on two drachms each of tartar emetic and sulphate of iron every morning in a handful of feed half an hour before the first meal. After six doses he may take a second active purgative. In case of need the addition of 6 grains arsenious acid and a drachm of carbolic acid to each dose will render them much more effective. All water must be withheld that comes from streams running by farm-yards, from ponds or open wells in barn-yards, from uncovered cisterns and from any source which receives drainage or leaching from land occupied by solipeds or spread with their manure.

A course of vermifuge medicine should be given at intervals of two or three months to get rid of the worms which have passed in the interval from the cysts of the colon, into the intestine.

NON-VERMINOUS INTESTINAL CONGESTION IN SOLIPEDS.

Causes: sudden changes to green food, or leguminous fodder, newly harvested fodder, frosted food, iced water, microbial infection, toxin poisoning, intestinal fermentations, experiments, volvulus, invagination, strangulation, compression, atony. Symptoms: as in verminous aneurisms. Diagnosis: absence of worms, presence of other causes. Treatment.

Causes. Acute intestinal congestion apart from verminous aneurisms is ascribed to a variety of causes. Sudden changes of food especially to green food, in spring, or to some of the leguminous fodder plants (alfalfa, cowpea, clover, tares, vetches), newly harvested grain or hay, fodders covered with hoarfrost, iced water, and microbial infection or poisoning with toxins or other irritant products of intestinal fermentations. Experimentally the injection into the circulation of pyogenic toxins and putrid matters has determined intestinal congestion and hemorrhage. In the same way musty hay or grain have proved the occasion of these attacks. Finally mechanical blocking of the circulation of the intestine as by volvulus, invagination, strangulated hernia, or even compression by bulky food has seemed to operate in this way.

It ought to be borne in mind that the habitual microbes of the healthy bowel may become pathogenic when brought in contact with a mucosa which is the seat of irritation, atony or any condition of debility.

Symptoms and Lesions. The verminous aneurisms and thrombosis aside, the symptoms and lesions of this form of congestion so closely resemble those of the verminous affection that it seems needless to repeat them.

Diagnosis is difficult but the absence of worms in the affected animals and their fellows, and the presence of some one of the other recognized causes may lead to a fair conclusion.

Treatment of the affection is more hopeful than in the verminous affection, and may be conducted on the same general lines.

PSEUDOMEMBRANOUS (CROUPOUS) ENTERITIS IN SOLIPEDS.

Definition. Causes: As in ordinary enteritis, with added infections or toxins. Symptoms: As in enteritis, nervous symptoms, diarrhoea. Lesions: Congested mucosa, whitish or grayish false membranes, in patches or tubular casts, granular, mucous, albuminoid, fibrinous. Diagnosis: False membranes in stools. Treatment: Glauber salts, calomel, alkaline carbonates or tartrates, oils, antiferments, demulcents, careful diet, bitters.

Definition. An inflammatory affection of the bowels characterized by the ejection with the fæces of false membranes.

Causes. It has been long attributed to the causes which produce other forms of enteritis and indigestions, as youth, rich stimulating feeding, sudden change to green food in spring, sudden chills, over-fatigue, confinement indoors, and prolonged costiveness. In man it is found as a sequel of infectious diseases (pneumonia, pyæmia), in Bright's disease, cirrhosis of the liver and cancer, and in poisoning by lead, mercury or arsenic (Osler). Cadeac, who found great numbers of streptococci in the false membranes in animals, is certain it is a microbial disease, and this is doubtless true, if qualified by the statement that the microbe as is so often the case with other intestinal affections, requires an occasion in the form of a diseased or debilitated condition of the mucosa to enable it to become pathogenic. The disease is not known to propagate itself indefinitely or without such a predisposing occasion.

Symptoms. There are dullness, prostration, langor, hyperthermia, accelerated pulse, and colics which may be slight or very severe. In some cases nervous symptoms have been observed, such as irritability or stupor and somnolence with icterus and fœtid stools. The fæces are usually semi-liquid, implying an excessive liquid secretion as well as the exudation of the membranous matter.

Lesions. There is a pink congestion of the intestinal mucosa more or less generally distributed. Whitish false membranes cover patches chiefly on the terminal portion of the small intestine, but frequently also on the cæcum and colon, covering an especially red and angry mucosa. They may occur as simple patches, as ribbon shaped pieces, or as hollow cylinders lining the entire circumference of the intestine. They appear as if fibrillated, but contain abundance of granular matter and seem to be composed mainly of mucus with albuminoid matter and probably a little fibrine. The deeper layers, in contact with the inflamed surface are soft and gelatinoid. It is alleged that coexisting wounds on other parts of the body become covered by a soft pultaceous false membrane.

Diagnosis is based on the presence of the false membranes of a considerable thickness, so that they can be distinguished from the film of mucus which covers the fæcal balls in constipation or enteric catarrh.

Treatment. Facilitate the secretion from the mucosa, and the separation of the false membrane by giving 1 lb. Glauber salts, or give this agent in doses of 5 or 6 ozs. per day. Calomel 1 dr. may be used instead and has the additional advantage of acting as a disinfectant. The alkaline carbonates or tartrates or even olive or castor oil may be used as substitutes. Antiferments like salol, naphthol, salicylic acid, and salicylate of soda have been prescribed to check the multiplication of the germ. Flaxseed tea, elm bark, and other mucilaginous agents may also be given. An easily digestible and laxative diet and a course of bitters may follow.

PSEUDOMEMBRANOUS (CROUPOUS) ENTERITIS IN CATTLE.

Causes: as in solipeds, youth, overfeeding, plethora, dietetic blunders, temperament, over-exertion, chill when heated, gestation, foul water, irritants, drastics, infections. Symptoms: as in enteritis with false membranes, complications. Duration. Lesions: false membranes, extent, color, structure, composition, congested mucosa. Treatment: Glauber and other salts, pilocarpin, potassium iodide, antiseptics, sulphites, sulphides, borax, bismuth, naphthol, creolin, muriatic acid, bitters.

Causes. The same causes are quoted as in solipeds, youth, extra high condition, rich feeding, sudden change to the green food of spring, climatic vicissitudes of the same season, a sanguineous (Reynal) or lymphatic (Friedberger and Fröhner) temperament, overwork, exhausting travel, suppressed perspiration, gestation, plethora, foul drinking water, special irritant plants (chicory, Huzard), and drastic purgatives. Cadeac suggests bacteria, quoting instances of a fifth or a fourth of a herd suffering at once. The same would come from any other cause acting on the whole herd and it seems probable that a microbial factor is present but can find occasion for its pathogenesis only in given morbid conditions of the mucous membrane. This would explain the failure of the affection to propagate itself like a plague, and at the same time its tendency to manifest itself extensively in given herds with a common predisposing condition.

Symptoms. There are indications of enteric inflammation and fever, rigors, slight hyperthermia, drying up of the milk secretion, impaired or suspended appetite and rumination, constipation, colicky pains, increasing dullness and prostration. As the disease advances the excrements become soft, pultaceous or watery, with floating hard baked pieces, dark and even glistening on the surface and more or less false membranes. These are sometimes stained with blood, which may also be mingled with the liquid debris. As in solipeds these membranes constitute the only true diagnostic symptom. They may appear as shreds, bands or complete cylindroid casts of the intestine.

Other complications, like pseudomembranous exudate on wounds, abortions and profound weakness are sometimes noted. The disease may last eight days before ending in recovery. When death takes place it is about the fourth or sixth day.

Lesions. The false membranes are found on the ilium and colon, in thin films or in thick masses, or tubular casts. In extreme cases the membrane has covered an extent of 24 feet in length, and if recent it is soft and friable. If older it may be firm, consistent and yellow or stained by the blood or ingesta. As in solipeds it shows a reticulated network and a fine granular structure, and is composed mainly of inspissated mucus with albuminoids and fibrine. The exudate covers a surface of extreme redness, with points of darker blood-staining and even abrasion or ulceration. The surrounding mucosa is also congested, the villi hypertrophied, the mucous follicles swollen.

Treatment. In the early stages a laxative of soda sulphate is of especial value in depleting from the inflamed mucosa, liquefying the secretions and dissolving and loosening the false membranes. Epsom salts, cream of tartar, Rochelle salts, calomel, and pilocarpin are more or less valuable substitutes. Iodide of potassium is most valuable in dissolving the exudate and acting as a microbicide (dose 3–4 drs.).

Other alkaline salts may be substituted or as antiseptics the sulphites, hyposulphites, or sulphides of potash or soda. Borax, bismuth, naphthol and creolin have also been recommended. Enemata of warm water are desirable.

In very adynamic conditions, muriatic acid ($\frac{1}{2}$ dr. doses) may be given with vegetable bitters and the same may be allowed during convalescence.

PSEUDOMEMBRANOUS (CROUPOUS) ENTERITIS IN SHEEP.

Causes: As in cattle, draughts in folds, overfeeding. Symptoms: fever, inappetence, weakness of hind parts, diarrhoea, tenesmus, false membranes, blood in stools, tympany. Treatment: change diet of dam, exercise, Glauber salts, potassium iodide, bismuth, flaxseed, elm bark, mallow, gum, carminatives, bitters, antiseptics.

Causes. The same causes are claimed as for cattle. Clavel attributed it to too rich milk, and exposure to cold draughts, in folded lambs.

Symptoms. To the general symptoms of fever are added refusal of the teat, weakness or paresis of the hind limbs, looseness of the bowels and the ejection of false membranes with an unusual amount of straining. The dejections may be watery and mixed with blood. In some cases defecation is suppressed, the intestines being blocked by the membranes, and then acute indigestion and fatal tympany may follow.

The pathological anatomy and lesions resemble those seen in the ox.

Treatment. Change the diet of the ewe, and allow more outdoor exercise. Give the lamb Glauber salts ($\frac{1}{2}$ to 1 oz.) with potassium iodide (10 grs.), and bismuth (1 dr.). Decoctions of flaxseed, or solutions of elm bark, mallow or gum arabic are desirable, and infusions of aromatic plants or oils of peppermint, anise, or fennel may be added with quinia. As in the other animals such antiseptics as salol, naphthol, naphthalin, boric acid, or salicylate of soda may be administered.

PSEUDOMEMBRANOUS (CROUPOUS) ENTERITIS IN DOGS.

Complication of other diseases like distemper. Symptoms: fever, retching, vomiting, tense, tender, tympanitic abdomen, irregular bowels, false membranes. Lesions: stomach empty, congested, croupous exudate, extravasations. Treatment: sodium sulphate, boric acid, sodium salicylate, salol, bismuth, by mouth or enema, strychnia, vermifuges.

In dogs the formation of false membranes on the intestinal mucosa seems to have less of an individual character, and is found associated with other affections, like canine distemper and parasitism. In the absence, however, of accurate knowledge of the specific cause of croupous enteritis in other animals it seems permissible for the present, to arrange the whole in one class characterized by the presence of false membranes.

Symptoms. Along with the general symptoms of fever and the special ones of the existing specific disease there is more or less disturbance of the digestive organs, anorexia, vomiting, tense, tender, perhaps tympanitic abdomen, irregularity of the bowels and the passage of the false membranes. A morose disposition and tendency to snap has been noticed by Röhl.

Lesions. The stomach is empty with red or dark mottled mucosa, the intestinal mucosa is congested covered with a layer of mucopurulent exudate, and at intervals patches of false membranes which are also found in shreds floating in the glairy contents. The exudates are of a yellowish gray color, more or less streaked with blood, and the mucosa infiltrated, swollen, highly congested and with spots of extravasation of blood.

Treatment. Small doses (1 to 2 drachms) of sulphate of soda may be given by the mouth, or boric acid (1 scruple), salicylate of soda (10 grains), salol (5 grains), or bismuth nitrate ($\frac{1}{2}$ drachm). Injections of boric acid, borax, sodium hyposulphite, or even Glauber salts prove useful, and powdered nux vomica (1 grain twice daily) may be added.

In case of intestinal parasites vermifuges must be resorted to.

PSEUDOMEMBRANOUS (CROUPOUS) ENTERITIS IN BIRDS.

In pigeons: *Æ*robie, non-motile bacillus, in lesions, membrane and internal organs, pathogenesis, in chickens pathogenesis differs, also in man, parts attacked, exudate, other symptoms, mortality early and late in outbreak, American form, pathogenesis to rabbits and Guinea-pigs. Prevention: Avoidance of infection, quarantine of birds, separation of sick, disinfection, accidental bearers of infection, pigeons, buzzards, carrion crows, dogs, men, cleanliness. Treatment: Locally antiseptics, phenol, boric acid, generally, phenol.

This has been especially seen in pigeons in which it has been studied by Löffler, Cornil and Megnin, and Babes and Puscarin. Löffler found an *æ*robie, non-motile, non-liquefying bacillus in the false membranes, inflamed tissues, liver, lungs and blood, even in the leucocytes. It formed irregular masses, and grew in nutrient gelatine, blood serum and potato. It proved pathogenic to pigeons, linnets, rabbits and mice, but not to hens, Guinea-pigs, rats or dogs. Chickens, however, suffer from an acute diphtheritic affection caused by a nearly allied bacillus, and it remains to be seen whether the varying pathogenesis may not be due to the habit of long continued growth in a particular genus and an acquired unfitness for growing in the other. The pathogenesis is also different from the bacillus of diphtheria of man, and the two diseases are not usually inter-communicable, in spite of the fact that in rare instances infection has appeared to have taken place from man to birds.

In pigeons and fowls the upper parts of the air passages and digestive tract are mainly involved, the tongue, fauces, corners of the mouth, nares, larynx, and conjunctiva. The bowels suffer less frequently and mostly concurrently with the mouth, nose and throat. The mucosa is deeply congested and in part covered by a yellowish exudate which may accumulate in masses, and dry into a firm substance. The disease affects particularly high bred birds, kept in close warm houses, and is often imported by prize animals returned from a show. There may be dullness, listlessness, sunken head, trailing wings and tail, erect plumage, diarrhoea, and, if the nose and throat are affected, a modification of the voice as in roup. Death may occur from asphyxia from the second to the fourth day near the beginning of an outbreak or the illness may last twenty days, after the more susceptible birds have been killed off.

In investigating a series of outbreaks of roup in chickens in America, Dr. V. A. Moore found a non-motile bacillus allied to the colon bacillus which proved much more deadly to rabbits and guinea pigs than to chickens, and which was not found in the blood nor internal organs but only in the local lesions where inoculated. The disease tended to assume a chronic type in place of the acute form as seen in Europe. Three inoculated chickens escaped the disease altogether. It would appear therefore that we have here a disease distinct from that described by Loeffler, or that there was an absence of some unknown predisposing or contributing conditions that were present in the European outbreaks. In both diseases however infection is an undoubted factor and similar measures of prevention and even of treatment may be followed.

Prevention. The first consideration is the seclusion of flocks from outside animals in affected localities. Newly purchased birds or those returning from a poultry show should be placed in strict quarantine for a few weeks until the absence of infection shall have been demonstrated. Different flocks should not be allowed to mingle, nor the members of a healthy flock to wander where the manure of another flock has been laid. Birds having diarrhoea, or any discharge from eyes, nose or beak, or any false membrane on such parts should be excluded from the flock, and the house and yard disinfected. It should not be forgotten that rabbits, guinea pigs and mice may be bearers of the infection, and that it may be introduced on the feet of dogs or their masters. Pigeons, buzzards, and carrion crows are especially dangerous as possible bearers of the infection. Cleanliness as regards food and water, buildings and yards is of vital importance.

Treatment. The sick birds should be strictly secluded and handled by a special attendant. When the lesions appear on visible mucosæ they should be painted several times a day with a 5 per cent. solution of phenol, or a saturated solution of boric acid, or salicylic acid, potassium permanganate, iodine, or some other germicide may be used. For the bowel affection one or two drops phenol in water may be given daily, and the drinking water should be slightly charged with the same. Cleanliness, pure air, warmth, dryness and sloppy food are all important.

ACUTE CATARRHAL ENTERITIS IN SOLIPEDS.

Definition. Causes: Irritants swallowed, debility, improper, insufficient food, congestions, parasitisms, impaired innervation or circulation, iced water, chills, perspirations, fatigue, hot, damp weather, overfeeding, cryptogams, bacteria, newly harvested fodder, septic, or fermented food, leafy fodder, toxins, stagnant, septic water, lack of pepsin, muriatic acid and bile, diseased teeth or jaws or salivary glands. Lesions: Gastritis, congestion of small intestine and colon, in striæ, thickening, ecchymosis, ulceration, necrosis, excess of mucus with pus, villi, follicles and glands swollen. Symptoms: Fever, high colored urine, costiveness, coated tongue, red eyes, inappetence, sluggishness, emaciation, weakness, unthriftiness, colics, rumbling, diarrhœa; or more fever, suffering, anorexia, icterus, hurried breathing, pleuritic ridge, arched back, tender abdomen, rumbling, flatus, diarrhœa, critical or bloody, anxiety, debility, prostration, collapse. Prognosis. Treatment: In mild cases, careful diet, and laxatives with antiferments, in severe cases, laxatives, anodynes, antiseptics, demulcents, stimulants of peristalsis, enemata, counter-irritants, fomentations, compresses, mustard, in profuse diarrhœa antiseptics, anodynes, demulcents, calomel and chalk, bismuth, astringents, boiled flour or starch, gums. Dieting during convalescence.

Definition. Inflammation of the intestinal mucosa.

Causes. Irritants of all kinds taken in with food, or as medicine or otherwise and acting on the mucosa. Debilitating conditions (chronic disease, starvation, overwork, close indoor life) which lower the tone of the system at large, and local debilitating conditions like coarse, dry, fibrous, innutritious food, congestions, parasitisms, impaired innervation and troubles of the circulation are strongly predisposing. Drinking iced water may operate by lowering the tone of the intestines but seems to habitually act rather by inducing reaction and congestion. Chills of the surface, especially when perspiring and fatigued, act in the same way. The relaxation and atony attending on long continued hot weather, predisposes to enteritis, but is doubtless even more injurious by the abundance of ferments which it propagates in food and water.

Overfeeding and stimulating aliments thrown on an alimentary canal in such an atonic condition become especially hurtful. The injury, however, comes most commonly from food that contains an excess of cryptogams or bacterial ferments or from water similarly charged. Newly harvested fodders in which the microbes are still in a state of vigorous life, when added to the poisonous principles in certain immature seeds (leguminosæ, gramineæ, etc.); fodders that have undergone fermentative changes (rotten potatoes, turnips, musty hay or oats); fodders that are leafy and harbor an excess of microbes (alfalfa, sainfoin, cowpea, clover) are especially dangerous at times. If musty or otherwise altered they often contain besides, dangerous toxins.

In taking into account the fungi and microbes in spoiled foods, we need not give exclusive attention to the particular species of microbe present. An extended observation shows that the same ferments may be present in the dry, well cured, wholesome fodder, and in the musty or spoiled specimen, the main difference being in the excess found in the latter case as compared with the former. With the excess too, there is always present a large amount of toxins, ptomaines and other more or less poisonous products, which, acting on the intestinal mucosa or even on the system at large, tend to reduce its vitality and to lay it open to the attacks of bacteria which had otherwise remained perfectly harmless. Porcher and Desoubry, Achard and Phulpin and Wurz have shown experimentally that intestinal microbes can enter the chyle and blood from even a healthy bowel. The streptococcus of pneumoenteritis equi of Galtier and Violet appears to be a common fodder and intestinal microbe, which has become pathogenic, because of its excess or on account of a lack of resistance on the part of the animal. In the same way the various intestinal cocci and the common colon bacillus may become pathogenic when the normal antagonism of the bowels and their contents is lessened.

In the same way stagnant and septic water may be harmless to one animal of great vigor and good tone and pathogenic to another which lacks these qualities; or the excess of the ferment and its toxins may overcome the natural resistance of the animal. The lack of the natural antiferments of the intestine, pepsin and hydrochloric acid, on the one hand and bile on the other, will also conduce to multiplication of the microbes and their products, so that they can successfully attack the mucous membrane.

Other accessory causes operate more or less, thus any impairment of the process of mastication,

through diseased teeth or jaws tends to the escape of undigested food through the stomach, as a specially favorable culture media for the microbes, and irritants of the mucosa.

Lesions. As the disease very often implicates the stomach (gastro-enteritis), the usual lesions of gastritis will be seen. Most commonly the lesions are best marked in the small intestine, and again in other cases in the colon, but usually there is more or less change in all parts of the intestinal canal. The small intestine is almost devoid of aliments and the mucosa deeply congested in patches or striæ, with at points thickening, softening so that it crushes under the finger, hemorrhagic discoloration, and even ulceration, and necrotic changes. It is covered with a layer of mucus, thin and mucilaginous or thick and glutinous, containing many granular and pus cells. The villi are swollen, the follicles of Lieberkuhn puffed up, and the agminated and solitary glands widely dilated, filled with exudate, and surrounded by an area of congestion. Proliferation of small, round cells has produced embryonic tissue in the mucosa and especially between the glands.

Symptoms. In the *mildest form* there is hyperthermia, thirst, insensible loins, scanty, high colored urine, costive bowels, a few small pellets only being passed at a time, hard, dry and covered with a mucous film, hot, clammy mouth, coated tongue, with redness along the edges and tip, yellowish red eyes, impaired appetite, dull, sluggish habit, a tendency to hang back on the halter, and a steady loss of flesh and increased dryness and unthriftiness of the coat. Slight, intermittent colics occurring especially after meals and attended by loud rumbling of the bowels are marked features. This may be followed by slight relaxation of the bowels and recovery in about a week, unless it should become complicated by intestinal indigestion or impaction, or should merge into the acute form.

In the *more intense forms* all symptoms are aggravated. There is anorexia and even refusal of water, dullness and prostration are well marked, the head carried low, and the gait is unsteady. The mouth is hot with tenacious mucus, and a foetid odor; the tongue is furred and red at the tip and margins, the eye is sunken, the conjunctiva icteric, the face pinched, and the pulse accelerated. Hyperthermia may reach 104° F. Breathing may be almost normal, or with fever, may become rapid and accompanied by a pleuritic ridge on the flank. The back is slightly arched and rigid, the belly drawn up and tender, after meals it may be tympanitic, and colics set in or are aggravated, pawing, uneasy movements of the hind limbs, and lying down to rise again shortly, with frequent looking at the flanks being noticeable.

Defecations are at first abundant and coated with mucus, later the balls are small and scanty and expelled with much effort. From the first the everted rectum is of a very deep red. Toward the end of the first day or later the intestinal rumbling increases, flatus passes freely, and diarrhœa may set in and prove critical. This usually indicates disease in the colon and tends to recovery; it may be entirely absent if the inflammation is confined to the duodenum, the effused liquid being re-absorbed from the cæcum and colon. If the diarrhœa should prove critical there is a return of appetite and spirit, the fæcal discharges become firmer and recovery takes place in a week. If, however, the diarrhœa becomes more profuse and bloody, the colics more intense, the eyes more sunken and hopeless, the face more pinched and anxious, and the temperature reduced to or below the normal, with great weakness and debility, the near approach of death may be feared. This state of collapse may be further marked by extreme coldness, or dropsy of the limbs, increased icterus, hurried breathing and rapid loss of flesh.

A prominent icterus indicates implication of the liver from the ascent of the infecting germs through the bile ducts, or the passage of microbes or their products or both through the portal vein. In either case it is a serious complication.

Prognosis. In its uncomplicated form the disease is not very fatal to vigorous, mature horses, though more trying to the young. If infective germs or their products implicate the liver producing marked jaundice, or if the general system is poisoned by the microbes or their toxins, producing marked depression and prostration the danger is enormously enhanced.

Treatment. In the *mildest cases* a limitation of the food to moderate bran mashes, and a dose of ½ lb. of sodic sulphate, with salicylate of soda (3–4 drs.) or bismuth will usually suffice.

In *severe cases*, at the outset, while constipation exists give 3 or 4 drs. of cape aloes, or ½ lb.

Glauber salts or ½ pint olive oil, combined with 2 drs. of extract of hyoscyamus or belladonna and 3 drs. salicylate of soda. This serves to deplete from the inflamed vessels and the whole portal system, to soothe suffering, to expel much of the offensive and infective matters from the bowels, and to check fermentation in that which remains. They should be given with, or followed by mucilaginous liquids like solutions of slippery elm or gum arabic, flaxseed tea, or well boiled farinaceous gruels.

Pilocarpin, 3 grs., or eserine, 2 grs., or both have been recommended and may be resorted to when action of the bowels is urgently demanded. They need not supersede the other laxatives. In manifest impaction of the large intestines, salts, aloes, pilocarpin and eserine may form an effective combination.

Copious enemata with mucilaginous liquids or warm soap suds should be given at frequent intervals.

Counter-irritants and derivatives to the abdomen are most important. Hot fomentations may be persisted in for an hour at a time, or a damp compress around the abdomen covered closely by dry blankets and held in place by elastic circingles. Mustard pulp made with cold water rubbed in against the hair and at once covered by paper and a thick blanket is often of great value as drawing blood and nervous action to the skin and relieving the suffering intestine.

In all cases the diet and drink must be carefully supervised. A little thoroughly scalded wheat bran, or farina, and decoctions of flaxseed, farinas, slippery elm or mallow, or a solution of gum arabic will refresh the animal without overloading the digestive organs or favoring further fermentation.

In case of the onset of diarrhœa which threatens to prove excessive and persistent, the giving by mouth and anus of antiseptics and anodynes with mucilaginous agents may be resorted to. Calomel may be given in 10 grain doses twice daily mixed with five times the amount of chalk. Or 2 drs. each of nitrate of bismuth and salicylate of soda and ½ oz. of laudanum may be given three times a day. Or quinine, 2 drs. and nux vomica 10 grs. may be added to the above. A choice may be made of other anodynes, (hyoscyamus, belladonna), antiseptics, (salol, chloral, naphthol, naphthalin, creolin), and bitters, (gentian, calumba, cascarilla).

Antiseptic and even astringent injections must be given, and well boiled farinas and mucilaginous agents may be given by the mouth. Wheat flour boiled for several hours; starch prepared with boiling water as for the laundry, (1 pint); gum arabic, or slippery elm may suffice as examples.

The patient should have a dry comfortable box and warm clothing according to the season of the year. He must be kept for a week on linseed gruel or other equally simple demulcent agent and brought back to his customary food by slow degrees.

CHRONIC CATARRHAL ENTERITIS IN SOLIPEDS.

Causes: As in acute: troubles of circulation, heart, lungs, verminous embolism, parasitism, skin disease. **Lesions:** thickening of mucosa, pigmentation, rigidity, hypertrophy of villi, follicles and glands, ulceration, polypi. **Symptoms:** impaired appetite, buccal foetor, retracted flank, unthrifty skin, pallid mucosæ, colics, tympanies, rumblings, irregular bowels, emaciation, perspiration, fatigue. **Treatment:** dietetic, tonic, bitters, salines, aromatics, enemata, bismuth, laudanum, calomel and chalk, iron, astringents, counter-irritants, electricity, sunshine.

Causes. This may occur from a continuance of the same causes as in the acute, or from an imperfect recovery from the acute form. It may result from troubles in the circulation, as valvular disease of the heart, or emphysema of the lungs, which forces the blood back on the venous system, including the liver and portal vein. Or the lesions that come from verminous embolism may leave such alteration in the intestinal walls as entail chronic congestion of the mucosa, or intestinal parasites may be the cause. Severe and inveterate skin diseases appear to affect the intestinal mucosa by sympathy, just as diseases of that mucosa usually entail skin diseases.

Lesions. Attenuation of the coats of the small intestine and thickening of the mucosa of the large have been noticed. The mucosa is darkly pigmented and covered with excess of mucus. The thickening of the mucosa may extend into the submucous tissue, giving a firm leathery feeling to the part, and entailing a loss of elasticity. The villi are hypertrophied and the follicles of Lieberkuhn and Peyer's patches may be congested, ulcerated or otherwise altered. Polypoid growths are not uncommon on the mucosa, and the mesenteric glands are enlarged and pigmented.

Symptoms are by no means very definite. Disturbance of the digestive functions, capricious or impaired appetite, dry foetid mouth, tucked up abdomen, dry hair and skin, pallor of the visible mucous membranes, slight intermittent colics and tympanies, loud rumblings in the bowels, and relaxed bowels, or alternate costiveness and diarrhoea, with some tenderness on manipulation of the abdomen are the usual symptoms. The animal loses flesh, has dry, unthrifty coat, and sweats and is easily exhausted at work.

Treatment. Dietary care is the first essential. Boiled oats, barley, rye or bran, in small amount and flaxseed tea may indicate the kind. These should be given in small amount often, and at regular intervals.

A failing appetite may be stimulated by nux vomica (10 grains) twice daily, or by gentian or other bitter, along with common salt and aromatics.

Constipation may be combated by fresh green food in small quantities, or by an ounce each of Glauber salt and common salt given every morning before feeding, in a drink of water (half to a bucket, if possible), and 10 to 20 grains of nux vomica may be advantageously added. Soapy injections with salt or glycerine may also be given.

Diarrhoea may be moderated or checked by nitrate of bismuth (2 drachms), with laudanum (1 ounce), repeated as may be demanded. A combination of calomel and chalk (1:12) will often serve a good purpose in drachm doses several times a day. For persistent diarrhoea Cadeac recommends the following: Iron carbonate 4 drachms, lime water 10 ounces, alum 1 drachm, powdered oak bark 1 ounce, given in water and farina.

Sepsis and fermentation must be combated by the same means as in the acute type, and the same counter-irritants may be resorted to. A life in the open air or sunshine, but without undue exertion is of great importance.

ACUTE CATARRHAL ENTERITIS IN CATTLE.

Causes: atony, debility, starvation, overfeeding, innutritious food, close, foul buildings, ill health, over-exertion, hot weather, sudden changes, chills, privation of water, irritants, spoiled and newly harvested grain, foul water, parasitism, chest diseases, thrombosis. Lesions; in small intestine mainly, tympany, congestion, thickened mucosa, epithelial degeneration, desquamation, enlarged villi, follicles and glands, erosions, ulcers, perforations. Symptoms: solid masses in rumen, impaired rumination and appetite, rumbling, tenderness, costiveness, fever, arched back, tender, tucked up abdomen, colics, in severe cases, agalactia, tremors, rigors, drooping head, ears, eyelids, tender abdomen, straining, expulsion of mucus, foul eructations, later diarrhoea, critical or exhausting. Death from tympany, bleeding, infection, inanition. Diagnosis: by concurrence of symptoms, hyperthermia, tender abdomen, no blood nor coccidia in stools, no frothy bloody mucus with tenesmus. Treatment: dietetic, friction, synapism, atropia, chloral hydrate by rectum, salines, demulcents, nux, tartar emetic, eserine, pilocarpin, sulphites, salol, sodium salicylate, naphthol, etc., bitter tonics, carminatives, stimulants, sodium chloride, ipecacuan, hygiene during convalescence.

Causes. As in solipeds the various conditions which lower the general tone, and those which especially debilitate the bowels predispose to catarrhal inflammation. Underfeeding and overfeeding, fibrous, innutritious, indigestible food, an indoor life in close, foul stables, chronic and debilitating diseases, overwork, overdriving, long continued hot weather, sudden changes of weather, chills, long railway journeys without water, exposure in hot stockyards in midsummer, all lessen the resisting power of the system and of the bowels.

As more direct irritants, may be named irritant plants, weedings and culls from gardens, musty and spoiled fodders of all kinds, newly harvested grain, and putrid drinking water. Also intestinal parasitism, diseases of the heart and lungs, and thrombosis of the mesenteric arteries.

Lesions. These predominate in the small intestine in catarrhal enteritis as they do in the large intestine in dysentery. The small intestine and cæcum may be distended by gas, and reddened more or less deeply on their outer surface. The mucosa is the seat of congestion, punctiform and ramified redness, thickening, infiltration and softening so that the epithelium breaks down into a pulp under the pressure of the finger. Desquamation may be extensive leaving a raw angry surface. The villi are infiltrated, erect, and ulcerated showing dark bloody points, and ecchymoses, and circumscribed sloughs and eschars are present. The solitary glands are congested, hypertrophied and projecting. The submucosa is infiltrated with a gelatinoid material and the same may be found around the swollen and congested mesenteric glands. Perforations have been met with in some cases, and coexistent inflammatory lesions in the stomachs are common.

Symptoms. In the *mildest forms* there is inactivity of the rumen, aggregation of the contents into hard masses, easily felt through the surrounding gases, appetite and rumination are greatly impaired, and there is much rumbling and considerable tenderness of the right side of the abdomen, and more or less costiveness, with hard, glazed mucus-covered fæces. There is some rise of temperature, ardent thirst, injected mucous membranes, dry, hot muzzle, weeping eyes, a small, hard, weak pulse, arched back, tender to pinching, and tucked up abdomen. There may be slight colicky pains, uneasy movements of the hind feet and tail, and sometimes lying down and rising at short intervals.

In *more severe cases* the impaction and tympany of the rumen are more marked, the hyperthermia runs high, appetite and rumination cease, the milk dries up, rigors and tremors appear, the head and ears droop, the eyes are sunken, the mouth is clammy and foetid, the colicky pains are severe or extreme, the right side of the abdomen is very tender, defecation may be altogether suspended and rumbling in the right side of the abdomen ceases or becomes rare. Straining may continue but seldom is anything but mucus passed. Eructations from the rumen are distinctly foetid.

After the third day the violence of the pains may abate, and sometimes diarrhoea sets in and may be regarded as critical, and portending recovery. If rumbling in the right side is resumed, if the fever subsides, the spirits revive, and some appetite and rumination return they will herald improvement.

If on the other hand the pulse becomes smaller, the temperature higher, the eyes sunken and fixed, the urine scanty, red and acid, the animal constantly recumbent on its left side, if when

raised it omits the healthy stretching of its hind limbs, and walks sluggishly and painfully with frequent moaning, if when down it rests its head on the ground, the prospects are very unfavorable.

Death may occur early from tympany and asphyxia; it may follow profuse intestinal hæmorrhage; or it may be the result of general infection and inanition.

Diagnosis must depend on the combination and succession of the above-named symptoms. From acute intestinal congestion it is distinguished by the more moderate type of the colic, and the more gradual advance of the disease. From acute indigestion and tympany of the rumen by the early and marked tenderness of the right side of the abdomen, and the decided hyperthermia. From hemorrhagic enteritis by the absence of the black sanguineous discharges from the bowels at an early stage of the malady, and of coccidia from the droppings. From dysentery it is distinguished by the absence of the mucous and bloody discharges, which are passed with much straining in that affection from the beginning.

Treatment. The first consideration is dietetic and hygienic. If the animal will still eat, he ought to have boiled flax seed or other well boiled gruel, rendered palatable by salt. Even if he refuses food, this may be diluted largely and will be taken on account of the thirst. If he refuses all, a bottle may be given at intervals to refresh him. Or better—milk may be given from a bottle in the same way. Active friction to the abdomen with straw, or the application of oil of turpentine or mustard may abstract blood to the skin and favor the restoration of the intestinal functions. To calm the pains and control spasms, sulphate of atropia ($\frac{1}{2}$ gr.) may be given subcutem and repeated if there is no action on the pupil in fifteen minutes. Or extract of belladonna (2 drs.), or chloral hydrate ($\frac{1}{2}$ oz.) may be given by rectal injection.

To overcome the intestinal torpor 1 lb. each Glauber and common salt may be given in four to six quarts of warm water and followed by frequent mucilaginous drinks, as much as the animal will take, but only two or three quarts at a time. The addition of $\frac{1}{2}$ dr. nux vomica will serve to rouse peristaltic action. Harm advises $1\frac{1}{2}$ dr. tartar emetic by rectal injection for the same end. Next to Glauber salts, Castor oil (1 qt.) is to be recommended. Along with these or independently of them sulphate of eserin ($1\frac{1}{2}$ gr.) or pilocarpin (2 gr.) may be employed subcutem.

Frequent rectal injections of soap or mucilaginous liquids, with or without laxatives will be useful.

As antiferments beside the salt may be used bisulphite of soda in $\frac{1}{2}$ oz. doses, salol $3\frac{1}{2}$ drs., salicylate of soda 3 drs., betol $3\frac{1}{2}$ drs., or naphthol $3\frac{1}{2}$ drs., by the mouth and rectum.

When free movement of the bowels has been secured, attempts should be made to restore appetite and rumination by tonics and stimulants: gentian $\frac{1}{2}$ oz., nux vomica $\frac{1}{4}$ dr., ipecacuan 2 drs., common salt 1 oz., may be given three times a day.

The diet should at first be restricted to flax seed gruel or that of other farinas, with a mere handful of fresh grass or bran mash and the restoration of the previous diet should be slow and gradual, care being taken meanwhile that no costiveness of the bowels supervenes.

CHRONIC CATARRHAL ENTERITIS IN CATTLE.

Causes: As in acute chest diseases, abdominal tuberculosis. **Lesions:** Thinning, discoloration, degeneration of mucosa, foetid, mucous contents, black baked masses, lymph glands pigmented. **Symptoms:** Impaired appetite, irregular bowels, tympanies, lies with nose on right flank, unthrifty coat, prostration, emaciation, weakness, tender flank. **Treatment:** Dietetic, laxative, stimulant of peristalsis, bitters, antiseptics, aromatics, muriatic acid, treat concurrent disease.

Causes. This may result from a continuance of the causes that are operative in the acute, or from the latter merging into the chronic form. Chronic diseases of the heart and lungs, local disturbances of the circulation, and tumors or tubercles of the intestines or mesentery are additional causes.

Lesions. These embrace attenuation of the intestinal walls at Peyers' patches, a dark, slaty discoloration of the mucosa, more or less congestion, an accumulation of foetid mucus in the small intestines, of mucus and black baked faecal matters in the large, and discoloration of the mesenteric glands. Inter-dependent diseases of the heart, lungs and liver are not uncommon.

Symptoms. Following the acute form there remain impaired or capricious appetite and rumination, costiveness alternates with relaxation of the bowels, intermittent slight tympanies occur, the subject inclines to lie much with his nose in his flank, has dull coat, erect on back and neck, sunken eyes, drooping ears, and rapidly loses flesh and strength. Tenderness of the right side of the abdomen when the fist or knee is pressed into it is a marked feature.

Treatment. The diet must be cared for as in the acute form, yet fresh green grass, a little at a time, is calculated to stimulate appetite and rumination and to prove laxative to the bowels. The same purgatives may be given in one-fourth the doses and repeated daily or reduced as may be found best to secure a moderate secretion and discharge from the bowels; eserine or pilocarpin may be used for the same purpose; the bitters and antiseptics may be given in the same way. As calmative aromatics, oil of peppermint 30 drops, powdered anise $\frac{1}{2}$ ounce, or ginger $\frac{1}{2}$ ounce, may be given twice or thrice daily.

Cadeac strongly recommends a drink slightly acidulated with hydrochloric acid to assist the digestion and stimulate the stomach to action.

Attention must of course be given to any curable concurrent or inter-dependent disease.

DYSENTERY OF CATTLE.

Definition. Attacks ox mainly. Causes: accessory causes, chills, rain storms, night dews, hoar frost, foul or iced water, alimentary irritants, spoiled fodder, over exertion, hot damp weather, odors of carrion, crowding, swamps, foul stables, germs or pathogenic ferment, in man catarrhal, diphtheritic and amœbic, amœba dysenterica, other microbes, effect of better hygiene. Symptoms: attack sudden, languor, trembling, weakness, weeping eyes, fever, buccal epithelial softening, erosions, tenesmus, foetid, liquid stools, involuntary defecation, hemorrhoidal congestion, open anus, colics, tender right flank, splashing on handling, anorexia, salivation, unthrifty skin, hide-bound, cracked muzzle, later prostration, low temperature, sunken glazed eyes, drooping head, ears, eyelids, weakness, emaciation, alkaline, foetid, frothy, bloody, mucous stools, with sloughs, saliva acid, gastric liquids alkaline, bile suppressed. Duration: three days to three weeks or chronic. Mortality 50 to 80 per cent. Complications: mostly septic, abscess, gangrene of other organs, lungs, joints, glands, etc. Lesions: rapid sepsis, blood deep red, coagulum loose, venous congestion, large intestines congested, tumefied, softened, desquamated, eroded, sloughing, necrotic, folds perforated, cicatrizing, contents mucopurulent, bloody, putrid, microbes, glandular lesions, implication of small intestines, stomach mouth, liver, spleen, hepatic abscess. Diagnosis: from rinderpest by tardiness and comparative weakness of contagion, absence of general mucous congestion and epithelial concretions, from toxic enteritis by same. Prevention: avoidance of causes, separation of sick, disinfection, careful feeding. Treatment: Demulcents, antiseptics, astringent tonics, opiates, ipecacuan, calomel, sodium sulphate with antiseptics, antiseptic enemata of glycerine, phenol, creolin, iron sulphate, silver nitrate, salicylic and boric acids, rest, gravitation, careful dieting.

Definition. An infective, ulcerative inflammation of the large intestine but especially of the colic and rectal mucosæ.

It is well recognized as a disease of the ox, and the older writers allege its existence also in horses, swine and dogs, though this is not admitted in all modern veterinary works.

Causes. It was formerly ascribed to improper hygiene, chills, cold rain storms, night dews, hoar frost, malarious emanations, putrid, stagnant or iced water, irritants in food, green, fermented or musty food, a too liberal diet after starvation, overtaxation in very hot weather, and bad odors from decomposing carcasses. These can only be accepted as predisposing causes begetting a general debility, or debility of the alimentary canal and laying that open to the attacks of specific microbes. The close aggregation of cattle on ship-board, in besieged cities, and in the parks of armies in the field, has apparently contributed to the propagation of the dysentery. The removal of a victim to a herd with free healthy range seldom starts a new center of the disease. In all infected herds huddled in small compass there is every facility for the propagation of a germ already present, and especially in the commissariat of a belligerent army there are enough privations, over-exertions and other trying conditions to favor predisposition. Faulty food like stale bread, musty hay, have been supposed to cause it. For man and beast alike dysentery is preëminently a disease of the tropics, and of hot seasons, and will often subside on the advent of cold weather.

Its propagation on given (swampy) soils, and in particular (foul) stables strongly suggests a special germ, though for the cow this has not been perfectly identified. Gerlach vainly attempted to inoculate it, and it does not often propagate itself beyond the foul and infected localities or stables, yet its persistence in them for years bespeaks unequivocally the operation of a special pathogenic ferment.

It may also be fairly assumed that it is not necessary that the same factor should be present in all cases alike, but that one operates predominantly in one case and another in another. In other words dysentery must be recognized as not one disease, but several, of which the true pathogenic microbes have not yet been fully demonstrated, but which are classed together because of the similarity of the attendant lesions.

In man three distinct forms are recognized. 1. **Catarrhal dysentery**, with frequent small stools of rosy mucus, and blood; and later pus, scybala, passed with tenesmus, but no sloughs and little odor; 2. **Diphtheritic dysentery**, with thin watery bloody discharges having a pronounced cadaveric odor; also tenesmus, sloughs, and increasingly offensive smell; 3. **Amœbic dysentery** with frequent bloody mucus stools, tenesmus sloughs and foetor, but with distinct remissions or intermissions. With the latter, amœbæ are found abundantly and more so in the more acute cases with alkaline stools. They are found in the fresh warm stools, 5 to 8 times the size of a red blood

globule and oval, pyriform or irregular in form, with nucleus and nucleolus. Kartulis and Hlava succeeded in inducing dysentery in cats and dogs by injecting pure culture of the amoeba, and the former testifies that dogs in Egypt take the disease spontaneously, and their stools contain the same amoeba coli as is found in man.

Cunningham who investigated the subject in India found amoeba in the bowels of healthy men, and also abundantly in the fæces of horses and cows, which have naturally the requisite alkaline reaction.

The mere presence of the amoeba therefore may not be sufficient to cause the disease, but with the requisite predisposition and an alkaline condition of the intestinal contents, it is manifestly an important factor in the disease.

The causative microbes in other forms of dysentery have not been identified, but under the requisite irritation and local debility one can easily conceive of the ordinary bacterial ferments of the intestine, concurring with others introduced from without, in determining the morbid condition. With better hygiene the disease is steadily diminishing in man and beast, though violent epizootics (in cattle) still appear in connection with wars (siege of Belfort, 1870, Zundel), and carriage by sea in hot climates (Mediterranean trade, Bouley).

Symptoms. The disease sets in suddenly, yet prodromata may occasionally be observed, such as dullness, langor, trembling over the flanks and elbows, weakness, prominent, weeping, congested eyes, and low moans when moved. Then follow hyperthermia, at first slight, heat of the mouth without injection, epithelial concretions or erosions and diarrhoea (sometimes there is straining without passage at first). Then follows a period of profuse and foetid discharge, with relaxed or open anus, the liquids escaping involuntarily and smearing the tail, perineum, thighs and hocks, and the protruded mucosa showing dark red congestions and even commencing erosions. Colicky pains, slight at first, have now become intense, and the right side of the abdomen is very tender to the touch and fluctuates noisily when manipulated. Appetite and rumination are lost. Salivation may be present, the saliva falling in films to the ground. The buccal epithelium is softened, loosened and easily detached by the finger, leaving raw sores. The temperature which has risen slowly (not as in rinderpest abruptly) may reach 106°. The hair becomes dry, the skin harsh, rigid, and firmly adherent to the deeper parts, and often cold, while the muzzle is hot, dry and even cracked.

At a still more advanced stage, the pulse is small, the temperature lowered, the animal very weak and unsteady and inclined to lie, great emaciation, sunken glazed eye, drooping head, ears and eyelids, and a general foetor from the skin as well as the dejections, which attracts crowds of flies. By this time there may be passed only bloody mucus mixed with eschars, and having a most repulsive odor.

It is a noticeable fact that the fæces are alkaline, and in man the saliva is acid and destitute of its glycogenic properties, the stomach secretions are alkaline and no longer peptogenic, and the secretion of bile is arrested until improvement sets in.

Course. Duration. Some mild cases recover in two or three days, and in violent cases death may occur at this early date. More commonly the disease continues for two or three weeks before ending in death or recovery. Some merge into the chronic form and may last for months and die in a condition of marasmus. These last cases become mere walking skeletons, with pallid mucosæ, sunken eyes, scurfy hide-bound skins covered with vermin, and the frequently everted rectum is congested and covered with ulcers and eschars. *Mortality* is from 50 to 80 per cent.

Complications. Most complications are in the direct line of septic infection. Among the most common are hepatic abscess, gangrenous pneumonia, and extensive gangrene of the intestinal walls. In man arthritis, paralysis, parotitis and other secondary affections are seen. In the animal as well we may expect necrotic centres in any organ, from the supervention of a general septicæmia.

Lesions. The carcasses putrefy with extraordinary rapidity being as a rule pervaded by septic microbes and their toxins. The blood is of a deep red, loosely and imperfectly coagulated, and accumulated in the veins and subcutaneous connective tissue.

The large intestines are the special seat of the disease, the walls being found hyperæmic, with

concentration especially on the mucous surface which is red, congested, infiltrated, tumefied so that it is easily detached under pressure by the finger, or broken down into a putrid pulp. At other points the epithelium or the whole mucosa has been detached leaving ulcers, varying from mere erosions to the deep and even perforating sores, through which the putrid contents escape into the abdominal cavity. At other points the surface is covered by necrotic masses surrounded by a swollen margin of living mucosa. Elsewhere the eschars have been detached and the granulating base and margin have contracted into more or less perfect cicatrices. The contents of the affected bowel may be largely mucopurulent, or it may be mixed with blood, with eschars and putrilage from the sores and at times with small packed masses of food, but in all cases it is very putrid and repulsive. It always contains the elements of blood and exudation, together with many microbes, the predominance of individual species suggesting the main factors in the pathogenesis. Peyer's patches and the solitary glands are often the seat of infiltration and ulceration. Virchow points out that in man the lesions are largely concentrated in the flexures of the colon in which the ingesta is longer delayed, and the very convoluted arrangement of the viscus in cattle may be one reason of their special predisposition to the disease.

The small intestines are exceptionally implicated, and the abomasum red, congested, ecchymosed and even ulcerated. The mouth and pharynx are often congested, with erosions and ulcerations on the gums and tongue.

The mesenteric glands are red and swollen or pigmented of a dark gray color, and these or the adjacent connective tissue may be the seat of abscess. The liver is enlarged congested, of a yellowish red color, with granular degeneration and softening. Hepatic abscess is rare in cattle. The spleen is engorged with black blood and distinctly enlarged.

Diagnosis. Dysentery is distinguished from rinderpest by the slower advance of the hyperthermia, by the absence of buccal concretions of epithelium, and of vaginal congestion, and above all by the absence of the virulent contagion by which the rinderpest spreads widely and rapidly, irrespective of special unwholesome environment. The lesions of dysentery are concentrated on the large intestines, while in rinderpest they are extended over the whole alimentary canal, and do not spare other mucosæ.

From toxic gastro-enteritis it is distinguished by the absence of any history or lesions showing the ingestion of a poison and by the concentration of the lesions on the large intestines whereas in poisoning with irritants, the stomach and small intestines are the most liable to suffer.

Prevention. Precautions against overcrowding on board ship in hot seasons, and the maintenance of cleanliness are prime considerations. In commissariat herds close crowding must be avoided, feeding should be done at different points for six or eight cattle at each, the yards should be frequently changed, or carefully cleared of manure and sprinkled with a solution of 3 per cent. sulphuric acid. In case an animal is attacked it should at once be removed and killed, and its offal and the yard where it has been, disinfected. Food and drinking water should be specially wholesome.

Treatment. Mild cases may be successfully treated by the use of mucilaginous draughts, (boiled flaxseed, mallow, slippery elm, gum arabic) with antiseptics (salicylic acid or salicylate of soda 2 to 3 drs., salol 1 dr., creosote 1 dr., sulphate or chloride of iron 1 dr.) every three hours.

In the more severe cases the most varied treatment has been resorted to. Formerly opiates and astringents were largely employed, practically shutting up the poison in the intestine. Ipecacuan in large and frequently repeated doses had the advantage of soliciting the action of both liver and bowels, but however useful, it has gradually declined in public confidence.

An eliminating and antiseptic treatment has at present the most general acceptance. Seven grains of calomel has been given every two hours for two or three days in succession. Still more recently 2 to 4 ozs. sulphate of soda every three hours with hot water and antiseptics (salicylates, sulphites, salol, boric acid, creolin, naphthol) have been substituted. Even more important is the washing out of the large intestine by antiseptic enemata. These should be very copious and frequently repeated so as not only to render the contents of the rectum and colon antiseptic but to secure the discharge of the offensive matters as they are produced. The agents may be glycerine, carbolic acid, creolin,

sulphate of iron, nitrate of silver, salicylic or boric acid. If the animal can be made to stand on an inclined plane with its hind parts elevated it will favor the penetration and retention of the liquid. Perfect rest is a most important accessory.

In case of recovery the return to ordinary diet should be gradual.

CATARRHAL ENTERITIS IN SHEEP AND GOAT.

Causes: Symptoms: Moping apart, inappetence, tympany, hard masses in rumen, constipation, colics, straining, stretching, fever, no rumbling nor defecation, diarrhoea, critical, or exhaustive. Treatment: Laxative, antiseptic, counter-irritant, demulcent antiseptic injections, in diarrhoea antiseptics, carminatives, bitters, diet, well boiled farinas.

Causes. These are unknown apart from those given for the ox.

Symptoms. Moping alone in place of following the flock, inappetence, suspended rumination, tympany, hard masses in the rumen, cold ears, horns and limbs, obstinate constipation, weary movements of the hind limbs, lying down and rising, looking at the flanks, and tenderness of the right side of the abdomen, frequent straining, stretching, hyperthermia, rapid pulse, and suppression of movement and rumbling in the bowels are prominent symptoms. The constipation may be followed by diarrhoea, which may presage recovery or hasten death, the symptoms of improvement or aggravation indicating which. The diarrhoea may be simply serous or mucopurulent and inoffensive or it may be black, bloodstained and foetid. It may cause extensive losses, with pathological changes as in the ox.

Treatment in the early stages with inactive bowels, must be laxative and antiseptic, sodic sulphate 4 ounces with 1 drachm chloral hydrate in a quart of warm water will serve to largely expel offensive ingesta and bacterial ferments and pave the way for recovery. A counter-irritant of aqua ammonia to the abdomen, under the wool, will have a good effect, and soapy or mucilaginous and antiseptic injections are desirable.

If the bowels are already acting freely, $\frac{1}{2}$ drachm each of boracic acid and nitrate of bismuth may be given at least thrice daily, with the addition of 60 drops of laudanum, and antiseptics (salol, naphthol, naphthaline). Carminatives like chamomile, anise, cardamons, peppermint may be added, and bitters such as quinia or nux vomica.

Well boiled gruels of barley, oats, linseed, or rye should be given and little or no solid food until appetite and digestion have been restored.

CATARRHAL ENTERITIS IN SWINE.

Causes: decomposing swill, ferments from snout and feet in swill, toxins, salt, brine, powdered soaps, etc. Symptoms: dullness, inappetence, fever, burrowing under litter, stiffness, drooping head, ears and tail, arched back, tender abdomen, constipation, diarrhoea, petechiæ, rumbling, tympany, weakness, emaciation, paraplegia, not actively infectious. Duration: one to eight days or more. Lesions: mucopurulent exudation in alimentary canal, congestion, extravasation, hemorrhages, ulcerations, congestion of peritoneum, mesenteric glands and other organs. Diagnosis: presence of alimentary causes, many attacked at once, no spread to better kept herds, no germ fatal to guinea-pigs nor rabbits. Treatment: eliminate, oil, calomel, flaxseed, gruels, enemata, antiseptics, revulsives, diet during convalescence, antidotes.

Causes. The enteritis of swine has been long considered as infective or septic, yet this is perhaps mainly due to the nature of the food too often furnished to this animal in a state of domestication. Not only is he fed on swill containing all kinds of over-kept food, in a state of more or less advanced decomposition, but he is kept in a pen or yard which soon becomes offensive, and following his natural instincts he roots around with his nose in the accumulating filth. When fed he plunges this foul snout in his liquid food, and as if this were not enough the fore feet follow, and if the trough is long enough the hind feet as well. Every available saprophytic microbe therefore finds its way down his throat, and the toxins from their growth outside the body accompany them to irritate or benumb the mucosa and fit it for the attacks of bacteria, which would otherwise have proved harmless. But in addition to all this, chemical irritants get into the swill and pave the way for the microbes. Salt, brine, and the various caustic washing powders used in washing dishes and tables find their way into the swill barrel often in quantity sufficient to irritate and poison. In experiments conducted at the N. Y. State Veterinary College the washing powders have been proved to be most deadly, and upon different farms, a mortality attributed to hog cholera, has again and again been arrested by preventing the entry of these powders into the swill barrel.

Symptoms. There is dullness, inappetence, hyperthermia (105°), a tendency to lie under the litter, leaving the herd, when raised the pig arches his back, moves stiffly with grunting, the tail and ears pendent, and the belly tender to the touch. The bowels are at first constipated, and thirst ardent, later diarrhoea may set in, and the skin and snout may have red blotches or patches as in hog cholera. Tympanies and abdominal rumblings are frequent. The animal becomes very dull, weak, emaciated, staggers in walking or is completely paraplegic.

It may end fatally in twenty-four or forty-eight hours, or death may be deferred for six or eight days or the disease may merge into the chronic form, or recover. The usual foul surroundings of the pig and the abundance of microbes and toxins taken in, serve to make enteritis in this animal much more redoubtable than in other domestic animals.

The *lesions* are usually common to stomach and intestine, and consist in abundant mucopurulent discharge, extensive congestion, points and extensive patches of blood extravasation, and of lymph infiltration, ulcers, congested peritoneum and mesenteric glands, and congestions and ecchymosis in distant organs.

The *diagnosis* between this affection and hog cholera on the one hand and swine plague on the other is not always easy, but it occurs in herds where no introduction of infection can be shown, there is always the evidence of a foul or unwholesome food, drink or environment, and the history of a number having been attacked at once and not one by one at varying intervals, as in infection, the disease does not spread to neighboring herds kept in better conditions, and there is an absence of the specific germ of hog cholera, motile, ærobic, non-liquefying, asporogenous, gas producing with glucose and fatal to guinea-pigs; or of that of swine plague, non-motile, ærobic, non-liquefying, asporogenous, not gas producing and not fatal to guinea-pigs, but fatal to rabbits.

Treatment. If the bowels are costive give castor oil 2 ounces, with great care to avoid choking, or shake 15 to 30 grains of calomel on the tongue and give flaxseed tea, or solution of slippery elm or gum which the animal will usually drink to slake its thirst. Or well boiled gruels may be substituted. Injections of soapsuds or Glauber salts with salicylate of soda should be added until the bowels respond, after which the salicylate alone may be given by both mouth and anus, or it may be replaced by one of the other non-poisonous antiseptics. Oil of turpentine alone or with ammonia may be applied on the abdomen and covered up until the skin is red and angry.

When appetite returns, gruels, linseed tea, boiled milk and other easily digested food may be given for some days until the stomach reacquires tone, when the patient may be slowly returned to its accustomed diet.

If the disease can be traced to alkaline washing powders, these should be first neutralized by vinegar, after which laudanum, antiseptics and mucilaginous gruels will be in order.

CATARRHAL ENTERITIS IN DOGS.

Causes: faulty feeding, close confinement, youth, distemper, over-exertion, meat diet, chills when heated and fatigued, ferments, bacteria, debility, ill health. Lesions: congested, softened, ulcerated, thickened mucosa, mucopurulent exudate, swollen intestinal and mesenteric glands, hyperplasia, polypi, follicular degeneration. Symptoms: dullness, segregation, inappetence, fever, arched back, abdominal rumbling, tenderness, retraction, constipation, diarrhœa, colics, vomiting, tenesmus, swollen red excoriated anus, icterus, weakness, debility, paralysis, if chronic, unthrifty skin, with eruptions, pallor, foul breath, tongue and gums, emaciation. Course: may recover in vigorous, or run down and die in old, young, weak, debilitated or ill. Relapses. Treatment: dietetic, laxative, in icteric, calomel, manna, enemata, warm bath, synapism, antiseptics, bismuth, sodium salicylate, in diarrhœa, bitters, astringent antiseptic tonics. Demulcents.

Causes. The dog is susceptible because of its varied, irregular, stimulating, often excessive diet, and its close confinement and lack of wholesome outdoor exercise. The conditions which predispose to or excite gastritis tend equally to enteritis. Youth, canine distemper, over-exertion, an exclusive meat diet, and chills from plunging into cold water when exhausted with hunting are to be specially noted. Spoiled meats charged with the germs of infection, or with putrefactive bacteria and their toxic products are common causes. Then any old standing disease or other cause of general debility will predispose to the attacks of such otherwise harmless germs. The irritation caused by intestinal parasites is an occasional factor.

Lesions. The mucosa is congested, ramified, spotted or dark and slaty; it together with the submucosa is swollen and infiltrated, and with points of ecchymosis, extravasation or even ulceration. The surface has a thick layer of mucopurulent matter. The solitary glands are swollen and charged with small lymphoid or pus cells and have a congested areola. The mesenteric glands are congested and the liver usually congested softened or mottled. In chronic cases there may be hyperplasia, polypi, cystoid degeneration of the follicles, etc.

Symptoms. There may be dullness, drowsiness, a seeking of seclusion, inappetence, hyperthermia (102 to 104°), ardent thirst, arched back, abdominal rumbling, tympany or tucked up tender abdomen, and a very temporary constipation, early giving place to diarrhœa. If however the inflammation is confined to the rectum and floating colon, constipation may persist. There are intermittent colics, frequent lying down and rising, or rising on the hind limbs only and when the belly is handled the patient may whine. The mouth is sour, hot, clammy, and red, and the dorsum of the tongue furred. The nose is dry and burning, the eyes congested, sunken and watery.

The fæces may be early soft or liquid and bilious, then as the defecations increase they pass through shades of gray to black or reddish black from the admixture of effused blood, and show bubbles and fœtor. Vomiting is usually an early symptom (septic matters in septic poisoning) and as the disease advances there is much fruitless straining and even protrusion of the congested and excoriated rectum. Icterus is common from infection of the gall ducts or the absorption of toxins into the portal vein. Weakness and debility amounting even to paresis may appear in the end.

In chronic cases there are emaciation and debility, unthrifty hair and skin, cutaneous eruptions, pallor of the mucosæ, fœtid breath, foul teeth and tongue, with irregular appetite, alternate constipation and diarrhœa, and, in case of implication of the duodenum and liver, icterus. The abdomen is retracted and tender.

Course. In strong vigorous dogs a spontaneous recovery is the rule, which is greatly favored by elimination through vomiting and purging. Fatal cases occur in puppies, in old, debilitated animals, or in such as have disease of the heart, lungs, or liver. Relapses are common and dangerous.

Treatment. Mild cases may respond to a purely dietetic treatment. Boiled milk, hot soup, or well cooked mush, and biscuit, are indicated, and with ½ oz. castor oil and 5 to 10 drops laudanum may suffice for treatment.

In the more severe cases, with some icterus, calomel 1 to 2 grs. with the oil, or with manna 5 drs. may be followed by emollient or soapy injections, and a warm bath or fomentations, the body being

afterward carefully dried and warmly covered. This may be followed by a mustard poultice.

The usual antiseptics (salol 10 grs., naphthol 20 grs., creoline 20 drops, creosote 7 drops, or naphthaline 20 grs.) may be given with the laxative and should be given by both mouth and anus several times daily, in combination with nitrate of bismuth. In case of icterus give a mixture of calomel 5 grs., chalk 60 grs., in doses of three to five grains three or four times a day. Or salicylate of soda (10 grs.) may be given at the same intervals.

Quinia sulphate 5 grs., nux vomica 1 gr., tannic acid 1 gr., or silver nitrate $\frac{1}{2}$ to 1 gr., or iron chloride 3 to 5 grs., may be employed when the bowels are much relaxed. Injections of well boiled rice or starch, or of gum or slippery elm, may be employed as adjuvants.

HEMORRHAGIC GASTRO-ENTERITIS OF THE DOG.

Definition. Causes: Spring, toxins, irritants, inflammation. Symptoms: Vomiting, diarrhoea, pendent head, arched back, retracted belly, black, bloody glairy frothing fæces, circulation excited, mucosæ red, yellow, or brown, death in two or three days. Diagnosis. Lesions: Stomach and intestines empty, mucosa of a dark blood red, thickened, liver and kidneys congested. Treatment: Little successful, intestinal disinfection, elimination, laxatives, wet compresses, enemata, heart stimulants, ergot, iron chloride.

Definition. A special form of septic enteritis, occurring as an epizootic and not transmissible by ingestion.

Causes. These are not well known. Occurring in a few dogs at one time in the same place, and time (by preference in spring) and then disappearing for months, and not being appreciably communicable by contagion or ingestion, it has the aspect of being caused by poisons, probably of the nature of toxins taken in with the food or water. Preëxisting inflammation has been alleged as a predisposing cause, the attack having followed superpurgation, or the administration of a handful of salt. Guinard found that the intravenous injection of tuberculin, mallein and other products of microbial growth produced lesions analogous to those of this disease.

Symptoms. The attack is usually sudden. The dog is seized with vomiting and diarrhoea and stands with head depressed, back arched and belly tucked up. The vomit is at first of alimentary matters, then of glairy mucus, or black and bloodstained material. The fæces are black, bloody, glairy, frothy, and abundant, fouling the tail and the hips. The abdomen is at first tense and extremely tender. The pulse is accelerated, the heart beats tumultuous, the breathing slow and temperature elevated. Redness of the visible mucosæ, often tinged more or less deeply with yellow, implies hepatic disorder or destruction of blood globules. Death may occur in two or three days.

The abruptness and violence of the attack, the violent vomiting and purging and the staining of the discharges with blood are to a large extent diagnostic.

Lesions. These are most noticeable in the stomach and intestines which are empty and of a dark blood red. The mucosa is thickened, gorged with blood, and showing deeper shades here and there where extravasation has occurred. The liver and kidneys are also deeply congested.

Treatment has been almost constantly a failure. Cadeac recommends disinfection of the intestinal canal, with salol, cresyl or dinaphthol, but in the absence of contagious germs this seems the less called for. The indications would seem to point to elimination of the chemical poisons, the soothing of the irritation and the tiding the animal over the period of weakness. Agents that will at once eliminate the poisons and sustain the heart's action appear to be called for, hence digitalis 2 grs. or tincture of strophanthus 10 drops are particularly indicated. Laxatives are rather hopeless considering the congestion and paralysis of the bowels, and yet if they can be made to operate on any comparatively healthy part of the intestine they will serve a good purpose in eliminating the poisonous contents, in securing secretion and elimination of poison from the blood, and in depleting from the overcharged portal system. Glauber salts by mouth and anus, or jalap, or castor oil may be resorted to. In the absence of these a free use of watery enemata and the ingestion of water by the mouth may be resorted to. A damp compress around abdomen and loins will be at once soothing and stimulating to the secretions of both kidneys and bowels.

To counteract congestion and extravasation Cadeac advocates ergotine 2 grs. subcutem, or iron perchloride, 2 ozs., to 1 quart of water to be given in doses of two or three tablespoonfuls every two hours. It might also be used in enema.

CATARRHAL ENTERITIS IN BIRDS.

Causes: microbes, diagnosis from fowl cholera, less virulent rapid and deadly, and comparatively harmless to the rabbit, debility, youth, age, unsuitable food, ill health: bacillus gallinorum, bacillus coli communis, bacillus of duck cholera, spirillum Metchnikowi. Symptoms: dullness, fever, langor, inappetence, thirst, pale comb, greenish faeces, erect plumage, drooping wings and tail, sunken head, gaping, staggering, somnolence, bloody faeces, violet comb, low temperature, death in one to three weeks. Diagnosis: by restriction to one flock, or species, and immunity of rabbits. Mortality 80%. Prevention: separation of sick and healthy, disinfection of roosts and yards, pure food and water boiled or acidified, immunization. Treatment: boiled food with antiseptics, antiseptic enemata, stimulants, tonics.

Causes. A number of different microbes are implicated in producing and maintaining catarrhal enteritis in the domestic poultry. All forms of the disease are therefore closely related to the well known fowl cholera, which is however to be differentiated, by its more intense virulence, rapid progress, and its deadly effect when inoculated on the rabbit. As in other forms of microbial enteritis, that of fowls is undoubtedly favored by general and local debility, youth, old age, unsuitable food and other health depressing causes, yet as the specific pathogenic microbe has been in many cases identified, it is well to consider some of the different species.

Bacillus gallinorum found by Klein (1889) in the blood of chickens suffering from an infective diarrhoeal enteritis, is ovoid, with rounded ends, from 0.8 to 2μ long and 0.3 to 0.4μ thick; often in pairs. Stains in the aniline colors. Aërobic (facultative anaërobic) non-liquifying, non-motile, asporogenous. Culture easy in neutral, alkaline or slightly acid media at room temperature, or better in thermostat. On gelatine plate films, it forms grayish white, superficial colonies, becoming flat homogeneous white discs, brownish under transmitted light. The deeper colonies are small spherical and brown or yellowish by transmitted light. On agar it forms a thin gray layer with irregular margins, which extends over the entire surface. In bouillon it causes turbidity and in 24 hours a precipitate of bacilli to the bottom.

Pathogenesis. Chickens inoculated hypodermically or intravenously die in 1 to 5 or 6 days with peritonitis and intense intestinal congestion. Fed in vegetable food it is harmless, but with animal food virulent. Rabbits and pigeons are immune.

Pond water is a common source of casual infection, also dung heaps in which carcasses of little chicks have been buried. Summer is the period of greatest prevalence, as there is the best opportunity for the multiplication of the germ, and the drying of the ponds concentrates the product.

The bacillus is found on the intestinal mucous membrane, and in the mucus and in advanced stages, in the blood, spleen, liver and kidneys.

Bacillus Coli Communis, the familiar bacillus of the healthy bowel, is charged by Lignieres with causing a fowl enteritis and probably does so as in mammals when the mucosa has become diseased and non-resistant. At the same time there are so many closely allied forms or varieties of this bacillus found in different intestinal diseases, that it may well be that the pathogenic agent is a modified form or "sport" from the parent microbe, though no clearly defined peculiarities can be established by cultures.

The typical colon bacillus is 2 to 3 μ long by 0.4 to 0.6 μ broad, with rounded ends, but it may be ovoid or even round, or it may be 5 μ long. It stains readily with aniline colors, bleaches with iodine. It is aërobic, facultative anaërobic, non-motile, non-liquifying, and asporogenous. It ferments all sugars producing gas, acidifies its culture fluids, and coagulates milk. It grows freely at room temperatures in peptonized gelatine, agar and bouillon and on potato. Stab cultures in gelatine have a moss-like tufted appearance.

Pathogenesis. Injections subcutem, and into the veins and ingestion with food all failed to infect the chicken, while the pigeon died in 24 hours from intravenous injection and in 12 to 18 days from 1 c.c. given subcutem. In rabbits and guinea-pigs hypodermic injection caused abscess, while

pleural and peritoneal injections killed in 24 to 48 hours. Rabbits are unaffected by intravenous injection, while guinea-pigs die in 1 to 3 days.

Bacillus of Duck Cholera found by Cornil and Toupet in the blood of ducks suffering from a diarrhoeal enteritis, is 1 to 1.5μ long, by 0.5μ broad, with rounded ends. It is aerobic, non-liquifying, non-motile and asporogenous. Stains in the aniline colors and bleaches in iodine.

Pathogenesis. In morphology and cultures it resembles the bacillus of fowl cholera, but it fails to infect chicken, pigeon or rabbit. It infects ducks readily by ingestion or hypodermic inoculation.

Spirillum Metchnikowi was found in 1888, by Gamaleia in the ingesta of chickens dying in Russia of a choleraic enteritis. It resembles the cholera spirillum, but is shorter, broader and more curved. Its size varies, being twice as broad as the cholera spirillum, when found in pigeons. It may be 0.8μ long, by $.5\mu$ broad, furnished with one polar flagellum and very motile. It stains in aniline colors and bleaches in iodine. Grows readily in common media at room temperature, and is killed in five minutes by 122° F.; renders milk strongly acid, coagulating it, and perishes in the acid. In eggs turns the albumen yellow and the yolk black. In gelatine it forms transparent colonies and in potato pale brown.

Pathogenesis. By inoculation it infects chickens, pigeons and guinea-pigs, while rabbits and mice are refractory except to large doses. By ingestion it infects chickens and guinea-pigs but not pigeons. Infection takes place easily by the air passages. In all cases alike the lesions are concentrated in the intestines.

Lesions. These are very similar in the different forms. The intestine is violently congested and contains a quantity of yellowish green mucopurulent or serous fluid. The mucosa is infiltrated, softened and even abraded by the desquamation of epithelium. The liver is greatly enlarged and softened and gorged with blood, and the gall bladder filled. The spleen is enlarged and pale, contrary to what is seen in fowl cholera, and the kidneys are congested. The heart is flaccid, soft, petechiated, and the pericardium is the seat of serous effusion.

Symptoms. In the *acute form* there is dullness, langor, inappetence, ardent thirst, pale comb, and greenish faeces. Later the feathers are erect, the wings and tail droop, the head sinks, the patient gapes frequently, walks unsteadily, and a liquid bluish green diarrhoea sets in, which later becomes yellow and bloody. The somnolence increases, the walk becomes more unsteady, or the patient sinks down with eyes half closed and refuses to rise. As the disease advances the comb becomes violet, the dark shade constantly increasing and a glairy grayish mucus is discharged from the nose and bill. The temperature which was at first raised 1° or 2° , falls 2° or 3° below the normal prior to death, which may be deferred to near the end of the second week.

In the chronic cases the disease may drag along for three weeks, the emaciation, pallor and weakness constantly increasing and the feathers around the anus soiled and matted together by the foetid liquid discharges. There may be remissions which go on to complete convalescence but more commonly an exacerbation occurs which proves fatal in a day or two.

Diagnosis. From fowl cholera this may be distinguished by the fact that it is confined to one farm or flock of turkeys, chickens or ducks, proving most deadly in early summer, to the broods of the same spring, and at the commencement of the epizootic, and proving less and less so as time passes. The immunity of rabbits even when inoculated is a further distinguishing feature. From intestinal parasitism it is distinguished by the color of the discharges, and the absence of worms and their eggs from these liquids.

Mortality is often very high. Klein found it 80 per cent.

Prevention. Remove the infected from the flock (with ordinary fowl it is often best to kill and burn or bury them), keep the poultry house and yard scrupulously clean of droppings and sprinkle it occasionally with a 3 per cent. solution of sulphuric acid. The poultry house may be fumigated with sulphur (1 ounce to the cubic yard), or the walls and roosts may be washed with a solution (1:12) of bisulphide of carbon in liquid vaseline. The diseased must be removed as soon as they are detected and food and water must be given pure. If pure water is not available, boil it or render it acid by sulphuric acid (1:33), and feed grain, cooked roots, bran and bread with more or less green food.

In the case of valuable birds immunization may be secured by inoculating with the virulent blood or culture so diluted that not more than one or two of the germs shall be inserted in each case or the virulent liquid may be heated for 20 minutes to a temperature of 55° C (121° F) and then injected in a dose of 2 drops.

Treatment. If it is decided to treat the sick they should be placed together in safe seclusion from all others. Feed with mush or cooked roots or vegetables adding salol $\frac{1}{2}$ dr. naphthol 1 dr. and quinia 1 dr. to the food of 15 or 20 fowls. Nitrate of bismuth and powdered charcoal may be added in moderate quantities. As drink give water containing 2% of sulphuric acid. Antiseptic enemata may be added in the case of very valuable birds, salol, naphthol, boric acid, salicylate of soda, or solution of carbolic acid or creosote. Stimulants and tonics are highly esteemed by some, and Cadeac recommends the free use of the following mixture: powdered fennel, anise, coriander and quinia of each 5 drs., gentian 10 drs., ginger 12 drs., ferric sulphate $2\frac{1}{2}$ drs.

COCCIDIAN ENTERITIS IN CATTLE. COCCIDIOSIS. RED DYSENTERY.

Definition. Distribution: Switzerland in summer. Causes: weakness, debility, youth, cold, heats, spoilt fodder, protozoa, coccidium oviforme, coccidium perforans. Lesions: reddening, thickening and desquamation of alimentary mucosa and lungs, congested mesenteric glands, liver and spleen, coccidia in discharge and epithelium, staining, anæmia. Symptoms: chill, fever, grinding teeth, foetid diarrhoea becoming bloody, tenesmus, stiffness, red ulcerated rectum, emaciation, false membranes. Duration: death in one to fourteen days or more. Complications. Diagnosis by coccidia. Prevention: avoidance of affected soils, water and fodder. Treatment: antiseptic, by mouth and as enemata, demulcents. Flesh safely eaten by man. Coccidium bigeminum in dogs, coccidium oviforme in rabbits, coccidium tenellum and gregarina avium intestinalis in birds.

Definition. Enteritis affecting chiefly the colon and rectum, and due to the presence of the protozoa, coccidium oviforme, and coccidium perforans.

Distribution. This affection was found in 1885 in the cantons of Berne, Lucerne and Argovi where it attacked 5 per cent. of the cattle and destroyed from 2 to 4 per cent. of those that suffered. It prevailed mainly in the summer (May to October) on the pastures, though not unknown at other seasons. Sucking calves were immune and the ages of a year to two and a half years were the main sufferers.

Causes. Predisposing causes embrace such as induce weakness or debility, youth, low condition, cold intemperate weather, extreme heats, musty or spoilt fodders.

The essential cause, the coccidium, appears to be taken in with food or water as sucking calves are largely exempt. The parasite is found in the gastric and intestinal epithelium of the diseased animals, in numbers proportionate to the severity of the attack, being very abundant when the disease is at its height, diminishing during convalescence, and disappearing entirely on recovery. Two species are found in this disease.

Coccidium Oviforme is 40 to 50 μ long by 22 to 28 μ thick, ovoid, with a double outlined limiting membrane enclosing a refrangent protoplasm with a nucleus two or three times as large as that of an epithelial cell, and staining with hæmatoxylin or aniline colors. The young coccidia appear as round granular protoplasmic masses, without a capsule but provided with a nucleus. At first of a diameter of 9 to 10 μ they increase to 26 μ retaining the spherical form and acquiring the membrane of the mature parasite. As it grows the protoplasm separates from the wall and forms a globular nucleated mass, which after fifteen days divides into two and later into four masses or sporoblasts. Each sporoblast in its turn divides into two falciform corpuscles lying in contact but in an inverse sense to each other. Each of these under favorable conditions becomes a new amœboid individual capable of invading an epithelial cell and passing through the same stages of development as its predecessor.

The coccidium oviforme is found in the epidermis in cutaneous psorospermiosis of birds, and in the coccidian hepatitis in rabbits.

Coccidium Perforans. In its mature form this is 25 μ to 35 μ long, by 14 μ to 20 μ broad. Its different stages of evolution are essentially the same as for the Coccidium Oviforme. It is a cause of intestinal Coccidiosis in both dog and rabbit as well as in cattle, usually killing the rabbit in from eight to ten days.

Lesions. In the first reported cases in calves in 1877 (Proger and Zurn), there was catarrhal inflammation of the upper air passages as well as the bowels. There was thickening, redness and desquamation of the abomasum near the pylorus of the small intestine and colon. In the intestines there were patches of thickening and softening of the mucosa and miliary ulcers with yellowish contents. The mesenteric glands, liver and spleen were enlarged, softened and ecchymosed. In all the lesions coccidia were found. In the cases reported by Hess, Zschokke, Guillebeau and Cadeac the abomasum and large intestine principally suffered. There was a diffuse inflammation of the mucosa and in the rectum and portion of the colon a great degeneration and desquamation of the epithelium. The columnar cells of the mucous glands especially suffered. A single cell would contain five or six psorosperms, in different stages of development. If the parasites have escaped, the cell walls are pressed together. Among the diseased glands others with healthy epithelium were

found, their orifices plugged with mucus. Both forms of coccidia above described are found in two conditions, in the epithelial cells with granular nuclei staining in hæmatoxylon, and violet or black with iodine (Lugol's), and outside the cells with granular nucleus or simple hyaline contents which do not stain. Zschokke considers the latter as in process of degeneration. Other organs are anæmic.

Symptoms. The disease is ushered in by cold extremities, weakness, dullness, suspended rumination, ardent thirst, hyperthermia, 102° to 104° and even 106° , small, weak thready pulse beating 100 to 140 per minute, sunken eyes, grinding of the teeth, and defecation in small quantity only. Toward the fourth day or earlier a fœtid diarrhœa sets in, watery, bloody and fibrinous. The bloody discharges last to the seventh day, the diarrhœa to the end of the second or fourth week. Straining may be violent, exposing or everting the irritated or ulcerated rectum, the hind parts may be stiff, the patient rises with difficulty, he winches if pressed on the back or right side of the abdomen, loses flesh rapidly and becomes a walking skeleton. when the discharges are less profuse cylindroid croupous casts are sometimes expelled.

Course. Duration. In the weak and young a violent attack may prove fatal in 24 hours. In others the malady lasts for two or more weeks and sometimes relapses, and the patient becomes very weak and anæmic. Complications of various kinds may also supervene and cut off the animal, the lesions and debility alike favoring the introduction of the germ. Thus black quarter, bronchitis, pneumonia, convulsions, paralysis, phthiriasis and ring-worm have been noted as sequelæ.

Diagnosis can always be made by microscopic examination of the fresh warm fæces.

Prevention. This will consist in the avoidance of water and of green food from soils where the disease has been found to prevail.

Treatment. Antiseptics given by the mouth have proved of very little avail having as a rule lost their power through dilution in the contents of the stomachs. They may still be given to keep in check the propagation of the coccidia in the fourth stomach. Silver nitrate, iron sulphate, lead acetate, tannic acid, oak bark, carbolic acid, cresol, lysol, salicylic acid, hydrochloric acid, sulphuric acid, hyposulphite of soda have been used in this way. The same agents may be used with better effect as enemata being forced as far as is consistent with safety and frequently repeated. Quinia and iodide of potassium may also be given as parasitocides. Mucilaginous gruels and decoctions should be given at first, followed by mashes as they can be borne, but the food must be moderate and easily digestible until full convalescence has taken place.

The flesh of animals killed while suffering from this affection has been eaten by man with impunity.

COCCIDIAN ENTERITIS IN THE DOG. INTESTINAL COCCIDIOSIS.

Causes. Two parasites have been found in connection with this disease in the dog; the **Coccidium penetrans**, already described, and the **Coccidium bigeminum**.

Coccidium Bigeminum, var. Canis. This is elliptical and from 12 to 15 μ long by 7 to 10 μ broad. They are usually found in pairs lying side by side, and sometimes both in the same envelope indicating multiplication by division along the longitudinal axis.

The bigeminum is held to be harmless, but the perforans, as in the cow and rabbit is very injurious and even fatal. The *symptoms* are those of digestive disturbance, dullness, loss of appetite, retching, vomiting, colic and foetid and bloody diarrhoea. Irritability, and a morose disposition and loss of control over the hind limbs have been set down as rabiform indications.

Diagnosis depends on the inveteracy and sanguineous nature of the diarrhoea, but especially on the discovery of the amoeboid organisms in the recent, warm discharges.

Treatment should be along the same line as in cattle.

COCCIDIAN ENTERITIS IN RABBITS. INTESTINAL COCCIDIOSIS.

Causes. In rabbits the coccidium perforans is the psorosperm which usually attacks the bowels. The coccidium oviforme which produces hepatic coccidiosis is also occasionally found in the intestines. The perforans is smaller than in cattle being 15 to 25 μ long, by 12 to 15 μ broad, approximating to the variety found in man.

Lesions. There is extensive congestion of the intestinal mucosa, with thickening and softening of the epithelium so that it breaks down into a pulp under pressure, also free desquamation with the formation of abrasions, sloughs and ulcers. In some instances extensive croupous casts of the intestine are found. As in the other animals the coccidium is found abundantly in the epithelial cells of the affected parts which swell up and degenerate. When the parasite has escaped from the cells it lives free in the abundant mucopurulent and sanguineous secretions of the bowels.

Symptoms. There are loss of appetite, swelling of the abdomen, profuse diarrhoea, the fæces yellowish in color and containing mucopurulent matters and blood. The disease may prove fatal in a few days without much loss of flesh, but if protracted it leads to extreme anæmia, emaciation and debility and the animal dies in marasmus.

Diagnosis is always to be certified by the profusion of coccidia found in the fresh liquid discharges.

Prevention must be secured if possible by the removal of the healthy rabbits from the infected and from the hutch or warren in which the latter have been. The greatest care must be taken to prevent them from obtaining access to the droppings of the sick, or to streams, ponds or wells, into which the drainage from such manure can have found its way. The safest course is to destroy the sick and burn up them and all their droppings, as the latter ground into powder can blow on the wind.

Therapeutic treatment has proved unsatisfactory but may be attempted along the same lines as for the larger animals.

COCCIDIAN ENTERITIS IN BIRDS. INTESTINAL COCCIDIOSIS.

Two sporozoa are known to be pathogenic in the intestines of birds: the **coccidium tenellum** and the **gregarina avium intestinalis**.

Coccidium Tenellum. This has a nearly globular body 21 to 25 mm. long by 17 to 19 mm. broad, a very thin, delicate investing membrane, and has been found in the mucosa of the cæca of birds, producing a fatal typhlitis. The sporoblasts are developed in water outside the animal body and when taken in with the food colonize in the intestinal mucosa.

Gregarina Avium Intestinalis. When mature this is in form of a granular body with hollow spaces or utricles, is oval, or globular and measures 40 to 48 μ in diameter. The spores are 11 to 14 μ . They are found in the submucosa of the intestines in chickens, being taken in with food or water, and attack not the intestines only but the skin, the buccal and pharyngeal mucosa and even the liver and lungs. They traverse the mucous membrane and become encysted in the submucosa as white isolated or confluent points, disturbing the circulation and nutrition and destroying the nutritive and other functional activities of the mucosa.

Lesions. From the coccidium tenellum these are mainly found in the cæca and consist in intense inflammation, white lines formed by the parasitic colonies, desquamations of the epithelium and erosions and ulcers. In the early stages and in the absence of diarrhœa there may be simply thickening and induration of the mucous membrane and whitish colonies of the parasites. When there has been diarrhœa the contents are serous, or seropurulent, brick red, and filled with epithelial cells, red globules, leucocytes, fat globules and coccidia.

The gregarinæ are not confined to the cæca but scattered over the whole intestinal canal as white spots in the submucosa surrounded by congestion and degenerative changes. It has been found complicated with false membranes.

Symptoms. Dullness, anorexia, ruffled feathers, sunken head, trailing wings, slow uncertain gait, plaintive cries, with diarrhœa, passing through serous, brick red, and bloody. In small chicks it may be whitish and followed by constipation. Death is usually an early result.

Treatment. Hyposulphite of soda with carminatives (fennel anise, coriander, ginger, and gentian) has been given in boiled milk or bread. Quinia and iodine might be tried.

Prevention. Avoidance of infected roosts and runs, and above all of infected streams, wells and ponds, and the removal and cremation of the sick, followed by thorough disinfection, are much more promising than therapeutic treatment.

GASTRO-ENTERITIS FROM CAUSTIC ALKALIES AND ALKALINE SALTS.

Ammonia: Counter-irritation, fauces, larynx, bronchia, congestion and softening of the gastric mucosa, loose blood clots, solution of globules, dysphagia, salivation, in carnivora and omnivora vomiting, diarrhoea, white fumes with muriatic acid.—**Ammonia carbonate:** less corrosive, same symptoms.—**Potash lye,** deep corrosion, gastric congestions, fluid, blackish red blood.—**Soda lye.**—Carbonates of potash and soda, less violent, tests for potash and soda. Treatment: weak acids, demulcents, anodynes.

Aqua Ammonia. Lethal dose pure, horse 1 oz. and upward; cattle 2 ozs.; dog 1/2 dr. There may be sudden death from abstraction of water and cauterization of the fauces and larynx including at times the bronchial mucosa. In other cases death occurs later from gastro-enteritis, the mucosa of the alimentary canal being congested, softened and covered with bloody mucus. The blood in the vessels is of a dark red, coagulates imperfectly and the blood globules are dissolved changing the color to black, and then brownish red. Prior to death there is great distress, salivation, inability to swallow, swollen tongue, frequent pulse and respiration, cough, spasms, and sometimes the odor of ammonia. Retching and vomiting may be a feature in carnivora and omnivora, and diarrhoea if the case is not promptly fatal. The urine is not rendered alkaline. White fumes with muriatic acid indicate ammonia.

Ammonia Carbonate gives rise to the same symptoms and lesions with the exceptions that there is less corrosion of the mucosa, and no pure ammonia exhales in the breath.

Caustic Potash, Lye. Lethal dose, 5 grs. dog intervenous. In strong solution this is one of the most potent caustics, which penetrates deeply into the tissues and abstracting water cauterizes everything with which it comes in contact. It therefore produces the most destructive changes on the walls of the stomach, and intestines, with violent gastro-enteritis, retching, or vomiting of alkaline matters. The congested, ashen and even black color of the tongue, and (post mortem) of the gullet, stomach and intestines, and the intense alkalinity of contents are characteristic. The blood is fluid, gelatiniform, and blackish red. Later, erosions and contractions are common.

Caustic Soda is only less destructive than potash, and produces the same general lesions and symptoms.

The **Carbonates of Potash and Soda** have the same general properties only they act with very much less energy. Potassium can be recognized by its purple color in an alcohol or Bunsen flame and sodium by an intense yellow.

Treatment. Weak acids (acetic, vinegar, citric, malic, lactic, boric, benzoic, salicylic) or the stronger mineral acids largely diluted. Mucilaginous solutions (flaxseed, elm bark, gruel, mallow). Anodynes (opium).

GASTRO-ENTERITIS FROM CAUSTIC ACIDS.

Sulphuric acid, corrodes, blackens, dysphagia, salivation, retching, vomiting, colics, collapse. Lesions. Test, barium nitrate. Nitric acid, corrodes, stains yellow, or brown. Test, reddish fumes with copper and sulphuric acid. Muriatic acid, corrodes, whitens. Test, chlorine odor, white curdy precipitate with silver nitrate. Oxalic acid, colic, emesis of black bloody matter, gastric mucosa red or black, blood bright red, lowered respiration, innervation, temperature. Acetic acid, congestion, softening of gastric mucosa, may stop heart. Treatment: weak bases, magnesia, lime or their carbonates, soap, lime water, demulcents.

Sulphuric Acid. This acts on stomach and intestine as on the mouth abstracting water and blackening the tissues. It produces dysphagia, salivation, retching, vomiting in carnivora and omnivora, colics, and collapse. Sometimes the urine becomes albuminous or bloody. The post mortem blackness of the contents and walls of stomach and intestine and their intense acidity are characteristic. Nitrate of baryta will precipitate the insoluble sulphate.

Nitric Acid. In concentrated state this acts in the main like the sulphuric acid, but stains the lips yellow, and the mucosæ white changing to citron yellow or brown and does not precipitate baryta. It gives reddish fumes with copper and strong sulphuric acid.

Muriatic Acid. This is less caustic than nitric or sulphuric acid, and may be recognized by its white cauterized patches on the mucosa of the mouth, stomach and intestines, its chlorine odor, and the curdy precipitate which it throws down with silver nitrate. It does not corrode the skin. In the stomach this acid is normally present in the free state.

Oxalic Acid. Lethal dose, dog 15 grs., cat 2 grs. When swallowed this causes colics, emesis in vomiting animals, the rejected matters being black and perhaps bloody. After death the gastric contents and walls and those of the bowels are congested and more or less blackened, and the blood of a bright red color. Heart is arhythmic, respiration slow, paresis of limbs, spasms, temperature subnormal.

Acetic Acid. This causes congestion and softening of the gastric and intestinal mucosa, colics, emesis in vomiting animals. It may kill by suddenly arresting the heart's action.

Treatment for the acids consists in alkaline or basic antidotes; magnesia, or its carbonate, lime, or its carbonate, soapsuds, carbonates and bicarbonates of potash and soda. Lime water is the one appropriate antidote to oxalic acid, precipitating the insoluble oxalate. Mucilaginous drinks may be added freely.

POISONING BY SODIUM CHLORIDE.

Poisonous with privation of water. Dose: horse, ox, pig, dog, hen. Symptoms: anorexia, thirst, emesis, colics, diarrhoea, dysuria, weakness, spasms, palsy, death in six hours to two days or more. Lesions: gastric congestion, ecchymosis, blood fluid, bright red, cerebral congestion. Treatment: emesis, water, demulcents, enemata, oils, cold to head. Brine: salt, ptomaines, toxins, from salt meat and fish. Strongest at three months old. Dose: horse, pig, dog. Symptoms: nervous, disorder, mainly spasmodic. Lesions: congestion of encephalon. Treatment: anodynes, stimulants, wine, camphor.

Common salt is especially irritant if given in concentrated solution and with subsequent deprivation of water. The poisonous dose for the horse is 2 to 3 lbs. (Gohier), for cattle 4 to 5 lbs. (Hertwig), for the pig 7 to 8 ozs. and for the dog 6 to 7 ozs. Chickens are poisoned by picking up broken pieces of salt instead of pebbles, or by salted food.

Symptoms. Anorexia, intense thirst, dullness, emesis in vomiting animals, colics, watery diarrhoea, frequent urination, muscular weakness, spasms, paralysis, weak pulse, red buccal mucosa, dilated pupils. Death may take place in six hours or it may be delayed two days or longer. In chickens giddiness and rotary movements are common.

Lesions. Congestion of the stomach and intestines with points of ecchymosis. The mucosa of the bladder is reddened. The blood is fluid and of a bright red. There is more or less congestion of the cerebellum and medulla and their meninges.

Treatment. Emetics (tepid water, tickling fauces) and the stomach pump. Abundance of water or of mucilaginous drinks. The same liquids by the rectum. Bland oils may be given as emollients and eliminating agents. Cold to the head is usually desirable.

POISONING BY BRINE.

This is partly due to the toxic effects of common salt but also to the ptomaines and toxins formed in old brine. It has been seen most frequently in hogs fed on salted kitchen waste and on the liquids from salt meats, (beef, pork, fish). Herring-brine is a common source of poisoning for hogs and dogs, also the brine from the salted meats of the butcher's shops. Reynal found it to be especially poisonous when at least four or five months old. He gave as the fatal doses for horse 3½ pints, for pig ½ pint, for dog 6–7 ozs. The lethal dose however will vary with the concentration of the fluid and its age.

Symptoms. In addition to the direct irritation caused by the sodium chloride there are marked nervous symptoms, nervous irritability, spasms, rolling of the eyes, convulsive winking, dilated pupil, blindness, vertigo, staggering gait, epileptiform seizures, trismus, oposthotonos, pleurosthotonos, stupor.

Lesions. In addition to those of the stomach and intestines there is marked congestion of the encephalon, especially the medulla and cerebellum.

Treatment. In addition to that for common salt, anodynes and stimulants (wine, camphor) may be demanded.

NITRATES OF POTASH AND SODA.

Lethal dose. Symptoms: Colics, tympany, emesis, salivation, pupillary dilatation, diarrhoea, diuresis, hypothermia, stupor, palpitations, trembling, spasms, palsy. Lesions: Congested gastro-intestinal mucosæ, erosions, ecchymosis, congested kidney and bladder. Blood fluid, bright red. Treatment: Emesis, stomach pump, water and mucilaginous liquids, stimulants.

Nitrate of soda being used largely as a top dressing for grass and other crops is more liable to be taken in toxic doses than nitrate of potash. The former is also given in mistake for common salt. The toxic dose is high. Morton gave 2 pounds saltpeter to a horse with only a temporary purgative and diuretic effect. Huzard found that 3 doses of 16 ounces each, given at intervals of 8 days sufficed to kill a horse. Cattle are said to have died from taking 5 to 6 ounces.

Symptoms. There are colics, tympany, emesis in vomiting animals, salivation, dilated pupil, diarrhoea, diuresis, hypothermia, stupor, palpitations, weak pulse, trembling, convulsions, tetanic symptoms, paralysis.

Lesions. Congestion of a cherry red, brown or purple of the gastric mucosa and that of the small intestine, erosions and ecchymosis, congestion and ecchymosis of the kidney and of the mucosa of the bladder. Blood uncoagulated and of a bright red.

Treatment. Favor emesis or use stomach pump according to the species of animal. Give abundance of water or mucilaginous fluids by mouth and anus. Stimulants may be resorted to in case of sinking or collapse.

ACUTE ARSENICAL POISONING, ARSENIOUS ACID, PARIS GREEN.

Acute poisoning. Sources. Lethal dose, horse, ox, sheep, dogs, pigs. Endermic. Symptoms: colic, weak pulse, rapid breathing, red eyes, dilated pupils, purging, weakness, trembling, stupor, convulsions, paralysis, green or yellow vomit, test of urine. Lesions: gastro-intestinal inflammation, ropy, bloody mucus, ecchymosis, extravasation, ulceration. Treatment: emesis, stomach pump, hydrated iron oxide, calcined magnesia. —*Chronic poisoning.* Excessive administration, arsenical fumes from smelting furnaces. Symptoms: indigestion, emaciation, hide-bound, depilation, red eyes, diarrhoea, agalactia, weakness, paraplegia, salivation. Lesions: as in acute, fatty liver, test of urine. Tests: on hot charcoal garlic odor, Marsh's test by zinc and sulphuric acid. Treatment: avoidance of cause, antidotes demulcents, diet.

Arsenious acid given recklessly as medicine, rat poison, arsenite of soda made into a sheep dip and left within reach of animals, and arsenite of copper used for potato bugs, or other insect pests and carelessly left where animals can get it are the most common sources of acute arsenical poisoning. Horses die from 140 grains in solution, or 3½ drs. in the solid form, cattle from 3½ to 7 drs., sheep are killed by 2 drs., dogs may die from 2 grs., but larger doses usually cause vomiting and the animal is saved. Hogs may die from 15 grs. but they often save themselves by vomiting with much larger doses. The poisonous effects may be induced by putting arsenic on a raw sore.

Symptoms. Violent colic, quick, feeble, irregular pulse, hurried respiration, emesis in vomiting animals, ardent thirst, purging, tenesmus, lowered or unevenly distributed temperature, red eyes, dilated pupils, and nervous symptoms, weakness, trembling, stupor, convulsions and paralysis. The urine is albuminous and may be bloodstained. In case of Paris green, the green color of the vomit, and of arsenious acid, a yellow color may be looked for. Test urine for arsenic.

Lesions. Inflammation of the stomach and small intestine, with ropy, often bloody mucus. Ecchymosis, and extravasations appear in the stomach. Ulceration is not common in very acute cases, but in protracted cases, it is usually present in the stomach (abomasum in cattle). Petechiæ may be met with in different internal organs and in protracted cases, fatty degeneration of liver, heart, or kidney.

Treatment. In vomiting animals encourage emesis by tepid water, tickling the fauces, or giving ipecacuan. For non-vomiting animals the stomach pump may be tried. The available antidotes are hydrated oxide of iron, or a solution of calcined magnesia. To make the first, mix 100 parts sulphate of iron in solution with 250 parts magnesia in solution. This should be given liberally and often: horse or ox 1 qt., sheep or pig 2 ozs., dog 1 oz. A simple aqueous solution of calcined magnesia also forms with arsenic an insoluble combination.

CHRONIC ARSENICAL POISONING.

This comes from continuous injudicious dosing with arsenic, or from the condensing on the grass of the vapors from the smelting of ores containing arsenic.

Symptoms. There is chronic indigestion, emaciation, hide-bound, depilation, red or weeping eyes, chronic diarrhoea, suppression of milk in cows, muscular weakness, paraplegia, soreness of the gums, salivation.

The *lesions* are essentially the same only less intense than in the acute form. Hugo found in the intestines of poisoned animals a slimy, serous, grayish white fluid, and a false membrane like a frog spawn streaking the intestine. Later this may be dense like a diphtheritic membrane. There was fatty degeneration of the liver and of the gall bladder epithelium. Injection of the capillary vessels of the brain and pia, and effusion into the ventricles and on the surface of the brain were found in dogs.

Elimination. Arsenic is eliminated mainly in the urine, but also in part in the bile and perspiration.

Tests for Arsenic. When taken in the solid form it may be found undissolved on the gastric mucosa.

If burned on red hot charcoal or iron it gives out the odor of garlic.

Marsh's test consists in evolving arseniureted hydrogen from zinc and sulphuric acid to which a little of the suspected liquid has been added. A flask is taken having a cork conveying two tubes, one a funnel reaching nearly the bottom of the flask, and the other a delivery tube of some length and provided with a chloride of calcium bulb, and at its end turned up at right angles and drawn out to form a narrow orifice. Pieces of zinc are placed in the bottom of the flask, and sulphuric acid is poured upon these through the funnel. This causes the evolution of hydrogen. The suspected liquid is now added, and the gas issuing from the delivery tube having been lighted, a piece of cold white porcelain is held above and a short distance from the flame. A dark metallic spot of arsenic is obtained. The stain obtained by antimony differs in being formed more closely to the flame, in volatilizing less rapidly under heat, and in forming a black or orange instead of the canary yellow sulphide when subjected to a stream of H_2S . with gentle heat. Chloride of lime dissolves the arsenic stain but has no effect on the antimonial one.

Treatment. Avoid the causes. Employ the antidotes in small doses once or twice daily. Check the diarrhoea by mucilaginous agents, and nourish the animals well.

POISONING BY PHOSPHORUS.

From matches, rat poisons, fatal dose, horse, ox, pig, dog, chicken. Symptoms: Anorexia, dysphagia, swelled tongue, thirst, colic, emesis, vomit phosphorescent, or bloody, diarrhoea, icterus, agalactia, trembling, weakness, choreic spasms, hemorrhages. Lesions: Buccal-gastro-intestinal inflammation, fatty degeneration of epithelium, liver, kidneys, heart and muscles, black blood, ecchymosis, hemorrhage, phosphorescent ingesta. Treatment: Emesis, stomach pump, oil of turpentine in mucilage.

Phosphorus is usually taken in the form of matches or more frequently as one of the pastes sold for the destruction of vermin. Almost all of the latter contain 1 to 2 per cent. of phosphorus, in combination with flour, sugar, and fatty or oily matters. The lethal dose of phosphorus is: Horse or ox 7 to 30 grains; pig 2 to 4 grains; dog $\frac{3}{4}$ to $1\frac{1}{2}$ grain; chicken $\frac{1}{3}$ grain.

Symptoms. Anorexia, dysphagia, swelling of the tongue, intense thirst, colic, emesis in vomiting animals, vomited matters may shine in the dark, and may be tinged with blood, diarrhoea is common though not constant, icterus, and suppression of the milk. Trembling, weakness, acceleration of the pulse and breathing and hyperthermia may be noted. Chickens have been noticed to have choreic movements in walking. Hemorrhages are common.

Lesions. Inflammation of the mucosa of the alimentary canal from the mouth to the stomach and intestines. Fatty degeneration of the epithelium very noticeable in the gastric glands. Enlargement and fatty degeneration of the liver and kidneys, and degeneration of the heart and muscles. The blood is black and ecchymosis and hemorrhages appear on various internal organs. The contents of stomach and bowels shine in the dark. Phosphorus may be recognized by its luminosity when distilled at a very low heat from an acid solution and received into a refrigerated condenser. This must be done in perfect darkness, and the phosphorescence will be seen in the condenser or connecting tube.

Treatment. Empty the stomach by emetic or stomach pump, and then give oil of turpentine in mucilaginous liquid: Horse 1 to 2 ounces; ox 2 to 3 ounces; pig $\frac{1}{2}$ ounce; dog 20 to 30 drops; chicken 5 to 10 drops. This may be repeated several times and if used early enough will probably succeed.

POISONING BY TARTAR EMETIC.

Fatal doses, large in herbivora. Symptoms: vomiting, diarrhoea, ulcerative stomatitis, salivation, thirst, colic, tremors, palsy. Lesions: gastro-intestinal inflammations, ulcerations, lung infarctions. Treatment: emesis, antidote tannic acid.

This is mainly seen in the smaller vomiting animals. To kill horses or cattle much larger doses are necessary than are likely to be taken. Barlow and Dun gave 4 drs. to a horse thrice a day, and in all 10 ozs. in 10 days, yet it improved in condition. Ten and a half ounces given in 6 days proved fatal. An old sow was killed by 2 drs., a 5 months pig by 80 grs., (Hertwig). Dogs were killed by 3 to 7 grs.

Symptoms. Vomiting, diarrhoea, ulcerative stomatitis, salivation, vertigo, thirst, dullness, cold surface, colicky pains, trembling, paralysis of the hind quarters, and early death.

Lesions. General inflammation of the gastric and intestinal mucosa, sometimes ulceration especially if the agent has been taken in the solid form. Congestions and infarctions of the lung are not unknown.

Treatment. Encourage vomiting by tickling the fauces, and by the ingestion of tepid water. The best known antidote is tannic acid in any one of its combinations. Solution of tannin, decoctions of oak bark, oak galls, catechu, kino, rumex, sumac, or even strong tea will serve to render it insoluble and non-irritant.

POISONING BY CORROSIVE SALTS OF MERCURY.

Calomel with muriatic acid, corrosive sublimate, mercuric chloride, iodide, nitrate, cyanide. Fatal dose. Symptoms: anorexia, salivation, thirst, emesis, colic, diarrhoea, rumbling, debility, tremors, stupor, death. Lesions: corrosive whitening of gastro-intestinal mucosa, congestion, ulceration, blackening, bloody, glairy ingesta. Treatment: albumen, emesis, demulcents, chlorate of potash, bitters, iron sulphate. Test: copper and muriatic acid.

Calomel in itself cannot be looked on as corrosive, but in ruminants in which it is retained in the system for 3 or 4 days it is largely resolved into mercuric chloride by the free gastric acid and alkaline chlorides. It has therefore been largely excluded from the materia medica of these animals. When in these or other animals it produces corrosive action, the operation is essentially that of corrosive sublimate.

The corrosive salts of mercury likely to be taken by animals are corrosive sublimate now so largely used as an antiseptic, the nitrates and iodides, and cyanides of mercury used as local applications or as antiseptics.

Mercuric chloride may be taken as the type. It has proved fatal to the horse in a dose of 2 drs.; to the ox in 1 to 2 drs.; to the dog in doses of 4 to 6 grs.

Symptoms. Loss of appetite, salivation, thirst, emesis in vomiting animals, colics, diarrhoea, often bloody, weak perhaps imperceptible pulse, hurried breathing, much rumbling of the abdomen, debility, trembling, stupor and death.

Lesions. Escharotic whitening in patches of the mucosa of the mouth, throat, gullet, stomach and intestines, with acute congestion, ulceration and ecchymosis, and sometimes blackening by the formation of the sulphide. The contents of the bowels may be serous or bloody and more or less glairy. Like arsenic, mercuric chloride concentrates its action on the intestinal canal by whatever channel it may have entered the body.

Treatment. The mercury should be precipitated in an insoluble form and then eliminated by emesis or by the stomach pump. White of eggs is usually the most available agent producing the albuminate of mercury. This is, however, still soluble in acid and alkaline liquids, in chlorides of potassium, sodium or calcium and even in excess of albumen. Vomiting may be favored by tickling the fauces, or by hypodermic injection of apomorphia. This may be followed by boiled flaxseed or copious drinks of rain water. When the mercury has been largely eliminated the salivation may be controlled by chlorate of potash, and the digestive disorder met by bitters and iron sulphate.

Test for Mercury. Place a few drops of the suspected solution on a clean surface of copper; acidulate with muriatic acid; then touch the copper through the liquid with a piece of zinc; a silver colored stain will be formed easily dissipated by heat.

POISONING BY SULPHUR.

Fatal dose, horse 16 ozs. Symptoms: dullness, anorexia, colic, diarrhoea, sulphur odor. Lesions: congestion, desquamation, ulceration of gastro-intestinal mucosa, sulphur odor in flesh, blood nearly fluid, blackens silver. Treatment: chloride of lime, oil, puncture rumen or colon, stimulants.

In excessive doses this is irritant. The horse is poisoned by 16 ozs. (Tabourin): violent colics follow a dose of 12 ozs. (Collaine). Cattle are less susceptible.

Symptoms. Dullness, anorexia, colic, pulse small and quick, skin cold and clammy. Foetid flatus and profuse diarrhoea, are marked symptoms.

Lesions. Injection of the gastro-intestinal mucosa, shedding of the epithelium, ulceration, and sometimes gangrene. Sulphur is found in the ingesta and faeces and a sulphur odor is prominent, not only in the bowels but also in the flesh. Tympany from H_2S is common. The blood is mostly fluid, and ecchymosis is shown on heart, lungs and other internal organs. The tissues blacken silver.

Treatment. Chloride of lime, oleaginous laxatives, use trochar and canunla for tympany. In prostrate conditions give stimulants (alcohol, ether).

POISONING BY BROMINE AND IODINE.

Poisoning by these agents is rare in domestic animals. Bromine 2 drs. killed a dog in 5 hours: 10 to 12 drops in 1 oz. water intravenously killed a dog suddenly, and 5 to 6 drs. of iodine by the mouth killed in a few days (Orfila). A horse had colic from taking $\frac{1}{2}$ oz. iodine (Tabourin), others died from the effect of 2 drs. doses intravenously (Patu).

Symptoms. Violent colicky pains, salivation, emesis in vomiting animals, diarrhoea, iodine or bromine odor, acute coryza, red eyes, dilated pupils, weakness, debility, vertigo, convulsions. If the patient survives the glandular system undergoes atrophy, with emaciation and scaly skin eruptions.

Lesions. After large doses there are congestion, ulceration, corrosion and sloughing of the œsophagus, gastric and intestinal mucosa, and more or less yellow discoloration of the parts. The odor is characteristic. With iodine there may be blue iodide of starch in the ingesta.

Treatment. Favor emesis by tickling the fauces and giving tepid water, or apomorphia subcutem. Boiled starch is the best antidote and may be given freely, both by mouth and rectum. Opium is often called for to relieve suffering.

POISONING BY COPPER.

Copper sulphate: Fatal dose, horse, dog. Symptoms: Dullness, colic, blue or green vomit, diarrhœa, straining, weakness, spasms, palsy, albuminuria, icterus, hæmoglobinuria, impaired appetite, emaciation, spasms. Lesions: Redness, softening, ulceration, sloughing of alimentary mucosa, methe-globinæmia, fatty liver, enlarged spleen, ingesta give copper film on polished iron. Treatment: Albumen, milk, mucilage, iron filings, sulphur, magnesia, laxatives, opium. Avoid acids.

The common copper poisons are the sulphate and acetate. Copper alum, oxide or carbonate of copper and paints with a copper base are less frequently taken.

Sulphate of copper 1 ounce has proved fatal to the horse. In dilute solution or with mucilaginous liquids it is much less injurious. Ten grains to 2 drachms subcutem have killed the dog. (Tabourin.)

Symptoms. Dullness, colics, emesis of blue or greenish matter in vomiting animals, diarrhœa, tenesmus, weakness, trembling, spasms, trismus, paralysis, small, weak pulse, hurried breathing. In experimental chronic poisoning in the sheep, albuminuria, icterus, hæmaglobinuria, hæmaturia, impaired appetite and rumination, constipation followed by diarrhœa, great emaciation and weakness and finally convulsions. (Ellenberger and Hofmeister).

Lesions. Redness and softening of the alimentary mucosa, ulceration, sloughing, perforation. In the chronic forms methe-globinæmia, free hæmatin as crystals in liver, spleen and kidney, nephritic extravasation, fatty degeneration of the liver, enlarged spleen, and catarrhal changes in the intestinal mucosa. Polished iron placed in the gastric or intestinal contents becomes coated with copper.

Treatment. White of egg, milk, mucilage, iron filings, sulphur, calcined magnesia, laxatives. Opium may be required to calm suffering, but acids must be carefully avoided.

POISONING BY ZINC.

Sulphate and chloride poisonous, less potent oxide and carbonate. Symptoms: colics, emesis, congested alimentary mucosa, diarrhœa, cramps, weakness, paresis, anæmia, emaciation. Lesions: white, leathery, sloughing or ulcerated alimentary mucosa, strictures. Treatment: emesis, demulcents, tannic acid, sodium carbonate.

The **sulphate** and **chloride** are the most likely to be taken in dangerous amount, the former being mistaken for Glauber salts. Three ounces of sulphate intravenously in the horse has proved fatal, or 10 to 50 grains in the dog. In the vicinity of zinc ore furnaces the agent is taken in on the fodder as oxide or carbonate.

Symptoms. There is much abdominal pain, emesis in vomiting animals, quick pulse, congested mucosæ, diarrhœa, cramps, weakness, and paresis, and if the patient survives, anæmia and emaciation.

Lesions. The mucosa of mouth, gullet, stomach and perhaps duodenum is white, opaque, hard, corrugated, leathery, sloughing, or ulcerated. Congestion is well marked. Strictures may appear in chronic cases.

Treatment. Give tepid water and tickle the fauces. Use white of egg or milk freely and mucilaginous agents. Tannic acid, or carbonate of soda are antidotal by tending to precipitate insoluble compounds.

POISONING BY SILVER.

Toxic doses of silver come mostly from materials used in the arts. The photographer uses chiefly the nitrate, iodide, bromide, cyanide and chloride. Taken into the stomach the silver salts are less poisonous because they are largely precipitated as insoluble chloride or albuminate. The chloride and albuminate are, however, soluble in solutions of alkaline chlorides and hence even they may poison.

Symptoms. Colic, emesis in vomiting animals the vomited matters blackening in the light, diarrhoea, great muscular weakness, paresis, weak clonic spasms, and disturbed respiration. The nervous symptoms are very prominent (Rouget and Curci). Chronic poisoning produces emaciation and fatty degeneration of liver, kidneys and muscles (Bogoslowsky).

Lesions. Patches of congestion and of white corrosion on the buccal oesophageal and gastric mucous membrane, the presence of the curdy white chloride of silver adherent to the gastric mucosa. In chronic cases the visible mucosæ and white skin may have a slaty color.

Treatment. Emetics in vomiting animals. White of egg, common salt largely diluted and followed by milk as antidotal, demulcent and nutritive agent.

POISONING BY BARIUM.

Poisonous salts. Symptoms: spasms, peristalsis, defecation, urination, restlessness, prostration, emesis, weak pulse, coma. Lesions: Moderate congestion of gastro-intestinal mucosa. Treatment: alkaline sulphate, anodynes, demulcents.

The salts of barium are irritant with a special action on the nervous system shown by weakened action of the heart and spasms or paresis of the muscles. The chloride is used in staining wool, the nitrate and chlorate in producing green colors in fireworks, the oxide and carbonate in glassmaking, the chromate by painters, and the sulphate for giving weight and body to various white powders. The chloride is now largely used to stimulate intestinal peristalsis in animals.

Symptoms. Barium chloride hypodermically produces tonic and clonic convulsions, increased peristalsis, discharges of urine and fæces, great restlessness, muscular prostration, emesis in vomiting animals, hurried, shallow respiration, weak, thready pulse, asthenia, coma, and death.

Lesions. There is congestion of the gastric and intestinal mucosa, but this is rarely violent, and corrosion and ulceration are almost unknown. The agent indeed seems to act more energetically upon the nervous system than on the mucosa of the alimentary tract.

Treatment. This consists in giving an alkaline sulphate (sulphate of soda, potash, or magnesia), to precipitate the insoluble barium sulphate, with anodynes (opium) and mucilaginous agents.

POISONING BY IRON.

Sulphate and chloride on empty stomach poisonous. Symptoms: Colic, emesis, rumbling, purging. Treatment: Alkaline or earthy carbonates, tannic acid, albumen. Opium.

Sulphate and chloride are the principal poisonous compounds. Both are comparatively harmless even in large doses taken on a full stomach, while on an empty stomach they may cause violent gastro-enteritis.

Symptoms. Colicky pains, purging, emesis in vomiting animals, more or less tympany and rumbling of the bowels, and surface coldness.

Treatment. Give carbonates of the alkalies, magnesia or lime to precipitate the comparatively insoluble carbonate or oxide; or tannic acid or infusion of oak bark or galls. White of egg, milk and mucilaginous agents, and opium may be required to allay irritation.

POISONING BY CHROMIUM.

Chromic acid, chromate and bichromate of potash. Corrosive. Cause: Gastro-intestinal inflammation, albuminuria, hæmaturia, emaciation, digital ulcers and sloughs, colic. Diarrhœa, vertigo, stiffness, weakness. Lesions. Treatment: Emesis, stomach pump, demulcents.

Bichromate of potash is used extensively in dyeing, calico printing, in the manufacture of porcelain, in chemistry and photography, and to a slight extent in medicine, while lead chromate (chrome yellow) is a valuable pigment. Chromic acid is one of the most potent caustics, at a moderately high temperature dissolving all animal products that may be subjected to it. The chromate and bichromate of potash are only less violently caustic, producing deep and fistulous sores on the hands of the dyers, and acting in a similar manner on the mucosa of the alimentary canal. Twenty-eight grains of the bichromate given by the stomach killed a rabbit in two hours, while 45 grains of the chromate had no such effect (Gmelin). Pelikan found that the bichromate acted like arsenic or mercuric chloride, producing violent irritation of the stomach and intestines, followed by albuminuria, hæmaturia and emaciation: 1 to 5½ grains proved fatal to rabbits and dogs.

Workmen inhaling the bichromate dust, have inflammation, ulceration and finally destruction of the nasal septum, together with skin eruptions and ulcerations.

Horses working at the factories have intractable ulcers of the skin and sometimes shed the hoofs (B. W. Richardson).

Symptoms. Taken by the mouth the bichromate causes colicky pains, emesis in vomiting animals, diarrhœa, great prostration, cold extremities, vertigo, stiffness or weakness of the hind limbs, dilated pupils, weak pulse and death. If protracted the urine may be bloody and albuminous.

Lesions. There is more or less intense congestion of the stomach and intestine, yellow shrunken mucosa, abrasions, sloughs, and ulcers, and congested kidneys with yellow cloudiness of the epithelium of the convoluted tubes and congestion of the glomeruli.

Treatment. Wash out the stomach by emesis or the stomach pump and use albuminous and mucilaginous agents freely.

POISONING BY CARBOLIC ACID.

Susceptibility of cat and other animals. Fatal dose. Effect of dilution and gastric plenitude. Concentrated is corrosive. Symptoms: Salivation, dysphagia, anorexia, thirst, emesis, colic, arched back, odor of the acid, dark, greenish brown, albuminous urine, tremors, lowered temperature, debility, stertor, stupor, coma. Lesions: Whitened corroded mucosa, inflammation, ecchymosis, dark, liquid blood, brain effusions or extravasations, nephritic congestion. Treatment: Vinegar, alcohol, emesis, demulcents, Glauber salts.

The cat is the most susceptible to this poison. For dogs, cats and rabbits from 3 to 4 drops per pound, is the minimum fatal dose. For an ordinary dog the lethal dose is $\frac{1}{2}$ to 1 drachm. The horse has taken 3 ounces without fatal results, and 15 ounces in a week (Munk). Much depends on the dilution of the agent and the plenitude of the stomach. In a concentrated state it acts at once on the mucosa as a caustic abstracting the water and forming a white eschar.

Symptoms. The concentrated acid causes salivation, dysphagia, anorexia, thirst, emesis in vomiting animals, colics, arched back, retracted abdomen, odor of the acid in the breath, dark or greenish brown albuminous urine, trembling, lowered temperature, debility, stertorous breathing, clonic or tonic spasms, paralysis of the hind limbs, stupor, coma and death.

Lesions. In the mouth, throat, stomach and intestines are whitish, cauterized patches, with active inflammation beneath and around them, and ecchymosis. The blood is fluid and dark colored, extravasations or effusions on the brain or in the lateral ventricles, with pulmonary congestion and parenchymatous nephritis.

Treatment. If available, give vinegar proportionate to the amount of carbolie acid taken. Alcohol is a good substitute. Emesis should be encouraged when available. Next mucilaginous agents and bland oils to dilute the acid are required. Glauber salts may assist in neutralizing and expelling the acid.

POISONING BY CREOSOTE.

Irritates, coagulates albumen, and blood, arrests heart, colic, emesis, salivation, labored breathing, vertigo. Lesions: Congestion, corrosion, dark clotted blood, odor. Treatment: Mucilaginous agents, emesis, oily laxatives.

On the mucous membrane creosote has a very irritant action, coagulating the albumen, causing violent inflammation. It also coagulates the blood and when injected into the veins, stops the action of the heart. Taken by the mouth it causes violent colics, emesis in vomiting animals, salivation, laborious breathing, convulsions, vertigo and death. At the necropsy the stomach is found congested of a dull red color, and corroded, and the vessels contain dark clotted blood. A dog died from a dose of 2 drachms. The odor of creosote is marked.

Treatment. Emesis, white of eggs, mucilaginous liquids, and oily laxatives.

POISONING BY SEEDS OF RICINUS COMMUNIS.

Superpurgation. Poison in seeds. Diluents, demulcents, stomach pump, laxatives if necessary.

An overdose of castor oil may kill by gastro-intestinal congestion and superpurgation. Apart from the oil however the seeds contain a very active poison, which has been fatally ingested by horses with grain or otherwise. Five and a half ounces of the seeds have proved fatal. (Pelletier. Wende).

The *symptoms* are those of acute colic and gastro-enteritis. The indications are to wash out the stomach by abundant demulcents and the stomach pump, and if necessary to hasten the expulsion of the offensive matters by bland laxatives (olive oil, Glauber salts).

POISONING BY CROTON SEEDS AND OIL.

Fatal dose without water. Vomiting animals. Superpurgation. Lesions. Treatment: diluents, demulcents, stomach pump. Opiates.

One drachm of croton seeds given to a horse, without water proved fatal in 24 hours (Morton); 2 drachms followed by all the water the horse would drink produced most violent catharsis followed by recovery (Hughes). Twenty to thirty drops of croton oil proved fatal; 8 drops in the jugular vein caused death (Hertwig). It is much less fatal to cattle. Dogs and pigs vomit it so readily that they usually survive with profuse catharsis.

The *symptoms* are profuse watery diarrhoea with tenesmus, congested mucosæ, rapidly increasing weakness and small pulse, becoming imperceptible.

The *lesions* are violent congestion of the mucosa of stomach and intestines, concentrated very largely on the cæcum and colon.

Treatment consists in abundance of mucilaginous liquids, which the animal readily drinks, and washing out the stomach with the stomach tube or pump. Opiates may be demanded to calm the pain.

POISONING BY EUPHORBIA.

In Europe *euphorbia lathyris* has been found to produce in animals, colic, constipation, tympany, followed by bloody diarrhœa, stupor and hæmaturia. In America the *euphorbia corollata* (large flowering spurge) and *euphorbia ipecacuanhæ* (ipecacuanha spurge) though less potent have a similar action. *Treatment* consists in favoring elimination by emesis, and abundant mucilaginous and demulcent agents.

POISONING BY BOX LEAVES.

The leaves of *buxus sempervirens*, used as a border in gardens, contains an acrid principle. After eating 1½ lb. a horse had colic, tympany and enteritis. After death the lesions of gastro-enteritis were found (Weiss). *Treatment* would be by stomach pump, laxatives, demulcents and anodynes.

POISONING BY DAFFODILS. (NARCISSUS POETICUS, AND NARCISSUS PSEUDO NARCISSUS).

These common denizens of gardens produce intense gastro-enteritis, profuse diarrhoea, spasms, stupor and weakness. *Treatment* will not differ materially from that advised in box poisoning.

POISONING BY RANUNCULUS.

The *ranunculus acris* (tall crowfoot), *repens* (creeping crowfoot), *sceleratus* (cursed crowfoot), and *bulbosus* (butter cup), are all more or less acrid and liable to produce gastro-enteritis when taken in quantity. They are usually avoided by animals but will sometimes be taken by accident with other vegetation. Sheep are said to eat *ranunculus bulbosus* with impunity (Daubenton). Both cattle and sheep suffered from the other species (Delafond, Lipp, Brugnone, Delplanque). There were salivation, colic, emesis in vomiting animals, diarrhoea, vertigo, spasms, grinding of the teeth, arched back and staring coat. *Treatment* would be emesis or the stomach pump when available, mucilaginous drinks and enemata. Johnson (Medical Botany of North America) had a herd of cows abort for years on a field thickly set with *ranunculus acris*, but which ceased to abort when removed to a field from which this weed was absent.

POISONING BY VERATRUM. VIRIDE. AMERICAN HELEBORE.

Used in the early days by New England farmers to destroy birds in the cornfields (Osgood). Taken internally it reduces the fullness and frequency of the pulse, and if the dose be large excites nausea, vomiting and purging with great prostration. In the horse I have found anorexia, irritability of the bowels, and frequent retching. The action is primarily on the heart and nervous system and incidentally as an irritant on the gastro-intestinal mucosa. *Treatment* consists in evacuation of the stomach and the free use of mucilaginous drinks and diffusible stimulants. Helleborus Niger, viridus and foetidus have analogous effects.

POISONING BY CICUTA MACULATA.

The American water hemlock is an energetic poison acting not only as a narcotic but as a violent irritant to the gastro-intestinal mucus membrane.

POISONING BY COLCHICUM AUTUMNALE.

This agent expends its energy mainly on the digestive and urinary systems. The symptoms are suppression of appetite and rumination, thirst, ptyalism, grinding of the teeth, colic, emesis in vomiting animals, profuse, watery foetid and often bloody diarrhoea, the frequent passage of a clear urine, abortion in pregnant females, with short difficult breathing, weak pulse, pale mucosæ, coldness of the extremities, trembling, muscular weakness, sunken eyes, dilated pupils, spasms and death. The activity of the plant is greatest in July and August. On post mortem examination the gastric and intestinal mucosa are violently congested and the lumen of the bowel filled with a thin bloody mucus. Congestion of the kidneys and bladder is usually present.

Treatment. Evacuation of the stomach, and abundance of demulcents.

POISONING BY SAVIN.

The tops of the *juniperus sabina* when powdered and given to dogs produce violent colic, vomiting, bloody fæces and urine, spasms, paralysis, and death, with lesions of gastro-intestinal and uro-genital inflammation. In cattle and sheep they caused tympany, anorexia, colic, hyperthermia, and constipation followed by a bloody diarrhœa. Horses took 4, 8 and even 12 ozs. twice daily for eight days without any ill effect (Sick).

Treatment. Evacuate the stomach and give demulcents.

OTHER VEGETABLE IRRITANTS.

List of gastro-intestinal irritants. Common Symptoms. General treatment: emesis, stomach pump, diluents, demulcents, laxatives, enemata, anodynes, antiseptics, tannic acid. Prevention.

Among vegetables which produce more or less disturbance of the digestion, or congestion of the digestive organs Cadeac names the following: **Acorns** in horses (Morton); **tares**; **bird's trefoil** (lotus corniculatus, Colin); **vetches** at ripening (Gerlach); **laburnum** (cytissus) horse and ox (Cornevin); **hybrid** and **sweet trefoil** (Pilz); **officinal melilot** (Carrey); the **field poppy**, **digitalis** and **snapdragon** often mixed with wheat and rye (Cornevin); **conium maculatum**, **cicuta virosa**, **yew leaves**, **lolium temulentum**, and other forms of **ryegrass when ripening**; **chickweed** (stellaria) killed 60 horses in 200 (Semmer); **clematis**, **aconite**, **tobacco**, **male fern**, **aloes**, **horsetail** (equisetum) when full of silica; **mercurialis annua**, **wild radish**, **resinous plants**, **potato tops**, **potatoes in excess**, or **green** from exposure to the sun; **Ænanthe Crocata** (water dropwort); **giant fennel**, **anemone**, **phytolacca** (poke root); **buckwheat** in flower (Moisant); **St. John's wort**, various species of **lathyrus**, **rhododendron**, **artichokes** in excess, **spurry seeds**, **galega**, **bryony**, the fruit of **melia azedarach** (in pigs) (Dreux); **nux vomica**, **podophyllum**.

It may be added that the plants credited with causing the "loco" disease (**Astragalus mollissimus**, **Hornii**, and **lentiginosus**, the **oxytropis Lambertii**, **mutifloris** and **deflexa**) cause diarrhoea and sometimes ulceration of the intestines.

The farina of **mustard** is sometimes mixed with linseed cake and (developing the active principles of that agent) produces a severe or even fatal gastro-enteritis in cattle and sheep. The wild mustard of the fields, being allowed to grow with the flax, or rape, the seeds mingle when harvested and thus the cake comes to contain an injurious quantity of the mustard.

Symptoms. These will vary much according to the predominating action of the individual poison on other organs, but when they irritate the gastro-intestinal mucosa they have this in common, that they impair appetite and rumination, produce colicky pains (perhaps salivation and vomiting), and constipation or diarrhoea of varying intensity.

Treatment. Apart from the individual treatment demanded by the special symptoms of disorder of other organs, it may follow the same general line for all: Unload the stomach by tepid water, ipecacuan, with tickling of the soft palate, or by the stomach pump or tube, and follow this by abundance of mucilaginous drinks. In cases attended by constipation a laxative of Glauber salts, or aloes may be demanded, or assiduous mucilaginous injections. With an excess of irritation anodynes may be indicated. When there is tympany and foetor of the discharges these must be met by non-irritant antiseptics, such as naphthalin or salol. For many of the vegetable poisons tannic acid proves advantageous, being at once an antiferment, and fitted to unite with organic alkaloids, rendering them less soluble and otherwise often changing their properties.

Prevention should be sought by removing all such poisonous plants from pastures, or land used for raising fodder crops.

POISONING BY CANTHARIDES AND OTHER INSECTS.

Action on genito-urinary and alimentary tracts. Dysphagia, congestion, retching, diarrhoea, with mucus and blood, diuresis, enuresis, albuminuria, retracted testicles, prostration. Lesions: urinary and gastro-intestinal congestion, ecchymosis. Treatment: emesis with ipecacuan, demulcents, avoidance of oils, alcohol and chloroform. Other vesicant beetles and larva.

Spanish flies have a primary physiological action on the genito-urinary organs, but when introduced by the stomach they prove direct and violent irritants to the gastro-intestinal mucous membrane. They cause redness of the buccal mucous membrane, difficulty of swallowing, retching, emesis in vomiting animals, diarrhoea with mucus and bloody fæces, diuresis or enuresis with albuminous urine, retraction of the testicles, prostration, perspiration, paresis and death.

Lesions. Active gastro-intestinal congestion with ecchymosis, marked congestion of the genito-urinary mucosa especially that of the bladder.

Treatment. Emesis with ipecacuan and tepid water, followed by abundant mucilaginous or albuminous liquids. Flaxseed tea, gum arabic, and white of eggs are useful. Avoid oils, alcohol and chloroform which dissolve the cantharides.

Among other insects which act as vesicants may be named the **cockroach** (*blatta orientalis*) and the **potato beetle** (*cantharis vitatta*), also the *cantharis cinerea*, *cantharis marginata*, *cantharis atrata*, and *cantharis nuttalli*. The larvæ of various lepidoptera, thus **army worm**, **Cnethocampa primivora**, **Cnethocampa processionea**, **liparis auriflua**, **lithosia crinola**, and the larvæ of the **artica cassus ligniperda**, and **pieris brassica** are covered with stinging hairs charged with formic acid and perhaps an enzyme, which are shed with the skin in passing into the state of chrysalis, and getting mixed with fodders produce violent stomatitis, hemorrhagic gastro-enteritis and nephritis.

POISONING BY FUNGI, BACTERIA AND THEIR PRODUCTS IN FOOD.

Poisons in spoiled food: Moulds, rust, smut, bacteria, toxins. Action of moulds on rabbits, on alimentary and nervous systems. Smuts, ergots and their congeners. Tetanizing and paralyzing products. Duration of symptoms.

Food is usually spoiled by the growth of moulds, rust, smut, bacteria and the toxins which they produce.

Kaufman has experimented with moulds on rabbits. He found that **aspergillus glaucus** (green mould) grown on bread produces a fatal infection in the rabbit even in very minute doses ($\frac{1}{10}$ milligramme); that it will attain this in a neutral or even slightly acid medium as well as in an alkaline one; and that the spores retain this pathogenic activity for six months at ordinary temperatures. The *aspergillus glaucus*, *penicillium glaucum*, and *mucor mucedo* affect the intestinal organs only, while *ascophora oidium aurantiacum* affect the nervous system as well. The **smuts** (*ustilago*) and **ergots** (*claviceps purpurea*) vary considerably in their potency according to the conditions of their growth and the stage of their development, yet experiment has shown a special action on the vaso-motor nerves leading to nervous disorders, circulatory troubles, and trophic disease. In connection with **ustilago maidis** (corn smut) there are usually found bacteria, such as **bacillus maidis** and **bacillus mesentericus fuscus**, and the combined products of these and the *ustilago* have been studied by Lombroso, Dupre and Erba. These observers isolated a red oil with the tetanizing action of strychnia, and oleo-resinous substances having bases which they named *maisine* and *pellagrozeine*, and which had a paralytic action on the nerve centres. Pellizi and Tirelli cultivated the bacteria of damaged maize and found that the sterilized cultures, introduced into rabbits hypodermically or intravenously caused muscular jerking, exaggeration of the reflexes, tetanic spasms and paralysis which lasted for fifteen days after the injection. This is exactly in line with the causation of contagious bacteridian diseases in which the ptomaines and toxins are, as a rule, the immediate pathogenic factors.

CRYPTOGAMIC POISONING IN SOLIPEDS.

Prominent symptoms, asthenia and vertigo. Vary with cryptogam, merge into zymotic diseases. Causes: grain harvested damp and moulded, bluish or greenish, hay greenish white, brown or black, clover reddish, musty fodder, and diuresis, indigestion, gastric intestinal and systemic paresis, somnolence, delirium. Rusts, spring and summer, their evolution. Bunt, smut, produce fever and paralysis, spasms, abortions and dry gangrene, buccal erosions; evolution of ergot, honey dew on leguminous plants causing skin disease, bacterial ferments, diplococcus, streptococcus from foul water, causing enteritis. Symptoms: *adynamic*, dullness, blunted sense, pendent head, ears, eyelids, congested, yellow, ecchymosed conjunctiva, fever, tympany, colic, constipation, dung small, round, coated masses, vertigo, sometimes fatal diarrhoea, or colliquative diuresis; *vertiginous*: fever, anorexia, yellow mucosæ, tardy breathing, costiveness colics, stupor, somnolence, giddiness, heavy steps, stumbling, delirium, push head against wall, clinch jaws, grind teeth, make walking or trotting or plunging motions, or pull on halter and fall, amaurosis, paralysis, coma. Remissions. Death in one day or upward. Resumption of functions and recovery. Diagnosis: from meningo-encephalitis. Lesions: gastro-intestinal congestion, infiltration, ecchymosis, fermenting ingesta, congestion of mesenteric glands, liver, brain and meninges. Leucin and tyrosin in urine. Treatment: stomach pump, antiferments, potassium iodide, purgatives, enemata; for brain, bleeding, sedatives, ice, snow, elevation, derivatives, prevent mechanical injury.

The most prominent features of cryptogamic poisoning in these animals are asthenia and vertigo. In dealing with such poisoning, however, we must bear in mind that we have in hand, not one particular disease but a group, differing among themselves according to the cryptogam and its products which may be present:—a group moreover which overlaps more or less the true zymotic diseases.

Causes. Oats, barley and other grain or fodder which has been put up damp, and especially ground feed, becomes speedily overgrown and permeated with moulds, especially **penicillium glaucus**, **aspergillus flavus** and **glaucus**, **mucor racemosus**, and **ascophora mucedo** which give a bluish or greenish color and heavy odor, rob it of its nutritive constituents and charge it with toxic products. On mouldy hay it is common to find **aspergillus candidus**, **botrytis grisea**, **torula herbariorum**, and **euotium herbarium** which form a greenish white or brownish dust. The **spoeria herbarium** is characterized by small black or brown spots with yellowish, brown or black spores. The peronspora trifolium attacks growing clover (clover sickness) and **isaria fuciformis** the fescue grasses. The latter has a red color and mucous consistency and is charged with producing fatal poisoning in cattle.

Mouldy or **musty** oats, or other grain or fodder have long been notorious for producing diuresis in horses with excessive elimination of phosphates, extreme emaciation, weakness and death. In other seasons, and probably because of a difference in the fungi or their products they have caused widespread enzootics of indigestion with paresis of stomach and bowels, and of the systemic muscles. Paraplegia is a common manifestation, suggesting lesions of the spinal cord, and in other cases there are general paresis, somnolence and delirium suggesting cerebral lesions, (Staggers).

Gillespie records an enzootic of gastro-intestinal indigestion and tympany among the horses of a battery of artillery in Afghanistan from eating mouldy grass. Fröhner, Martin and Varnell have seen cases of poisoning by moulds without digestive, urinary or febrile troubles.

Of **rusts** growing on grain crops there are two chief varieties; the **spring rust** (*uredo rubigo vera*) which commences as light yellow patches on the leaves and stems, which change to black as the fungus approaches maturity; and the **summer rust** (*uredo linearis*) which grows to a larger size and assumes a browner color. Each of these passes through an evolutionary cycle, the small preliminary patches (**uredo**) passing into the mature fungus (**puccinia rubigo vera** and **puccinia graminis**.) Then it must pass through an alternate generation on another family of plants before it can again grow on the gramineæ.

Bunt produced by the *tilletia caries* attacks the grasses and small grains. The growing seed

(wheat especially) is changed into a black or olive colored powder, having a fishy odor. If the stems are attacked the leaves become pale, withered and dry. It can only be detected by carefully examining the individual seeds.

Other forms of smut are the **ustilago carbo** and **ustilago maidis** the familiar black smuts of small grains and maize. These develop by preference in the growing seeds, but also in the stem and leaves. The *tilletia caries* is as a rule more poisonous than the *ustilago* the effects being mainly hyperthermia and paralysis. There is, however, a tendency to spasmodic contractions, and abortions, and dry gangrene will occur from smut. The author has seen a large herd of cattle attacked with gangrenous sores around the coronet, which were promptly stopped when the light, smutty ears of corn were no longer given. In other cases the hoof was in part separated from the quick and creaked when the animal walked.

In connection with the gangrenous ergotism of cattle, the author has found on the same farms and feed, horses with ulcers on the buccal mucosa and gastro-intestinal indigestion. **Ergot** affects the seeds in nearly all the small graminæ and is produced by the *claviceps purpurea* which first attacks the ovary of the seed (*sphacelia* stage), then it invades the whole seed which grows out from the glumes as a hard, dark or purple spur-like process (*ergot* stage), then falling on the ground it grows up as a minute stalk with rounded head containing spores.

Honey dew growing on leguminous plants is reputed to cause skin disease in white horses and on the white spots of dark horses, from which those not eating the diseased plants escaped.

Bacterial ferments have an equally bad reputation. Bastin records the poisoning of five foals by fermented rye; Dieckerhoff describes an acute gastro-enteritis with congestion and swelling of liver, spleen and kidneys, as the result of microbes and their products in the fodders. Galtier traced a pneumoenteritis in the horse to two cocci, a motile *diplococcus* and non-motile *streptococcus*. Both stained in aniline colors, and were bleached by iodine. They grew in ordinary culture media above 50° F. but most freely at 98° F. The animals were infected by drinking putrid water or spoiled fodders in which the microbes were contained. The change to boiled water in the former case led to their prompt disappearance. Reynal, Cailleux and Foucher have also adduced instances of severe enteritis in the horse from drinking putrid water. These animals showed active congestion of the intestinal mucosa with abundant infiltration of the submucosa.

Bouley found 14 cases in one stable, the owner of which had marketed the good fodder and kept the spoiled for home use. Barthelemy, Alasonniere, Lombroso and Hausmann, Clichy, Rey, Gamgee and others give similar examples.

A large number of observations show the dangerous results on the horse of mouldy bread, inducing colics, vertigo, profuse sweating.

Symptoms. When **Adynamia** prevails there is great dullness and depression, the senses are blunted, the head depressed resting on the manger, the eyes sunken, weeping and half covered by the drooping upper lid, the conjunctiva is congested, sometimes yellow or marked by petechiæ. The mouth is hot, the lower lips pendent, the tongue furred, the abdomen somewhat tympanitic, with slight colics, but with little rumbling or indication of peristalsis. There is a primary constipation, a few small, hard pellets being passed with effort. The temperature may be 102° to 104°, breathing short, pulse small, weak; the walk unsteady, the animal preferring to stand, completely apathetic. In some cases a profuse diarrhœa sets in and may prove fatal.

In the cases attended by diuresis, the weakness is extreme, emaciation advancing rapidly, but the other symptoms of nervous depression are less marked, the poisons being apparently eliminated by the kidneys (see *diuresis*).

In the **Vertiginous form** the disease may set in with more or less hyperthermia, anorexia, a dislike particularly of the spoiled fodder, yellowness of the visible mucosæ, slow breathing, small accelerated pulse, costiveness, tympany, colics more or less intense, tenderness of the belly, and sooner or later marked nervous disorder. This may be in the form of stupor, the head resting in the manger, the senses are manifestly clouded, the animal walks unsteadily, staggers, steps heavily, striking the feet against obstacles, and stumbling. At the end of a variable number of hours (2 to 6 or 8 after feeding) nervous excitability and vertigo may supervene. He may push the head against

the wall, the jaws clenched, grinding the teeth, the eyes fixed, pupils dilated, facial muscles contracted, respirations hurried, heart palpitating and the skin perspiring. He may continue in this position, moving his feet as if walking, or he may rear plunging his feet into the manger or fall back over, and rising push anew against any object he may come in contact with. Coulboux speaks of rabiform symptoms such as attempts to bite but any such deliberate purpose is rare.

There may follow complete amaurosis, insensibility to pricking of the skin, and even paralysis or coma. Hyperæsthesia may also be temporarily present.

Course. Remissions and exacerbations usually alternate, the duration of the former furnishing some criterion by which to establish a favorable prognosis. Death may take place in 24 hours or it may be delayed for several days. Recovery is usually heralded by the resumption of defecation and urination, and the return of appetite. It is liable to be at first only partial, some of the senses remaining dull, or a general stupor persisting.

Diagnosis. In all such forms of poisoning there is the history of the ingestion of the toxic matters, and in any suspicious looking cases a careful examination of the food should be made. From meningo-encephalitis the presence of the abdominal disorder will serve to identify and to incriminate the food.

Lesions. These vary much with the poison. There is always, however, inflammation of the gastrointestinal mucous membrane, usually with ecchymosis, and infiltration of the submucosa. The contents of the bowels are imperfectly digested, the mesenteric glands congested and enlarged, the liver congested and softened, and the brain and its meninges hyperæmic or infiltrated. The leucine and tyrosine present in the urine during the acute attack is said to disappear when improvement sets in (Pellagi, Azzaroli).

Treatment. The first object must be the removal or neutralizing of the poison. In some instances the stomach pump or tube might be tried. Usually one must fall back on antiferments such as naphthol, naphthalin, salol, salicylic acid, and above all iodide of potassium. The last checks the growth of the fungi or bacteria and favors elimination of the toxins. It may be given freely to act on the kidneys. Creolin, 1¼ drachm, repeated three times a day has been found effective (Albrecht). In addition the action of the bowels may be solicited by full doses of sulphate of soda and abundance of water.

When the brain is implicated Cadeac recommends bleeding as an eliminating as well as a sedative measure. In any case use cold water, snow or ice to the head, elevation of the head, and purgatives which may as a rule be doubled. Potassium iodide or other antiseptics should be pushed, and diuresis as well as a relaxed condition of the bowels maintained. Counter-irritants such as mustard may be applied to the abdomen, and enemata used at frequent intervals. It is important to fix the patient to a ring in the centre of a box stall or barn to keep him from injuring himself.

CRYPTOGAMIC POISONING IN RUMINANTS.

Moulds and bacteria in brewer's grains, or the marc of beet sugar or cider factories derange digestion, or cause abortion. Spoiled potatoes cause enteritis, vertigo, palsy, in sheep, nephritis and cystitis. Mouldy bread causes indigestion, urinary and nervous disorder. Mildew. Musty grain and fodder as in the horse. Ergot causes winter and spring gangrene of skin, feet, limbs, ears or tail, lethargy, palsy, spasms, delirium, abortion; variation in toxicity with stage and condition of growth, privation or liberal supply of water, or succulent vegetables. Symptoms: varying, mouldy bread causes digestive and urinary trouble, with marc or ensilage, develops slowly, impaired appetite, salivation, tympany, colic, diarrhoea, debility, paresis, spasms, delirium. Duration, 5 hours to 2 weeks. Gangrenous ergotism, necrotic sore, slough hard, dry, leathery, black, living parts at demarcation line pink or purple, puffed up, tender, necrosis involves all soft tissues and bone; nervous form: abortion form. Lesions: congestion of stomach, bowels, mesenteric glands, brain and meninges, petechiæ. Diagnosis, from anthrax, from coccidian hemorrhagic dysentery, from foot and mouth disease, from rinderpest. Prevention, stop or regulate the injurious fodder, salt and pack the fresh grains or marc. Treatment: antiferments, potassium iodide, saline purgatives, stimulants, oil of turpentine, injections, derivatives.

Causes. The growth of **moulds** on or in brewer's grains, which have been preserved without salting and close packing, has at times rendered them dangerous poisons (Duvieusart, Wehenkel, Schütz). The refuse or marc of beet sugar factories, or of cider works may act in a similar manner. These products, at first neutral or only slightly acid, undergo an acid fermentation, with an abundant production of acetic, lactic or butyric acid which adds materially to their action in deranging digestion. These agents usually require a large amount to prove deleterious, about 150 to 200 lbs. a day. Arloing found three active microbial ferments in the pulp of the sugar factories, and four in that of the distilleries. The marc of apples has even caused abortion (Cornevin).

Spoiled potatoes have caused adynamic enteritis, with vertigo and paralysis (Zimmermann, Grabin, Holme) and in sheep symptoms of nephritis and cystitis as well (Kloss).

Mouldy bread has been found to cause indigestion and cerebral disturbances in cows (Cagny) or nervous disorders without digestive, urinary or febrile trouble (Fröhner, Martin and Varnell).

Mildew on the leaves of a grapevine has also poisoned six cows (Bisseauge).

Musty grain and fodder has the same general action as on the horse and produces paraplegia and other nervous disorders with or without digestive troubles.

The **isaria fuciformis** has caused the death of cattle which ate the grasses infested by it.

When we come to the ergots and smuts we find even more evidence of poisoning than in the horse. Toward the end of our long winters in the Northern States we occasionally find widespread **gangrenous ergotism** from eating infested hay, the lesions varying from simple sores around the top of the hoofs, in the interdigital spaces or on the teats and mouth, to loosening of part of the sole or wall, shedding of the entire hoof or sloughing of the entire limb—just above the hoof, at the fetlock, or in the metatarsal region. Portions of the tail or ears will similarly slough. This appears to be mainly due to the lessening of the calibre of the capillaries by contraction of their walls, under the action of the ergotin and secalin, seconded by the cold of the season. Cold is, however, by no means essential to its production. The other most common form of ergotism is the action on the nervous system. The contraction of the cerebral capillaries and disturbance of the circulation lead in some cases to a condition of lethargy and apathy in which the animal fails to eat or ruminate and gradually falls into marasmus, or paralysis may be induced, or delirium and spasms. Then finally there is the familiar form of abortion induced apparently by the contraction of the involuntary muscles of the womb and of its capillary vessels.

There is, however, a great difference of opinion as to the deleterious action of ergot. Various experiments with large doses of ergot on pregnant animals have failed to produce any sign of abortion. The agent, however, varies in its nature according to the conditions under which it grew and the stage at which it was collected, so that the failure to produce the expected result in a given case can by no means be accepted as disproving its pathogenic properties under other conditions.

The same remarks apply largely to the action of the smuts, which are often eaten in large quantities with impunity, especially if plenty of water or succulent vegetables are allowed, whereas under other conditions as in winter, under the action of cold, with the usual water supply frozen up,

and no succulent food, it proves very destructive.

Symptoms. These vary with the particular poison: With mouldy bread the symptoms may come on promptly with indigestion, tympany, constipation, marked irritation of the urinary organs, and it may be nervous disorder. Sometimes, as noted above, the narcotic action is shown with paresis or paralysis and stupor without any manifest disorder on the part of the digestive or urinary functions.

Most commonly with mouldy fodders, grains, marc, or ensilage the results are tardily developed and only after long continued use of the spoiled food. There is then loss of appetite, and rumination, drivelling of saliva, some tympany, and abdominal pain shown by frequent movement of the hind limbs, lying down and rising. The bowels may be costive at first, but this early gives place to a foetid diarrhoea, with weak rapid pulse (100 per minute) palpitations and hurried breathing. The walk becomes weak, unsteady, staggering or stumbling, and there may appear marked paresis especially of the hind parts. When nervous excitement sets in there may be twitching of the muscles of the neck, shoulders or thigh; the eye rolls or becomes fixed and the pupils are dilated; the muscles of the face are contracted and the jaws clinched, with grinding of the teeth. Bellowing or pushing of the teeth and nose, the forehead or horns against the wall or other obstacles, or the dashing violently against obstacles is occasionally observed, and indicates in most cases an unfavorable termination.

The duration of the malady is uncertain. It may not be over five or six hours in acute cerebral cases, and especially in sheep, and again it may be prolonged for one or two weeks. Death often takes place in convulsions.

In **gangrenous ergotism** a necrotic sore with more or less surrounding swelling may be seen, and a line of demarcation forms of a pink or purplish aspect along which the separation of the dead tissue takes place. The slough is usually of a dark red or black color, the red globules having apparently migrated into the tissues and piled up in the capillaries in the early stages of stagnation. When the line of separation is higher, the line of demarcation completely encircles the limb, the inflammation and swelling is very marked just above this line, the skin and soft tissues beneath drying and withering up into a dark red leathery mass, and this is gradually separated by the formation of a granulating surface above. The process of separation takes place much more slowly through the bony tissues, and not unfrequently the soft tissues having become detached, the lower part of the limb is separated at the first joint below the line of demarcation and the bone from that line down to its free end remains as a projecting necrosed stump. In the ear or tail the necrotic portion withers up into a stiff rigid shrunken slough which becomes detached sooner or later by mechanical violence.

In the **nervous ergotism** the symptoms are largely those of the adynamia, paresis and convulsions already described.

In **abortion from ergotism** there are usually few premonitory symptoms, and the occurrence is to be explained by the number of victims in a herd eating ergot or smut.

Lesions. These vary greatly. Usually the congestion and inflammation are most prominent in the abomasum and small intestine, complicated by ecchymosis and even extravasation which may so thicken the mucosa as to block the intestine (Walley). The mesenteric glands are usually gorged with blood and of a deep red. The brain may be nearly normal or violently congested and with its meninges covered with petechiæ.

Diagnosis. From anthrax this affection is distinguished by the absence of the specific large bacillus in the blood and of the marked enlargement of the spleen, by the great prominence of the nervous symptoms in many cases, and by the history of a dietetic cause.

From the coccidian hemorrhagic dysentery it is diagnosed by the absence of the coccidia in the stools and the predominance of the nervous systems.

From foot and mouth disease, the gangrenous ergotism is distinguished by the facts that the sores are in the nature of sloughs, and not vesicles, and that some members of the herd are almost certain to show sloughing of the limb at some distance above the hoof. More important still is the fact that the daintily feeding sheep and the pig kept in the same yards do not suffer from the ergotism.

From rinderpest it is differentiated by the fact that the sores on the mouth (when present) are not of the nature of epithelial concretions, and they do not appear on the vulva, and more significant still there is no indication of the introduction of the disease by contagion nor of its rapid progress from herd to herd. The immunity of sheep from gangrenous ergotism is another significant feature.

Prevention consists in putting a stop to the supply of the altered food or, if it must be given, in giving it in small quantities only with abundance of water or fresh succulent aliment. In the case of grains or marcs the fermentation may be checked by adding $\frac{1}{4}$ per cent. of common salt and packing the material firmly in a close box or silo.

In ergotism, succulent food, water ad libitum, stimulants, poultices, fomentations or wet bandages, a warm building and pure air are all important. Usually ergot and smut can be safely fed in relatively large amount with a liberal ration of potatoes, turnips, beets, green food or ensilage.

Treatment does not differ materially from that advised for the horse. Antiferments including potassium iodide, and saline purgatives stand at the head of the list. Stimulants may be demanded to rouse the torpid bowels and nervous system and unless contraindicated by gastro-intestinal inflammation oil of turpentine offers itself as at once stimulant, antiseptic and eliminating. Injections and counter-irritants are of use. Then cold (ice, snow, water) to the head, and the confinement of the patient so that he cannot injure himself or others are not to be neglected.

CRYPTOGAMIC POISONING IN SWINE.

Spoiled foods. Toxic rye. Botulism. Brine poisoning. Flesh of overdriven (leucomaines). Symptoms: from mouldy bread, brain disorder, gastric, urinary, hæmatic: from brine, restlessness, convulsions, eructations, vomiting, weakness, paresis. Lesions: congestion of stomach, bowels and mesenteric glands; with brine, heart and skin charged with black blood, congestion of brain, kidneys and bladder. Petechiæ. Treatment: empty stomach, emetics, purgatives, potassium iodide, antiferments, sedatives, cold to head, enemata, counter-irritants. Prevention.

Causes. Spoiled foods of all kinds such as mouldy bread, musty meal, spoiled grains of all kinds, or rotten potatoes, or apples are common causes of poisoning. In specimens of toxic rye Woronin found four fungi:—**fusarium roseum**, **gilberella Sanbinetti**, **belminthosporium**, and **cladosporium herbarium**. Prilleux and Delacroix have obtained a fifth, **endoconidium temulentum**. Rye so affected produced nervous disorder in swine, dogs, birds and even in man. Pigs often die from botulism, being fed flesh in a more or less advanced stage of putrefaction. Old meat brine is also a source of toxin and ptomaine poisoning since its toxic property increases with age which is not the case with a mere solution of common salt. Yet this is very deadly to the pig, a half a pint often proving fatal. Kuhnert records that the flesh of an overdriven horse proved fatal to the pigs that ate it.

Symptoms. From mouldy bread the pig becomes dull, stupid, sluggish, stiff, stilty and staggering in his gait, and usually costive. There are usually signs of colicky pains, inappetence, frothy lips, champing of the jaws, yawning, retching and vomiting. The mucosæ become yellow and the urine red and albuminous.

The symptoms caused by old brine are more violent. There are dullness, prostration, and inclination to lie, but at short intervals the animal gets up and wanders round, moving stiffly, is seized by tremors and finally convulsions, in which he falls to the ground, dashes his head from side to side, champs his teeth, rolls his eyes and froths at the mouth. There are usually eructations and often vomiting which does not, however, give the desired relief. The animal becomes rapidly weaker and though unable to rise has frequent paroxysms of trembling and other nervous symptoms.

Lesions. With mouldy bread the congestion of the gastric and intestinal mucosa, and of the mesenteric glands are marked features. With brine there is in addition the heart gorged with black, tarry blood, the skin and subcutaneous connective tissue shows a similar dark congestion, also the brain and its membranes and the kidneys and bladder. Petechiæ are abundant on the serous membranes and other parts of the body.

Treatment. Evacuate the stomach by tickling the fauces, giving tepid drinks, or ipecacuan. Follow with purgative of sulphate of soda and diuretics especially potassium iodide. Potassium bromide, sodium salicylate or salol may be given to calm nervous excitement and check fermentation. Degoix recommends chloral hydrate, enemata, cold to the head, and counter-irritants and in very prostrate conditions stimulants may be employed.

The most important measure, however, is the cutting off of the supply of the poison and the administration of a wholesome diet. Medicinal treatment is too often unsatisfactory and will seldom pay for the trouble; prevention is the one rational and economical measure.

CRYPTOGAMIC POISONING IN CARNIVORA.

Botulism. Racial immunity. Acquired immunity. Gastric secretions, protective. Bacteria in septic beef, old brine, stale fish, privy air. Symptoms: sudden onset, colic, nausea, retching, vomiting, septic vomit, fever, tender abdomen, bloody, foetid, mucous diarrhoea, prostration, death in three to twenty-four hours. Lesions: of gastro-enteritis, ingesta foetid, congestions and petechiæ of other organs. Treatment: emetic, laxative, diuretics, potassium iodide, antiseptics, demulcents, stimulants.

Causes. What is called *botulism* in man is not uncommon in carnivora. Eating carcasses of animals that have died of disease, of poisoning, etc., in a raw condition and too often in an advanced state of putrefaction, they are liable not only to infection by the pathogenic or poisonous microbes, but also to direct poisoning by the ptomaines and toxins. There are however certain protective conditions. Accustomed as this class of animals is to the consumption of such food, they have by selective evolution acquired an insusceptibility to many such poisons which would prove deadly to the herbivora. Again the mature dog or cat has already been subjected to many of these poisons and having survived them has acquired an immunity which renders it comparatively safe for the future. The antiseptic power of the gastric secretion of the dog may be easily shown, by letting him bolt a moderate piece of putrid meat, and killing him ten or fifteen minutes later when the mass will be found in the stomach without odor of decomposition. He succumbs to the poison either when the quantity ingested is too large, or when the digestion is from any cause deranged and the gastric secretion impaired. Anthrax flesh may produce pharyngeal or intestinal anthrax in the dog. In the animal with temporarily impaired immunity the more virulent septicæmias may prove infecting. From putrid beef the following pathogenic bacteria have been isolated: 1. Gärtner and Basenau have independently isolated **bacillus bovis morbificans** which forms clear colonies on the surface of peptonized gelatine, and brownish ones beneath the surface, and coagulates milk in 24 hours. This is short with rounded ends, very motile, non-liquefying, and capable of surviving the freezing temperature. Its toxins are not destroyed by boiling. It causes enteritis, or, subcutem, debility, somnolence, and stupor. This bacillus was pathogenic to mice, rats, guinea-pigs, rabbits, goats and calves but had little effect on dogs or cats. 2. The **bacillus of Poels** which produces paralysis. 3. The **bacillus of Gaty** which performs very rapid gyratory movements only, and is not found in the muscles.

Old Brine is as poisonous for the dog as for pigs. Six to nine ounces proved fatal, (Reynal).

Stale Fish has been known to prove poisonous in the same way.

Three dogs that were made to breathe the air over a foul privy were seized with vomiting, fever and diarrhoea.

Symptoms. These usually develop six or more hours after a feed. The earlier symptoms are those of digestive disorder. There is colic, nausea, retching, vomiting of septic or bilious matter, ardent thirst, dry mouth, furred tongue, redness of the buccal mucosa, and tenderness of the abdomen. Diarrhoea sets in early and may become bloody, foetid and dysenteric. There is usually much prostration and debility so that apart from his compulsory movements under the colics the animal remains most of the time curled up. Fever is usually slight but the temperature may rise to 105° F. Death may take place in 3 hours, or may be deferred 24 hours or longer.

Lesions. There is usually gastro-enteritis, the mucosa being congested, red oedematous, with petechiæ, and congestion and swelling of Peyer's patches, the solitary glands, the mesenteric glands, the liver and kidneys. The contents of the bowel are brownish red, and an effusion into the peritoneum is common. The spleen is often engorged and enlarged. Congestions of the lungs and brain are common.

Treatment. Evacuate the stomach by ipecacuan, etc., and the bowels by a purgative. Next seek elimination of the toxins by potassium iodide and other diuretics. Antiseptics (calomel, salol, naphthalin) to counteract the further formation of toxins, and demulcents by draught and enema are indicated. In cases of great prostration, heart and nerve stimulants may be useful.

DIARRHŒA, SCOURING.

Definition. Concomitant of other affections. Causes: Congestion, effusion from small and large intestine, irritants in bowels, or blood, chill or other shock acting as reflex, cold drink and violent exercise, aqueous food, cooked, pulped; irritants, feculent concretions, parasites, fermentation products, diseased teeth, jaws or salivary glands, drink after grain, gastric hepatic or pancreatic disease, spoiled food, purgative agents in food, fever products, purgative waters, rains, dews, damp stalls, etc., fear, “washy” horses, nervous animals, root diet, œstrum, hepatic torpor, equine susceptibility. Symptoms: with root diet, with much or little bile, slight cases do not affect system, in severe cases, tympany, pawing, straining, foetor, in infective diseases; complications, laminitis, enteritis, pneumonia. Treatment: remove or expel cause, demulcents, laxatives, anodynes, antiseptics; chronic cases, iron, bitters, antiseptics, astringents, dietetics, rest.

Definition. A frequent discharge of fluid or semi-fluid evacuations from the bowels without excessive griping or painful straining.

This is a common condition attending many diseases, rather than a specific disease of itself yet it is such a prominent feature of these various affections, and one so very characteristic that it seems well to give it a special place, even at the risk of repeating much of what must necessarily appear elsewhere.

The immediate cause of diarrhœa is a congestion of the intestinal mucous membrane and a profuse secretion into the intestinal canal. When such congestion occurs in the small intestine alone, it may be counterbalanced by increased absorption in the large, so that the secretion must be excessive to produce liquid alvine discharges. When on the other hand it occurs in the large intestine or in both large and small, the product is likely to escape in the liquid form.

In its turn the congestion of the intestinal mucosa may result from irritants in the bowels, from the presence in the blood of irritant agents which being secreted stimulate the intestinal glands to excessive secretion, and from reflex nervous action, starting from a distant point as in chilling or irritation of the skin or other organ.

Among direct irritants of the intestinal mucosa may be named a full drink of cold water especially if the horse is trotted or galloped for twenty minutes immediately after;—soft, juicy, rapidly grown green food, to which the animal is unaccustomed, as the first grasses of spring;—cooked or pulped food or ensilage in hard worked animals;—many irritant and acid plants;—accumulations of hard feculent masses in the intestines;—irritation caused by intestinal worms especially the blood-suckers;—the presence in the intestines of undigested matters, and resulting fermentations, the result of diseased teeth and jaws and imperfect mastication, of disease of the salivary glands or ducts and imperfect insalivation, of a drink of water after a grain feed, washing a part out of the stomach in an undigested condition, of disease of the stomach, liver or pancreas interfering with their proper functions; unwholesome and fermenting food like spoiled grain, or fodder, or decomposed potatoes, apples, turnips, pumpkins, carrots, cabbages, etc.;—stagnant and putrid water;—tumors, ulcers, volvulus, invaginations, adhesions and other serious lesions of the bowels may act in the same way.

As examples of the secretion of irritant matters from the blood may be mentioned almost all the different agents used as purgatives, and purgative agents accidentally taken in, these being as a rule absorbed and later secreted again on the intestinal surface, increasing the secretions in their passage:—also the morbid products of fevers which irritate the intestinal mucosa and glands as they are thrown out by them (rinderpest, lung plague, Southern cattle fever):—the purgative waters on certain “scouring lands” act in a similar way. Under the head of reflex action may be named the chills from exposure to cold rains, night dews, damp stalls or beds, and damp, hot buildings, seasons and localities. Under the head of nervous causes must be included strong emotions as excitement, fear, etc., which lead to increase of both secretion and peristalsis. Some horses are very subject to this and are known as “washy”. These have usually a slim abdomen and long loin, and scour whenever they are put to hard work. Other nervous animals with good conformation, but which fret under saddle or in harness will scour under specially severe work or under excitement. This is especially common in young colts while being “broken”, and will occasionally show in mares which are in *heat*. Cattle that have been on a specially succulent diet (turnips, beets, ensilage, grass) are liable to scour profusely if driven far or fast, and stock men seek to obviate this by feeding some

dry bran, meal, and above all fresh dry brewer's grains just before starting. Cows running at large when in heat are very liable to scour. An exclusive diet of turnips or beets will keep cattle in a chronic condition of mild diarrhoea, though not enough to interfere with rapid improvement in flesh. Chronic diseases of the liver by obstructing the flow of blood through the portal vein, cause intestinal congestion and predispose to diarrhoea.

Of the various domestic animals horses are the most liable to superpurgation, from an undue dose of aloes acting on the very large colon and cæcum. Hence the importance of using such an agent carefully in the young, fat or debilitated especially, of the avoidance of cold drinks or exercise to excess after the aloes has been given, and of keeping from work during its operation or immediately after.

Symptoms. These are of all degrees of severity from the frequent pulpy evacuations of animals fed exclusively on roots, (beets, turnips, potatoes), to the excessive and almost constant discharge of a dark colored liquid mingled with more or less mucus. The discharge may be of a light color and foetid, indicating deficiency of bile, or of a dark yellowish brown and odorless.

Slight diarrhoea does not affect the appetite nor general health, nor check improvement in condition. In the more severe and continued forms there is loud rumbling in the abdomen, loss of appetite and condition, a rapid small pulse, accelerated breathing, pallid mucous membranes, sunken glassy eye, and increasing debility even to an unsteady gait. Distension of the abdomen with pawing and other indications of abdominal pain may appear in bad cases. In the milder cases due to simple irritation and congestion there is no tenesmus, no excess of mucus, no formation of bubbles or froth in the stools, as occurs in active intestinal fermentation and dysentery. In symptomatic cases on the other hand there are superadded the marked symptoms of intestinal inflammation, or fermentation, and the fæces become putrid and offensive, which they do also in the different infectious diseases (influenza, contagious pneumonia, rinderpest, lung plague, hog cholera, swine plague, canine distemper, fowl cholera), when the toxins and waste matters of the food and decomposing tissues are being thrown off by the bowels.

Diarrhoea may be complicated with other diseases and especially in the horse with laminitis.

In mild cases it tends to a spontaneous recovery, and is followed by some slight costiveness, and if this should prove extreme there may be some danger of complicating sequelæ such as indigestion, enteritis, pneumonia or laminitis.

Treatment. The first consideration for the practitioner is to discover if possible the immediate cause of the diarrhoea. If this is found to reside in some infectious or other disease aside from the bowel, the attention must be directed to that even more than to the diarrhoea. If it depends on an overdose of some purgative agent or of acrid purgative plants taken with the food, any further laxative is to be avoided, and yet astringents and other agents which tend to lock up the offending material in the alimentary canal must be equally guarded against. An abundance of mucilaginous and demulcent liquids (mallow, flaxseed gruel, boiled starch, etc.) may be given both by the mouth and anus, to sheathe and protect the irritable mucous membrane and to dilute and carry off the irritant contents. Moderate doses of opium may be required to allay the violence of the spasms and peristalsis, but this should not be pushed to the extent of locking up the irritants. Sometimes antiseptics (naphthalin, salol) are useful to check fermentation, and pepsin may be given to assist digestion.

In ordinary cases due to the presence of an irritant the first object must be to relieve the bowels of this, and the second to soothe the irritated mucous membrane. A laxative is usually all that is required, but it must be a mild one so as not to add to already existing irritation. Olive or castor oil are to be preferred as a rule (horse and ox 1 pint; sheep and swine 4 ounces; dog ½ ounce), alone or with a moderate dose of laudanum. Or rhubarb or aloes may be substituted if desired. A dose of whisky or brandy, or oil of turpentine will often do much to allay the secretion and peristalsis. These should be followed by moderate doses of flaxseed gruel, or solution of slippery elm or mallow, or simple well boiled gruels.

If the discharge persists after the laxative has had time to operate, these mucilaginous agents may be replaced by solutions of boiled starch, or of gum arabic, and small doses of calmatives such

as laudanum (horse or ox 1 ounce, sheep or pig 2 drachms, dog 20 drops), or prussic acid or cyanide of potassium (30 drops of the acid or grains of the salt for horse or cow). Sub-carbonate of bismuth, chalk, and carminatives and antiseptics may also be given. According to the indications the practitioner must combat persistent intestinal fermentation, or a relaxed adynamic condition of the intestinal mucosa, or general weakness and exhaustion, with such agents as seem best adapted to the individual case.

Chronic cases will demand the exercise of much judgment. After a gentle laxative, salts of iron (sulphate, chloride) and pure bitters may be given with antiseptics. Or vegetable astringents (catechu, kino) with freshly burned charcoal and essential oils (peppermint, cloves, cajeput) may be employed. In some instances calomel and chalk (1:12) will serve a good end. In others silver nitrate, or arsenite of copper succeeds. Quinine, nux vomica, pepsin, may be used to improve tone. The diet is usually all important. Well boiled gruels, boiled milk, arrowroot, pulped or scraped raw flesh may be demanded in different cases. The patient should be kept at perfect rest, and all excitement avoided.

COLIC, ENTERALGIA, INTESTINAL SPASM.

Definition. Colic pains from spasm, enteralgia, tympanitic indigestion, overloading of bowels, impaction, calculi, concretions, sand, foreign bodies, intestinal and arterial parasites, irritants, enteritis, catarrhal, bacteridian, protozoan, chemicals, strangulation, adhesion, volvulus, invagination, hernia, trauma of stomach or intestine, peritonitis, pleuritis, metritis, ovaritis, hepatitis, biliary calculus, nephritis, urinary calculus, neoplasms, lead poisoning. Causes of enteralgia or spasm, idiosyncrasy, nervousness, cold, wet, high condition, debility, cold, rain, dew, perspiration, fatigue, indigestion, rheumatism. Symptoms: horse—sudden attack, paws, kicks, anxious look at flank, crouches, goes down, rolls, sits, rises, shakes himself, feeds, repeats at intervals, rumbling, defecations. Complications. Diagnosis, symptoms violent, transient, completely intermittent, no fever, no tenderness; from acute indigestion by absence of faulty feed, loaded or tympanitic abdomen, crepitation, continuous pain, and of careful decubitus; from constipation by complete intermissions and freer passages; from intestinal worms by absence of fur on anus, of rubbed rump, and of parasites in stools; from verminous thrombosis by complete intermissions and absence of prostration, cold sweats and of bloody stools; from enteritis and other inflammations by absence of fever; from intestinal anthrax by the intermissions, the absence of brownish mucosa, and perhaps of anthrax from the district; from hepatitis by absence of icterus, tender hypochondrium, and fever; from kidney affections by lack of stiff, straddling gait, tender loins, stretching; from pleurisy by absence of catching breathing, tender intercostals, and friction sound; ruminants—similar symptoms except sitting up or rolling; swine—sudden starting with grunt or scream, vomiting, etc.; Carnivora—frequent moving, yelps, snapping, straining, looking at flank. Treatment: solipeds, morphia subcutem, anodynes, laxative, friction, walking, enemata, chloral hydrate: ruminants, walking, enemata, morphia, laxative; swine, morphia, laxative, antispasmodics, injections, derivatives; dog,—purgative, injections, chloral hydrate, ether, olive oil.

The term colic is loosely applied to all abdominal pains from whatever cause they may arise. It is thus allowed to embrace all diseases of the abdomen. In its more restricted sense in which it will here be considered it may be held to indicate **abdominal pain without inflammation or any structural lesion**.

It may however be well to note the most common causes of abdominal pain so that the distinction may be more definitely reached by a process of exclusion.

1st. **Simple spasmodic colic.** 2d. **Enteralgia or neuralgia of the intestines.** 3d. **Colics from indigestion, a tympanitic, b from overloading with ingesta, c from impaction or constipation, d from calculi or concretions or from sand, or gravel taken with the food or from foreign bodies swallowed, e from worms in the intestines, f from worms in the mesenteric vessels (thrombo-embolic), g from irritants taken with the food or otherwise.** 4th. Colics from **structural lesions of the intestines; a from inflammation of intestine, b from bacteridian inflammation of the bowels, c from protozoan inflammation, d from chemical or other irritants, e from intestinal strangulations, f from adhesions, g from volvulus, h from invagination, i from mesenteric omental or phrenic hernia, j from strangulated inguinal, femoral ventral or umbilical hernia, k from wounds, ruptures or perforations of stomach or intestines, l from peritonitis or pleuritis, m from metritis or ovaritis, n from hepatitis or biliary calculus, o from pancreatitis or pancreatic calculus, p from nephritis, nephritic, uretral, cystic or urethral calculus, q from neoplasms affecting any of the abdominal organs.** 5th. Colic due to **lead poisoning**.

Causes of enteralgia and spasmodic colic. Enteralgia may be defined as a neuralgic pain of the bowel which may therefore be free from spasm or any other appreciable structural or functional change. Its existence in the lower animal is necessarily somewhat problematical, as it can only be inferred from the analogy of the animal with man, and of the enteron with the superficial parts that are more frequently attacked with neuralgia, and also from the absence of visible spasmodic contractions in the bowel which has been the seat of intense pain, yet shows no inflammatory lesion. But whether this is accepted or not, the occurrence of spasm is undeniable and as both are functional nervous disorders the same causative factors will apply to both.

In some nervous animals, especially high bred horses and dogs, there is undoubtedly an idiosyncrasy which shows itself in a special susceptibility of the nervous system. In such animals an exposure to cold or wet, or the presence of a local irritant which would have been without effect under other circumstances, lights up the nervous disorder and produces an explosion, it may be as spasm or it may be as nervous pain. Animals that are kept under the best care, that are least

accustomed to exposure and neglect, that are highly fed, and maintained in high spirits and are impatient of control are more susceptible than those that become inured to change and exposure, yet are kept in moderately good condition. On the other hand the subject which has become debilitated by overwork, underfeeding, exhausting disease, or the generation in the system of some depressing poison is likely to show a similar nervous susceptibility, so that at the two extremes of plethora and nervous susceptibility, on the one hand, and anæmia and neurasthenia on the other, we find a corresponding tendency to nervous disorder under comparatively slight causes. Thus it happens that a drink of ice cold water, an exposure to a cold blast or a drenching rain or a heavy night dew may seem to be the one appreciable cause of the trouble. If the animal has been perspiring and fatigued the attack is more likely to occur. In other cases a slight indigestion unattended by impaction or tympany, or the ingestion of an irritant which on another occasion, or in another animal would have been perfectly harmless, will induce a violent nervous colic. In some instances the attack is supposed to be of a rheumatic nature the causative action of the cold giving color to the theory.

Symptoms. The attack usually comes on suddenly especially if it has followed on a drink of cold water or a cold exposure.

Solipeds. The *horse* leaves off feeding or whatever he may have been engaged in, paws with his fore feet, moves uneasily with his hind ones or kicks with them, one at a time against the abdomen or out backward, he looks back at the abdomen with pinched, drawn, anxious countenance, bright anxious eye, and dilated nostrils, he moves uneasily from side to side of the stall or box, crouches for a few seconds with semi-bent knees and hocks and then throws himself down violently with a prolonged groan. When down he may roll from side to side over the back, and struggle in various ways, he may start to rise, sit for a moment on his haunches, then go down and roll as before. Or he may get up, shake himself and resume feeding as if entirely well. Soon the spasms reappear, suddenly as at first, and after a time subside as before. Thus the disease proceeds, each succeeding paroxysm diminishing in violence until they permanently subside, or increasing until the animal dies worn out with shock, suffering and exhaustion. If the paroxysms are severe the skin is usually bathed more or less in perspiration. Usually the peristalsis continues more or less, a rumbling is heard in the bowels and more or less fæces are passed in small solid balls or semi-liquid.

The *course* of the disease is usually rapid and followed by recovery. When prolonged it may become complicated by volvulus, invagination, indigestion or even enteritis.

Diagnosis. The characteristic symptoms are the suddenness of the onset, the extreme suffering during the paroxysm, the reckless manner in which the animal throws himself down, the intermissions with complete absence of pain, the natural condition of the pulse and temperature in the intermissions, the comfort with which the patient shakes himself, and the absence of all abdominal tenderness, manipulation and friction seeming to give relief rather than discomfort.

In the colic of **acute indigestion** there is the previous excessive or unwholesome meal, or the full drink after feeding; there is tympany, or a loaded state of the abdomen proving flat on percussion, there may be crepitation on auscultation, there is continuous pain with exacerbations (not complete intermissions), and there is rather a careful mode of lying down.

In **intestinal constipation** or other **obstruction**, fæces may be passed at first in small pellets coated with mucus or they may be at first passed freely but in steadily lessening quantities until they stop altogether. The pain is constant but worse at one time than another and in case of external hernia the swelling will be visible.

In **helminthiasis** there is the general unthriftiness, irregular appetite, frizzled broken hair on the base of the tail, a fur of dried mucus around the anus and the presence of parasites in the droppings.

In **verminous thrombosis**, to the symptoms just named there are added the reckless method of throwing himself down, hyperthermia, constancy of the pain, rapidly running down pulse, cold sweats, and profound prostration. When blood is passed per anum it is all the more significant.

In **enteritis** or **peritonitis** the hyperthermia and the constancy of the pains are sufficiently pathognomonic.

In **intestinal anthrax** there are the dusky brownish yellow mucosæ, the marked prostration, the hyperthermia and the constancy of the suffering. There is also the fact that the region is subject to anthrax and bacilli may be present in the blood.

In **acute hepatic disease** there is hyperthermia, dusky or icteric mucous membranes, great tenderness when percussion is made over the short ribs, and sometimes lameness of one shoulder (usually the right).

In **disease of the urinary or generative organs** the stiff or straddling gait, tender loins, and the frequent stretching as if to urinate, are nearly pathognomonic.

In **pleurisy** the hyperthermia, the transient duration of the colic, and the tenderness on manipulating the intercostal spaces will usually differentiate.

Ruminants. In cattle, as in the horse, the symptoms of spasmodic colic are restlessness, constant movement, looking round at the flanks, wriggling of the tail, uneasy lifting of the hind feet, kicking at the abdomen, and abruptly lying down and rising again. The animal does not roll on the back nor sit on the haunches. Fæces may be passed in small quantity or entirely suppressed, and there may be a slight tympany of the paunch.

Swine. The animal is attacked abruptly, starts with a grunt or scream, moves around uneasily, lies down, rolls, gets up, and repeats the motions. Vomiting is not uncommon, and the belly may be tense, tympanitic and even tender. The bowels may be confined or relaxed.

Carnivora. The colicky dog is very restless, changing from place to place, sitting on his haunches, lying down curled up, starting up suddenly with a yelp, and repeating the restless movements. He looks anxiously at his flank, sometimes bites at it, and cries plaintively. The bowels are usually torpid, and defecation effected with straining.

There are distinct intermissions but these are cut short by a new accession of pain.

The attack is usually transient and ends in recovery.

Treatment. **Solipeds.** For nervous colic the hypodermic injection of sulphate of morphia (2 grs.) is very effective. This will commonly bring relief in less than five minutes. Should there be no effect at the end of this time it may be repeated with advantage, but should a second dose fail, it is well to resort to other measures. Eserine and barium chloride are contraindicated as being liable to increase the spasm, and if there is no irritant to expel there is no object in their exhibition. The old prescriptions of laudanum and turpentine; laudanum and ether; sweet spirits of nitre with belladonna, or hyoscyamus, and other stimulants and narcotics are of little avail as they are not absorbed from the horse's stomach and cannot operate until they have reached the duodenum. If given at all, their action may often be hastened by injecting them into the rectum.

When the morphia fails it is the safest treatment to give a moderate dose of aloes or other laxative, in combination with extract of hyoscyamus or chloral hydrate. This takes time to pass into the duodenum, and be absorbed and secreted anew in order to have its full effect, and therefore it may be necessary to keep up a moderate action of the morphia as a palliative. In four hours, however, at the latest, the aloes can be counted on to bring permanent relief. This appears to come as soon as the active principles have been absorbed, the nauseating effect operating at once on the overexcited nerve centres. The action is more perfect still when a free secretion has been started from the intestinal mucosa, and the circulation and innervation in the intestinal walls are essentially changed. This measure which was long successfully practiced and advocated by the late Joseph Gamgee, is even more perfectly adapted to the colics of indigestion and irritation, of impaction and fermentation. There are of course cases of complete obstruction in which it must fail, but it is probably the most successful method for colicky affections in general.

In addition to the above, other methods of correcting the disordered innervation are available. Active friction of the abdomen with straw wisps is often effective, also fomenting the abdomen with hot water. Simply leading the animal around acts as a nervous derivative, and may be employed to prevent his dashing himself down so suddenly as to injure himself. Then copious injections of warm water soothe the rectum, solicit its peristalsis and by sympathy affect the other intestines in the same way. They may often be made more effective by the addition of antispasmodics (extracts of belladonna or hyoscyamus or chloral hydrate).

In all cases a soft bed should be provided to secure the animal against injury in his sudden reckless movements.

Ruminants. Simple spasmodic colic is usually transient and may be successfully treated by driving around, giving copious warm water injections, and using morphia subcutem. Frictions to the abdomen with straw wisps, or with oil of turpentine should be tried. Should these fail there is a presumption of further trouble and no time should be lost in giving a laxative (Glauber salts) 1 to 2 pounds, or castor oil 1 quart, with antispasmodics and stimulants as for the horse.

Swine. The antispasmodic treatment may be tried on the pig, but usually it is well to give a purgative at once in combination with the narcotic. Castor oil 4 ounces, laudanum $\frac{1}{2}$ drachm, or jalap 2 drachms, and extract of hyoscyamus 20 grains in electuary. Warm injections and embrocations to the abdomen are desirable.

Dog. It is usually well to give a purgative at once (jalap $\frac{1}{2}$ drachm) with 10 to 20 drops laudanum according to size. Copious injections of warm water and a warm bath may follow. Chloral hydrate 20 to 60 grains, may be exhibited by the rectum; also ether 1 drachm, in olive oil.

CONSTIPATION FROM INTESTINAL ATONY.

Definition. Symptomatic. Causes: habit of retention in horse, dog and cat, indigestible matters in colon, calculi, dry food, lack of water, fever, diuresis, diaphoresis, milking, bleeding, hepatic torpor, verminous thrombi, old age, debility, nervous disease, matting of hairs, hæmorrhoids, abdominal suffering. Lesions: dilatation and catarrh of bowel, disease of rectum. Symptoms: solipeds—small, dry, coated, infrequent stools, straining, inappetence, tympany, colic, stretching as if to urinate; dog, fruitless straining, or dry, earthy looking fæces, coated, bloody, foetid, anus swollen, tender, moist, palpitation, colic, vomiting, diarrhoea may fruitlessly occur, male urinates like bitch, foul, foetid mouth; may last days or months; complications, sequelæ. Treatment: solipeds—exercise, pasturage, cold water before morning feed, regular regimen, bran, flaxseed, carrots, turnips, ensilage, common salt, Glauber salts, nux, cold injection, glycerine, barium chloride, purgative, enemata; dog:—mechanical unloading of the rectum and colon, injections, exercise, water, regular habits, laxative food, eserine, oil, calomel, jalap, podophyllin, colocynth, belladonna, nux vomica, abdominal massage, electricity.

Definition. Constipation consists in dryness, hardness, and undue retention of the fæces. It has of course many grades and may lay claim to many different causes, so that it might, like its antithesis diarrhoea, be held as merely a symptom of another disease. It has however such a definite character, that it is convenient to retain the name to designate those cases in which the torpor or atony is the most prominent and dominating feature, while other forms of obstruction will be treated under different heads.

Causes. Defecation is immediately due to the active peristaltic movements of the rectum overcoming the resistance of the sphincter. There is also the concurrent closure of the glottis and contraction of the diaphragm and abdominal walls, and it is usually the voluntary operation of these forces that rouses the rectum to effective peristalsis. The excitability of the rectum depends greatly on habit, hence the habitual retention of fæces, gradually dulls that organ and renders it less and less disposed to respond. In *horses* this is seen largely in connection with abundant dry feeding and lack of exercise. In house *dogs* and *cats* on the other hand, inculcated habits of cleanliness, compels the suppression of the natural instinct, and the habitually overloaded bowel becomes less and less responsive. The retained excrements meanwhile give up more and more of their liquids until they become so dry, and incompressible as well as massive that they can be expelled only by violent efforts. Inflammation of the mucosa naturally follows the retention and this in its turn adds to the weakness and torpor.

Acting in a similar way the partial obstruction, by accumulations of bones and other undigested matters in the colon, and by calculi, tends to continued accessions of new material and to gradually increasing intestinal paresis. So with the other forms of obstruction which will not be further referred to here.

In all animals dry feeding and a lack of water are potent causes of inspissation of the ingesta and torpor of the bowels.

All or nearly all febrile affections, leading as they do to suppression of secretions, cause drying and tardy movement of the contents of the bowels.

The excessive loss of liquid through other channels,—by diuresis, by profuse perspirations, by excessive secretion of milk, or by bleeding—has a similar tendency.

The suppression of the biliary secretion through liver disease, or obstruction of the biliary duct, withholds from the intestine, the most important of the stimuli to peristalsis, and tends to constipation, unless the resulting irritation should cause excessive secretion.

The derangement in the circulation in the intestinal walls caused by verminous thrombi in the horse, acts in the same way, the imperfectly nourished walls not only losing the normal power of peristalsis, but sometimes contracting so as to cause a stricture. Parasites encysted in the walls of the bowels, like catarrhal and other inflammations of these parts tend to atony and tardy peristalsis.

A weakness of the nervous system attendant on old age, or debility, or chronic lead poisoning often tells with force on the alimentary canal, and the loss of nervous power through disease of the great nerve centers (ganglionic system, brain, spinal cord) impairs the vermicular motion. This is notoriously the case in paraplegia, chronic hydrocephalus, and vertigo.

Finally among the causes of constipation must be noted the matting of the hairs around the anus (in dogs), and painful affections of the anus, or the abdominal walls, which render efforts at defecation painful and deter the animal from attempting them.

Lesions. These are as varied as the diseases which give rise to constipation, or result from it. Permanent dilatation or sacculation of the intestine, and the structural changes attendant on intestinal catarrh are the most common local lesions. But proctitis, hemorrhoids and ulcers of the anal follicles are met with in canine patients and ulcers of the colon in the seat of impaction are common.

Symptoms. In **Solipeds** the fæces are passed, at long intervals, in small quantity, usually only a very few balls at a time, firm, dry, moulded smooth and black on the surface, often covered with mucus, or with streaks of blood. They are passed with unusual effort and straining, and even with groaning, and one or more balls that may be exposed in the act are often drawn back and retained by the inversion of the rectum and closure of the sphincter. It is liable to be complicated by impaired appetite, tympanies, slight recurrent colics, and dryness, scurfiness and unthriftiness of the skin. Not unfrequently the pressure of the impacted colon (pelvic flexure) irritates the bladder causing stretching as if to urinate, and the passage of urine often in small quantity. There may be the symptoms of any one of the different nervous affections that lead to impaired peristaltic action, or of the local diseases which tend to obstruction of the bowels.

In **dogs** there are violent and painful efforts to defecate, which may be fruitless, or may lead to the expulsion of small masses of dry, earthy looking fæces, smoothly moulded on the surface, coated with mucus, streaked it may be with blood and highly offensive in odor. The anus may be puffy and swollen with mucopurulent secretion from the anal glands, which soils the hair of the hips and tail. If the abdomen is flaccid, manipulation with both hands on opposite sides usually detects a solid mass representing the impacted rectum and colon, and extending from the pelvis forward, often to the sternum. The same mass will be reached by the oiled finger introduced into the rectum. Both methods of exploration are painful and may call forth cries from the patient. The abdomen is usually distended, largely from the impacted fæces, in which case it gives a flat sound on percussion, or from gaseous emanation, in which case it is tense, resilient and resonant. Colicky pains are liable to appear, and vomiting at first of food only, then more or less yellow and bilious, and finally of distinctly feculent matters. For a time appetite may be retained, but this is gradually lost. There may supervene diarrhoea, which in favorable cases may lead to expulsion of the impacted mass, but in others it fails to completely dislodge it. The patient is dull and spiritless, inclined to lie curled up in dark corners, and when raised walks slowly and stiffly, with the tail carried straight or slightly to one side. The male urinates like a bitch without lifting the leg. The nose is dry, the tongue furred, the teeth usually covered with tartar, and the breath foetid. There is at first no hyperthermia, but some rise of temperature attends on the advance of the disease, and the auto-poisoning by absorbed products of the putrefaction of retained fæces.

The disease may last a few days only or it may continue for weeks or months. In the last case intestinal catarrh, ulceration, and circumscribed necrosis are likely to supervene and the animal may die of auto intoxication, acute peritonitis or enteritis. Yet the majority of cases in the dog reach a favorable termination, or recover with remaining cicatrices, strictures or dilatations.

Treatment. In **solipeds** accustomed to an idle or pampered life, plenty of daily exercise will often correct the torpor. A run at pasture will often effectually counteract the tendency. If the patient must be kept in the stable a full drink of cold water every morning before feeding will often succeed. Regularity in feeding and watering is of the utmost importance, and the addition of a little wheat bran or flaxseed to the grain is often of material advantage. Next may be added a moderate allowance of carrots, turnips, or ensilage to furnish the needed succulence and organic acids. If in addition medicinal measures are wanted, a small handful of common salt, or of Glauber salts, in the morning drink to be taken ten or fifteen minutes before the first feed, will usually operate well. This may be continued for a length of time if necessary, without the ill effects of purgatives given at other times. It may be rendered slightly more effectual by the addition of 10 grains nux vomica on each occasion. A morning injection of a quart or two of cold water with one or two ounces of glycerine may be tried. Another resort is 2 or 3 grains of barium chloride in the morning drink or

hypodermically repeated daily for some time.

In the more severe cases with already existing impaction of the colon, purgatives and copious injections will be demanded as advised under that disease.

In **dogs** the first object is the unloading of the rectum and colon and this usually demands direct mechanical intervention. (See Intestinal Indigestion with Constipation.) In case of hypertrophied prostate this may be rendered somewhat difficult, yet with a free use of oily, soapy or mucilaginous injections it can usually be accomplished.

The further treatment is on the same line as for the soliped. An abundance of exercise in the open air is a prime essential, together with a free access to fresh water. House dogs must be taken out for urination and defecation at regular times that are not too far apart. The food must be of a laxative nature. At first fresh whey or buttermilk only may be allowed, but as some action of the bowels is obtained well salted beef tea, pulped or scraped red muscle seasoned with salt, or milk treated in the same way is permissible. If the bowels fail to respond when the dog is taken out at the regular times an injection of cold water may be given. Sulphate of eserine ($\frac{1}{5}$ gr.) may be given daily by the mouth or hypodermically, or castor oil ($\frac{1}{2}$ to 1 oz.) may be administered at one dose to be followed by careful dietary and hygienic measures. Or sweet oil, calomel and jalap, podophyllin, or colocynth may be substituted. When the bowels have been freely opened a daily morning dose of a drop of the fluid extract of belladonna and $\frac{1}{2}$ gr. of nux vomica will often materially improve the peristalsis. Active manipulation of the abdomen may be employed, or, if available, a current of electricity through the torpid bowels for 10 or 15 minutes daily.

CONSTIPATION IN BIRDS.

Causes: Matted feathers, impacted cloaca, arrest of eggs, debility, catarrh, parasites, nervous disorder. Symptoms: swelling of anus, pendent abdomen, waddling gait, straining without effect. Treatment: remove obstruction by mechanical means, cut off matted feathers, egg matter may demand laparotomy, castor oil, tincture of rhubarb, enemata, green food, ensilage, roots, onions.

In birds torpid and obstructed bowels may come from the effects of a previous diarrhoea, which has led to the matting together of the feathers over the anus at once obstructing defecation and rendering it painful. It may result in and be aggravated by a slow accumulation of indigestible matters in the intestine or cloaca (pebbles, feathers, etc.), and the arrest of eggs in the oviduct, pressing upon and obstructing the bowel. In a recent case the author removed 18 ozs. of impacted egg matter from the oviduct of a hen, which when divested of this load weighed barely 2 lbs. Debility of the general system and particularly of the walls of the bowels, and its various causes (old age, exhausting disease, intestinal catarrh, parasites, nervous diseases, etc.) retard defecation and favor impaction as in the mammal.

The *symptoms* may be; hard dry droppings, matting of the feathers over the anus with feculent matters, a firm swelling surrounding the sphincter, a pendent condition of the abdomen which when manipulated is felt to be firm and resistant, ruffling of the feathers, drooping of the head, wings and tail, walking sluggishly with legs half bent and a waddling gait, and ineffectual attempts to defecate.

Treatment. As in dogs remove the obstructing mass by mechanical means. Matted feathers may be clipped off, and feculent accumulations may be dislodged by the aid of the finger, or in small birds of a blunt prob. This may be favored by manipulation through the abdominal walls, and the injection of soapy or oily enemata. Accumulations of impacted egg matter may be similarly removed, or, failing this, by an incision made through the abdominal walls and oviduct. As a purgative give one or two teaspoonfuls castor oil according to the size of the hen, or a few drops to a small cage bird. For the latter Friedberger and Fröhner advise a few drops of tincture of rhubarb in the drinking water. Injections of warm or cold soapsuds or water may be continued as symptoms demand. Green food, ensilage, roots, worms, snails and insects are indicated to correct the tendency to costiveness and may be continued until the bowels have acquired their proper tone. A moderate allowance of onions is often of great value.

HAIR BALLS IN THE INTESTINES—HORSE. EGAGROPILES.

Seat, colon, cæcum; hair of oat seed, clover leaf, vine tendrils, hair of horse, nucleus, calcic admixture, straw, in horses on dry food, with depraved appetite, or with skin disease. Symptoms: none, or torpid bowels, colics, recurring, fermentations, tympany, obstruction, rupture, peritonitis, rectal exploration. Lesions: impacted ball, with excess of liquid and gas in front, rupture, ragged bloody edges. Treatment: extraction, enemata, eserine, barium chloride.

Hair balls, received the name of egagropiles because of their discovery in the alimentary canal of the wild goat, but they are found in various forms in all the domestic animals. In *horses* they occupy the cæcum and colon and are most frequently composed of the fine vegetable hairs that surround the grain of the oat, or the leaf of clover, of the woody tendrils of vines, and of the hairs of themselves and their fellows taken in at the period of moulting. They sometimes contain a nucleus of leather or other foreign body which has been swallowed but in many cases no such object can be found, the hair having become rolled and felted by the vermicular movements of the stomach and intestines. An admixture of mucus assists materially in the felting, and calcareous and magnesian salts may make up the greater part of the mass, rendering it virtually a *calculus*. They may further have a large admixture of straw and vegetable fibres of larger size than oat or clover hairs. They are most frequent in horses kept on dry food, (sweepings of oatmeal mills) and at hard work, and which show depraved appetite and lick each other. Omnibus horses suffer more than army horses. Skin diseases, by encouraging licking, contribute to their production.

Symptoms. In the great majority of cases hair balls do not seriously incommode the horse. They do not attain a large size, and being light do not drag injuriously on the intestine and mesentery. They do, however, retard the movement of the ingesta, and when grown to a considerable size they may block the intestine, more particularly the pelvic flexure, the floating colon or rectum. Under such conditions they produce colics which may be slight, transient, and recurrent, or severe and even fatal, having all the characteristics of complete obstruction from other causes. Fermentations, tympanies, and straining without defecation are common features. When the obstruction takes place in the pelvic flexure, the floating colon or rectum, it may often be detected by rectal exploration. When complete obstruction occurs all the violent symptoms of that condition are present, and these may pass into those of rupture (Peuch, Leblanc, Neyraud), and shock or peritonitis. If the animal has passed hair balls even months before, the colics may with considerable confidence be attributed to other balls of the same kind.

Lesions. In case of death there are the usual lesions of gaseous indigestion, with or without enteritis, but with the accumulation of a great quantity of liquid contents, above the ball, which is felt as a firm body impacted in the gut. In other cases the distended bowel has given way and the liquid contents and often the hair ball as well are found free in the abdominal cavity. In such a case the edges of the laceration are covered with blood clots and thickened with inflammatory exudation, and there is more or less peritonitis.

Treatment. Relief may sometimes be obtained by the extraction of a hair ball lodged in the rectum or adjacent part of the floating colon. In other cases abundant soapy or oily enemata, and the employment of eserine or barium chloride subcutem are indicated.

HAIR AND BRISTLE BALLS IN DOG AND PIG.

From licking in skin disease. Symptoms: of obstruction. Treatment: manipulation, enemata, oil, antispasmodics, eserine, barium chloride, laparotomy, diet in convalescence.

The hair balls of *dogs* come mainly from licking themselves when affected with skin diseases or parasites. In *pigs* they are mostly attributed to depraved appetite.

The hair balls of the *dog* are small, open in texture, and easily disintegrated, having little mucus and no earthy salts in their composition.

The bristle balls of *pigs* take the form of straight or curved rods of firm consistency, but without earthy salts. The projecting ends of the bristles render them particularly irritating.

The *symptoms* are those of obstruction of the bowels, and the *treatment* consists in efforts to dislodge them. If situated near the anus they may sometimes be reached with the finger, or copious oily injections may facilitate their passage. Manipulations through the abdominal walls may be helpful in the dog. Oleaginous laxatives and antispasmodics may be tried, or these failing, eserine or barium chloride. As a last resort laparotomy may be performed, the ball abstracted and the intestine and abdominal wall carefully sutured (Siedamgrotzky). In such a case the diet should be restricted for a week to beef soups, buttermilk, and well boiled gruels, especially flaxseed.

INTESTINAL CALCULI. ENTEROLITHS. BEZOARS.

Earthy basis, nucleus, stratification, in cæcum or colon, multiple, size, number up to 1000. Composition, phosphates of lime, magnesia, and ammonia, silica, mucus, epithelium, organic matter. Ammonio-magnesian tend to crystalline form, common phosphate of lime to smooth forms. Concretions. Source in food. Ammonia from bacteridian fermentation, action of colloids, varied nuclei, rapid growth. Lesions: catarrh, dilation, obstruction, rupture, peritonitis. Symptoms: intermittent colics with obstruction, tympany, bowel distension, liquid and gaseous, before obstruction. Diagnosis: by hand in rectum, hard obstruction with distension in front. Treatment: purgative dangerous, but exceptionally successful, extraction, oleaginous enemata, laparotomy.

Horse. Intestinal calculi have an earthy basis (ammonio-magnesian phosphate, or oxalate of lime, and more or less silica) glued together by mucus and having a central nucleus usually of some foreign body, (a particle of sand, pebble, morsel of hair, lead, cloth, nail, coin, blood clot, or inspissated mucus) around which the earthy salts have been deposited layer after layer. They are usually formed in the cæcum or double colon and may be multiple and moulded upon each other, so that they become discoid, angular or otherwise altered from the globular shape. The worn, flattened surface in such cases shows concentric rings representing the layers as deposited in succession.

The *size* of the masses may be from a pea or smaller, up to calculi of six inches in diameter.

In *number* there may be a single calculus or there may be an indefinite quantity. Zundel counted 400 in a single colon, and Gurlt 1,000.

Composition. They are usually composed of phosphate of lime and of magnesia, of ammonio-magnesian phosphate, with a little silica, mucus, epithelium, and organic matters from the ingesta. Traces of sodium chloride, and iron oxide may also be present.

The phosphates of lime, magnesia, and of ammonia and magnesia usually constitute the main part of the calculus. Fürstenberg found specimens in which the ammonio-magnesian phosphate amounted to 72 to 94 per cent.

The calculi containing an excess of ammonio-magnesian phosphate tend to assume a crystalline or coralline form which causes them to be specially irritating to the mucosa. When broken they show a radiated structure from the centre to the circumference in addition to the concentric rings. These are usually of a yellowish brown or a gray color and have a specific gravity of 1694 to 1706.

Calculi in which the common phosphate of lime abounds are likely to be smooth on the surface and on section show the concentric rings more distinctly and the radiating lines less so. The brownish calculi of this variety are much more compact, and harder than the crystalline or mulberry calculi, and have a higher specific gravity—(1823).

Bluish calculi with a smooth glistening surface and lower specific gravity—1681—, have been found of small size and in great numbers in the colon (1000 in the colon, Gurlt).

In some calculi there is a large admixture of alimentary matters, and a low specific gravity (1605 to 1674). These were designated as *pseudo calculi*, by Fürstenberg.

In still other cases a calculous looking mass, when broken into, is found to be composed of a mass of dried alimentary matter enclosed in a thin layer of lime salts. These have a low specific gravity (1446 to 1566) and have been named *concretions* by Fürstenberg.

Causes. As a large proportion of the calculus is phosphate of lime or ammonio-magnesian phosphate, we must look for the source of these in the food and then at the conditions which determine their precipitation.

The percentage of ash and of phosphoric acid in the common foods of horses may be seen in the following table:

	Ash. Per cent.	PO ₅ in the Ash. Per cent.	PO ₅ in the entire food. Per cent.
Wheat bran	7.3	50	3.65
Wheat grain	3.0	46.38	1.3914
Oats grain	2.50	26.5	0.6625
Barley grain	3.10	39.9	1.2276
Bean grain	3.10	31.6	0.9864
Pea grain	2.75	34.8	0.957
Tare grain	3.00	36.2	1.086
Indian corn grain	1.5		
Rye grain	1.6	39.9	1.0384

The source of the magnesia may be found to a large extent in the grains represented in the following table:

	Ash. Per cent.	Mg. in Ash. Per cent.
Oat, grain	2.50	7.3
Barley, grain	3.10	8.5
Rye, grain	1.6	2.4
Wheat, grain	2.12	9.98
Wheat, bran	7.3	11.2
Bean	3.1	6.6
Pea	2.7	5.6

The amount of magnesia in each of these grains is amply sufficient to furnish the material for the constant growth of a calculus. Wheat bran is preëminent in the amount of its magnesia and therefore wheat bran has been charged with predisposing to calculi. In the perisperm as a whole, Fürstenberg found 1 per cent. of phosphate of magnesia, and in coarse bran not less than 2.5 per cent.

The ammonia which is essential to the precipitation of the phosphate of magnesia in the form of the compound salt (ammonio-magnesian) can be found wherever proteids are in process of septic fermentation. The slightest failure to peptonize every particle of such proteids, implies septic change and the evolution of ammonia, which on coming in contact with magnesia phosphate instantly precipitates the insoluble salt.

This fully agrees with the doctrine of the formation of urinary calculi through the agency of bacteria, since the ammonia is essentially a fermentation or bacterial product.

It may also be noted that the experiments of Rainey and Ord showed that in the presence of colloids (mucus, epithelium, pus, blood) the earthy salts are precipitated as minute globular bodies which by further accretions become calculi. In the absence of colloids the salts tend to precipitate in angular crystalline forms, so that the mulberry and coralline calculi may possibly have been precipitated in the absence of such bodies. From the solvent quality of ammonia, however, the contents may easily pass from a fermenting liquid containing colloids to a non-fermenting and noncolloid mixture.

The presence of a solid body which may act as a nucleus is an essential element, and the condition of the food or drink will often supply this. It has been noticed that army horses in the field, feeding from the ground and taking in sand and pebbles, are unusually liable to intestinal calculus. Horses which lick earth in connection with acidity of the stomach or other dyspepsia are specially subject to it. Horses watered from shallow streams with sandy bottoms, where they take

in sand with the water, have been similarly affected. Millers' horses, in the days of old process milling, suffered not alone because of the abundance of oat hairs in the feed but also on account of the grit from the millstones. Hay and other fodders that have lain on the ground and which contain earth and sand furnish other sources of such nuclei. Shingle nails and other small nails, pins, needles, coins, etc., which have mixed with the feed are common causes of trouble, and indeed any foreign body may become the centre and starting point of a calculus.

Catarrhal affections and other lesions of the mucosa, which furnish excess of mucus, beside pus, lymph and even blood as nuclei, are invoked as starting points of the calculi, but however true this may be in particular cases, irritation and catarrh appear to be much more frequently the result than the cause of the calculus.

Attempts have been made to estimate the time taken in the formation of a calculus by allowing a ring for each feed and successive deposit therefrom (Fürstenberg, Colin). Thus a calculus of 14 pounds with 720 layers, it was estimated could be formed in one year at two feeds per day. More definite evidence was found in the case of Pastore in which a coin with the mint mark of 1847 was found as the nucleus of a calculus the size of the fist in 1848.

Lesions. Formed in the most spacious parts of the colon and cæcum, calculi usually rest there for a length of time without visible injury, and it is only when they are moved onward and get arrested at a narrow part of the gut (pelvic flexure, floating colon, rectum) that they cause appreciable trouble. Yet it is claimed that by their weight they drag upon the yielding walls of the bowel, causing dilatation and attenuation, weakening the peristalsis and predisposing to rupture. The compression of the vessels also tends to anæmia and atrophy. In the case of rough crystalline calculi the mucosa is subjected to attrition, irritation, and inflammation. The more serious and urgent trouble is that of obstruction of the narrower portions of the colon and rectum, which may be absolute and persistent, leading to rupture and death or a fatal inflammation on the one hand, or may end in recovery on the other, in connection with a displacement onward or backward of the calculus as the result of peristalsis or anti-peristalsis.

Symptoms. These are intermittent colics, each reaching a climax and followed by a sudden recovery as the calculus is displaced into a more spacious part of the colon. A significant feature is the complete obstruction, fæces being passed for a short time at first and then suddenly and absolutely stopped. Coincident with this are tympany, violent colics, straining, rolling, sitting on the haunches, perspirations, anxious countenance, and all the symptoms of obstruction.

Diagnosis is never quite certain unless the practitioner with his oiled hand in the rectum can detect a hard stony mass obstructing the pelvic flexure of the double colon with a tense elastic distended bowel immediately in front of it, or a similar hard obstruction of the terminal part of the floating colon with a similar distension in front of it. The pelvic flexure may usually be felt below and to the right at the entrance to the pelvis, and the floating colon above, under the right, or more commonly the left kidney. Calculi in the more spacious parts of the double colon or in the cæcum are inaccessible to manipulation. The feed (bran, ground feed) will be suggestive, as will the occupation of the proprietor (miller, baker).

Treatment. This is rather a hopeless undertaking. No effective solvent of the calculus can be given, and purgatives usually increase the danger by increasing the peristalsis and dangerously distending the bowel above the point of obstruction. It is true that this is sometimes followed by a temporary recovery the calculus being loosened and falling back into the dilated portion of the bowel. Less frequently the increase in the peristalsis forces on a moderately sized calculus to complete expulsion. It is a desperate though sometimes successful resort. A more rational course of treatment is the dilation of the bowel back of the obstruction by copious mucilaginous, soapy or oleaginous enemata. Trasbot suggests CO₂ produced by injecting sodium bicarbonate and tartaric acid. This may be seconded by the hypodermic injection of barium chloride or of atropia. When the calculus is lodged in the floating colon or rectum it may be possible to reach it with the hand and extract it at once. The last resort, is by laparotomy for the removal of the calculus. One such successful case is on record in which Filizet removed a calculus as large as an infant's head. In other cases the horses failed to survive. Desperate as the resort may be it is not to be neglected in a case of undoubted calculus, solidly impacted and of such a size that its passage is impossible. A fatal result

is imminent, and even if the present attack should pass off it can only be looked on in the light of an intermission, so that there is practically nothing to lose in case the result should prove fatal. Anæsthesia and rigid antiseptic measures should of course be adopted.

FOREIGN BODIES IN THE INTESTINES OF SOLIPEDS.

Sand, pebbles, earth, lime, nails, pins, needles, coins, shot, cloth, leather, rubber, sponge, tooth, bone, wood, twine. Symptoms: as in intestinal indigestion or calculi, or sand or pebbles in fæces; peritonitis, phlegmon. Lesions: congestion, catarrh, ulceration, abscess, needles may travel to other organs. Treatment: laxative, enemata, or as for calculi.

All sorts of foreign bodies are taken in with food and water and find their way to the intestines. Sand from drinking from shallow streams with sandy bottoms, from browsing on sandy pastures where the vegetation is easily torn up, or from feeding grain from sandy earth will sometimes load the intestines to an extraordinary extent so that such horses will pass sand for some weeks after leaving the locality. Small stones and gravel are taken in in the same way or from the habit of eating earth or licking crumbling lime walls. Nails, pins, needles, coins, shot, pieces of cloth, leather, caouchouc, sponge, and even a molar tooth and a piece of a dorsal vertebra have been thus taken. Recently the author saw a small twig of hard wood transfixing the pylorus and duodenum with fatal effect. In another case were balls of binding twine which had been taken in with the fodder on which it had been used.

The *symptoms* are usually those of intestinal indigestion or calculi. In some cases, however, they are peculiar, thus there may be a constant passage of sand, there may be indications of peritonitis, or there may form a phlegmonous swelling of the abdominal walls in the abscess of which the foreign body is found.

Lesions. Pechoux found 56 lbs. of a brownish earth in the cæcum and colon. Congestion and ulceration of the intestines are common, with occasionally abscess. All the lesions that attend on or follow obstruction may be met with. Boullon saw a remarkable case of the ingestion of needles in which these bodies were found in the small intestine, liver, pancreas, diaphragm, kidney and lung.

Treatment varies with the character of the bodies ingested, sand and gravel may be passed on by a laxative diet and even by the use of mild laxatives. Bernard gave 10 quarts of water and 4 oz. Glauber salts every hour for eight days, and the same amount by enema. For the larger solid bodies which obstruct the intestines the treatment is the same as for calculus. For sharp pointed bodies causing abscess and fistulæ, we must follow the indications, ever aiming at the discovery of the whereabouts of the offending object and its removal.

FOREIGN BODIES IN THE INTESTINES OF RUMINANTS.

Foreign bodies are usually arrested in the rumen of cattle and unless sharp, pointed or rough so as to cause mechanical trouble or caustic so as to act chemically, rarely do much harm. The most extraordinary objects that have found their way into the intestine are snakes. Gherardi claims that he found in the intestines a snake of 25 inches long; Jager found one of 21 inches in length, in an advanced state of decomposition, in the rectum of a calf. It is supposed that both had been taken in with the food. In each case there was obstruction of the intestine with severe colicky symptoms.

FOREIGN BODIES IN THE INTESTINES OF CARNIVORA.

Small bodies, especially playthings, feathers, hair, bristles, bones of prey. Lesions: congestion, inflammation, hemorrhage, ulceration, perforation, invagination. Symptoms: colic, vomiting, tucked up belly, straining, palpitation, rabiform symptoms, cough, convulsions. Course: emaciation, prostration, death in five days or two weeks according to seat of obstruction. Treatment: Oleaginous injections, laparotomy.

Causes. The dog is especially liable to this form of trouble, in consequence of his habit of carrying objects in his mouth and of playing with different objects especially the playthings of children. Marbles, pebbles, spinning-tops, corks, coins, nuts, peach stones, pieces of rubber, cloth or leather, bits of wood, sponge, needles, pins, potato, bone, cord, hair, bristles, feathers, wire, and a number of other objects. Some of them like feathers, hair, and bones are swallowed with food, and when that has been digested, they are either vomited or failing in this, are passed on into the intestine. Lately the author made a post mortem of a house dog with over 24 inches of the jejunum virtually blocked with fragments gnawed from a caouchouc ball and pieces of twine.

Cats also swallow a variety of objects. Benjamin and Megnin record three cases of intestinal obstruction by the crystal drops of shades.

Lesions. When the lumen of the intestine is blocked with a round solid body like a marble or peach stone there occur active congestion, inflammation, blood stasis and hemorrhage, with in many cases necrosis, ulceration and perforation. Similar lesions occur from cord. In a recent case of impaction with gnawed fragments of caouchouc and cord, the 24 inches of the bowel implicated were the seat of extended patches of necrosis and of deep, and even perforating ulcers on the lesser curvature of the intestine, evidently caused by the tension of the stretched cord on the shorter attached border of the gut. Cadeac says the lesions from cord are always at the point of attachment of the mesentery, whereas those coming from round or cubical solid bodies are mainly on the greater curvature. Mathis found at the pylorus a piece of net from which a cord extended through the small intestine and ended in a ravelled mass near the ileo-cæcal valve. The dragging of the cord on the intestine often causes invagination at one or several points.

Symptoms. There may be slight colic, dullness, a disposition to lie curled up in some secluded place, loss or caprice of appetite, vomiting, tucked up abdomen, arching of the back, straining, and unless the bowels are distended with gas, the obstruction can usually be felt by the two hands applied on opposite sides of the abdomen. The matters vomited are at first alimentary, then bilious and in the advanced stages always feculent.

The French veterinarians assure us that rabiform symptoms are very common as the result of obstruction of the intestines with foreign bodies. The indications are signs of fury without the barking which characterizes genuine rabies. The patient becomes wicked, cross and excitable, sometimes dull and morose, and snappish, his eyes glittering and his mouth frothy. He has alternate paroxysms of fury and torpor, at one time flying at and biting any living thing he meets, or tearing some object to pieces, and at another hiding away in secluded and dark corners. Massenat saw two dogs supposed to be affected by rabies, but which recovered promptly after having vomited the foreign bodies which they had swallowed. In a country where rabies is so prevalent as in France, it would be interesting to see the results of inoculation with some of the most pronounced of these rabiform cases.

Beside the rabiform symptoms cough and epileptic seizures occasionally result from the foreign bodies.

Course. Termination. Unless relief is obtained by vomiting or purging, appetite ceases altogether, emaciation advances rapidly, the animal becomes dull and stupid, being evidently poisoned by the absorbed toxins, and death may ensue in four or five days if the obstruction is near the stomach, or in one or two weeks if in the large intestines.

Treatment. The general treatment advised for the horse is applicable to the carnivora. Purgatives are always dangerous as threatening the overdistension and rupture of the bowel above the obstruction. Oleaginous and mucilaginous injections with manipulations are more promising if the obstruction is in the colon or rectum.

In many cases laparotomy is the only hopeful resort. Felizet and Degive have been quite successful in removing corks in this way, and Fröhner advises the operation to be performed under opium narcosis, and with antiseptic precautions. Make an incision of $1\frac{3}{4}$ inch near the umbilicus and parallel to the linea alba, extract the blocked loop of intestine, ligature it in front of the foreign body and behind it, incise, remove the offending mass and carefully close by sutures, bringing the muscular and serous coats in accurate opposition. Remove the ligatures, disinfect, return the bowel into the abdomen, close the abdominal wound with sutures and apply an antiseptic bandage.

If such cases are to be operated on it is important that it be done early, before the occurrence of necrosis, ulceration, perforation, or general infection.

RUPTURE OF THE INTESTINE. SOLIPEDS.

Causes: overdistensions in front of obstructions, softening, friability, necrosis, suppuration or ulceration, Duodenum from worms or perforation by pointed bodies, exudate in verminous embolism, petechial fever. Jejunum and ileum, by disease of walls, ulcers, abscesses, neoplasms, caustics in umbilical hernia, clamping of hernia. Cæcum, falls, blows, kicks, blows of horn, tusk, stump, calculi, abscesses, cauterizing of hernia. Colon, external traumas, calculi, worms, verminous thrombosis, neoplasms, abscesses, overdistensions, violent straining, arsenic. Symptoms: follow accident, signs of obstruction, no rumbling, tympany, stiffness, great prostration, fever. Death in short time.

Causes. Ruptures occur as we have already seen from overdistensions of the bowel in front of some obstruction, by ingesta, concretions, calculi, foreign bodies, etc., and this may take place in the most healthy organs. In other cases, however, there has been some pathological process at work rendering the intestinal wall soft, friable, necrotic, suppurative or ulcerative, by which its substance is attenuated or its consistency or cohesion reduced.

Duodenum. Lacerations of the duodenum are often connected with obstruction by tumors or the ravages of worms. These latter are mostly the *ascaris megalocephala*, accumulated in mass, and sometimes engaged in pouches outside the walls of the gut. In other cases, the walls of the intestine have been perforated by hard woody stalks of straw or hay (Mollereau) or of still more woody plants as in a case observed by the author, and in which the pylorus was perforated. Sometimes the exudate or blood extravasation attending on petechial fever, or verminous embolism will pave the way for the rupture. Perforations by pieces of wire (Schmidt) or other metallic bodies are also observed. Adhesive peritonitis has also rendered the walls friable and predisposed to rupture.

Jejunum and Ileum. Lesions are most frequent toward the termination of the ileum and resulting from obstructions of the bowel or the weakening of the walls by disease, or both. Ulcerations, abscess of the closed follicles opening into the peritoneum, and neoplasms of various kinds are to be especially noted among the causes. The impaction of the cæcum, blocking the ileo-cæcal valve is also among the observed factors. Other instances have been traced to deep cauterization of an umbilical hernia, the enclosed loop of small intestine becoming inflamed and perforated. The author has observed one instance from clamping of a hernia in which the contained intestine was adherent to the hernial sac.

Cæcum. From its position on the lower part of the abdomen and from its habitual plenitude with food or water, this organ is especially exposed to direct mechanical injuries and ruptures. A sudden fall, more especially if the umbilical region strikes on a stone or other projecting solid body, kicks with heavy boots or with the feet of other animals, blows with a cow's horn or a boar's tusks, and violent contact with stumps, poles and other objects may be the occasion of the rupture. These are usually found near the base of the viscus and across its longitudinal direction.

Inflammations, connected with punctures, calculi, parasites, etc., may render the walls so friable that they give way under slight strain or injury. Abscesses have been found in the walls of the viscus leading to perforation, and extension of inflammation from an umbilicus cauterized for hernia has determined adhesion and perforation.

Colon. The loaded colon is even more liable to mechanical injury than the cæcum. Occupying as it does the more lateral parts of the abdominal floor, it is even more exposed to kicks and blows, and extending as it does back toward the inguinal regions, it is especially in the way of blows of horns so often delivered in this region. From the solid nature of its contents the presence of calculi, the presence of blood sucking worms, and its implication in the congestions and extravasations of verminous thrombosis, this organ is especially liable to degenerations and inflammations which render its walls particularly friable. Neoplasms of various kinds, cancerous, tubercular, etc., have been found on its walls as occasions of rupture. Abscesses of strangles have ruptured into the viscus. Overdistensions in front of an obstruction in the pelvic flexure, floating colon or rectum are the most frequent causes of rupture. Again, cases have been seen as the result of violent exertions, as during straining in dystokia. It has been a complication of phrenic hernia, of volvulus of the double colon, and of ulceration caused by the prolonged ingestion of arsenic. In severe impaction the necrosis of the intestinal walls has proved a direct cause of laceration. The seat of these ruptures may be at any point, but it is most frequent in front of the pelvic flexure, or in the floating colon, or

directly in the seat of impaction.

Symptoms. The attack comes on suddenly, perhaps in connection with some special accident or injury, and is manifested by violent colicky pains which show no complete intermission. In many respects the symptoms resemble those of complete obstruction of the bowel, there is a suspension of peristalsis, rumbling, and defecation, a tendency to roll on the back and sit on the haunches, an oblivion of his surroundings and pain on pressing the abdomen. Usually the shock is marked in the dilated pupil, the weak or imperceptible pulse, the short, rapid breathing, cold ears, nose and limbs and the free perspirations. Tympany is usually present as the result of fermentation. Signs of infective peritonitis and auto-intoxication are shown in the extreme prostration, unsteady gait, dullness and stupor, and general symptoms of collapse. The temperature, at first normal, may rise to 105° or 106° as inflammation sets in, and may drop again prior to death.

Termination is fatal either by shock or by the resulting peritonitis and auto-intoxication. Exceptions may exist in case of adhesion of the diseased intestine to the walls of the abdomen and the formation of a fistula without implication of the peritoneum.

RUPTURE OF THE INTESTINES IN RUMINANTS.

From blows of horns, tusks, etc., from rectal abscess. Symptoms: colic, resulting in septic peritonitis and sinking. Treatment.

Lesions of this kind usually come from blows with the horns of others. They may lead to artificial anus as in a case reported by Rey, or the formation of a connecting sac as in that of Walley. In a case seen by the author a large abscess formed above the rectum, from injuries sustained in parturition. This ruptured into the gut leaving an immense empty cavity in which the hand could be moved about freely, but which gradually contracted so that the cow made a good recovery.

André furnishes an extraordinary record of rupture of the colon, blocked by a potato. It seems incredible that a potato could have traversed the stomachs and intestine without digestion.

The *symptoms* are those of violent colic suddenly appearing in connection with some manifest cause of injury, and going on to septic peritonitis and gradual sinking.

Treatment is manifestly useless excepting in the case of some such fortunate condition as in the case of abscess of the rectum in which the free use of injections and the antiseptic of the abscess cavity proved successful.

LACERATION OF THE INTESTINE IN SWINE.

This is rare and appears to have been observed only in connection with scrotal and ventral hernias, with adhesion. It may lead to an artificial anus which in its turn may cicatrise and close, or to the discharge of fæces into the peritoneal cavity with fatal effect. If seen early enough, laparotomy with suture of the bowel and careful antisepsis will be indicated.

LACERATION OF THE INTESTINE IN CARNIVORA.

Obstruction and overdistension, necrosis, ulceration, feculent impaction, kicks, parasites, caustics, abscess, tubercle, cancer. Symptoms: peritonitis following accident, vomiting, no defecation. Treatment: laparotomy.

The most common cause of intestinal rupture is obstruction by foreign bodies, with overdistension of the bowel immediately in front, or necrosis and ulceration of the portion of the bowel pressed upon. Feculent impaction acts in a similar way. Kicks and other external injuries sustained on a full intestine will lead to rupture. Perforation by parasites, by caustic agents swallowed, by abscesses, and by tubercle or cancer is also to be met with.

The *symptoms* are those of sudden peritonitis, with marked abdominal tenderness, tucking up of the abdomen, bringing the legs together under the body, vomiting, suspension of defecation and peristalsis. Rabiform symptoms have been noted.

Treatment. As in swine there is every hope of success by suture of the intestinal wound if done early. The same general method may be followed as in closing the wound after extraction of a foreign body.

ABSCESS OF THE BOWEL IN SOLIPEDS.

In strangles, from puncture, kicks, blows, foreign bodies in food, larva, cysts, large or small, creamy or cheesy, open into bowel or peritoneum, infective peritonitis. Symptoms: rigor, ill health, unthrift, colics, tender abdomen, tympany, painful movements, lying, rising, turning, going downhill, rectal exploration, phlegmonous swelling, pus passed by anus. Treatment: open when it points on abdominal wall, or when near rectum, antiseptics, support strength, careful dieting, antipurulent agents.

This is most common as an irregular form of strangles, the abscess forming in connection with the mesenteric glands or on the walls of the intestine. Small abscesses may also implicate the mucous glands or Peyer's patches as a result of catarrhal enteritis. Less frequently an abscess forms in the seat of the puncture of the colon for tympany, or in connection with blows, kicks, punctures with stable forks, nails and other pointed objects. Foreign bodies entering with the food and the cysts of the larvæ of the sclerostomata will also give rise to suppuration.

These abscesses may attain a large size, especially in strangles, and involve adhesions between the bowel and other viscera, or the walls of the abdomen. Or they may be small like peas or beans scattered along the coats of the intestine or between the folds of the mesentery. They may be inspissated to the consistency of thick cream or rich cheese, and they may rupture into the intestine, through the abdominal walls or into the peritoneum. In the last case infective peritonitis sets in usually with fatal results.

Symptoms. These are generally obscure. There may have been noticed a rigor, and there are always marked indications of ill health, dullness, lack of spirit or appetite, dryness and erection of the hair, hide bound, insensibility of the loins to pinching, colics after meals, tenderness of the abdomen, tympany, groaning when lying down or rising, when turned around short, or when walked down hill. Sometimes the abscess can be distinctly felt by the hand in the rectum. When it implicates the abdominal walls there is usually a diffuse phlegmonous swelling, at first soft and pasty, then firm and solid, and finally softening and fluctuating in the center. Sometimes there is the evacuation of pus by the anus or of the investing membrane of the abscess, and this may be expected to herald recovery. In case of infective peritonitis there are the usual symptoms of stiff movement, the bringing of the feet together under the belly, abdominal tenderness, trembling, hyperthermia, cold ears and limbs, cold perspirations, great dullness and prostration, small, weak or imperceptible pulse, hurried breathing and gradual sinking.

Treatment. This is most favorable when the abscess approaches the surface so as to be punctured through the abdominal walls. In other cases it is so situated that it can be punctured with trochar and cannula through the rectum. In such a case it may be evacuated and injected with a nontoxic antiseptic, the puncture and injection being repeated as wanted. In the internal and deeply seated abscesses we must seek to support the general health, give pure air, easily digestible and nourishing food, and agents that may be hoped to retard suppuration. Hyposulphite of soda in ½ oz. doses, or sulphide of calcium in scruple doses, may be repeated two or three times a day.

ULCERATION OF THE INTESTINES.

Symptom or sequel of other disease, or from traumas, caustics, neoplasms, peptic ulcers, verminous thrombosis, tubercle. Catarrhal erosion, peptic, deep, round ulcer, calculi with irregular ulcers, cord ulcer at mesenteric attachment, small, follicular, grouped ulcers, sloughing ulcers of infectious diseases, circular projecting, button like ulcers of hog cholera, microbes. Symptoms: diarrhoea, black, or red, sloughs, fever, blood stained vomit, manipulation. Treatment: for foreign body, poison, or infectious disease, careful diet, antiseptics.

Ulceration of the intestines is commonly a symptom or sequel of other intestinal disorder, such as intestinal catarrh, impaction, calculus, foreign body, parasites, petechial fever, influenza, glanders, rinderpest, Southern cattle fever, hog cholera, pneumoenteritis, rabies, canine distemper. Then there are ulcers, caused by sharp pointed bodies, by caustic agents ingested, and by obstructive changes in neoplasms. Peptic ulcers may occur in the duodenum as in the stomach. Finally local disturbances of the circulation and especially such as attend on verminous thrombosis, are at once predisposing and exciting causes of ulceration. Tuberculosis and other neoplasms are additional causes.

The ulcers may vary in different cases. In catarrh there is usually superficial desquamation of the epithelium, and erosions rather than deep ulcers. The peptic ulcer forms on the dependent wall of the gut, where the gastric secretions settle, and assumes a more or less perfectly circular outline (round ulcer). Those due to calculus or impaction, may be irregular patches mostly on the unattached side of the intestine and resulting from necrosis of the parts most exposed to pressure. The ulcers resulting from cords stretched along inside the bowel, are in the form of longitudinal sores on the attached or mesenteric side of the intestine, where the wall being shorter the cord continually presses. Follicular ulcerations are usually small, deep excavations, commonly arranged in groups. Ulcers connected with neoplasms have an irregular form determined by that of the morbid growth. In infectious diseases the ulcers are round or irregular, resulting from circumscribed sloughs. In most of the infectious diseases the tendency appears to be to attack the intervals between the folds of the mucosa, probably because the bacteria of ulceration find a safer lodgement in such places. In the hog cholera ulcers the older ulcers tend to the circular form with thick mass of necrotic tissue in the form of plates or scales imbedded in the bottom and projecting above the adjacent surface of the mucosa. As a rule the microbes which in the different cases preside over the necrobiosis are found in the depth and walls of the ulcers.

The *symptoms* are largely those of the diseases of which the ulcers are a concomitant or result. There is usually diarrhoea, which is generally black from extravasated blood, and may be marked by fresher red bloody striæ. Sloughs of variable size are not at all uncommon in the fæces. Hyperthermia is usually more intense than in ordinary chronic enteritis, indicating the action on the heat producing centres of the necrosing microbes and their toxins. In pigs and dogs there may be vomiting of dark blood stained material or of feculent matter. In the small animals it may be possible to feel through the walls of the abdomen the thickening of the intestine at and around the seat of any extensive ulcer.

Treatment. So far as this is not the treatment of the foreign bodies, poisons, or specific fevers which cause the ulcers, it consists mainly in careful dieting and the use of antiseptics such as subcarbonate of bismuth, salol, salicylic acid, sodium salicylate or naphthol.

DILATION OF THE INTESTINE.

Capacity adapted to ingesta, rich and nutritious food improves breeds, excessive filling renders paretic, dilates; obstructions, impactions, strangulations, hernias, invaginations, twisting, tumors, compressions, calculi, lowered innervation, impaired circulation, verminous aneurism, peritonitis, persistent umbilical vesicle in horse and ox, hernia of mucous through muscular coat, cæcal dilatation, colic, rectal, with atresia ani, diseased end of cord, retained fæces. Symptoms: colics after meals, abdominal and rectal exploration, softer than impaction. Treatment: empty mechanically or by laxatives, demulcents, kneading, stimulants, nuxvomica, ergot, barium chloride, eserine, rich concentrated food, electricity, enemata, laxatives.

It is a physiological law that the intestine develops in ratio with the demands made upon it, provided these demands are not too sudden and extreme. Thus the domestic pig and rabbit have intestines at once longer and more capacious than those of the wild varieties. The same is true of cattle and even of horses, heavy, rich feeding, generation after generation, increases the capacity to take in and utilize more, and to attain to a larger size and earlier maturity. In such a case the walls of the intestinal canal retain their primary thickness and strength and the whole change is in the direction of physiological improvement for economical ends.

When, however, the retention or habitual accumulation of food in the alimentary canal exceeds the self-adapting powers of its walls a true pathological dilatation takes place, and attenuation or thickening and paresis or actual paralysis of the walls ensues.

Whatever interferes with the normal active movement of the ingesta predisposes to this. Thus partial obstructions of all kinds, strictures, impactions, strangulations, hernias, invaginations, twisting, tumors, compressions, calculi, contribute to the overfilling of the bowel in front of them and to its more or less speedy dilatation. Whatever weakens the muscular walls of the bowels or the nerves presiding over these has a similar effect. Thus pressure on the solar plexus or its branches from any cause, or degeneration of the same, a tardy and imperfect circulation resulting from verminous aneurism and thrombosis, and a circumscribed peritonitis extending from the serous to the muscular coat of the bowel act in this way.

The persistence of the canal of the umbilical vesicle has been repeatedly observed in solipeds, in the form of a pouch or dilatation connected with the ileum three or four inches in front of the ileo-cæcal valve. Rauscher records one of these of thirteen inches long and having a capacity of seven quarts. These have been noticed in cattle as well.

Another form of sacculation results from rupture of the muscular coat through which the mucous forms a hernial sac in the peritoneal cavity. On a small scale these sacs are not uncommon, the size of a pea, a bean, or a marble, and very often containing larval or mature worms. Degive records an enormous dilatation of the horse's cæcum, Peuch, one of the pelvic flexure of the colon having a capacity of forty pounds, and Simonin one of the floating colon. Dilatations of the rectum always take place in the new born affected with atresia ani.

Dilatation of the rectum into a cloaca is found in the horse and ox, often connected with disease or injury of the terminal part of the spinal cord, and is very common in dogs and cats in connection with the compulsory retention of the fæces indoors. Pigs also present instances of the kind.

The *symptoms* are in the main slight colics, with or without tympany and recurring after each meal. In the small animals the distended gut may often be recognized by palpation through the abdominal walls, and in the larger animals by rectal exploration. The distended viscus has not the firmness nor hardness of impaction or calculus and is mainly recognizable by its bulk and form. When the distension is in the rectum it may be easily reached and contents dislodged with the effect of giving complete relief for the time being.

Treatment. Treatment is necessarily mainly palliative and consists in the removal of abnormal accumulations. From the rectum this can be done with the hand, or in the smaller animals with the finger. For abnormal dilatations more anterior, purgatives and mucilaginous injections are required, with kneading of the bowels through the abdominal walls, or through the rectum in the

larger animals, and stimulation of the peristalsis by nux vomica, ergot, barium chloride or eserine.

Having unloaded the dilated portion of any undue collection, further accumulation should be guarded against by giving nutritious food in small compass, and of a laxative nature, by stimulating peristalsis by nux vomica or other nerve stimulant and by the daily application of electricity. Enemata and laxatives should be employed when necessary.

STRICTURE OF THE INTESTINE.

From healing of ulcers, inflammation or infiltration, neoplasms, ring like or sacculated, in small intestine in horse preceded by a dilatation, an effect of verminous thrombosis; in cattle; in dog. Symptoms: Progressive, if in duodenum can't eat full meal, belches gas, has colics and tympany; in cattle tympany, unthrift; in dog vomiting, tympany, colic, accumulations. Treatment: Gradual stretching by bougies if within reach.

Strictures of the intestine are in the main the result of ulceration of the intestinal walls which contract in healing, or inflammation, and infiltration which leads to contraction in their organization into tissue. Neoplasms of the walls (cancer, myxoma, lipoma, polypus, melanosis, actinomycosis, tubercle) are additional causes of constriction. If resulting from a lesion which completely encircled the bowel there is an uniform constriction in the form of a circular ring; if on the other hand it started from a longitudinal ulcer or lesion the bowel is shortened on that side and puckered.

In **solipeds** strictures are most frequent in the small intestine, or rectum. The pylorus is often affected. When on the small intestine there is constantly a dilatation just in front of the obstruction. The constricted portion is usually short, but as seen from outside of the gut may be duplicated a number of times. Cadeac mentions seventeen such strictures in the same animal, each preceded by a dilatation. The individual stricture may be less than two inches in length and so narrow as just to allow the passage of the index finger. The walls of many times their natural thickness, are still further thickened by an external layer of adipose tissue. It may be the seat of a small abscess, or of a tumor. Internally the mucosa may show ulcerations.

The stricture or strictures in solipeds often depend on the disturbance of the circulation which results from verminous thrombosis, the exudate into the intestinal walls, undergoing organization, at once thickens and constricts the tube, and determines as secondary result the dilatation in front of it.

Professor Mauri of Toulouse records the case of a horse with a rectal stricture 4 inches from the anus, and a great dilatation in front. The removal of the stricture, secured normal defecation, (whereas before this the fæces had to be removed by hand) and the colics entirely disappeared.

In **cattle** strictures have been found mainly at or near the pylorus, less frequently in the rectum, and on one occasion (Revel) in connection with a cancerous tumor, in the colon.

In the dog the pylorus is also the favorite seat of thickening and stricture, yet it may occur in the small intestine, the rectum, or the colon.

Symptoms. These are gradually advancing, as the stricture approaches more and more nearly to a complete stenosis. If the stricture is in the pylorus or duodenum, the patient can not eat a full feed of grain without discomfort. He stops, hangs back on the halter, plants the fore feet in front, arches the neck, drawing in the nose and eructating gas. If he cannot eructate he is liable to show colics, tympany, and the general symptoms of gaseous indigestion of the stomach.

In **cattle** there is tympany, partial loss of appetite, tardy rumination, and loss of condition.

Dogs show vomiting as a prominent symptom. When the stricture is in the rectum there is a gradual lessening of the amount of fæces passed at a time and an accumulation of feculent masses in advance of the obstruction, recognizable by rectal exploration. When in the terminal part of the small intestine or in the colon, a gradual lessening of defecation, with tympanies and colics, culminating in complete obstruction, may afford a suggestion of the trouble but no means of certain diagnosis. In the smaller animals some additional indications may be had from abdominal palpation.

Treatment is usually hopeless unless the stricture is in the terminal portion of the rectum. In the latter case gradual dilatation by the passage of the hand, the finger, or of bougies which are used larger and larger, as they can be forced through with moderate pressure may secure a sufficient dilatation. Forced dilatation, or even careful incision at several different points of the circumference of the stricture may give good results in certain cases.

INTESTINAL INVAGINATION. INTUSSUSCEPTION IN SOLIPEDS.

Definition. Seat: ileum into cæcum, rectum through sphincter, duodenum into stomach, floating small intestine into itself, cæcum into colon. Lesions: blocking, or tearing of mesentery, dark congestion, peritoneal adhesions, incarceration gut, necroses, sloughing of invagination. Symptoms: colics of obstruction, enteritis, and septic infection, eructation, emesis, tenesmus, signs of sepsis and collapse, death in seven hours or more, or recovery by disinvagination or sloughing. Diagnosis: by rectal exploration or passing of slough. Treatment: oily laxatives, demulcents, enemata, mechanical restoration of everted rectum, laparotomy.

Definition. The sliding of one portion of an intestine into a more dilated one, as if a few inches of the leg of a stocking were drawn within an adjoining portion which is continuous with it.

Seat. It is most commonly seen in the inversion of the small intestine into itself or into the cæcum, or next to this the passage of the rectum through the sphincter ani, to constitute eversion of the rectum. It would appear to be possible at any part of the intestinal canal in the horse, in which the bowels are more free to move than they are in ruminants. Peuch records a case of invagination of the duodenum into the stomach and Cadeac gives a woodcut of such a case, which one would suppose the fixed position of the duodenum would render impossible. It is conceivable that the jejunum could be invaginated into the duodenum, and that this should have continued until it extended into the stomach, but it is difficult to see how the duodenum itself could have passed into the stomach without tearing itself loose from its connections with the pancreas, liver and transverse colon.

Schröder, Serres and Lafosse describe cases in which the small intestine was everted into the cæcum and thence through the colon and rectum until it protruded from the anus.

The invagination of the floating small intestine into itself is common at any point, and extensive and even repeated. Marcout records a case in which 24 feet were invaginated, and Rey a case of quadruple invagination at the same point.

The invagination of the cæcum into the colon is frequent, the blind end of the cæcum falling into the body of the same organ, and this continuing to increase until it passes on into the colon, and even carries a portion of the small intestine with it. This lesion is more rare in solipeds because the cæcum has its blind end lowest and gravitation opposes its invagination.

Resulting Lesions. In any case of invagination it must be noted that it is not the intestine alone which slips into its fellow, but it carries with it its attaching mesentery, which, dragging on one side of the invaginated gut, shortens and puckers that and turns its opening against the wall of the enclosing gut so as to block it, while the opposite or free side passes on and tends to form convolutions. If the outer and enveloping intestine is too small to allow of this, the detaining mesentery of the invaginated mass must be torn or stretched unduly and its circulation and innervation correspondingly impaired. When the invagination occurs of one portion of the small intestine into another of nearly equal size, the resulting mass is firm like a stuffed sausage, and this enlargement and consolidation ends abruptly at the point of visible entrance of the smaller contracted portion, into the larger dilated one.

If recent, the invaginated mass is still easily disengaged from the enveloping portion, though considerably congested and dark in color in proportion to the duration of the lesion. When it has been longer confined the incarcerated portion is the seat of extreme congestion, and extravasation, and has a dark red or black color. The exudation into its substance, which is especially abundant in the mucosa and submucosa, produces a thickening which may virtually close the lumen, and on the opposing peritoneal surfaces leads to adhesions which prevent the extraction of the imprisoned mass. The interruption of the circulation and the compression of the invaginated mass, leads soon to necrosis and thus a specially offensive odor is produced, and if the animal survives the whole may be sloughed off and passed with the fæces, the ends of the intussuscepted portion and of that receiving it meanwhile uniting and becoming continuous with each other.

Symptoms. These are the violent colic of obstruction of the bowels, soon complicated by those of enteritis and finally of septic infection.

The animal looks at his flank, paws, kicks with his hind feet, lies down, rolls, sits on his haunches, waves the head from side to side, and sometimes eructates or even vomits. Straining may

be violent, with the passage of a few mucus-covered balls only, and rumbling may continue for a time if the small intestines only are involved.

The partial subsidence of the acute pains, the presence of tremors, dullness and stupor, the coldness of the ears and limbs, the small, weak or imperceptible pulsations, the cold sweats, dilated pupils, and loss of intelligence in the expression of the eye and countenance may indicate gangrene, and bespeak an early death which may take place in seven hours.

The subsidence of the acute symptoms with improvement in the general appearance and partial recovery of appetite may indicate a spontaneous reduction of the invagination, an issue which may happily arrive in any case in the early stages, but especially in those implicating the cæcum and colon.

An absolutely certain *diagnosis* is rarely possible, unless the lesion is a protrusion of the rectum, or unless as the disease advances the invaginated part is sloughed off and passed per anum.

Treatment. The failure to make a certain diagnosis usually stands in the way of intelligent treatment. Oleaginous laxatives and mucilaginous gruels are advised to keep the contents liquid, and favor their passage through the narrowed lumen of the invaginated bowel. In cases implicating the floating colon and rectum abundant watery or mucilaginous injections may assist in restoring a bowel which has not been too long displaced. In case of eversion of the rectum, the hand should be inserted into the protruding gut and carried on till it passes through the sphincter ani. Then, by pushing it onward, the arm carries in a portion of the invaginated gut and usually of the outer portion next to the anus as well, and this should be assisted by the other free hand, and even if necessary by those of an assistant, and whatever is passed through the sphincter should be carefully retained, while the arm is withdrawn for a second movement of the same kind, and this should be repeated until the whole protruding mass has been replaced.

Invaginations situated more anteriorly and which can be correctly diagnosed by rectal exploration or otherwise, will sometimes warrant laparotomy, especially those of the cæcum into the colon, where adhesion of the peritoneal surfaces is less common or longer delayed. The patient should be given chloroform or ether, the abdominal walls should be washed and treated with antiseptics, and the incision made back of the sternum and to one side of the median line, and large enough to admit the exploring hand. It has also been suggested to introduce the hand through the inguinal ring, or behind the posterior border of the internal oblique muscle.

INTESTINAL INVAGINATION IN RUMINANTS AND SWINE.

Double colon cannot be invaginated, floating small intestine, cæcum and floating colon can. Causes. Lesions. Symptoms: Acute, violent, persistent colic, palpation of right flank causes gurgling, rectal exploration, prostration, collapse. Duration 1 to 5 weeks. Treatment: Laxative, enemata, injections of sodium bicarbonate and tartaric acid, laparotomy.

In these animals the double colon is rolled around itself between the folds of the great mesentery the free border of which supports the small intestine. The arrangement is as if a piece of rubber tubing were first doubled upon itself, and the end of the loop were then turned inward and the remainder wound round it as a centre. If this were then sewed between two pieces of cloth, the stitches passing between the different windings of the tube at all points, we would have an arrangement fairly representing that of the double colon of ruminants, and, for our present purpose, of swine as well. It must be evident that no portion of a tube arranged in this way can slide into another. It would also appear that the small intestine cannot become invaginated to any extent into another portion or into the cæcum without extreme stretching or laceration of the small portion of mesentery left between it and the coils of the double colon above. The anatomical arrangement is therefore opposed to the formation of invaginations in a way that is not the case in the horse.

Yet invaginations are by no means unknown in these genera. The small intestine can be invaginated into itself or into the cæcum. The cæcum, which floats loose at the right side of the mesentery that envelopes the double colon, can be invaginated into the colon, and the floating colon can be invaginated into the double colon on the one hand and into the rectum and through the anus on the other. Invagination into the rectum, for eight inches, in a bull calf, of six days old, is reported by Cartwright in the *Veterinarian* for 1829. In a similar case of Youatt's the intussuscepted portion sloughed off and was discharged per anum.

The *causes* are like those acting in solipeds, and which give rise to excessive and irregular peristalsis. A drink of ice cold water, indigestions and colics of various kinds, diarrhoea, chills, the irritation caused by poisons or parasites, and the paresis and dilatation of portions of the intestine into which the more active portions can easily pass. Almost any irritation or congestion may cause intussusception, and young animals in which peristalsis is most energetic are the most liable.

Lesions. The intussusception is usually found in the ileum and to a less extent in other parts of the small intestine, or involving the cæcum and colon, or again the floating colon and rectum. The successive conditions of congestion, exudation, adhesion, obstruction, necrosis, sloughing, and repair by union of the remaining ends are the same as in the horse.

Symptoms. There is acute, agonizing and dangerous colic in an animal in which these troubles are usually comparatively slight and transient. The animal looks at the right flank, paws or stamps with fore feet as well as hind, lies down and rises often, strains to pass manure but passes only mucus or a few small hard masses, if anything. If pressure is made on the right side of the abdomen and the hand suddenly withdrawn there is a significant gurgling and the corresponding hind foot is lifted or moved forward or backward, appetite and rumination are lost, the pulse becomes rapid and weak, and the animal becomes prostrate, dull and stupid, often remaining recumbent in spite of all efforts to raise him. Rectal exploration may detect the firm tender mass in the seat of the invagination. The disease may last from one week to five, according as the obstruction is complete or partial. The usual termination is a fatal one, though a certain number of spontaneous recoveries are met with.

Treatment. By a happy accident the peristalsis or anti-peristalsis determined by a purgative will sometimes disengage the intussuscepted bowel. Copious injections into the rectum may also prove useful in case of intussusception of the floating colon or rectum. Or the disengagement of carbon dioxide from the injection of solutions of sodium bicarbonate and tartaric acid may be tried. Laparotomy is however the most radical measure when a certain diagnosis has been made and this is less dangerous in the cow than in the horse in which peritonitis is so grave. Under antiseptic precautions an incision is made in the right flank and the invagination found and reduced. In case firm adhesions have already taken place, and above all if the included gut is apparently gangrenous,

the latter may be exposed by breaking down the connections at the side opposite to the attachment of the mesentery, or where the adhesions are least firm, then cutting out and removing the incarcerated gut and carefully closing the opening between the ends by suture. The use of a sublimate or carbolic acid solution and careful suturing and bandaging of the external wound with carbolated cotton wool will often give a successful issue.

INTESTINAL INVAGINATION IN DOGS AND CATS.

Anatomical conditions favor. Causes: as in other animals, common in icterus, and surgical operations from deranged peristalsis. Lesions: most common in small intestines, congestion, inflammation, necrosis, sloughing. Symptoms: may be colic, but not always, dullness, anorexia, vomiting, constipation, palpation, swelling firmer than from impacted twine. Treatment: shot, castor oil and exercise on hind legs. Demulcents. Laparotomy.

The intestines of the carnivora are more open to invagination than in other domestic animals for even the colon is free enough throughout its course to allow of one part sliding into another. The *causes* to which it is attributed are in kind the same as in other domestic animals. The swallowing of ice cold water in excess when heated, diarrhoea, superpurgation, intestinal worms, the active peristalsis of early life, and jaundice have been especially blamed. Reynal found intussusception twenty times in forty cases of icterus and Rancilla four times in five cases. It has been frequently seen after severe surgical operations, and it is surmised that in both cases alike the deranged peristalsis attendant on severe suffering was the cause of the accident.

Seat and Lesions. The most common seat of invagination is in the small intestines, and less so in the cæcum and colon, or rectum. The lesions are as in the other animals, congestion, infiltration, adhesion, necrosis, gangrene and sloughing.

Symptoms. There may be colics as in the larger animals, but in some instances there are simply prostration, dullness, inappetence, vomiting, constipation, or the passage of a little liquid and foetid excrement. Palpation of the abdomen detects a firm, cylindroid and very tender swelling on the line of the softer intestine which taken with the other symptoms is nearly pathognomonic. If situated in the small intestine and disconnected from the rectum and pelvis the diagnosis is more satisfactory. Impaction is most commonly in the rectum and floating colon and can be traced into the pelvis and even felt by the finger introduced into the anus. It might be confounded with obstruction of the intestine by the ingestion of twine, but the swelling is usually firmer and the cylindroid outline more uniform in intussusception.

Treatment. The measures recommended for the larger animals are applicable to the dog. Cadeac has had four recoveries in seven cases after the use of leaden shot and castor oil. Three balls of No. 16 calibre are dipped in castor oil and given to the dog. This is followed by ½ oz. of castor oil slightly heated, and walking or running exercise, or take the dog by his fore limbs and walk him around on his hind. No drink is allowed for 24 hours, and a quart of decoction of flax seed on the day following.

Should these measures fail, laparotomy is available, yet it is more promising in proportion as it is resorted to early, before ulceration, or gangrene has set in. The manipulations are practically the same as in the ox and the outcome is even more promising. The diet should be restricted to milk or mucilaginous gruels for a week after the operation.

VOLVULUS (TWISTING) OF THE INTESTINE IN SOLIPEDS.

Definition: rolling of a loop on its mesenteric axis, bending in a vicious direction, rolling of one loop round another. Causes: laxity of mesentery in hernia, relaxation or rupture of linea alba, pot belly in old breeding mares, sudden movements in falls, leaps, draught, galop, slipping, mounting, warm weather, casting, rolling, rising, sudden filling of a loop, heavy feeding and fermentescible food, cold drinks, chills. Lesions, most in double colon, next in jejunum, cæcum wrapped in small intestine, floating colon, tympany and pallor of obstructed loops, later congestion, infiltration, extravasation, adhesions, necrosis, sloughs, infective peritonitis. Symptoms: sudden severe attack, violent, reckless colic movements, pain constant with exacerbations, fever, prostration, collapse. Diagnosis: only by rectal exploration and exceptionally. Treatment: by rectal manipulation, eserine, castor oil, laparotomy.

Strictly speaking this lesion consists in the twisting of a loop of intestine upon its mesenteric axis, so that the portion which is drawn spirally over the mesentery of the other is more or less completely obstructed. The term has, however, been applied as well to the turning of a viscus at a sharp angle from its normal direction so as to interfere more or less with the passage of its contents and with its circulation. This has been especially seen in a vicious direction given to the cæcum, but also at times to the double colon. The simple twisting on the mesenteric axis is common to the floating portion of the small intestines, the double colon, floating colon and rectum. A third form of twisting which is, however, rather a strangulation, consists in the rolling of one loop of intestine round the loop of another, the mesentery of which has become unduly long.

Causes. The predisposing cause is a certain laxity or undue lengthening of the mesenteric attachment of the intestine. This is sometimes formed in connection with the existence of hernia, into which the bowel protrudes, or short of this a relaxation or rupture of the linea alba so that the whole of the intestinal mass hangs down unduly, or finally in unthrifty pot-bellied animals and in breeding mares in which the abdomen is unduly pendent.

Next comes the question of sudden movement as in falls, in leaping, in violent exertions of draught, or galop, in sudden slipping upon wet or icy ground, and of stallions in mounting mares. In the Omnibus Company's (Paris) horses Palat found 35 cases in entire horses, 23 in mares, and 11 in geldings. The stallions in the stables were fewer than the mares and geldings put together, but it does not appear that the difference was sufficient to make the above figures very significant. These stallions it should be added, are not used for breeding, so that the statistics have no bearing on the effect of mounting.

Palat's figures show a greater number of cases in summer, than in winter. There were 58 cases from April to October and but 21 from November to March. It would seem as if the relaxation of the system and mesentery in summer more than counterbalanced the combined effect of slipping on ice and sudden chills.

A large proportion of the cases have been found in horses that have died of colic, or which have been cast for operation, and the recent character of the lesions has often shown that we must look upon them as the result of the tumultuous peristalsis, and the lying, rolling, sitting and other sudden and unwonted movements performed. A heavily loaded portion of bowel, occupying a position slightly lower than a lighter portion or parallel to it, suddenly moves by gravitation when their relative positions are altered as in rolling, decubitus, or rising, and it thereby becomes twisted upon itself. Or a portion of intestine filled with liquid or gaseous contents is suddenly emptied by the passage of these onward into another and the latter portion of intestine or some other lodged alongside it, in moving to fill the place, rotates upon itself and establishes a volvulus.

Hard worked horses which are subjected to stimulating feeding are much more frequently attacked than those which have light work and feeding. In the same way newly harvested hay or oats, spoiled or otherwise indigestible food have apparently been productive causes. Cold drinks, and exposure to cold draughts have been similarly charged. Indeed any cause of indigestion and

colic may be held to predispose to volvulus.

Lesions. Seat. Twisting of the small intestine is impossible in the duodenum, and for the floating portion it is far more common in the ileum and terminal portion of the jejunum where the mesentery is long than in the anterior jejunum where it is short.

The double colon from the sternal portion to the pelvic flexure being free from any restraint by omental or mesenteric bands is especially liable to torsion. Palat found four cases of torsion of the colon to one of the small intestine, and Schutze gives the ratio as 56 of the first to 13 of the second.

The cæcum has been frequently found twisted upon itself with a portion of the small intestine rolled round it.

The floating colon like the small intestine is rolled around its mesenteric axis, but cases are much more rare than in the case of the small intestine.

In a recent and complete twisting with obstruction of the bowel, the loop of intestine is distended with gas the result of fermentation of its contents, and its walls may be thin and pale. Much more commonly and when the lesion is of longer standing there is hyperæmia, and infiltration and thickening with inflammatory products, and blood extravasations. At the seat of torsion the compressed intestine is congested, covered with petechiæ, and its peritoneal surface with fibrinous exudate tending to bind the parts together. Later there may be seen spots of necrosis and perforating sores and semi-detached sloughs, or the whole mass of twisted bowel may be gangrenous. The patient usually dies before this last stage has been reached. If the animal survives long enough the lesions of infective peritonitis are constantly present.

Symptoms. The disease usually sets in suddenly with intense severity. In exceptional cases there is an insidious onset, the twist being at first but partial and gradually increasing and for a time the contents pass on in a restricted but still physiological manner. Colics at first slight become by degrees more and more intense until all the symptoms of obstruction and acute inflammation are developed.

More commonly symptoms of extreme gravity appear at once, the patient stops, paws, kicks at his belly, tries to lie down, strains to defecate or urinate, lies down, rolls, sits, gets up and moves round uneasily trying to lie down again. He looks at the flank with anxious eye and countenance and has all indications of the most violent colic. Pain is constant, but worse at one time than another, the pulse is from 50 to 90 and becomes weak and even imperceptible as the case advances, and hyperthermia, at first slight or absent rises with the onset of inflammation. Finally great prostration, depression and stupor, sunken, glazed eye with dilated pupil, and cold sweats and extremities bespeak collapse or general infection.

Diagnosis is rarely certain. The sudden onset, extreme violence of the symptoms, and rapidly fatal progress are significant and in exceptional cases rectal exploration will detect obstruction in the rectum or floating colon, or a tympanitic condition of the pelvic flexure.

Treatment. A rational treatment is only possible in those rare cases in which the diagnosis is certain. When the lesion can be reached in the rectum or adjacent part of the floating colon, the oiled hand may be made to drag on the interior of the viscus so as to restore it to its normal position.

Cadiot has had recoveries from supposed volvulus by the use of eserine hypodermically, and Trasbot by the use of castor oil and it is just possible that active peristalsis, and plenitude of the bowel running into the volvulus, may serve to unwind slight cases. When the diagnosis is certain and the case, as usual, intractable the resort of laparotomy and the attempt at untwisting is fully warranted. The case is a fatal one if unrelieved.

VOLVULUS IN RUMINANTS.

This condition is almost unknown in ruminants, Reichert recording a single case of volvulus of the ileum involving 8 inches of the gut. The reason for the habitual immunity is to be found in the arrangement of the small intestine at the end of a mesentery which is strengthened and stiffened by the winding folds of the colon until twisting is practically impossible. There remains, therefore, only the floating colon and rectum at all exposed to the lesion. The isolated case of Reichert only proves the rule.

VOLVULUS IN DOGS.

Rare. Short mesentery hinders. Occurs with hernia. Symptoms: obstruction, prostration, colic, tense, painful abdomen, retching, anorexia, exhaustion. Treatment: laparotomy.

The carnivora seem to be protected against volvulus by the shortness of their mesentery, the comparative lightness of the intestinal contents, and the restricted area of the abdominal cavity. Cadiot, Müller, and Friedberger and Fröhner agree in ignoring the subject as a canine disease, while Cadeac mentions only such cases as are complicated by mesenteric hernia, the protruding loop becoming twisted in the wound through which it has protruded.

“The *symptoms* are those of invagination or intestinal obstruction; sometimes the animal is dull, anxious, resting almost constantly down on his belly, and this prostration dominates the table of symptoms; sometimes, on the contrary, the subject manifests signs of excitement and intestinal pain; it trembles, lies down, glances at its flanks; sometimes it even lets itself fall abruptly, straightens out stiffly its limbs and head, clenches its jaws and rolls its eyes.”

“In all cases the belly is hard, drawn up, painful to pressure or palpation; but these means of exploration are insufficient to feel the intestinal knot.”

“Constipation is persistent, obstinate, and efforts at vomiting continuous; anorexia is incomplete, or the animal rejects the solid and liquid aliments immediately after their ingestion. Vomited matters, when they exist, become glairy, bilious toward the end of the attack; but sometimes the animal becomes exhausted in his fruitless efforts; one is rendered uncertain and hesitates to confirm his diagnosis by laparotomy.”

The only treatment advised is by laparotomy.

INTESTINAL STRANGULATIONS BY ADVENTITIOUS BANDS.

Result of circumscribed peritonitis. Extends from one part of abdominal wall to another, around or between intestines, on omentum, or adhesion of omentum to inguinal ring. In cattle tubercular products strangulate bowels. Symptoms: the colic of obstruction, rectal exploration. Treatment: laparotomy, removal of constricting band.

Adventitious fibrous bands in the abdomen are the result of a pre-existing peritonitis of a circumscribed area. The fibrinous exudate thrown out on the parietal peritoneum is detached in its median part but remains adherent at the ends, and when organized into tense resistant white fibrous tissue remains as a trap to entangle and strangulate the intestinal folds. In other cases they form on or between intestines or other viscera which are for the moment torpid and inactive, and contracting as they become organized, they bind these together and hamper their movements, and endanger the integrity of all movable viscera in the vicinity. Again they form on the omentum and threaten the integrity of the bowels by strangulation. The omentum protruding at castration becomes adherent to the inguinal ring and forms a dangerous band.

In *cattle* the growth of peritoneal tubercle often binds the small intestines together in an inextricable tangle leading to considerable compression, and obstruction without a complete stenosis.

Symptoms. These vary with the organ constricted, but they are usually those of a more or less perfect obstruction of some portion of the intestine small or large, which has passed over or under the constricting band and fails to return, often undergoing a torsion which renders its imprisonment still more secure. The author has found the double colon fatally constricted in this way, and Leblanc furnishes a similar case. Cases are on record of the binding of the duodenum to the right lobe of the liver by a strong fibrous band, of the binding of the double colon to the right flank and in another case to the floor of the abdomen, and of the ileum to the meso-rectum and in another case to the left flank. These give rise sooner or later to obstruction of these viscera or, to incarceration of adjacent ones so that the animal dies of intestinal obstruction with its attendant symptoms.

Treatment. If the true nature of the case can be made out by rectal exploration the appropriate treatment is by laparotomy and the removal of the constricting band.

PELVIC HERNIA IN THE OX. GUT TIE.

Cause: laceration of parietal peritoneum by tearing through the spermatic artery, a loop of intestine is strangulated in the resulting pouch. Diagnosis: patient is a steer castrated by the method named, rectal exploration confirms. Treatment: turn on back, jump from a height, trot down hill, dislodge incarcerated mass by hand in rectum, pass cannula through rectum, introduce probpointed bistoury and cut through the band, or laparotomy. Second Form: adhesion of stump of spermatic cord to intestine, or abdominal wall or formation of a pediculated tumor, and resulting strangulation of intestine. Third Form: weight of testicles tears the peritoneum from the abdominal wall forming pouch. Treatment: laparotomy.

Causes. This is a peculiar affection said to be connected with a faulty mode of castration in calves. When the testicle has been exposed, the spermatic cord is torn through by sheer force, or the posterior portion having been cut across the anterior division is dragged upon violently until torn apart. The gelders usually estimate the quality of the operation by the length of the artery which can be torn out. The artery which is the most resistant portion of the cord and the last to give way takes its origin from the posterior aorta opposite the posterior mesenteric artery, extends outward by a curvilinear course just in front of the brim of the pelvis, and when stretched violently it is straightened out and carries with it the peritoneum, tearing it from the portion immediately in front and forming a sac, or tearing it completely apart from the abdominal wall in its median part and leaving it attached above and below. Into this sac, or above this band, which is just below the sacral transverse processes, a loop of intestine may pass, and becoming strangled there, produces all the symptoms of intestinal obstruction.

Diagnosis is helped by the fact that the animal is a steer, and in a locality where the above-named mode of castration is in vogue. It is completed by feeling the imprisoned intestine and the constricting cord just under the right or left transverse processes of the sacrum.

Treatment. The gut will sometimes escape from the sac if the animal is turned quickly on its back. Another method is to invoke the influence of gravitation by jumping the animal from a high step down to a lower level, or by trotting him down a steep incline. A still more effective method is to introduce the hand into the rectum and press the palm upward and forward against the soft mass of the imprisoned intestine. In this way the gaseous, liquid and solid contents are passed over into the portion of the gut in front of the constriction, and by continuing the process the intestine itself can usually be pressed out and the suffering relieved. It is further suggested to press the thumb or the whole hand forward against the constricting band and tear it in two. A certain amount of to and fro movement is usually required and in exceptional cases the cord is so strong that the measure has to be abandoned.

Another resort is to pass a cannula and trochar through the adjacent part of the rectum, and withdrawing the trochar, to pass a probpointed bistoury through the cannula, and beneath the cord and cut it in two.

These measures failing an incision must be made in the right flank, following the line of the fibres of the external oblique, and the hand being introduced and passed round the posterior border of the omentum the seat of the disease is found and the constricting cord is cut with a probpointed bistoury or a bistouri caché. The wound is then sutured, disinfected and covered with antiseptic gauze or cotton and bandage. The sounds of peristalsis are resumed and in five or six hours defecation is restored.

2nd Form. Another alleged condition vouched for by English and continental veterinarians depends on drawing down the spermatic cord as far as possible, cutting it off and allowing it to be retracted into the abdomen. Having been detached by the traction from the abdominal wall it is alleged to float free, sometimes establishing an adhesion to one of the intestines of which it later limits the movements; sometimes forming a connection with the abdominal wall and forming a sling in which the intestines may be snared, and sometimes swelling at its free end to form a pedunculated tumor, and winding around a loop of intestine so as to strangle it. For these conditions incision of the abdominal wall and section of the offending cord are recommended.

3rd Form. Lobbe tells us that the disease is common in bulls as well as oxen in mountainous regions, being determined by violent exertions, or by the struggle to rise, when the animal has

accidently fallen. In this alleged form there is the suggestion of the heavy pendent testicles as factors in detaching the cord with its vessels and nerves from the abdominal wall, so as to form a loop or snare for the intestine.

DIAPHRAGMATOCELE. DIAPHRAGMATIC HERNIA. PHRENIC HERNIA.

Definition. Susceptibility by genera, horse, dog, ox. Enterocoele, epiplocele, gastrocele, hepatocele. Congenital, arrest of development and of closure of foramen or elsewhere, diaphragm absent. Traumatic, blows on false ribs by pole, shaft, buffer, gate, bars, beams, kicks, blows with horns, tusks, clubs, fractured rib perforating diaphragm, falls on projecting bodies, muscular strains in draught, plunging, falling, slipping, casting, parturition; trotting or galloping down hill, jumping to lower level, slipping to knees, dystokia, colic, tympany. Symptoms: extreme dyspnoea and asphyxia; or difficult breathing slowly increasing, colics, dilated nostrils, retracted angle of mouth, projecting eyeballs, shallow, rapid catching respirations, gurgling in chest, drumlike percussion sounds, with perhaps flat areas; in slight cases, listlessness, colics, double lift of flank, tender intercostals; in chronic cases, short wind on exertion, sluggishness, colics after meals. Hernia through intercostal space. Lesions: unduly large foramen sinistrum or dextrum, lacerations of all forms and sizes, edges ragged, thickened, bloody, broken rib, inflated stomach causes shreddy tear; post mortem lacerations show no inflammatory products; chronic cases have edges devoid of inflammation and often smooth, serosæ usually perforated, nature of hernial mass, omentum, intestine, colon, cæcum, stomach, spleen, liver, congestion of viscera involved. Prognosis: slight cases may survive, to be fattened or to breed, but are useless for work. Treatment: quiet, sedatives, antiferments, cathartic, concentrated food, lift by fore limbs, incline stall backward and downward, laparotomy in cattle, dogs, and swine.

As this lesion is shown by symptoms referable to internal organs only, and as it is considered irremediable by surgical measures, it may be properly considered in the class of medical affections.

Definition. A displacement of one or more of the abdominal organs into the cavity of the thorax.

Frequency in different animals. It has been found most frequently in the horse, and less so in the dog and ox. Severe exertions conduce to it in horse and dog, while in the ruminant the great bulk of the gastric cavities, covering the whole posterior surface of the diaphragm, tends to prevent protrusion even if a slight rupture has taken place. On the other hand, the weight of the gastric cavities in the ox and the tension upon the œsophagus, when the animals ride each other, sometimes cause laceration of the foramen sinistrum and hernia of the reticulum.

Hernial mass in different animals. In the horse the protruding organ is most commonly the small intestine (enterocoele), omentum (epiplocele), colon, or less frequently the cæcum, stomach (gastrocele), or liver (hepatocele). In the ox the reticulum most commonly protrudes, and after that the liver, abomasum, the omentum or the small intestine. In the dog the mass is usually formed by the stomach or liver, or less frequently by the small intestine or omentum.

Causes and mode of formation. The hernia is either congenital or acquired after birth. Again, it may result from imperfect development or from a trauma. Congenital cases usually depend on an arrest of development, the foramen sinistrum fails to close and the abdominal organ passes through by the side of the œsophagus into the chest; or the diaphragm is left imperfect at some other point, and the two cavities, abdominal and thoracic, communicate. In some instances there is left not a distinct opening but a mere relaxation of the parts and under traction, as, for example, by the gullet, an orifice is torn and a hernia protrudes. Such cases are very rare. Other lacerations may be almost all referred to external injuries on the posterior ribs, violent muscular exertions, sudden shocks in connection with falls, or throwing for operations, and overdistension of the abdominal viscera.

1st. External Violence. In the larger animals this may come from blows on the last ribs, by poles or shafts of carriages, the buffers of cars, the end of a gate and the gatepost, projecting ends of bars or beams against which they run, kicks by horse or ox, blows by the horns of cattle or tusks of boars. In the smaller animals in addition to the above, blows with heavy clubs and kicks with heavy boots. In all such cases there is usually a fracture of one or more ribs, the sharp broken ends of which are forced into the diaphragm, which they tear when they again spring outward. The same occurs as the result of falls on hard projecting bodies of any kind.

2nd. Muscular Strain. In heavy draught the fixing of the glottis, ribs and diaphragm and the extreme contraction of the abdominal muscles often lead to extraordinary tension of the muscular septum by the mere violence of which, or when there is superadded a sudden shock, (in plunging in harness, or displacement of the feet or slipping and falling,) the diaphragm is torn, usually in its tendinous portion, and the abdominal viscus protrudes into the chest. If the diaphragm is

momentarily relaxed the lesion may take place in the peripheral muscular portion. In still other cases the lesion is at one of the natural openings. The same accident occurs in animals thrown for operation, the abdominal viscera being full, the hind limbs drawn forward so as to further compress the belly, and the muscles being subjected to violent contraction in the efforts to get loose. Violent straining in dystokia is another cause which, however, usually partakes of a sudden shock on the diaphragm when a violent pain sets in.

3rd. Sudden Shocks on the diaphragm, and pressure by the abdominal organs. In the horse especially the weight of the abdominal viscera is very great and the floor of the abdomen inclines downward and forward so that the whole mass presses with great force against the concave diaphragm. In cattle the great weight of the stomach and liver is especially important and in dog and pig of the liver mainly. In trotting or galloping down hill or jumping from a higher to a lower level, or in slipping back on the fore feet so that the horse falls on the knees or shoulders, this pressure is suddenly greatly enhanced and the tense diaphragm may give way in its tendinous portion or the relaxed organ through its muscular tissue. A similar danger attends on the violent straining which attends on difficult cases of parturition, and even in cases of overloaded stomach and tympany and especially when the tortured animal throws itself suddenly and recklessly on the ground. In cattle and sheep this is usually the result of tympany of the rumen, and in solipeds of stomach and intestines.

Rupture by simple overloading or overdistention is, however, a rare occurrence, and many cases attributed to this are in reality instances of *post mortem* lesions, to be identified by the seat of the laceration in the muscular portion, and by the absence of blood clots, exudate, thickening or other sign of inflammation on the torn border.

Symptoms. These bear a direct relation to the size of the laceration and the mass of abdominal organs that protrude into the chest.

In *very grave, recent cases*, with a great phrenic rupture and a most extensive protrusion of abdominal organs into the chest, there may be simply the indications of extreme dyspnoea, nostrils and chest widely and persistently distended, nasal mucosa darkly congested, countenance pinched, eyes protruding and fixed, pupils dilated, breathing rapid, shallow and oppressed, and in a few minutes the animal staggers and falls in the death agony.

In *cases which are less rapidly fatal*, the patient lasting for hours or even days, there occurs, after the accident, deep, difficult and oppressed breathing, but not so violent as to threaten instant suffocation, or more commonly, these symptoms increase slowly as more and more of the hernial mass protrudes through the narrow opening into the chest. This form is usually seen only in animals of a specially quiet disposition, and which have not been subjected to active exertion or excitement after the accident and the hernia has increased by slow degrees only. The patient becomes listless, or very restless, paws, looks at his flanks, shifts from one hind foot to the other or even kicks at the abdomen, lies down carefully, rolls, sits on his haunches (though no more than in other forms of colic), and manifests the anxious, pinched, colic-countenance. The advance of the pain is constant but slow, and usually it is not characterized by that intensity which drives the animal to throw himself down recklessly and to roll and kick with violence. There is also usually an absence of the weak running down pulse of hemorrhagic congestion (thrombosis) and of the pallor of the surface mucosæ which usually attend on the extensive blood extravasations of that disease. An exception may be made in those cases in which the hernial mass is strangulated, as these may closely resemble spasmodic colic or hemorrhagic congestion.

The respiration furnishes more distinctive symptoms. The breathing which may be hurried and almost panting in colic and acute congestion, is changed in this lesion to a condition of extreme oppression, the nostrils remain widely dilated in expiration as well as inspiration, the angle of the mouth is retracted so as to show the teeth and gums, all the facial muscles stand out, the eyes are protruding and fixed, with dilated pupils, the head is held extended on the neck, and the ribs are not allowed to fall in freely, as after ordinary inspiration, but like the nostrils they remain permanently drawn out. The efforts at inspiration are violent though shallow and marked by lifting of the flanks. There are usually one or two nervous catches in each expiration and sometimes in inspiration as well. This is partly due to the impotence of the deeply lacerated diaphragm as an

organ of respiration, but also to the pressure of the displaced and overdistended abdominal organs on the lungs, and to the profound nervous shock. The whole work has been suddenly thrown on the costal muscles, and the depressed nervous system proves unequal to sustaining them in the unwonted toil.

Still clearer indications may be obtained from auscultation and percussion. These are gurgling, rumbling and clucking, or a coarse mucous râle which seem abnormally close to the ear, and a drumlike resonance, much greater than that of emphysematous lung and enormously in excess of what is given out by the sound lung tissue. These may be heard at points where only pulmonary murmurs naturally occur or where abdominal sounds, if heard at all, are distant in health. There may also be areas of abnormal flatness on percussion by reason of the protrusion of a solid viscus like the liver or spleen or one with solid contents. These symptoms are only clear when there is a large intrusion of abdominal organs into the thorax, and they increase rapidly until asphyxia supervenes.

In cases which do not immediately threaten life the extent of the phrenic laceration is usually small and the orifice may be blocked by a bulky organ like the rumen, double colon, stomach or liver, so that any protrusion takes place only to a limited amount and the function of the diaphragm can still be carried on to a reasonable extent. In these cases there may be no very marked symptom at the outset, though the animal is dull, listless and without appetite, or, if he eats or drinks, it is liable to be followed by slight colics and a double action of the flank in expiration as in pulmonary emphysema (heaves). Pressure or percussion in the posterior intercostal spaces is painful. Cough when roused by pinching the larynx is broken and abortive. These symptoms are not distinctive, however, and unless there is a protrusion of a loop of small intestine, to give gurgling and drumlike sounds the diagnosis of the case is liable to fail. The fact of a recent injury may however assist in the recognition of the lesion.

The *chronic cases* are even more difficult to recognize as there is no record of recent injury and no fever. There may be *short wind*, the animal breathing hurriedly on slight exertion, and showing a double lift of the flank in expiration (Girard). In place, however, of the tympanitic bowels and frequent passage of flatus which characterize heaves, there is a tendency to colic, especially after meals, and in a certain number of cases there are all the symptoms of fatal strangulation, due to the contraction of the diaphragmatic wound. When the hernia is made by a loop of intestine there are the characteristic symptoms of thoracic gurgling and drumlike resonance.

Cases are on record in which the intestine protrudes through one of the last intercostal spaces or between the ends of the broken rib as a hernia and diagnosis becomes easy by auscultation, palpation and percussion. But in a large proportion of cases the lesion escapes recognition and is only found on *post mortem* examination.

Lesions. The lacerated orifice in the diaphragm varies much as regards situation, extent, form and the nature of its border. In congenital cases due to an imperfect closure of the natural openings there may be simply a round or ovoid opening, too spacious to be filled by the gullet, vena cava or aorta as the case may be, and capable in the different cases, of containing an organ of any size from the omentum to the liver or stomach. Its margins may be perfectly smooth and even, without any thickening, irregularity, fringe, clot or exudate. In traumatic cases on the other hand the orifice may be of almost any form, size or situation. It may be round, elliptical, triangular, or irregular in many ways. It may be so small as to admit nothing more than a small fold of the omentum, or it may be large enough to open the two cavities, thoracic and abdominal into one common space, and to practically abolish the function of the diaphragm. If the lesion is a recent one the torn margin is irregularly indented or fimbriated and marked by small black blood clots, and somewhat later by exudate and irregular thickening or swelling. When due to a broken rib, the existence of the fracture is patent and the laceration extends along two lines often radiating from point of perforation by the rib. When the laceration has resulted from tympany of the stomach or intestines or from other overdistension of the abdominal organs, the general and comparatively equable pressure has determined the independent laceration of numerous tendinous or muscular bundles all over the diaphragm, so that the divided ends stand out at intervals each bearing its little clot of dark blood, but without actual perforation. The actual orifice in such cases is confined to one point where the

tension has been greater or the resistance less. *Post mortem* lacerations, from tympany or other cause, are easily distinguished from those occurring during life, in that the edges of the wound are pale and bloodless, without clot or exudate.

When the hernia is chronic there is an absence of exposed fringes, and of indications of inflammation, the margins of the orifice being in some cases smooth, even and fibrous, and in others irregularly notched or indented with nodular, fibroid swellings of various sizes at intervals. In such cases the orifice is always relatively small and the hernial mass inconsiderable.

As a rule the peritoneum and pleura, being firmly adherent to the diaphragm, are involved in the laceration so that the hernial mass is not retained in a special sac, but simply protrudes into the pleural cavity, after the manner of an eventration. In exceptional cases they become detached from the muscle, and becoming distended, envelope the hernial mass in a distinct sac.

In hernia with a very small orifice the omentum alone may pass through, even the small intestine proving too large for admission. In such cases it is usual to find the band of omentum adherent to the callus formed by the repair of a fractured rib. When the orifice is somewhat larger a portion of small intestine or of the double colon or cæcum may be engaged, while with a still larger opening the stomach, spleen, or liver may form the hernia. In cattle the reticulum is most likely to be the hernial mass, as noted in an article below. In rare cases the small intestines, omentum or liver protrude (Youatt, Lafosse). The protruding organ is liable to be constricted and strangulated sooner or later by increase in its bulk in connection with its vermicular movements, the accumulation of its contents or the extrication of gas in its lumen, or by the gradual contraction of the orifice in process of healing. Then there may be dark red venous congestion, blood extravasation and effusion, friability and even rupture of the intestinal walls, and adhesions to the wound or the lungs. Once started this congestion and extravasation may extend backward into the abdominal cavity involving a great part of the contiguous intestinal canal.

Prognosis. The worst cases are promptly fatal, while others destroy life in one or two days and such are always to be recognized by the extreme dyspnœa which appears soon after the accident. The slight cases with small orifice and little protrusion may merge into the chronic form, and the animal may even be fit for work, notwithstanding existing dyspnœa, which closely resembles that of chronic emphysema (heaves), but is not benefited by the same treatment. In the ruminants even considerable lacerations and protrusions may not be incompatible with fattening provided the animal is kept from all causes of excitement or over-exertion.

Treatment. Surgical treatment has not proved successful. Expectant and medicinal treatment are the only available resorts and then only in the slighter cases. Quiet and the absence of all excitement is the first consideration to allow of an arrest of any increase of the hernia and the establishing of a healing process in the torn margins. Bouley strongly advises bleeding to allay abdominal pain. Chloral hydrate (1 ounce for the larger animals) will often meet the same end, with the additional advantage that it counteracts fermentation and tympany. The unloading of stomach and bowels by a cathartic, and the use of flaxseed meal or other concentrated food of a laxative nature are indicated. Small animals may be lifted by their fore limbs, and the abdomen may be meanwhile manipulated to favor the return of the hernial mass by gravitation. Large animals should be placed in a stall having an inclination downward and backward for the same reason. When it can be ascertained that the hernia consists of a loop of small intestine only, it is permissible, especially in cattle, carnivora and omnivora, to make an incision in the flank and with the disinfected hand to attempt the reduction of the hernia and the placing of a bulky viscus, like the rumen, stomach or liver in the way of its return. In case of violent abdominal pain Bouley advises active counter-irritation over the abdomen, but as strangulation is usually present in such cases, this measure may be held to be inferior in value to gravitation, anodynes, antiseptics, unloading of the gastro-intestinal organs and absolute rest or surgical interference.

DIAPHRAGMATIC HERNIA OF THE RETICULUM.

Anatomical reasons. Gullet, weight of stomach, riding, dystokia. Symptoms. Tendency to chronicity. Treatment.

In ruminants this is the most common phrenic hernia of a hollow abdominal viscus. On the right side the gastric and intestinal organs are separated from the diaphragm by the flat mass of the liver. A laceration in this region must therefore be extensive to allow of the protrusion of any abdominal organ into the chest. The left half of the diaphragm, however, comes in direct contact with the reticulum and any opening large enough to admit of this viscus is likely to entail hernia of the second stomach. The lesion is further favored by the fact that the gullet passes through this part of the diaphragm and is connected with the stomachs in the furrow between the first and second stomachs. In case, therefore, that the gullet is violently dragged upon by the weight of the contents of the overloaded or tympanitic paunch, or when the animal rises on its hind limbs, in riding its fellow, the foramen sinistrum is liable to be enlarged by laceration, and the second stomach most naturally protrudes through the opening. The strain thrown on the diaphragm in violent abdominal contraction, as in difficult parturition, is mainly expended on this left half, and the laceration takes place around the œsophagus, or as in cases reported by Schurinck and Siedamgrotzky through the aponeurotic portion. The protrusion may be composed of the second stomach alone, or together with portion of the paunch as observed by Schmidt in a goat, or of the third and fourth stomachs as in a case in a bull reported by Baraillé. The edges of the orifice may show, in recent cases, the fringed or irregular jagged outline with blood clots and thickening, or in chronic cases the pale, fibrous, smooth, even outline already described under diaphragmatic hernia.

The *symptoms* are like those of other forms of phrenic hernia, in ratio with its extent. The tendency to survival, and chronicity is greater than in the monogastric animals, 1st because the entrance of the smaller viscera is barred by the great gastric masses applied against the wound, and 2nd by the quiet uneventful life of the ox and the absence of active work and violent excitement.

Treatment will not differ from that of other forms of phrenic hernia.

HERNIA THROUGH THE MESENTERY, OMENTUM OR OTHER FOLD OF PERITONEUM.

Definition. Causes: Concussions in horse especially, anatomical conditions in cattle, pediculated tumors, dystokia, hernia of small intestine in horse, ox, broad ligament, pelvic hernia. Lesions: Congestion, hemorrhage of hernial viscus, adhesions, softening, thickening, strangulation. Symptoms: Intestinal obstruction, rectal exploration. Treatment: Laparotomy.

Definition. This consists in a protrusion through an opening, congenital or acquired, in the double fold of peritoneum (mesentery) which passes off from the abdominal wall to support a viscus, or that which passes from organ to organ (omentum).

Causes. The lacerations of mesentery or omentum are attributed to sudden concussions of various kinds (falls, blows, leaps, violent efforts), and have been especially found in horses in which the bulk and weight of the contents of the digestive organs furnish a special predisposition. In ruminants in which the contents of the abdomen are equally bulky and heavy the lesion is rarely seen, probably because the great bulk of the ingesta lies in the first three stomachs, and because the large intestines are folded up in the mesentery which supports the small, thereby strengthening this means of support and restricting the freedom of movement on the part of the intestines themselves. In carnivora the limited bulk of the intestines and their contents, and the relative shortness of the mesenteric folds largely obviate the predisposition.

Pediculated tumors of mesentery or omentum may drag on the delicate membrane so as to cause laceration, and circumscribed peritonitis, by producing softening and friability, may act as a causative factor. Violent straining in defecation or parturition is another cause of laceration.

In solipeds the loosely suspended and eminently mobile small intestine is the viscus which most commonly forms a hernia through such adventitious openings, either through the great mesentery, the great omentum, the gastro-splenic omentum, the mesentery of the umbilical vein (falciform ligament of the liver), or the gastro-hepatic omentum. Cases are on record, however, in which the floating colon, the double colon and even the cæcum formed herniæ through the peritoneal lacerations.

In *cattle* the most common lesion is the hernia of a knuckle of intestine through a laceration in the mesentery, but, the rupture has also occurred in the great omentum and exceptionally in the broad ligament of the uterus which is very extensive in these animals. Pelvic hernia or gut tie as usually described is dependent on a laceration of the mesentery of the spermatic artery.

Lesions. The fold of intestine which makes the hernia is liable to become strangled, and sometimes twisted in the opening, so that the circulation of blood and ingesta is interrupted, congestions and hemorrhages set in, and necrosis and general infection follow. Oftentimes a fibrinous exudate is thrown out, binding together the intestinal convolutions, and attaching them to the margins of the mesenteric or omental opening. Similarly the lips of the lacerated wound in the mesentery become covered with blood clots, or congested, or infiltrated, and sometimes the seat of extensive extravasations. The inflamed membrane may soften, become friable and tear more extensively, or if the patient survives, the exudation becomes organized, thickening and strengthening the margins of the wound and causing them to contract so as to strangle the enclosed loop of intestine.

Symptoms. The indications are those of intestinal obstruction, to which accordingly the reader is referred. The only possible indication of the exact nature of the lesion is to be obtained by rectal exploration. Herniæ through the meso-colon or broad uterine ligament may be reached in this way, and possibly diagnosed.

Treatment. Laparotomy alone gives any hope of success, and this will only be warranted when a certain diagnosis has been reached.

HERNIA THROUGH THE FORAMEN OF WINSLOW IN THE HORSE.

Anatomical considerations, small size and elevated position of foramen, length and freedom of mesentery of jejunum; spare diet, draught, straining, rolling, colic. Symptoms: of intestinal obstruction only, lesion found at necropsy.

The foramen of Winslow is a comparatively small opening between the lesser curvature of the stomach and the liver, and between the gullet and its cardiac ligament on the left side and the gastro-hepatic omentum on the left. With its elevated and anterior position in the abdomen it would seem to be little exposed to this kind of accident, yet a number of recorded instances in the horse, show that it is certainly not immune. The great mobility of the jejunum, owing to the extra length of its mesentery is believed to be the essential predisposing cause. A spare diet, or one which is in small bulk, allows the comparatively empty gut to pass more readily through the small opening. Severe efforts in draught and straining in defecation and parturition are also invoked as means of pressing the jejunum through the orifice. So with the concussions attendant on falls and the unwonted positions taken in decubitus and rolling on the back in wantonness or colic.

Symptoms of this lesion are essentially those of intestinal obstruction, with usually a rapid and fatal course. An accurate *diagnosis* is impossible during life.

OTHER FORMS OF HERNIA.

The other forms of hernia (umbilical, inguinal, scrotal, femoral, ventral, vaginal, ischiatic) are essentially surgical and need not be further referred to here, than to guard the reader against overlooking them as factors in producing intestinal and omental strangulation and colic. They are all to be recognized by the presence of a local swelling, which may often be obliterated by returning its contents into the abdominal cavity, which sensibly enlarges when the animal is made to cough, and which, if made up of intestine, is subject to gaseous distension, and gurgles when manipulated and returned. A violent colic occurring in a male animal should never be considered as certainly diagnostic until the scrotal and inguinal region have been carefully examined for hernia.

PARALYSIS OF THE RECTUM.

In solipeds, ruminants, swine, carnivora. Injuries to loins and croup. Palsy of tail and sphincters. Nerve centres in end of cord. Fractures and dislocations of the pelvis. Hæmoglobinuria. Advanced gestation. Sclerosis. Ovariectomy by vagina. Impaction of rectum. Neoplasms. Thrombosis of internal iliac artery. Ptomaines and toxins in fevers. Symptoms: tardy defecation, impaction, over distension, fæces firm, dry, roller like, liquid oozing, excoriation, foul tail and thighs, bulging of anus, incontinence of urine. Paresis and wasting of quarter. Treatment: remove cause, for sprains and fractures sling, treat myelitis, debility, poisoning, unload rectum often, oily or soapy enemata, strychnia, eserine, ergot, barium chloride, derivations.

This is much more common in the horse than in ruminants, swine or carnivora, mainly because the soliped is more exposed to traumatic injuries of the loins, croup, and pelvic bones. It is noticeable that in the majority of cases the paralysis of the rectum is connected with palsy of the tail, anus, and sphincter vesicæ. This comes from the anatomical fact that the centres presiding over the motions of these different parts are situated close to each other in the terminal portion of the spinal cord, and any lesion of that part by traumatism or disease is likely to affect all of these parts alike.

The condition has been especially noticed in fractures or severe sprains of the loins causing pressure on the spinal cord. In some cases injury to the nerves supplied by this part of the cord, leads to an extension of inflammation to the nerve centres, thus paralysis of the rectum has followed on fracture of the ischium, dislocation of the sacro-iliac joint, or even of the first bone of the coccyx. Again, congestions and effusions on the terminal part of the cord, which occur in certain cases of hæmoglobinuria and in old hard worked horses is a cause of these local paralyses. Advanced gestation appears at times to produce the disease through pressure on the nerves, though it has also been noticed in non-breeding animals, and is doubtless traceable to sclerosis or other degenerations of the cord. It sometimes follows vaginal ovariectomy.

Sometimes the condition is traceable to local lesions as over distension of the rectum in horse or dog or in rectitis, but the result in such cases is usually partial, a paresis rather than a paralysis. The same may at times result from the growth of the neoplasms, and from the debility of old age. In other cases thrombosis of the aorta or internal iliac artery, implicates the hemorrhoidal vessels and paresis occurs as a result of the limited blood supply.

It may further result from the action of toxins and ptomaines on the spinal cord as when it supervenes in the course of debilitating fevers. This usually shows itself first as paresis of the sphincter ani, and later implicates the rectum as well.

Symptoms. In the slighter forms defecation is retarded, the fæces accumulate and overdistend the organ, adding to the paresis; they escape only under violent straining and apparently by the peristaltic contractions of the anterior portion of the rectum; the ejected matters are discharged usually in the form of a cylindroid mass; and they are dry, and firmly compressed. In some cases the irritation caused by the impaction leads to a free secretion, which escapes through the widely open anus and runs down the thighs, leading to excoriation of the skin.

In the more severe cases the accumulation is more complete, the expulsion still more difficult, and as the tail is often implicated, it lies flaccid between the thighs, and is smeared with the discharges. The peristalsis in front and the forcible compression by the abdominal muscles may be entirely inadequate to effect defecation so that the fæces have to be removed by the hand. The pressure on the bladder often leads to incontinence of urine, if the paralysis of the vesical sphincter has not already brought this about.

The trouble is not unfrequently associated with paresis and wasting of the muscles of the quarter.

Treatment. The first consideration is the removal of the cause. If fracture of the loins or pelvic bones, or severe sprain of the lumbar region, slings are usually requisite to obviate renewed injury in lying down and rising. Simple inflammation of the cord or its membranes may be met by laxatives, blisters and perhaps bromides. In the old and debilitated, tonics, rich food, open air, and sunshine, with locally, blisters or stimulating embrocations, will be demanded. In case of poisoning by ptomaines or toxins the disease which produces them must be attended to, and elimination favored as far as compatible with existing weakness. In all cases the rectum must be frequently

unloaded with the hand or soapy injections, and its walls may be stimulated by giving of nuxvomica, or by the hypodermic use of eserine, ergotin or barium chloride. Blisters or stimulating embrocations may be applied over the croup or between the thighs, or mustard or tobacco may be added to the injections.

NEOPLASMS. TUMORS OF THE INTESTINE IN SOLIPEDS.

Lipoma: sessile or polypoid, pediculated, strangulation, stenosis, size. Sarcoma: on bowel or peritoneum. Fibroma: flat or pediculated, obstruction, stenosis, degeneration, fatty, calcic. Myoma: hypertrophy of muscular coat, degeneration, stenosis. Myxoma: small translucent, oval, intestine mesentery. Carcinoma: extension from urocyt in horse, intestine, lymph glands, spleen, stomach, liver, mesentery, omentum. Epithelioma: colon, stomach. Cystoma: may contain sand or sclerostomata. Lymphadenoma: rectum, colon. Undetermined neoplasms. Causes: foreign bodies, microbes, constitutional, hereditary. Symptoms: tardy defecation, obstruction; in malignant, pallor, anæmia, emaciation; peritonitis, ascites; foetid irregular stools, bloody, sloughs, rectal exploration. Treatment: surgical removal where possible, recovery by sloughing, laparotomy, useless in multiple or malignant tumors.

Tumors of the intestine are very varied in kind (lipoma, sarcoma, fibroma, myoma, myxoma, carcinoma, epithelioma, cystoma), and are found on all portions of the canal.

Lipoma or *fatty tumor* is most frequent in connection with the small intestine or rectum, and on the mucosa it may assume a pediculated or polypoid form and may more or less perfectly block the intestine. When situated under the peritoneal surface it is usually sessile and flattened apart from the mesentery, but if growing from the latter or at its connection with the bowel it tends to become pediculated, sometimes hanging at the end of a very long cord which may roll around a loop of intestine and strangle it. Similarly the sessile masses, as they increase press inward so as to diminish the calibre of the bowel and finally close it. They are often found no larger than a coat button, while in other cases they grow to enormous size (25 lbs. Vogt, 42 lbs. Semmer). Semmer's case bound the cæcum and colon to the abdominal wall. The structure is essentially fatty tissue, though in some cases the fibrous stroma is more dense than in others.

Sarcoma. Small round cell tumors have been repeatedly found in connection with the intestine or mesentery. Baranski found one over 4 inches in length in the wall of the large intestine producing a serious constriction. Lucet found one of 18 lbs. weight and Mouquet one of 7 lbs. attached to the omentum. They are found to follow in certain cases the irritation and exudation of peritonitis.

Fibroma. Pediculated fibrous tumors have been found in the rectum of the horse and when large may threaten obstruction. They are usually of a loose fibrous texture, soft and elastic, and are often situated between the mucous and muscular coats. Quite frequently they are already in process of fatty or calcareous degeneration at particular points, the debris tending to fall into the intestine and be discharged with the fæces.

Myoma in the form of hypertrophy of the muscular coat of the bowel, the muscular fibres being increased in number and the wall further thickened by an intermixture of fibrous tissue, with areas of fatty degeneration. This may be confined to one side of the bowel and assume a spherical form, but it tends to contract the lumen until it is little more than half an inch in diameter. Mollereau records one case of myoma of the double colon which measured nearly a foot in diameter.

Myxoma. These are small, pale, translucent, round or oval neoplasms having a sparse network of fusiform cells and fibres, filled in the main with small round mucus corpuscles. They are not frequent in man and appear to be less so in the lower animals, but Friedberger and Mollereau have recorded two cases of myxoma in the intestines of the horse, and Chassereaud one case connected with the mesentery. In one case a mare passed such a tumor three inches long and nearly an inch in thickness. Chassereaud's case caused torsion of the floating colon.

Carcinoma. Cadeac says it is not rare to see cancer spread from the urinary bladder to all the abdominal organs in the horse. Conté describes a case in the duodenum, Marty one on the pelvic flexure of the colon, Latour on the ileo-cæcal valve, Mario on the floor of the rectum, and Casper in the lymphatic glands of the spleen, stomach, liver, mesentery and omentum, the last showing a mass of 28 lbs.

Epithelioma. When in a growing neoplasm, epithelial cells are arranged not only on the surface, but also in the form of cylinders extending into the substance of the tissues the growth is looked upon as epithelioma. Morot records a growth of this kind on the horse's colon, and which had grown to enormous dimensions. Röhl also mentions it as occurring on the gastric and intestinal mucous membrane.

Cystoma. As seen in the horse these have usually been determined by the presence of foreign bodies. Redieus describes a cyst of the small intestine which enclosed 30 lbs. of sand. Charlot speaks of a cyst placed between the stomach and sternal arch of the double colon, furnished with a smooth serous lining. Vernant and the author have found on the walls of the cæcum and double colon small cysts, each communicating with the interior of the intestine by a narrow opening. Beside mucopurulent matter, these have often in our experience contained the sclerostomata or their larvæ.

Lymphadenoma. Wuth describes a case of obstruction of the rectum by an adenoid tumor as large as the closed fist, which induced a fatal hemorrhage. It was connected with the terminal part of the floating colon. Jobelot records another case.

Tumors of uncertain kinds. A large number of tumors are described in veterinary literature, the true nature of which has not been made out. If these could be successfully differentiated they would add to the above list materially, and other forms not referred to above.

Causes. The causes of these neoplasms are not always traceable. In the case of some an occasion may be found in the presence of an irritant, like worms, sand, gravel, oat seed, etc., found in their interior, in others like the carcinomata we may accept the presence of the protozoön of that disease, and in still others there is an unknown cause, perhaps constitutional or hereditary which predisposes to the new growth. This last cause was probably operative in producing the fatty neoplasm in the rectum of Pritchard's fat Hereford heifer, seeing that her grandsire also died from a similar lesion.

Symptoms. These usually culminate in the classic symptoms of obstruction of the bowels, but there is sometimes a train of significant symptoms leading up to this climax. Thus in the malignant tumors in particular, though in some other forms as well, there is anæmia, pallor or yellowness of the mucosæ, weakness and emaciation. In other cases there are symptoms of peritonitis and ascites. In others there is obstinate constipation or diarrhœa, the excretions having a peculiar fœtid odor, often suggesting the decomposition of animal matter. They may be mixed with fresh blood, or sloughs from the surface of a tumor, or the whole tumor may be passed at once. Rectal exploration may be intercepted by a rigid stricture, by a tumor blocking the lumen, or a mass may be felt projecting in from one side. Aside from these the situation, form and size of the tumor can sometimes be felt through the flaccid walls of the rectum. In other cases the blocked and distended bowels may be felt, without the identification of a tumor as the cause.

Treatment. For tumors situated in the rectum, the removal by surgical measures (torsion, ecraseur, etc.) is indicated. If the tumor is simple a permanent cure may be hoped for; if malignant it is likely to recur. Tumors situated more anteriorly are usually desperate cases. Yet a certain number of pediculated tumors are detached spontaneously and discharged. In the absence of this, and when a tumor can be certainly diagnosed, there remains a resort to laparotomy, which in the horse is too often unsuccessful. If the tumor can be made out to be malignant and multiple it is useless to resort to removal.

TUMORS OF THE INTESTINE IN CATTLE.

Sarcoma: usually near lymph glands. Lipoma: mucosa, serosa, pediculated, strangulation, obstruction. Fibroma: floating colon. Carcinoma: generalized cancer, colon. Cystoma; reticulum, omasum, abomasum. Undetermined tumors. Symptoms: discovery by rectal examination, in case of debility or digestive disturbance. Treatment: removal through rectum or by laparotomy, spontaneous sloughing. Hyperplasia of intestinal mucosa.

Sarcoma. Multiple sarcomata have been found in nearly all parts of the abdominal cavity but above all in or near the lymph glands. They are from a small size up to masses of 60 lbs. (Revel).

Lipoma. Fatty tumors are found attached to the mucosa, and peritoneum, but above all as masses with long elastic pedicles which are liable to wind round and obstruct the intestines. Tannehauer records a case of fatal obstruction of the small intestine from a lipoma of the mucosa, and Pritchard two cases of fatal obstruction of the rectum by a fatty neoplasm occupying its walls.

Fibroma. Furianetto furnishes an interesting case of a fibrous tumor connected with the mucosa of the floating colon, on which it had dragged so as to cause a fatal invagination.

Carcinoma. Mauri relates a case of multiple cancer involving not only the intestines but also the lymph glands, the heart, the brain, the muscles, etc. Landis also records the case of a cancerous tumor of six inches in circumference attached to the floating colon of a calf.

Cystoma. Tumors of this kind have not been found to any extent in the intestines of cattle. Reboul describes a large cyst weighing 20 lbs., attached to the reticulum, omasum and abomasum and containing a pultaceous foetid debris. There was attendant congestion of the colon.

Tumors of Uncertain Kind have been recorded by other observers.

Symptoms. As in the soliped the one diagnostic symptom is the discovery of the neoplasm and its effects by rectal examination, in those cases in which the tumor is within reach. Apart from this there are the general symptoms of ill health, anæmia, pallor of the mucosa and emaciation in malignant cases, and recurrent colics, tympanies, impaired rumination and appetite, with dark colored liquid or bloody fæces, or complete suspension of defecation. In Pritchard's cases one animal was in the highest possible condition, having been in preparation for the Smithfield (London) fat stock show.

Treatment. If the tumor has been diagnosed with certainty, it may be removed by surgical means from its seat in the rectum, or even from other parts if not multiple or malignant. Laparotomy is better borne in cattle than in horses.

Hyperplasia on the Intestinal Mucosa in Cattle. In a Jersey heifer presented at the clinic of the New York State Veterinary College a hyperplasia of the mucosa of the duodenum, which blocked the lumen, was removed and the bowel resected. On examination the mass was found to be the result of productive inflammation, and undergoing necrosis and separation.

TUMORS OF THE INTESTINES IN DOGS.

Sarcoma: lymph glands. Epithelioma, intestine. Adenoma, rectum. Undetermined tumors. Diagnosis by abdominal palpation or rectal exploration. Removal by rectum or laparotomy. Chickens: Sarcoma. Epithelioma.

Cadeac records the frequent existence of sarcoma of the lymph glands, and epithelioma of the intestines and Friedberger an adenoma close to the rectum. Born describes a tumor of uncertain kind, and with a number of smaller adjacent rounded masses.

The *diagnosis* is usually easier than in the larger animals, the flaccid walls of the abdomen facilitating a satisfactory manipulation.

In the case of isolated and non-malignant growths, laparotomy should be resorted to without hesitation. Even resection of the intestine may be resorted to for the removal of a neoplasm the ends being reunited by sutures or by Murphy's button.

TUMORS OF THE INTESTINES IN BIRDS.

In chickens it is no uncommon thing to find sarcomata, and other tumors of less determinate structure in connection with the intestine. Pommay and Bizard record a case of cylindroid epithelioma in an ostrich, almost completely blocking the intestine.

The *treatment* in birds may be very heroic, the tendency to infection by pyogenic and other common bacteria of wounds being reduced to a minimum.

STRANGULATION OF THE INTESTINE BY THE OVARIAN LIGAMENT IN SOLIPEDS.

Pediculated ovary in mare. Strangulation. Diagnosis by rectal exploration. Castration.

In most healthy animals the ovary is light and its situation in the anterior border of the broad ligament so firm that it is impossible for it to enwrap and constrict the intestines. In the mare, however, the healthy ovary may be almost as large as the closed fist, and when further enlarged by cystic or other degeneration, it drags upon and lengthens the ligament until that may form a long pedicle which can easily be wound round the floating colon or small intestine.

Diagnosis of this trouble can often be satisfactorily made by rectal exploration, and *treatment* will consist in the removal of the offending body by castration through an incision made in the anterior part of the roof of the vagina close behind the os uteri.

PERITONITIS.

Acute: Chronic; general; local; idiopathic; traumatic; surgical; accidental; perforation; strangulation; cachexia; microbial almost always; aseptic foreign bodies escape into bowel: Castration; gastric or intestinal rupture or ulcer, enteritis, obstructions, cold storms, draughts, chills, all lower resisting power of tissue; generalization through peritoneal serum. Non-infective peritonitis from chemical irritants. Paves the way for microbes of ingesta. Rheumatic peritonitis, tuberculous, actinomycotic, microbes differ.

All inflammations of the peritoneum go under this general name. At the same time clinical and pathological distinctions have been made with the view of distinguishing more precisely different classes of cases. Thus it is described as *acute* and *chronic*, *general* and *local*, *idiopathic* and *traumatic*, *surgical*, *accidental*, or by *perforation*, by *strangulation* and by *cachexiæ*.

The advance of bacteriology has greatly simplified our views of the disease, as microbes are found to be at work in practically all cases. If we could exclude microbes from this membrane, peritonitis would be practically abolished, portions of aseptic powdered glass, sponge or gauze can be left in the abdominal cavity with comparative impunity, the tendency being, as shown in the dog, to coat themselves with a fibrinous exudate, and to make their way into the intestines through which they escape (Sternberg, Jalaguier and Manclaire). Hence it is that peritonitis is to a very large extent the result of a traumatism (castration, penetrating wound of the abdomen, contused wound of the abdomen), or of a rupture or ulceration of the stomach or intestine, through which the microbes make their way into the peritoneal cavity. In enteritis, congestions, strangulations, intussusceptions and obstructions of the bowels, the cause is the same, the microbes making their way with greater ease through the coats of the bowels in which the circulation and nutrition are impaired and the power of resistance diminished. Finally the occurrence of the disease as a consequence of exposure to cold or wet, of exposure in a cold rain or snow storm, of standing in a draught when perspiring, or plunging into cold water when heated and fatigued, or drinking ice cold water when in a similar condition, may in most cases be explained on the same grounds. The germs in this case had already gained access to the blood, but were helpless to accomplish much harm, until by chilling, the resisting power of the system was lowered and an occasion furnished for their successfully colonizing the peritoneum. Parallel cases are found in the frog which is immune from anthrax until it is heated, and in the chicken which is immune from anthrax until chilled. Reduce the vitality of the system and the germ which was already present, and up till now harmless, takes occasion to colonize more or less destructively.

This view also furnishes an explanation of the tendency of local peritonitis to become generalized. In the scanty liquid which bedews the surface of the abdominal organs, the microbes grow, multiply and spread; by the constant peristaltic movements of the bowels and their rolling upon each other this extension is largely favored; and the tendency to generalization will be in ratio with the potency of the invading germ, and the general or local weakening of the invaded tissues. With a limited infection wound in an otherwise healthy peritoneum and system, and invasion by a pus coccus only, the infection may not succeed in extending from its primary centre, but with a debilitated system, an extended enteritis, or when the invasion is made by septic germs it is likely to become speedily and fatally generalized.

Pernice has shown, however, that peritonitis may occur independently of infection. The injection into the cavity of concentrated mineral acids, acetic acid, phenol, nitrate of silver and other powerful antiseptics determine inflammation by their purely irritant action. By weakening the tissues of the bowels these in their turn pave the way for the escape of the microbes from the contents, and to the occurrence of a secondary infective inflammation.

Cases occur as a manifestation of rheumatism, tuberculosis, actinomycosis and other affections which will be treated at greater length under these respective heads.

The microbes would seem to vary greatly. Soula attributes infection of castration wounds mainly to the bacillus of malignant œdema which is 3 to 3.5 μ long and 1 to 1.1 μ broad often bearing a refragent spore at one end (is sporeless in the peritoneal cultures), and growing out into chains in artificial cultures. They are anærobic, liquefying, motile, easily stained by aniline colors, but bleached by iodine.

In other forms of peritonitis the bacillus coli commune is found and probably comes from the intestinal contents where it is present in all our domestic animals.

In man Fränkel found the bacillus coli communis 9 times, streptococcus pyogenes 7 times, bacillus lactis aerogenes 2 times, micrococcus pneumoniae crouposa 1 time, staphylococcus pyogenes aureus 1 time. Flexner found the proteus vulgaris.

ACUTE PERITONITIS IN SOLIPEDS.

Susceptibility to pyogenic bacteria, infection simple or complex. Traumatic injuries: accidental, omphalitis, operations, strangulation, wounds in rectum or vagina, coition, hernia, castration of mare, or horse, infected from 2d to 6th day, later granulation protects, ruptured stomach, perforating ulcer, perforation of parturient womb, ruptured abscess, microbes in circulation, debility. Symptoms: trauma with spreading swelling, œdema, tenderness, stiffness, arched back, tucked up abdomen, fever, prostration, colics, careful decubitus and rising, tense tender belly, ridge along flank, breathing short, inspirations catching, straddles, steps short, costive or later diarrhœa, enuresis, abdomen fluctuates, death in 1 to 8 days. With ruptured stomach or intestine prostration extreme, collapse, vomiting. Resolution. Ascites. Diagnosis: trauma, gastric or intestinal lesion, followed by specific symptoms. Lesions: trauma, rupture and escape of ingesta, congestion, ecchymoses, false membranes, adhesions, liquid effusion, bloody, pink, or straw colored, albuminous, fibrinous, granules, cells, salts, bacteria, pus, fœtor, bowels tympanitic, later fibrous bands, strangulations, degenerations. Prevention: fatal in solipeds, avoid abdominal congestions, inflammations, traumas, infections, accumulation of serum, blood, etc., also debility, ill health, chill. Treatment: old methods, by anodynes and checking peristalsis. Modern method: antisepsis, iodoform, carbolic acid, mercuric chloride, irrigation with boiled water, drainage; internally, saline laxatives, eliminates from bowels, blood, peritoneum, favoring phagocytosis, and innervation, antiseptics, sodium salicylate, chloral hydrate, morphia, enemata, hot fomentations, or ice, in suppuration drainage and washing with normal salt solution at body temperature, derivatives, laparotomy, puncture in tympany.

Causes. Solipeds are especially subject to peritonitis in its acute and dangerous forms largely because this class of animals is preëminently obnoxious to the attacks of pyogenic bacteria. The disease may however be dependent on a great variety of different organisms, and these may cause different forms through invasion by one specific microbe or by a complex invasion. It is convenient to note the different channels of invasion.

a. Traumatic injuries. Wounds are not uncommon from pricks with forks, pickets, broken rails, prongs of stump fences, poles or shafts of wagons, nails, barbed wire, horns of cattle, tusks of boar, and other sharp or pointed objects, which carry infecting germs, or in any case make an entrance for those found in the dust of the stable, on the horses skin, comb, brush, rubber or clothing. Inflammation of the umbilicus and resulting abscess may prove an entrance way for the germs either by rupture into the peritoneum or by causing adhesions between two loops of intestines from which the microbes escape through the weakened tissues. Wounds made in operations on hernias may have a similar ending and as Dieckerhoff has pointed out the onset of the peritonitis may be delayed for one or two weeks while the abscess is maturing or the walls of the bowels are being traversed by the microbes. Strangulated hernias and those in which the intestine is congested are especially subject to such peritonitis, as the germs may enter by the external wound and through the intestinal wall as well. The author has seen artificial anus formed through inclusion in the clamps of an adherent loop of small intestine, and at such a point peritonitis is liable to start.

Wounds of the rectum or vagina are sometimes the starting point of the inflammation. The penis of a stallion entering and lacerating the rectum of a mare, or the large penis of an ardent male rupturing the roof of the vagina are occasional causes. The latter may occur without fatal consequences, yet the author has seen a generalized and rapidly fatal attack follow such an injury when the mare had at once thereafter made a journey of nine miles in a cold rainstorm. The horse was a Percheron with very large penis and the mare would weigh about 900 lbs. The castration of mares, even through the vagina may be followed by peritonitis from sepsis of the instruments, hands or arms.

The castration of the horse is more liable to be followed by this infection. Too often no attempt at asepsis is made, implicit trust being placed in the defensive power of the tissues. In other cases even a very careful local antisepsis fails, the germ being already present in the circulation and the extensive wound and resulting local congestion and debility are seized upon as an opportunity for colonization and growth. This infection usually takes place from the second to the sixth day while

the inguinal canal and vaginal sheath are still open to the cavity of the abdomen. Later when these have closed by adhesion, and when protective granulation has formed the implication of the peritoneum is rare.

b. Rupture of the Stomach or Intestine. This comes as already shown from gaseous distension, overloading, sudden shock or concussion, obstruction by dried ingesta, calculi, foreign bodies, parasites, etc., and by abuse of too powerful purgatives in cases of obstruction. The resulting infection is very abundant and varied, and the microbes accustomed to an anærobic existence in the intestines, multiply with extreme rapidity in the peritoneum and prove rapidly fatal. Beside the bacillus coli commune, there are usually staphylococcus and streptococcus pyogenes and not unfrequently the bacillus of malignant œdema.

c. Perforating ulcer. Though having a separate point of origin the effect of this is precisely the same as in rupture, the same bacteria escaping and the nature of the infection being identical. Inasmuch, however, as the perforation is usually small at first and the escape of contents very limited the symptoms advance more slowly and reach their acme later.

d. Perforation of the Parturient Womb. This usually depends on a case of dystokia in which the organ is torn by a foot of the foetus or by some ill-directed instrument. The infection has usually been carried in on the hands or instruments, or introduced as dust by an aspiratory movement in the intervals of labor pains. The healthy womb is usually sterile as regards microbes, yet Lignieres claims that he has found staphylococcus pyogenes albus and aureus and in contagious abortion the specific bacillus of this affection can always be found. In woman peritonitis following rupture of the womb usually shows streptococcus pyogenes.

e. Rupture of Abscess into the Peritoneum. As an abscess is nearly always the product of pus microbes it follows that its rupture into the abdominal cavity will determine infection. If the abscess contains some special infective germ like that of strangles or glanders the resulting inflammation partakes of their nature.

f. Penetration of Microbes through the Circulation. Healthy blood is free from germs, yet it is not uncommon to find a few circulating in the blood in given conditions. Debility, toxin and ptomaine poisoning and other conditions render it possible for bacteria to successfully invade the circulating blood, hence, many infective diseases are at first local, and later on become generalized. Under these circumstances any cause of debility operating especially on the peritoneum opens the door to their infection. Under such debilitating causes all those already referred to as chills must be recognized, together with kicks, blows, local congestions and other injuries.

Symptoms. The existence of a penetrating wound or sore of the abdomen, a kick, an open abscess, or a recent exposure to severe cold when heated and fatigued, or finally some serious affection of the abdominal organs will give definiteness to some of the symptoms which follow. There may have been noticed a rigor, or trembling of the muscles may still continue. There is swelling around the external wound, which in case of castration is usually œdematous and more or less tense, affecting the entire sheath and extending forward on the abdomen. In any such case there is tenderness to pressure around the margin of the wound, for a distance that constantly increases. The animal moves stiffly and the back is more or less arched. The temperature is raised two or three degrees and may go on till it has reached 107. The patient becomes dull and listless, with drooping head and ears, sunken, lustreless, pale eye, more or less fixed, lips drawn up firmly and muscles of the face contracted and prominent. He stands with back arched, loins insensible to pinching, and legs drawn somewhat toward each other under the belly. There are indications of colic, pawing, looking toward the flanks, and shifting of the hind feet without the violent kicking motion of spasmodic colic or intestinal congestion. When he lies down it is comparatively slowly and carefully and he is more inclined to lie on the side with hind legs, or at least the one on the affected side extended backward. The rolling on the back and the sudden jerking movement of the hind limbs, seen in spasmodic colic are rarely noticed. There are exceptions to this rule when violent spasms or acute congestion is present as well as in some cases following castration and with strangulated cord.

The abdominal walls are always tense from muscular contraction, and often also from tympany, in which case there is marked drumlike resonance, on percussion. An elevated ridge like that seen

in pleurisy extends from the outer angle of the ilium to the lower end of the last rib. The breathing is hurried and carried on mainly by the ribs, the diaphragm being kept as fixed as possible. The inspirations are short and catching as in pleurisy, the expirations a little more prolonged. In standing the hind legs are held apart, and in moving the animal straddles and moves them stiffly avoiding advancing them far forward. Constipation is the rule the rectum containing a number of small, round, dry balls, yet after a day or two diarrhoea may set in. Urine is usually suppressed, or passed in small amount and of a high color. The pulse is usually small, hard, and at times thready, the skin perspires more or less generally, prostration and dullness set in and death may occur within 24 hours or more commonly in 4 to 8 days. After the 1st day there may be fluctuation of the abdomen from liquid effusion.

In case of infection from perforated or ruptured stomach or intestine the symptoms are more severe from the first, and the issue is more rapidly fatal. With marked trembling, there occur extreme weakness and prostration, dull, sunken eyes, flaccid facial muscles, cold perspiration, chilly ears and limbs, entire cessation of defecation, small, weak accelerated pulse, breathing rapid, broken in inspiration or expiration, and more or less tympany. Yet the tenderness of the abdomen is less marked, and the animal may move with somewhat less stiffness, and gets up and down with less apparent suffering. The temperature is less elevated than in the external traumatism, and the whole aspect is that of collapse and sinking. These cases may die from shock or tympany in a few hours, or they may survive 24 or even as long as 48 hours but rarely longer. In case of rupture of the stomach there may be the usual feature of eructation or vomiting. Resolution may occur but non-fatal cases are liable to become chronic with ascites.

Diagnosis. Apart from traumatism, the evidence of some previous intestinal or gastric lesion, or abscess, succeeded by continuous dull colicky pains, the arching of the back and drawing together of the limbs, the tender abdomen, the careful decubitus and lying on the side, the tympany, obstinate constipation, and pale conjunctiva, the pleuritic ridge and breathing without the friction sounds or intercostal tenderness of pleurisy, the high temperature, the weak rapid pulse and rapidly advancing weakness, prostration and collapse furnish a combination which is very characteristic.

Lesions. In rapidly fatal cases there may appear to be little more than general peritoneal congestion and ecchymosis. In such cases, however, there is usually a mixture of the ingesta with the intestinal convolutions and omentum.

In cases that have survived twelve hours, false membranes are found, in the form of fine filamentous shreds on the surface of the congested serosa, which has become dull, opaque, and thickened. In twenty-four to thirty hours these have increased in thickness and solidity, binding together the convolutions of the intestines or floating free as shreds or membranous layers in the exuded liquid. At first yellowish white, these become gray, red, and finally white as they become organized into fibrous tissue. They may cover any of the abdominal organs and bind these together more or less firmly.

The liquid effusion collecting at the lower part of the abdomen, may be blood red, serosanguinolent, or straw colored, and contains a considerable amount of albumen, fibrine, granules and cells as well as the bacteria. It may attain to as much as 25 or 30 quarts. When purulent or septic the liquid is comparatively limited in amount and is usually connected with a ruptured abscess or external wound or intestinal perforation. The presence of alimentary matters, the foetid odor, and gaseous emanations are marked features in this last condition.

The intestines are usually distended with gas, and have thin walls infiltrated, pale and thickened, and often bound to other convolutions or to adjacent organs by false membranes. The liver and spleen are pallid, and their capsules swollen, thick and opaque, with more or less membranous exudate.

In case the patient survives, the effusion and neoplasm are slowly absorbed, but the false membranes only imperfectly, and they may be found later as organized bands attaching the intestines or other organs to adjacent parts, and limiting their motions or constricting and strangling them. Hence there is left a predisposition to relapse or to other disease of the abdomen. Röhl has noticed degeneration and softening of the false membranes, which extended to the wall of the bowel beneath and led to perforation.

Prevention. In solipeds especially this affection is so fatal that every precaution should be adopted to prevent its occurrence. In this class of animal the tendency to suppuration in wounds and inflammations of all kinds greatly exceeds what we see in other animals. A wound that in man will heal kindly by first intention will almost certainly suppurate in the horse, and an abdominal wound which in man, ruminant, or pachyderm might be viewed with confidence, must be treated as a very serious matter in the horse. But though thus differing in degree, all abdominal wounds must be considered as serious lesions. The peritoneal sac is, like other serous sacs, a dependency of the great lymphatic system of vessels, and the liquid present in it in health is, like the lymph, the most favorable culture medium of the body for microbian life, the greater the amount of such peritoneal fluid (as in inflammatory or other exudate) the more favorable it becomes to its growth and diffusion, and finally the enclosed intestine is teeming with micro-organisms, which, though held in check by the healthy mucosa, are ready when any congestion, inflammation or other morbid process gives occasion, to traverse these thin walls and start their deadly career in the peritoneum.

In every animal, therefore, but in solipeds above all, every precaution should be taken against the infliction of accidental wounds of the abdominal walls, and to remedy any serious derangement of the digestive organs. Above all, operations that involve the peritoneal cavity should be made only under careful surgical precautions. The introduction of pyogenic, septic or potentially septic material from hands, head, beard, floating dust, unboiled water, or surgical appliance of any kind, is a direct bid for a fatal peritonitis. Next to this the greatest care must be exercised to prevent unnecessary injury to the peritoneum or any abdominal organ, which would in any way impair its vital properties and resisting power. Again, to leave blood or exudate of any kind in the wounded peritoneum is a direct bid for the propagation of micro-organisms. These should be removed by means of aseptic agents. Finally, in case of enteric disease and abdominal wounds the patient should be guarded against chill, which would lower the vital and resisting powers and lay the system open to microbian invasion.

Treatment. The therapeutics of peritonitis furnishes a striking example of the transforming influence of bacteriological discovery. Systematic medical and veterinary works enjoin the time-honored method of treatment by opium to check intestinal peristalsis and the painful friction of inflamed surfaces on each other, and to keep the organs quiescent until nature shall have time to subdue the inflammation. The still older treatment by calomel and opium has essentially the same foundation. To the bacteriologist the latter has the recommendation of being to some extent antiseptic and of tending to secure depletion from the intestinal mucosa. Another cardinal principle of the old practitioner was to hail the liquid exudate as tending to separate the inflamed and painful surfaces, and as allowing them to move past each other without aggravating the suffering and inflammation. In short, the practitioner of the past had an especial dread of mechanical injury, and treated all other measures as secondary to this though by no means unessential.

Bacteriological considerations direct attention rather to the vital properties of the causative bacteria and seek to check the disease by checking this its most effective cause.

In simple local peritonitis, as in the infection following castration, the washing out of the infected wound with boiled water and application of an antiseptic (iodoform, iodoform or carbolic acid guaze), and the free use of carbolic acid solution (1:50) to the skin is of great value. If the sheath or inguinal region is swollen to any extent, puncturing it at intervals with a lancet to the depth of half an inch so as to drain it speedily and thus reduce the swelling and culture fluid, and to restore the vitality of the parts, and the frequent bathing with the carbolic acid lotion, will usually succeed in bringing about a healthy action.

The question of medical treatment comes forward mainly in cases that have invaded the abdominal peritoneum, and which are not already completely generalized, nor the result of extensive escape of gastric nor intestinal contents. In such forms and above all in the early stages of surgical cases sulphate of soda given to the extent of causing free purgation has been found to be incomparably more effective than the opium treatment. The explanation of its action may rest in part (1) on the expulsion from the bowel of a large proportion of the dangerous microbes which are simply waiting for that opportunity to pass into the peritoneum, which will be furnished by the

inflammation of the intestinal walls; (2). On the elimination from the blood and system of much of the deleterious ptomaines and toxins which have already been absorbed from the inflamed surface and the presence of which robs the tissues of their vitality and resisting power; (3). On the active depletion from the intestinal mucosa and (through the common capillary plexus) from the congested peritoneal coat, counteracting alike the effusion into the peritoneum which forms the culture fluid for the invading bacteria, and the infiltration of the serous and subserous tissues which beside tearing apart the tissue-elements, and robbing their leucocytes of their power of phagocytosis, furnishes within the invaded tissue itself the most favorable of culture media; and (4) on preserving a better tone of the nervous system and, locally, of the tissues the cells of which, can struggle more successfully against the small body of invading bacteria advancing slowly along the surface of the peritoneum, than with the countless myriads produced in and washed everywhere by the abundant liquid exudate.

Along with the soda sulphate may be given antiseptics, like sodium salicylate, or chloral hydrate. The latter serves to mitigate the pain without checking the secretion or peristalsis.

When the suffering is very acute, opium may still be resorted to, but preferably subcutem, in the form of morphia sulphate so as to lock up the poisons as little as possible.

Enemata are in order to facilitate the operation of the bowels, and may be made laxative and antiseptic. The danger of tympanitis speaks forcibly for a judicious use of antiferments, both by the mouth and anus.

Hot fomentations have long been in use but require persistent application and this is often difficult to secure. Recently cold applications to the abdomen in the form of ice or snow, or in the absence of these of cold water applied on a light rug, kept against the abdominal walls by elastic circingles, have been found of great service. This can be persistently applied, as all that is requisite is to keep the rug constantly wet.

When pus forms in the peritoneum or when extensive effusion has taken place, it should certainly be evacuated, as it is but a centre for the development of the deadly bacteria. It can be drawn off through the already existing traumatic orifice, or, if necessary, a new opening can be made by cannula and trochar, or by direct incision under suitable antiseptic precautions. The opening having been made, and the liquid having escaped, the peritoneum may be profitably washed out with a normal salt solution which has been recently boiled and which is used at near the body temperature (80° to 90°).

Blisters are sometimes of use in the advanced stages of the disease, in stimulating resolution and reabsorption, but hot or cold applications are preferable in the early acute stages.

Laparotomy in cases due to rupture of stomach or bowel, or of extensive perforation, has not been attempted in solipeds, and it could hardly be expected to succeed, yet in such cases, which are otherwise inevitably fatal, any measure giving even a remote hope of success is allowable.

When tympany sets in it may be met by using a fine cannula and trochar, and as soon as the gas has escaped, antiseptics like chloral hydrate, salicylate of soda, salicylic acid, or glycerine, can be injected into the fermenting mass by attaching a caouchouc tube and funnel to the cannula.

CHRONIC PERITONITIS IN SOLIPEDS.

Secondary: after acute, or with disease of liver, spleen, kidney, rheumatism, melanosis, lymphadenoma, epithelioma, carcinoma, or sarcoma. Gastric ulcer, infected punctures. Symptoms: poor health, tender abdomen, irregular bowels, slight colic, tense, fluctuating belly, pallid mucosæ, dropsy of sheath, limbs, etc., slight fever. Treatment: remove primary disease: saline laxatives, diuretics, drainage, antiseptic irrigation (boric acid, etc.), abdominal bandage, tonics, derivatives.

Causes. Chronic peritonitis is always a secondary disease, succeeding the acute, or dependent on some other affection of the abdominal organs, as chronic congestion of the liver, or spleen, Bright's disease, rheumatism, melanosis, lymphadenoma, epithelioma, carcinoma, or sarcoma. Tuberculosis and actinomycosis, so common in cattle, are rare in solipeds. Chronic ulcers of stomach or bowels and injuries and infections from punctures are exceptional causes.

Symptoms. The manifestations of the disease are indefinite, the acute form may have subsided so that the patient is supposed to have completely recovered, a moderate appetite and a certain capacity for work may be present, yet he is easily fatigued, there is some tenderness of the abdomen to pressure, some irregularity of the bowels—constipation and diarrhœa alternating—and occasional slight colics. Some weeks later may be noticed abdominal tension and tympany, with perhaps fluctuation in the lower parts, increasing pallor of the mucosæ, and œdematous swelling of the sheath, mammæ, abdominal walls or hind limbs. To detect fluctuation it is sometimes necessary to introduce one hand into the rectum. When present hyperthermia is slight, but assists in diagnosis from ascites.

Treatment should be directed to the primary disease. As that is too often irremediable the peritonitis will resist all other treatment.

In cases that supervene on the acute form, paracentesis, saline laxatives and diuretics with antiseptics, tonics, and counter-irritants will sometimes succeed. As in the acute form of the disease the removal of the effusion is the removal of the culture fluid, and may be followed by irrigation of the peritoneum with a normal salt solution, or with an antiseptic solution (boric acid or potassium permanganate), 1:20 warm water; Aluminum acetate, 1:2000. Sulphate of soda given to keep up a moderately laxative action, tends to counteract the contraction of the intestine by false membranes, and operates with diuretics in reducing the tendency to exudation or in causing its reabsorption. After removal of the liquid, support by a close (or elastic) abdominal bandage is often of value in preventing further effusion. As tonics, gentian, nux vomica and the iron salts may be profitably employed, and as antiseptics salicylate of soda and iodide of potassium. As counter-irritants mustard and cantharides may be named.

PERITONITIS IN RUMINANTS.

Causes: infection, chill, blows, wounds, debility, ill health, Chauveau's experiment with castration, dystokia, abdominal congestion and inflammation, bile or urine in absence of sepsis, gastric or intestinal ulcer or perforation, foreign bodies, abscesses, surgical wounds. Spoilt marc of beet sugar factories. Symptoms: fever, stiffness, dragging hind limbs, knuckling, arched back, shifting feet, moving tail, tense belly, pendent, fluctuating below, friction sounds, diarrhoea, later constipation, weakness, emaciation, death fourth to twentieth day. Recovery, often partial. After dystokia putrid vaginal discharge, and nervous depression, resembling parturition fever. Infection in ewes through shepherd's hands, oedematous swellings of vulva, perineum and abdomen. Lesions: as in solipeds, with abundant false membranes, foetid pus metritis, with putrid contents of womb. Treatment: saline laxatives, diuretics, demulcents, enemata, morphia, antiseptics, cold to abdomen. After dystokia, antiseptic irrigation of womb, elevation of head, with ice, strychnia, acetanilid. Tubercular peritonitis.

Causes. As in solipeds infection of the peritoneum and the increase of susceptibility by exposure to cold, blows, wounds, poor feeding or stabling, disease and other causes of ill health, operate together in inducing peritonitis.

The effect of debility or predisposition of the tissues is well shown in Chauveau's experiments with *bistournage* in rams. Healthy rams subjected to *bistournage* showed no infection, and rams subjected to intravenous injection of pus microbes without *bistournage* showed no infection, whereas if the rams were first subjected to injection of pus microbes, and then to *bistournage*, peritonitis set in. In the same way chills occurring after dystokia, when the womb is charged with microbes and more or less congested, may determine peritonitis, and congestions, impactions, tympanies, and other injuries of the gastro-intestinal viscera coöperate with cold to the same end. Rupture of the gall or urinary bladder does not usually cause prompt peritonitis, yet it irritates the serosa and lays it open to infection if the germs should reach it through the circulation. Otherwise the animal suffers only from uræmia or biliary poisoning and may survive one or two weeks. Ruptures of stomach or intestine, or ulceration or the perforation of their walls by hard, pointed or other metallic bodies are causes of peritonitis. The rupture of abscesses into the peritoneum and the escape of germs from the womb in case of rupture of the womb in difficult parturition, or in metritis, are additional causes. Surgical wounds as in castration of the male or female, and punctures and incisions of the rumen are occasional causes, but there is by no means the tendency to extension of such peritonitis that we see in solipeds. The self-protective power of the tissues is incomparably greater in the ruminant.

Nocard, Butel and others have recorded a gastro-entero-peritonitis of septic nature, occurring in cattle and above all in sheep, fed on the fermented refuse of beet sugar factories, which had been kept in silos through the winter. On the third and fourth days of this feeding many were attacked.

Symptoms. Beside the general systemic disorder and a very variable amount of hyperthermia (102° to 107°), there are the special indications of abdominal inflammation, stiff movement and dragging of the hind limbs, or if standing the back is arched, the head drooping and the legs drawn together and slightly bent, with uneasy shifting of the hind feet, and lateral movements of the tail. The walls of the abdomen are usually tense, often bulging laterally below, though fallen in beneath the lumbar transverse processes (pot-bellied); they are tender to pressure, and may be drum like to percussion above, while flat, dull, and fluctuating below. The tenderness is slightest below, where liquid effusion has settled, but is quite marked in the upper and resonant parts, where pressure will cause wincing and trembling. In this upper part on the left side may be heard friction sounds after the fourth or sixth day. This is especially observable in tuberculous peritonitis over the parts covered by tubercular growths. There may be at first diarrhoea which usually soon gives place to constipation, and weakness and emaciation advances rapidly, and death may take place from the fourth to eighth day, or may be deferred for some weeks.

In favorable cases the acute symptoms subside, the liquid effusion is absorbed, appetite and rumination are in great part restored, and a partial recovery is made. It is, however, very liable to

merge into the chronic form, and inevitably so in tuberculous cases.

In cases following on difficult parturition there are redness of the vaginal mucosa, with mucopurulent or putrid discharges, and swelling of the lower part of the abdomen, with liquid effusion and fluctuation, and tenderness of the right flank. The further symptoms are largely nervous, approximating somewhat to those of parturition fever. Temperature may rise to 104° or 106° , blindness, stupor, incoördination of muscular movement, staggering gait, if down she may lie on the side or sternum unable to rise, has frequent afterpains, tympany, sour eructations, and grinding of the teeth. The case may culminate in loss of vision, in stupor and coma, or improvement may set in and go on to a rapid recovery. This is a more common affection in ewes than in cows and is very destructive, the infection being carried by the hands of the shepherd. The most fatal cases are those in which the infection becomes generalized, and œdematous swellings appear round the vulva, between the thighs and beneath the abdomen.

In traumatic cases the external wounds can usually be found with active inflammation and surrounding tumefaction.

Lesions. The peritoneum, as in solipeds, shows the symptoms of congestion, exudation of a fine fibrinous network or shreds, of thicker and more extended false membranes in patches, of effusions more or less sanguineous, of formation of pus, usually fœtid, or the presence of decomposing ingesta which has escaped through a lesion of stomach or bowels. The peritoneal and subserous tissue are infiltrated with liquid, and the other gastric and intestinal organs are more or less tympanitic, and the mucosa of the latter is thickened, ecchymosed, or eroded, with black, fœtid bloodstained contents. In parturient cases, the uterine mucosa is congested, reddened and softened, the cotyledons swollen, perhaps gangrenous, and the membranes, if still present, float in a dark, putrid offensive liquid.

Treatment. As in the horse, morphia has been used to relieve pain and check peristalsis. The addition of saline laxatives, and diuretics, will assist in elimination and depletion, and in the removal of intestinal bacteria which become a source of danger. A laxative dose should be followed by frequent drinks of pure water or mucilaginous liquids, and sulphate of soda may also be given freely in enema. As diuretics, saltpeter or digitalis may be resorted to. Antiferments (salicylate of soda, bisulphite of soda) should not be forgotten nor cold applications to the abdomen. When effusion or suppuration has taken place evacuation by puncture may be followed by antiseptic irrigation.

If with septic metritis, antiseptic injections of the vagina and womb are the first consideration. With boiled water at a tepid heat the womb should be thoroughly washed out, followed by a solution of mercuric chloride (1:2000), or permanganate of potash (1:1000), or boric acid (1:25) until the liquid returns clear and odorless. This may be repeated several times a day. The symptoms of brain congestion, may be met by tying, or packing up the patient with straw so that the head will be somewhat elevated, and bags of ice or snow, or simple cold water may be kept applied to the upper part of the head and neck. When there is no great nervous excitement the nervous functions may be roused by nux vomica in enema, or strychnia subcutem. If on the other hand the temperature runs very high acetanilid may be tried with caution, or resort may be had to wet compresses.

In case of perforation or rupture, if the animal cannot be at once sacrificed for beef or mutton before inflammation has set in, the only hope lies in laparotomy, followed by the cleansing, disinfection and suturing of the wound.

In tubercular peritonitis which constitutes a very large proportion of bovine cases, treatment is undesirable, and the animal is unfit for consumption.

PERITONITIS IN CARNIVORA.

Causes: gastro-intestinal inflammations, metritis, trauma to walls of abdomen, pyæmia, septicæmia, tuberculosis, cancer, tumors, parasites. Symptoms: dullness, hiding away, movements tardy, painful, arched back, retracted abdomen, tense and tender, drags hind limbs, vomits, yawns, bloats, hopeless look, snappish, death in 2 to 8 days. Treatment: anodynes, saline laxatives by mouth and rectum, damp compress, warm bath, antiseptics, diuretics, in effusion, puncture, antiseptic irrigation, laparotomy.

Causes. All inflammatory and other serious affections of the stomach and bowels may be associated with peritonitis. Metritis and injuries to the womb, and all injuries to the walls of the abdomen (kicks, blows, penetrating and castration wounds), may have a similar complication. Pyæmia and septicæmia may also have localization in the peritoneum. It must be borne in mind, however, that purulent and septic infection are less likely to occur in the dog than in cattle, the leucocytes of the dog having much more resisting power. On the other hand the dog, and, still more so, the cat has a fair measure of susceptibility to tuberculosis, cancer and various forms of tumors, which show a strong tendency to localization in the abdomen. Parasites also penetrate and irritate the peritoneum.

Symptoms. The animal becomes dull, retiring, and inclined to lie in a quiet place, though his suffering may lead to frequent change of bed, he moves slowly, painfully, with arched back, retracted abdomen, and drooping head, and dragging his hind limbs stiffly. The abdomen is tense and firm, hot and very tender, drawing forth whines and yelps when it is handled. There are hyperthermia (104°), small, weak, accelerated pulse, hurried, catching breathing, vomiting, yawning, tympany and constipation. The face has a hopeless, stupid look and the eyes are sunken and at times glazed. Some patients become ill natured and snappish. The animal gradually sinks into a condition of prostration and finally of collapse and dies in two to eight days.

Treatment does not differ materially from that given for larger animals. Pain may be moderated by belladonna, hyoscyamus, chloral, or even opium, while the sulphate of soda is employed by both mouth and rectum. The abdomen may be enveloped in a damp compress, or a warm bath may be given. Diuretics will be in order and above all antiferments, the latter by enema as well. Distension of the abdomen with fluid may be relieved by puncture, followed by antiseptic irrigation. If there is good ground to suspect a gastric or intestinal lesion or tumors, laparotomy is a much more hopeful resort than in the larger animals.

PERITONITIS IN BIRDS.

From caponizing, accidental traumas, ruptured oviduct, perforations of bowels by foreign bodies or worms, pyogenic susceptibility slight. Symptoms: inappetence, drooping head, wings, tail, erect plumage, stiffness, straining, tense, tender, pendent belly. Treatment: unload cloaca, puncture and irrigate abdomen, laxatives. Prophylaxis, by laxative food, expulsion of worms, antisepsis in operations, unloading cloaca, etc.

Causes. Male birds contract peritonitis from caponizing, and other penetrating wounds of the abdomen, from rupture of the oviduct impacted with egg matter, from perforations of the intestines by foreign bodies, and from perforations by worms.

The danger from ordinary pyogenic germs is, however, at its minimum, since birds stand at the opposite extreme from the horse, and their wounds rarely suppurate.

Symptoms. The bird loses appetite, droops head, wings and tail, ruffles its feathers, walks stiffly and heavily, and expels fæces with much effort and even with cries. When caught the abdomen is found to be full, tense and pendent and very tender to the touch. There is more or less hyperthermia (108° and upward), and the subject becomes more and more dull, stupid and feeble until death.

Treatment. In certain cases relief may be had by the unloading of the cloaca, or the evacuation of peritoneal fluid, followed by antiseptic, irrigation of the cavity. Laxatives may also be resorted to. The most important measures are however prophylactic, and run in the direction of careful manipulation and antisepsis in caponizing, the unloading of impacted cloaca, before it has developed serious disease, the maintenance of a suitably laxative diet, and the prevention and treatment of worms. In case of tumors causing chronic peritonitis, laparotomy can be resorted to with great confidence.

ASCITES IN SOLIPEDS.

Causes: follows peritonitis, obstruction of portal vein, tumors, hepatic diseases, pressure on posterior cava, dilated right heart, heaves, ovarian disease, nephritis or kidney degeneration, hydroæmia. Symptoms: slow advance, pot-bellied, with fluctuation, hollow above, dropsy in limbs, sheath and under belly, percussion sound flat below, weakness, debility, no fever. Diagnosis: Absence of fever, and of fibrine, cells and granules in effusion. Lesions: those of primary disease, amount and composition of effusion. Treatment: treat primary disease glandular swelling or actinomycosis, iodide of potassium, remove diseased ovary or tumor, draw off fluid, compress abdomen, saline laxatives, diuretics, iodides, pilocarpin, electricity, bitters.

Causes. Ascites may be a remnant of a pre-existing chronic peritonitis, or it may occur from any obstruction of the portal vein, such as compression by organized false membranes, thrombus, in the vessel, or pressure by lymphadenoma in the portal fissure, melanosis, sarcoma and other tumors. It results from cirrhosis and other diseases of the liver which retard its circulation, from pressure on the posterior vena cava, from insufficiency of the right auriculo-ventricular valves, from dilatation of the right heart, and from heaves or other obstruction in the pulmonic circulation. Other causes are cystic or other disease of the ovary, diseases of the kidney and hydroæmia, the latter two tending to general œdema as well as ascites.

Symptoms. The disease comes on slowly and insidiously and at first it usually passes unnoticed. When more fully developed the abdomen is distended but somewhat pendent (pot-bellied), fluctuating below, with falling in beneath the lumbar transverse processes. Later the whole abdomen may be full, rounded, smooth and tense, and the hind limbs œdematous to above the fetlocks or hocks. There may be œdema of the sheath or lower wall of the abdomen. Fluctuation can still be felt as a shock when an assistant makes sudden concussion with the fist on the opposite side from that on which the hand is pressed. This may be felt even more distinctly by the hand introduced into the rectum. Percussion gives a flat sound below and more or less resonant above. The pulse is small, weak, and accelerated, heart beats irritable (sometimes palpitating), and respiration labored and with lifting of the flank. From first to last there is no hyperthermia.

If the cause is irremediable the issue is necessarily fatal sooner or later.

Diagnosis from peritonitis depends largely on the absence of hyperthermia, and of abdominal tenderness, and on the nature of the ascitic fluid which is incoagulable, and comparatively destitute of leucocytes cells and granules.

Lesions. The quantity of effused liquid is often enormous (50 qts. Reynal, 80 qts. Woodger, 150 qts. Friedberger and Fröhner). It is very watery and poor in salts and albuminoids, of a density near 1012, neutral or slightly alkaline, does not coagulate spontaneously, and is not associated with false membranes. The peritoneum shows no congestion, but is pale, and, like the abdominal walls, infiltrated. Tumors, cysts and venous obstructions referred to under causes may be found.

Treatment. When ascites depends on actinomycosis or glandular enlargement a course of iodide of potassium may remove the cause. In other cases an operation may remove the offending tumor or ovary. Too often, however, the cause is beyond remedy and palliative treatment only is available. The most urgent indication is the removal of the accumulated fluid, and paracentesis under proper antiseptic precautions is the readiest means to this end. Compression by a tight bandage is necessary to prevent the sensation of vacuity and tendency to fainting which come from the removal of the fluid and to counteract the disposition to the instant effusion of more. Even with the compress it is judicious not to draw off all of the fluid at once in bad cases, but to make two or three operations and allow the patient to become accustomed to the change in the intervals. These may be repeated as circumstances demand. Saline purgatives, or diuretics (saltpetre 1 oz., digitalis 25 grs., squill 3 ozs., iodide of potassium 2 drs.), are useful, and pilocarpin is the most efficient agent of this kind, but also dangerous by reason of the extreme depletion which it causes. Electricity has been employed with alleged advantage, also poultices of digitalis applied over the loins.

Cholagogues are also recommended especially in cases of liver disease. Bitters may prove useful.

ASCITES IN RUMINANTS.

Causes: as in horse, tuberculosis, in sheep distomatosis, chills when heated and fatigued. Symptoms: pot-belly, fluctuating on percussion, gives flat sound, debility, pallid mucosæ, sunken eyes, superficial dropsies on belly, in limbs, and under jaw, in distomatosis, great emaciation, weakness, paperskin, ova of distoma in fæces. Diagnosis: from ruptured bladder by passage of urine, and perhaps by sex, and absence of urinous odor in liquid, from hydrometra by fluctuation over whole belly. Lesions: those of solipeds, also tubercles or enlarged gall ducts with distomata. Treatment: as for solipeds. Tuberculosis demands separation or destruction, distomatosis, prevention.

Causes. These are in the main those which operate in the horse and need not be repeated. In cattle, however, the affection is to a large extent the result of abdominal tuberculosis, while in sheep it is a constant result of advanced distomatosis. Gellé says it is common in working oxen, which are turned out, hot and perspiring, to pass the night in cold and wet.

Symptoms. The belly is enlarged and pendent, bulging out back of the ribs, with fluctuation and dullness on percussion. The animal is in very low condition, the mucosæ pale or yellowish white, the eyes dull and sunken, panting and palpitations may be roused on the least exertion, and swellings often appear along the lower aspect of the body and between the branches of the lower jaw. In distomatosis it is common to find dropsy of the chest, pallor and attenuation of the skin, complete absence of the subcutaneous fat (paperskin), and great emaciation and weakness. Ova of the distoma can be found in the fæces. (See distomatosis). By turning the sheep on its back or setting it up on its croup the percussion dullness will be made to shift, always to the dependent part of the abdomen.

Diagnosis. From rupture of the bladder it is distinguished, by its occurrence in females as well as males, by the absence of fever, and of the complete suppression of urine and emptiness and tenderness of the bladder which characterize the latter. Liquid drawn from the abdomen has no urinous odor. From hydrometra, pyometra, and hydramnios it is distinguished by the fact that the water accumulates in the lower part of the abdomen, and is not confined to the womb. On rectal exploration the outline of the empty womb is made out.

Lesions. Besides the lesions described for solipeds, one finds in cattle, tuberculosis of the liver, spleen, and lymph glands, and extensive clusters of tubercles on the peritoneum. In sheep the white branching lines on the back of the liver may indicate the distension of gall ducts infested by distomata.

Treatment does not differ from that recommended for solipeds. In tuberculous cases, sanitary considerations demand the destruction of the animal and disinfection of the carcass. In distomatosis treatment must be preventive, as the distomata are difficult to reach with vermifuges.

ASCITES IN CARNIVORA.

Causes: obstructed flow of blood in hepatic, portal or renal veins, or in vena cava, renal, heart, liver or splenic diseases, pulmonary congestion, asthma, tuberculosis. Symptoms: pot-belly, hollow above, drooping back and loins, flat percussion sound and fluctuation, change of position changes area of flatness, anæmia, debility, scanty urine, diarrhoea, no fever. Diagnosis: absence of fever, general fluctuation changing its seat by turning the patient, not confined to a given organ like the bladder or womb. Lesions: quantity and composition of liquid, lesions of primary diseases. Treatment: Correct if possible the primary disease, evacuate the liquid, compress on abdomen, iodine solution for irrigation, saline purgatives, diuretics, pilocarpine, bitters, iron, sunshine.

Causes. Ascites is generally the result of some obstruction to the return of blood from some abdominal organ, but may also come from renal disease, or hydroæmia in which general dropsy is likely to occur. The dog is specially subject to heart disease, and disease of the right heart (tricuspid insufficiency, dilatation, hydro-pericarditis, fatty degeneration, etc.) throws the blood back on the whole venous system and the extensive and dilatable portal veins are especially liable to suffer. Diseases of the liver, so common in pampered house dogs, still more directly block the portal circulation and induce ascites. Tumors in the liver or spleen or in the lymph glands of the porta act in this way, also cirrhosis, tuberculosis, cancer, hepatic congestion, and degeneration. Constrictions of the vena portæ by false membranes the result of former peritonitis must also be recognized. As more distant causes, must be named obstruction to the pulmonary circulation, as in congestion, asthma, tuberculosis and diseases of the left heart. Seventy-eight cases were traced as follows: to diseases of the heart and pericardium, 10; to tuberculosis, 8; to pleurisy, 4; to malignant tumors of the liver and lung, 2; to hepatic disease without heart lesion, 3; to cancer of the liver, 1; (Cadiot).

Symptoms. Enlargement of the belly is marked and peculiar, the liquid accumulating below, pushing outward the lower ends of the ribs, and making the lower part of the abdomen baggy while the upper part, under the lumbar transverse processes, is flattened or hollow. The back and loins droop forming a concavity superiorly, so that the belly may almost drag on the ground. On palpation this pendent abdominal sac gives the sensation of a mobile fluid without the usual firm outlines of the intestinal masses, and when percussed it gives out a flat, dull sound and produces a fluctuation or shock at the opposite side of the abdomen. In the upper part of the abdomen over the hollow flank more or less resonance is found. If the animal is made to stand on his hind limbs the saccular dilatation and flatness on percussion are in the region adjoining the pelvis; if held up by its hind limbs they are transferred to the epigastric and hypochondriac regions and the respiration is seriously interfered with; if turned upon his back, the resonance is obtained on the linea alba and at each side, while the percussion dullness is next to the vertebræ. The clearness of the fluctuation is in ratio with the amount of liquid present.

As in other animals, there are anæmia, pale mucosæ, poor condition, thin, dry, unhealthy skin, weak pulse, irritable heart and interference with respiration proportionate to the amount of liquid. The urine is scanty, and there may be diarrhoea.

Diagnosis. From advanced or chronic peritonitis it is distinguished by the history or evidence of diseased liver, heart, or kidney, the absence of hyperthermia or abdominal tenderness, and the absence in the ascitic fluid, extracted with a hypodermic needle, of blood globules, or leucocytes in numbers, of false membranes, of excess of salts, or of a tendency to coagulate firmly.

From overdistended bladder it is diagnosed by its slow, and gradual development, and the change of fluctuation to the most dependent part no matter what position is given to the patient, whereas the tense bladder can be felt through the abdominal walls, extending forward from the pelvis under all circumstances.

From ruptured bladder there is the same distinguishing feature of slow development, the absence of symptoms of uræmic poisoning, of tenderness of the bladder, and of suppression of urine, and also of the urinous odor in the ascitic liquid obtained with the hypodermic needle.

From advanced gestation the differentiation is found in the general diffusion of the swelling and fluctuation, which is not confined as in gestation to the mobile uterine horns, with a series of enlargements each containing a solid nodular foetus.

From hydrometra and pyometra there are the same pathognomonic differential features of the general diffusion of the swelling among the intestines, and its accumulation in one fluctuating mass at the most dependent part of the abdomen.

Tympany of the bowels causes uniform drumlike resonance, and the swelling does not sag and fluctuate in the lower part of the abdomen.

Abdominal obesity in old dogs gives the rounded swollen abdomen, but there is an entire absence of the pendulous and fluctuating features, and when punctured with the hypodermic needle it furnishes no fluid.

From tuberculosis it is distinguished by the absence of nasal discharge, or of tubercle bacilli in such discharge, or in the ascitic fluid, and the latter inoculated on guinea pigs or rabbits does not cause tuberculosis. The tuberculin test may also be resorted to.

Lesions. The liquid exudate has been found to amount to 30 or 40 quarts in large dogs (Hordt). It is often clear and translucent, of amber tint, though in some cases it is slightly opaque, or reddish yellow. It may remain fluid after extraction or again it may form a loose jelly. It may be red in case of soft tumors or other neoplasms. The liquid is very watery but may contain a considerable amount of fatty globules or granules, and a few epithelial cells and leucocytes. The peritoneum is pale or in advanced cases dull white from fatty degeneration of the epithelium.

Treatment. The first consideration is the removal of the cause. If this is a mere vicious action of the peritoneum, or the presence of a thrombus, or of operable tumor, or even of curable disease of the liver or kidneys, success may be hoped for, while in dilatation of the heart, insufficiency of the cardiac valves, irremediable disease of the lungs, liver or kidney, or malignant or inoperable tumor no such result can be hoped for.

Apart from the removal of the cause the first indication is to evacuate the liquid and this may be done with a large hypodermic needle or small cannula and trochar inserted by preference on or near the linea alba while the animal is in a standing position. Skin and instrument should be rendered thoroughly aseptic, and a bandage should be wrapped round the abdomen and gradually tightened as the liquid escapes. This to a large extent obviates the tendency to faint, or to cerebral anæmia which has caused sudden death in a number of cases. It also to some extent counteracts the sudden effusion of blood in the abdomen, which is at times determined by the sense of vacuity.

Injection of a solution of iodine (tincture of iodine 1 pt., iodide of potassium 1 pt., boiled water 20 pts.) has been employed sometimes with success, but in other cases it has roused a fatal inflammation. It is best adapted to a simple morbid, relaxed state of the peritoneum.

Saline purgatives (sulphate of soda or magnesia) are especially useful in constipated cases and should be pushed in continuous action, as far as the strength of the animal will warrant. By depletion from the portal system they oppose the tendency to mechanical transudation, while by rendering the portal blood more dense they strongly solicit endosmosis from the adjacent peritoneum.

Diuretics have been used extensively and with benefit. They may prove injurious in a kidney that is already the seat of irritation and yet after all be the least of two evils. In some cases instead, the resulting dilution of a dense and irritating urine is directly soothing to the tender kidney. Saltpeter (10 to 15 grs.), acetate of soda (15 to 30 grs.), squills (1 to 2 scr.), may be repeated so as to keep up a free action. Pilocarpine (subcutem) ($\frac{1}{106}$ to $\frac{1}{30}$ gr. daily), has removed the ascitic fluid in 14 days (Zahn), but its action is always to be dreaded in a weak system, or with a diseased heart, or lungs.

A supporting bandage on the abdomen is always useful as counteracting the tendency to vacuity and further transudation.

A course of bitters and iron, and a supporting diet, and out door life (sunshine) are important elements in treatment.

DISEASES OF THE LIVER.

In veterinary and medical works the diseases of the liver have been accorded a minor place, ill in keeping with the great physiological importance of the organ. If the function of the liver were circumscribed by the mere secretion of bile there would be some excuse for the apparent neglect, as the gland is so deeply situated and so much enveloped in surrounding organs that physical exploration is difficult and somewhat unsatisfactory, and the one symptom of jaundice was long relied on as indicating hepatic disorder.

Taking into account all the varied functions of the liver we realize the wide-reaching nature of its physiological influence and the extensive and varied effect of its disorders. We can also deduce, with greater or lesser certainty, the existence of hepatic disorders from the morbid conditions of the blood or of organs, the functions of which are inter-dependent with those of the liver. To elucidate the subject it is well to trace some of the most prominent functions of the liver; the following considerations are submitted.

SANGUIFICATION IN THE LIVER.

Glycogenic function. Glycogen derived through glucose and laevulose from starch, glycerine, milk and cane sugars. Less from proteids. Peptones as a source. Its use in cell growth and heat production, in white blood cells, in contracting muscle, becoming lactic acid. Excess dissolves red globules, setting free hæmoglobin. Ammonia carbonate and asparagin increases it. Arsenic, phosphorus or antimony arrests glycogenesis. Liver increases leucocytes, and reduces size of red globules. Reduction of proteids. Fibrine formers reduced, urea formed; liver inactivity means less of soluble urea, and more of less soluble and more dangerous products. Urea increases with hepatic circulation. Hepatic disorder and suppression of urine dangerous. Red globules probably destroyed. Bile: Amount, uses, oil solvent, helps endosmosis, deodorant, stimulates glycogenesis, excretory. Source of bile pigments, tests. Bile acids, dissolve blood globules, antiseptic, tests. Bile increased by; bile absorbed from bowel, olive oil, salol, salicylates, benzoic acid and benzoates, turpentine, terpene, terpinol, euonymus, alkalies, arsenic, ether. Agents lessening biliary secretion, starvation, excess of fat, alkaline iodides, atropia, strychnia, hepatic diseases, septic duodenal fermentation. Arrest in liver of copper, iron, iodides, bromides, nicotine, quinine, morphia, curare, toxic bile products, ptomaines, toxins. Reduces toxicity of peptones, casein, ammonia salts, indol, phenol.

The liver is the goal to which most of the products of gastric and intestinal digestion are carried by the portal vein. In the hepatic cells large quantities of *glycogen*, $6 (C_6H_{10}O_5) + H_2O$, are stored up after each meal. This is believed to be derived largely from the transformation of glucose, $(C_6H_{12}O_6)$ and laevulose $(C_6H_{12}O_6)$ which have been produced from starch in the alimentary canal and conveyed by the portal vein to the liver. By the liberal use of starch, glycerine, or the sugars of milk, fruit or cane, (but not mannite, or glycol, or inosite) the glycogen is very greatly increased (to 12 per cent. in the fowl), but it is diminished on a purely albuminous diet. Yet it can be produced from albuminous food, as it is always increased in the dog after a meal of flesh, and is largely present in the livers of carnivorous animals that have been fed for a month on flesh only (Landois). The peptones are therefore decomposed in the liver with the production of glycogen and such waste products as leucin and tyrosin, which are finally resolved into urea. A purely fatty diet diminishes it enormously and during prolonged abstinence it practically disappears. It passes, not into the bile, but into the hepatic veins, and the general circulation, where it serves in its decomposition to generate heat, and probably to hasten cell growth. In the vegetable and animal world, in the germinating seed, and in cartilage, muscle and epidermis of the foetus and in the amnios, glycogen and glucose are found in abundance. The liver, too, the great center for the production of glycogen, is relatively much larger in the young and growing animal, and also in the adult animal which has great power of assimilation.

Glycogen is always present in the white blood globules so long as they maintain their vitality and amoeboid movements, but when they die, it is replaced by sugar (Hoppe-Seyler). The red blood globules give up a ferment which rapidly transforms glycogen into sugar.

Glycogen and sugar are evidently of use in muscular contraction as they are always diminished in the vessels of contracting muscles (Sanderson), being converted into lactic acid (Bernard).

Forced muscular movements soon expel glycogen from the dog's liver, passing it into the blood, and there the excess of glycogen dissolves the red blood globules. If glycogen is injected into the blood, achrodextrin and hæmaglobin appear in the urine (Landois).

Ammonia carbonate and asparagin, or glycin, with a carbohydrate diet produced in rabbits a considerable increase of glycogen (Rohmann).

Poisoning by arsenic, phosphorus or antimony destroys the glycogenic function of the liver, which then fails to respond even to diabetic puncture of the medulla.

There are important changes effected in the blood globules in passing through the liver. The *leucocytes* are increased, the hepatic veins containing 5 or even 10 times as many as the portal vein (Bernard, Lehmann, McDonald). Their ratio to the red globules is in the portal vein 1:524 and in the hepatic veins 1:136 (Hirt). The *red globules* undergo marked changes, having, in the hepatic

veins, a smaller size, sharper outlines, less flattening in the disc, a habit of massing together irregularly in place of adhering in rouleaux, and they dissolve less readily in water.

REDUCTION OF ALBUMINOIDS.

A large proportion of the fibrine formers are changed in passing through the liver (Lehmann, Bernard), in man as much as 2,690 grammes daily (Brown Sequard), a fact which goes to account for the increase of fibrine in inflammation when the liver is inactive. The change consists mainly in deoxidation and reduction into simpler compounds which can be more readily dissolved and eliminated. Arrest of the liver functions in fever is therefore liable to throw into the blood, products that are little soluble and often poisonous. The end product is largely urea, and this is always found in excess in the hepatic veins of dogs (in the portal veins 0.08 grammes, and in the hepatic veins 0.14 to 0.17 grammes). In man hepatic disorder is at once marked by the lessening or disappearance of urea from the urine, and the increase of the less oxidized uric acid (Parkes). In acute atrophy of the liver, urea disappears from the urine, being replaced by the less oxidized leucin and tyrosin (Frerichs, Murchison). In birds urea is replaced by uric acid and this is always found in the liver.

The increase of urea and allied products bears a direct relation to the activity of the hepatic circulation. Stimulation of the liver by electric current sent through the abdominal walls largely increased the secretion of urea (Sigrist, Stolnikow, Schröder and Salomon). Murchison, Perrin and Bruardel had a great increase of urea by stimulating the circulation in the liver. Certain agents ingested are transformed into urea, among which may be named glycocholate, brucin, asparagin, sarcosine, alanine, and ammonia muriate.

Any degeneration of the hepatic cells which impairs or arrests their functions lessens the production of urea. In fevers therefore and in hepatic degenerations the extent of the functional or structural derangement may be to a large extent gauged by the diminution of urea. A simple hyperæmia, without as yet any serious impairment of structure or function, may be attended by a marked increase of urea, whereas any destruction of the liver cells, or any serious modification which interferes with the normal function, brings about a decided decrease. A hepatic disorder accompanied by suppression of urine is always a grave disorder. On the contrary a free secretion of urine during liver disease is a favorable symptom.

There is reason to believe that red blood globules are destroyed in the healthy liver, producing bilirubin and urea (Landois). In diseased states this becomes excessive, and the resulting coloring matter is often modified, giving the strong tints, seen in the urine in fever and certain hepatic disorders.

SECRETION OF BILE.

The secretion of bile is but a small part of the function of the liver, and that is by no means a purely eliminating process. Man secretes in twenty-four hours about 10 parts per 1,000 of body weight, the dog 14 to 15, the cat 15 to 20, the sheep 25, the rabbit 130, the Guinea-pig 170, the goose 12 (Cadeac), the horse 12 (Colin). But the amount varies largely; Scott found that a dog yielded 21, and Kölliker that another yielded 36 per 1,000 of the body weight.

Only about one-fourth of the biliary acids (Bischoff, Voigt), and one-eighth of the sulphur (Bidder and Schmidt) of the bile can be found in the fæces. Most of the bile is re-absorbed from the intestine and secreted anew, so that, in the course of twenty-four hours, the material secreted serves the same purpose again and again. During this repetition of secretion and absorption, it becomes little by little metamorphosed into other products, which are eliminated by the lungs and kidneys (Parkes, Murchison).

The functions of the bile so far as known are:

- a. The solution of alimentary matters, and especially of fat, in the intestine, and the hastening of endosmosis, of fats and peptones;
- b. The stimulation of peristalsis in the bowel;
- c. Antisepsis and deodorization of the contents of the bowels;
- d. The determination of the formation of glycogen;
- e. The excretion of bile coloring matter, bile acids and cholesterine.

In regard to the glycogenic action it may be said that in cats, the bile ducts of which have been tied, no glycogen was formed, even when the diabetic puncture of the brain was made (Legg). Clinical observation seems to throw some doubt on the formation of bile coloring matter apart from the liver. In diseased liver with suspended secretion of bile (waxy and fatty degeneration, cancer, cirrhosis) the bile pigment was found in neither blood nor urine (Frerichs, Murchison, Haspell, Budd). Even after extirpation of the liver in frogs, neither biliary acid nor pigment could be found in the blood (Müller, Lehmann, Moleschott). These results must, however, be qualified by the observations of Hammersten who found bilirubin as a normal constituent of blood serum in the horse, and by Virchow's discovery that hæmatoidin (now held to be identical with bilirubin) is constantly found in old blood extravasations into the tissues.

The origin of the bile coloring matters may be traced in part to destruction of red globules in the liver. Quinke has shown that in the hepatic capillaries in post-embryonic life the leucocytes englobe and destroy the old and worn out red blood corpuscles which thus become a source of bile coloring matter. Such destruction is specially likely to occur in badly maintained conditions of the blood, and in hepatitis or other liver disease in which the white cells accumulate in the hepatic capillaries, and when the blood current is retarded. Hence the liability to jaundice in such conditions. The formation of new red blood corpuscles has been observed in the protoplasmic cells of the liver in the embryo, but this has not been established for post-embryonic life (Neumann, Lowit).

The two common coloring matters of the bile are **bilirubin** which colors the yellow bile of man, omnivora, and carnivora and **biliverdin** which tints the dark green bile of herbivora. **Bilirubin** ($C_{32}H_{36}N_4O_6$) forms transparent fox red clinorhombic prisms. It is insoluble in water but soluble in chloroform, and may thus be separated from the biliverdin which is insoluble in chloroform. United as a second basic acid with alkalis it is soluble in water. It is easily obtained from the red gall-stones of man or ox, and is chemically identical with hæmatoidin. **Biliverdin** ($C_{32}H_{36}N_4O_8$) is an oxidized derivative of bilirubin and is insoluble in chloroform, slightly soluble in ether and freely soluble in water. In addition to its presence in bile it has been found in the placenta of the bitch. The **test** for bile coloring matter is made by placing a drop of the suspected liquid on a white porcelain plate and adding a drop of impure, brown, nitric acid (nitric and nitrous acids). If bile is present there is produced a beautiful play of colors passing from the green of biliverdin, through blue, violet, red, and ending in yellow.

With regard to the formation of bile pigments in morbid conditions it may be noted, that agents which dissolve the red blood globules (such as bile acids or water), when injected into the veins determine the appearance of bile pigment in the urine (Frerichs, Kuhne). When we consider that an animal (dog) secretes $\frac{1}{50}$ of its body weight daily of bile, and that nearly all of this is re-absorbed from the intestines, we can realize this as an important source of bile and urinary pigments.

Of the **bile acids**, **taurocholic** is the most abundant in the bile of man, birds, and of many mammals and amphibians, while **glycocholic acid** is the more plentiful in the ox and pig. It is absent in sucklings. The taurocholic acid has been found to prove most destructive to red blood corpuscles, and in strong solution is distinctly antiseptic, checking the development of bacteria, of the alcoholic and lactic fermentations and of the tryptic and diastatic action of pancreatic juice.

These are conjugate acids, formed by the union of cholic acid with taurin and glycin respectively, and they are found almost exclusively, in combination with soda in the bile. They are found in the liver and do not accumulate in the blood when the liver has been removed. They increase under an albuminous diet.

The **test for bile acid**, is to take the suspected liquid from which all albumen has been precipitated, add a few drops of solution of cane sugar, shake into a froth, and pour sulphuric acid, drop by drop, down the side of the test tube. A *reddish purple* color appears in the froth, and shows two absorption bands at E and F. Any albumen left in the liquid will give the same color, but only one absorption band.

The **secretion of bile** is more abundant on animal than on vegetable food, and on albuminous than fatty. It ceases during hunger, but is increased by ingestion of water. Its solids are most abundant one hour after feeding. It increases under a copious and rapid blood supply, but is arrested by diminished blood flow, even under increased pressure (in ligature of the vena cava in front of the diaphragm). Vigorous exertion, drawing off blood to the muscles of the trunk, diminishes the secretion of bile, while transfusion of blood, up to a given grade of blood pressure increases it. Nervous conditions, which cause contraction of the portal vessels, increase the secretion by forcing more blood through the liver. Such are strychnia or other stimulation of the valve of Vieussens, of the inferior cervical ganglion, of the hepatic or splanchnic nerves, or of the spinal cord. Fever causes its arrest.

The **secretion of bile** is further stimulated by the following :

a. The ingestion of bile into the stomach and abdomen. This being absorbed and carried to the liver greatly increases the biliary secretion. It is not necessary that the bile shall be a product of the same genus of animal, the bile of the ox is an active stimulant of the liver of the dog.

b. Of medicinal agents the following increase and liquefy the bile: olive oil in large doses, phosphate of soda, salol, and salicylate of soda.

c. The following not only increase the bile, but through their purgative operation, expel it from the bowels: calomel, mercuric chloride, colocynth, aloes, jalap, rhubarb, podophyllin, and cold rectal injections. These accordingly lessen the secretion later, by removing the stimulus of the absorbed bile.

d. The following are comparatively mild biliary stimulants: benzoic acid, benzoate of soda, oil of turpentine, terpene, terpinol, and euonymus, and still less active are alkaline bicarbonates, bromides, sulphates and chlorides, arsenic and ether.

Secretion of bile is lessened by: starvation, a too fatty dietary, alkaline iodides, atropia, strychnina, hepatic degenerations, (fatty, cirrhosis), catarrh of the bile ducts, diseases of the liver, gall duct, or duodenum which interfere with the discharge of bile, the antiseptics of the bowels, or the reabsorption of bile. This work virtually moves in a vicious circle, as the action of septic ferments in the duodenum hinders the reabsorption of bile and of the food products which go to the production of bile, and in its turn the withholding of bile from the intestine removes the normal antiseptic (the bile acids) and favors septic fermentation and the inhibition of duodenal digestion and absorption. Another factor is found in the ptomaines and toxins absorbed from the alimentary canal and arrested in the liver. These debilitate the liver cells, impair the liver functions and lay the gland open to bacteridian infection. The bile in such a case is transformed into a pale or yellow,

viscid liquid, with more or less dark colored granular debris, and this proves a favorable culture ground for bacteria especially the *golden staphylococcus* and the *bacterium coli commune*. With septic condition of the liver the usual result of ligature of the bile duct is a peri- and intralobular sclerosis and the formation of minute biliary abscesses. In the absence of sepsis, ligature of the biliary duct, produces—not abscess but—necrobiosis, preceded by interlobular connective tissue hyperplasia, and granular or fatty degeneration of the hepatic cells. (Charcot, Legg, Lahousse, Dupre).

THE LIVER AS A DESTINATION AND DESTROYER OF POISONS.

The liver in the mature animal, being the one destination of the blood carried in the portal vein, necessarily becomes the recipient of all medicinal and poisonous agents absorbed by the capillaries and venous radicals of the stomach and intestines. This organ retains and lays up for a time the heavier metals, such as the salts of copper and iron, the iodides and bromides, the vegetable alkaloids such as nicotine, quinine, morphia, and curare, the toxic elements of the bile, the ptomaines and toxins produced by gastric and intestinal fermentations, indol, phenol, etc. Some agents it transforms, as peptones (which it renders non-poisonous), casein, the carbonate of ammonia and its salts with vegetable acids, also indol and phenol, which it combines with sulphuric acid as indyxol and phenyl sulphate, thus rendering them much less toxic. The destructions or new combinations established in the cases of the ptomaines and toxins may explain why such agents are usually much less poisonous when taken by the stomach than when generated in tissues or blood, or when injected hypodermically. Another interesting fact in connection with the ingestion of these bacteridian products (ptomaines and albumoses) is that, when the liver functions are normal as evidenced by the production of glycogen, the toxins are largely destroyed, and they fail to produce poisoning, whereas with a functionally deranged liver and no production of glycogen, they retain their potency, almost as if injected subcutem.

FUNCTIONAL DISORDERS OF THE LIVER.

MELLITURIA, GLYCOSURIA, DIABETES MELLITUS, SACCHARINE URINE.

Source of glucose in food. Glycogen: Its use: Enlarged liver means more glycogen. Glycosuric centre in medulla. Other glycosuric nerve centres. Reflex action, action of drugs and poisons, phlorizin. Disease of lungs or pancreas. Removal of pancreas in dogs. Removal of thyroid. Diseased, liver, fatty, fibroid, hypertrophy, congestion. Extreme fatty change arrests glycogenesis. In solipeds: 3 cases with liver hypertrophy; 1 case with adenitis; 6 cases with emaciation; 2 cases with hæmoglobinuria. Symptoms: Emaciation, debility, langor, fatigue, breathlessness, hollow flanks, unthrifty skin, ardent thirst, polyuria, urine saccharine, of high density. Diagnosis by analysis of urine, sweet taste, Fehling's test, Trommer's test, fermentation test. Prognosis: Grave, diet being carbonaceous, when functional resulting from curable disease is hopeful. Treatment: In poisoning cases, antidotes and eliminants, in curable disorders treat these, in more inveterate cholagogues, antiseptics, codeine, opium, croton chloral, strychnia, phosphoric acid, iodoform, ergot, skim milk or buttermilk, good hygiene, open air, shelter, carminatives, bitters, mineral acids, treat complications.

Grape sugar (glucose, $C_6H_{12}O_6$) is undoubtedly formed in the stomach and intestines by the action of saliva and pancreatic juice on starch ($C_6H_{10}O_5$), and glucose and laevulose ($C_6H_{12}O_6$) are also derived from the transformation of cane sugar ($C_{12}H_{22}O_{11}$). These sugars are absorbed, transformed into glycogen in the liver and passed into the circulation, where they serve to maintain animal heat through their decomposition into carbonic acid and water. They further assist in nutrition and growth, and if their metamorphosis is imperfect they pass out of the system in the urine, producing a temporary glycosuria. As shown above glycogen is produced in the liver cells, and stored up there, in greatest abundance during digestion of starchy and saccharine food, but it is also formed in animals kept on a purely albuminous diet, (flesh), and in the foetal calf and unhatched chick to which neither starch nor sugar has been furnished as food. It is produced during the decomposition of albuminoids, along with the other end products, leucin, tyrosin and urea. None of these last three is found in the portal vein nor bile ducts, but all four are found in the liver cells, and in the hepatic veins.

In health a physiological balance is maintained by the oxidation of the glucose, mainly in the lungs, so that in the blood of the pulmonary veins no sugar is found. There is an exception to this observable after a full meal, rich in starch and sugar, which produces such an excess of glycogen that a portion is carried to the kidneys and expelled by them causing temporary glycosuria.

A small amount of glycogen is also produced habitually by the white blood cells and stored up in them, but this is insufficient to determine its appreciable elimination by the kidneys.

In cases of persistent glycosuria the fault may be held to consist in one of three functional derangements:

- 1st. The failure of the liver to transform the alimentary sugar into glycogen.
- or 2d. The excessive production of glycogen in the liver.
- or 3d. The arrest of the destructive oxidation of sugar in the lungs and tissues.

In a diabetic patient who died suddenly of apoplexy Bernard found that the liver was enlarged, comparing with the average as 25:14 while the contained sugar bore the ratio of 37.5:22. This enlargement coming from malaria or other poison, such as alcohol, ether (Harley), arsenic, quinia (Aitken), ammonia, chloroform, or phosphoric acid (Murchison), is an established condition of glycosuria. A rich and abundant food (starchy and saccharine especially), or an unusually active hepatic circulation acts in the same way.

Bernard as early as 1849 showed that the glycogenic function of the liver was greatly increased and glycosuria determined by pricking the floor of the fourth ventricle in the median line just in front of the calamus scriptorius and near the root of the vagus nerve, or a few millimeters in front of this.

It follows that irritation of this part of the medulla however produced, whether from local disease, or by reflex action from some distant organ in a state of irritation, may serve as the starting point of diabetes in particular instances. That the cause may be a reflex stimulus is shown by the suspension of the glycogenic function after section of the vagus nerves, and its reappearance when the central end of the cut vagus is galvanized, or, the floor of the fourth ventricle is irritated, the

direct or efferent excitation being transmitted through the sympathetic nerve (Bernard). I can cite a case of glycosuria in a man supervening on a severe blow on the head from a falling ledger. Brain injuries which suspend animal functions, but not the nutritive ones, such as apoplexy, concussion of the brain or curare poisoning are liable to induce diabetes.

Traumatic injuries to other parts of the nervous system induce glycosuria. Thus traumatism of the optic thalami; of the cerebral lobes or peduncles; of the pons; of the cerebellum or of its middle or posterior peduncles; transverse section of the medulla or of the spinal cord opposite the second dorsal vertebra; traumatism of the superior or inferior cervical ganglion or the first thoracic (Eckhard); of the sympathetic twig which accompanies the vertebral artery (Pavy); of the brachial plexus; of the solar plexus (Munck, Klebs); or of the sciatic nerve (Schiff).

The explanation of these facts may be sought in a reflex action established by the conveyances of irritation to the true glycogenic centres in the brain and the transference of the efferent nervous impulse through the sympathetic nerve to the liver. It will be borne in mind that in the case of section of the vagus nerve electric stimulation of its detached peripheral part has no glycogenic effect on the liver, while galvanizing the central portion determines glycosuria.

In the case of glycosuria through stimulation of the sympathetic nerve or its ganglia the action may be concluded to be direct. Strangely enough, irritation of the sympathetic between the tenth and twelfth ribs or the splanchnic nerves fails to produce glycosuria, though the hepatic branches of the sympathetic pass through them.

In ordinary cases of reflex glycosuria it may be assumed that the existence of irritation at the peripheral ends of the vagus and of some other nerves, leads to an apparent glycogenic influence passing through these to the brain, and of the distribution of the efferent impulse through the upper portion of the spinal cord, as far as the fourth dorsal vertebra in the rabbit (Cyon, Aladoff, Schiff), and through the sympathetic nerve to the liver. This may account for the appearance of the disorder as a sequel of disease in any part to which the vagus in particular is distributed, and notably in the lungs. A number of poisons (malarial, alcoholic, ether, carbon monoxide, amyl nitrate, curare, or the nitro-propionic acid, methyl delphinin, morphia, chloral hydrate, arsenic, quinia, ammonia, chloroform, phosphoric acid, and phlorizin) produce glycosuria.

The intravenous injection of dilute saline solutions, or frequent blood letting materially increases the sugar, probably by causing solution of the red globules. Phlorizin is the most potent of all these agents. Whether given hypodermically or by the stomach it causes in three hours a marked production of glucose which continues to be eliminated for a period of thirty-six hours. The urine may become charged with glucose to the extent of from 6 to 13 per cent., and without any rise in the body temperature. This artificial glycosuria may be kept up indefinitely by the continued administration of phlorizin, and even in the fasting animal, or one on an exclusively albuminous diet, as well as in those on an aliment rich in saccharine or hydro-carbonaceous matter. In the frog it produces diabetes even after the extirpation of the liver showing that it stimulates other sources of sugar production beside the hepatic or that it inhibits the transformation of sugar derived from the alimentary canal and other sources.

Another suggestive source of mellituria is disease of the lungs, or any condition which interferes with the due æration of the blood and oxidation of the alimentary or hepatic sugar. But it cannot be assumed that the rôle is altogether or mainly chemical. The thoracic organs being supplied by branches of the vagus and sympathetic nerves there is the obvious suggestion of a reflex action through the diabetic centers in the brain. The frequent complication of diabetes with lung diseases (inflammatory, tubercular, syphilitic, and otherwise) is abundantly proved, whether it is to be explained on the above hypothesis or through other unknown changes in the blood.

Diabetes has been repeatedly found in connection with disease of the pancreas, and the complete extirpation of the pancreas in dogs gives rise to glycosuria (Mering and Minkowski, Thierloix, Lancereaux, Lepine). If a small portion of the pancreas remains glycosuria does not supervene. It has been suggested that the pancreas has a double function, and beside its secretion, produces a glycolytic ferment which passing into the portal blood determines the formation of glycogen in the liver. Arrest of the pancreatic secretions does not cause glycosuria, so it has been suggested that the glycogenic enzyme is a product of the connective tissue cells of the pancreas. Functional as well as

structural disease of the pancreas can be conceived of as inhibiting the production of this ferment and the consequent elaboration of glycogen. Chauveau and Kauffmann deduce from their observations that the action is a reflex one established through the glycogenic centres in the medulla. Pancreatic glycosuria is especially fatal (Harley).

Finally extirpation of the thyroid body in dogs has been followed by glycosuria (Falkenberg). This suggests a systematic examination of the urine in all cases of goitre, with extensive glandular changes.

Apart from experimental cases diabetes in the lower animals has been observed to be nearly always associated with diseased liver. Fatty degeneration has been the most frequent lesion, but cirrhosis, hypertrophy and congestion were present in other cases. In a number of cases as the fatty degeneration reached an extreme degree, the sugar disappeared from the urine, the hepatic cells being no longer functionally active, and death speedily followed. The same has been observed in the fatty degeneration attendant on poisoning by arsenic or phosphorus.

GLYCOSURIA IN SOLIPEDS.

Heiss records two cases of this disease in heavy Belgian horses ten and eleven years old, the urine of which showed a percentage of 3.75 of grape sugar, and which died in two months in a state of marasmus. The liver was enlarged and of a clay yellow color. Dieckerhoff reports one fatal case in which there were also yellow discoloration, congestion and hypertrophy of the liver. No lesion could be found in the pancreas nor nervous system. Perosino records a case in a horse suffering from contagious adenitis, which may be supposed to have been connected with the action of the toxins or the imperfectly oxidized albuminoids on the nerve centres or liver. Delprato relates six cases in the same stable in overworked, half starved and emaciated horses. Rueff and Mouquet each contributes a case occurring in paraplegia attendant on hæmoglobinuria and in which the amounts of sugar were respectively 5.85 and 1.01 per cent. These latter cases are manifestly complicated ones in which the reflex irritation (or inhibition of glycogenesis) is transmitted from the diseased or poisoned brain to the already disordered liver.

Symptoms. There is a profound interference with nutrition, a rapid loss of flesh and weight, of spirit and energy and an extreme muscular weakness in spite of an excessive appetite. The subject is fatigued and breathless under the slightest exertion, the flanks are retracted and hollow, and the hair dry, rigid and lifeless. Appetite is poor and fastidious, but an intense and consuming thirst is usually present, the animal drinking deeply at every opportunity, and passing urine with corresponding frequency and abundance. The urine is clear, yellow, neutral, and saccharine, the sugar varying from 1 to 12 per cent. (3.6 on an average). Notwithstanding the amount passed (55 litres per day, Cadeac) the density usually exceeds the normal (1052 and upward), normal being 1040 to 1050. There may or may not be hyperthermia, and in exceptional cases appetite has been retained to the last. Cataract and corneal ulceration are sometimes observed as in man.

Diagnosis. Presumption may arise from the above mentioned symptoms, and especially the bulimia, the polydipsia, the polyuria, the rapidly advancing emaciation, weakness, and prostration and the ocular troubles, but conclusive evidence is only found in the presence of glucose permanently in the urine.

Tests for Sugar in the Urine. For one who can go through it the touching of the tip of the tongue with a drop of the suspected urine will give a prompt and reliable test.

Fehling's cupric test is the next best for simplicity and availability. Dissolve 34.639 grammes ($1\frac{1}{5}$ oz.) pure cupric sulphate in 200 cubic centimeters of distilled water: 173 grammes (6 ozs.) of pure neutral sodio-potassic tartrate and 80 grammes of potassium hydrate in 500 cubic centimetres of distilled water. Add the copper solution slowly to the potassium one and dilute the clear mixture to one litre. One cubic centimeter of this fluid will be discolored by 0.005 gramme of sugar; or 200 grains will be discolored by 1 grain of sugar.

Trommer's test is even simpler for a mere qualitative test. Pour the suspected urine, freed from albumen, into a test tube and add a solution of caustic potassa or soda until distinctly alkaline. Should this throw down earthy phosphates or carbonates filter these out. Then add drop by drop a solution of pure cupric sulphate in distilled water (3.5:100) so long as it throws down a yellowish red precipitate of oxide of copper. When the supernatant liquid remains clear and assumes a distinctly bluish tint, the sugar has all been precipitated. The amount of precipitate is a criterion of the quantity of sugar, which may be otherwise estimated by the amount of copper salt used.

The fermentation test is made by adding a teaspoonful of liquid yeast to four ounces of the suspected urine, stopping the flask lightly and placing it in a temperature of 60° to 80° F. for 12 to 24 hours when the sugar will have been converted into alcohol and dioxide of carbon. The loss of weight will indicate the amount of sugar, as also will the lowering of the specific gravity. If before testing the urine was 1060, and after 1035, it contained 15 grains of sugar to the fluid ounce.

Prognosis. This is always rendered more grave in the horse than in man, because of the impossibility of putting him on a purely albuminous diet. The great tendency is to a rapidly fatal

issue, especially in cases of irremediable structural lesions in the brain and liver. Where the disorder is largely functional, as in connection with hæmoglobinuria or as the result of poisons ingested the prospect of recovery is often good.

Treatment. In cases due to poisoning the use of antidotes and eliminating agents will be effectual, and in transient and curable diseases like pulmonary disorder, hæmoglobinuria and paralysis the appropriate treatment will restore. In the more inveterate or constitutional cases all treatment is liable to prove ineffectual. At the outset some apparent amelioration may be obtained from salicylic acid, salicylate of soda, bicarbonate, acetate, citrate, sulphate or chloride of soda, nitro-muriatic acid and other cholagogues. Blisters to the perichondrium may also be employed. Later, when degeneration of the liver has reached an extreme point, these will be of no avail. Cadeac recommends acetanilid, antipyrine, and benzo-naphthol largely on their antiseptic merits, and Jong claims a recovery in a horse under daily doses of 12 grains of codeine. Opium has long been employed in man with partially good results, and croton chloral, strychnia, phosphoric acid, iodoform and ergot are recommended in different cases.

One of the most beneficial agents is skim milk or buttermilk as an exclusive diet, and this may be to a large extent adopted for the horse. Under its use the sugar may entirely disappear, and though rheumatoid pains in the joints may be brought on, these usually subside on withdrawing the source of lactic acid. They may further be met by the use of salicylates. The greatest care should be taken of the general health, an open air life, with protection against colds and storms, and a healthy condition of bowels, kidneys and skin being particularly important.

The impaired digestion and assimilation usually demand carminatives, stomachics, bitters, and mineral acids, particularly the nitro-muriatic. With the same intent a fair amount of exercise short of absolute fatigue should be secured. But each case will require a special study and treatment consonant to its special attendant lesions, its causative functional disorders, and its stage. One case may demand attention to bacteridian poisoning, one to a better regimen and diet, one to liver disease, and one to disease of the brain, etc. After this treatment specially directed to the abnormal function or structure, would come the more specific treatment for mellituria which would be more or less applicable to the general glycolytic disorder.

GLYCOSURIA IN CATTLE.

Accompaniment of parturition fever and apoplexy. Essential glycosuria. Dense saccharine urine, passed often, congested mucosæ, emaciation. Lesions uncertain. In parturition fever and apoplexy the congested medulla is the reasonable starting point. Toxic glycosuria. Treatment: addressed to the primary disease or poison; otherwise treat as in the horse.

In cattle this has been observed as a symptomatic affection in connection with parturition fever or apoplexy (Nocard, St. Cyr, Violet). One case of essential mellituria has been recorded by Darbas.

Symptoms. In the last mentioned case in a work ox, the animal, when at work, would stop every five or ten minutes to urinate, passing a small quantity of amber colored urine of a high density and containing a large amount of glucose. The conjunctiva was pink, the animal considerably emaciated, and rest and generous feeding brought about no improvement, so that the subject was finally sent to the butcher to anticipate a natural death.

The lesions in this case are altogether hypothetical. The red eyes might imply congestion of the encephalon (medulla), but the redness might be caused by active disease in the liver, pancreas or kidney. The failure to notice jaundice does not indicate a healthy liver, as some of the most fatal diseases of that organ are unattended by icterus. The frequent emission of urine in small amounts would imply irritation in kidneys or bladder, from which the glycogenic stimulus may have started. In the absence of any more definite evidence of disease in other organs it is, however, more probable that the fundamental disorder resided in the liver, the great glycogenic factor of the body.

In parturition fever, the presumption is in favor of considering the congested medulla as the starting point of the disease, yet in view of the manifest paralysis of stomach and bowels, it is not improbable that the vascular congestion and paralysis of the chylopoietic viscera constituted the initial step in the morbid process, while the glycogenesis was the result of a reflex operation on the liver.

Toxic mellituria would occur in cattle under the same conditions as in the horse.

Treatment is only hopeful in the sympathetic and toxic forms. These must be treated according to the nature of the primary disease or the poison. To these the general principles of treatment as recommended for the horse should be superadded. For essential diabetes an exclusively milk diet and any one of the agents that have given good results in man or horse can be tried, but with an animal in fair condition it will be better as a rule to turn him over to the butcher.

GLYCOSURIA IN THE DOG.

More common than in horses and cattle. Causes: pampered in diet, sweets, liver, disease of pituitary body, or mostly of the liver. Removal of pancreas. Brain and nervous lesions and reflex action. Symptoms: pampered asthmatic subject, with dysuria and lameness, dense, saccharine urine, bulimia, loss of weight, corneal ulcers, cataracts, palsy, coma. Duration: 4 to 8 months, sugar may disappear with complete liver degeneration. Diagnosis: by pampered condition, asthma, thirst, diuresis, later by loss of weight, troubles of vision, saccharine urine. Lesions: usually hypertrophied, fatty or caseated liver, thickened capsule, disease of thyroid, heart and eye. Treatment: skim or butter milk as sole diet, restricted diet of lean meat clear of fat, warmth, dryness, pure air, sunshine, gentle exercise only, cholagogues, sodium sulphate, or chloride, or carbonate, or salicylate, salol, nitro-muriatic acid, antithermics, ergot, codeine, bitters, mineral acids, derivatives.

Among domestic animals the dog has furnished the greatest number of cases, yet even in this animal the disease appears to be far from common.

Causes. The relative frequency of diabetes in the dog is probably dependent on his life in human dwellings and on gourmandizing on dishes prepared for man. Friedberger and Fröhner have produced the disease artificially by feeding a great quantity of sugar and W. Williams has met the disease in dogs fed exclusively and generously on liver. Thiernes records one case complicated by atrophy and steatosis of the pituitary body, but in all other instances the appreciable lesions were confined to the liver. In one case, reported by St. Cyr, the liver was hypertrophied, yellow, mottled, marked by irregular elevations of congested and hypertrophied hepatic tissue, and showing extensive degeneration—mucous, caseous and fatty. Thiernes found the liver of a yellowish white color, and the seat of fatty degeneration implicating the hepatic cells. Franzenberg in one case found fatty degeneration of the liver, and Fröhner and Schindelki, in four cases, met with extensive hepatic disease. The macroscopic lesions of the disease in dogs as in horses appear to be mainly hepatic.

On the other hand the complete removal of the pancreas in the dog by Mering, Minkowski, Thirolloix, Lancereaux and others was invariably followed by mellituria, so that even in the absence of clinical examples, we must recognize pancreatic lesions and functional disorders as possible primary causative factors in glycosuria. In the light of experimental medicine we must similarly recognize brain and nervous lesions and reflex actions as possible causes, even if as yet unsupported by clinical facts observed in the dog.

Symptoms. The disease usually appears in an old, fat, pampered dog, affected with dyspnœa or asthma, with dysuria and lameness. The urine is high colored, viscid, and of a high density (1055 to 1060, the normal canine urine being about 1020), and charged with glucose. The subject may have an enormous appetite but fails to gain in weight, and after a time loses flesh and becomes badly emaciated. The pulse is small and frequent, and the temperature which at the outset may reach 102°, falls to the normal as the end approaches. Watering eyes, corneal ulcers, and cataracts as well as hemiplegia and diabetic coma may precede death. The amount of sugar has been found to vary in different cases from 3.2 to 12 per cent. of the urine.

Course. Duration. The dog may live from four to eight months and, as in the horse, sugar may finally entirely disappear from the urine, in connection with the progressive degeneration of the liver. If the patient is unable to take exercise, the case reaches a more speedily fatal issue.

Diagnosis is deduced from the bulimia, pampered condition, breathlessness, thirst, and diuresis, the subsequent loss of condition, ocular troubles, and dropsy or coma, the whole being confirmed by the dense, high colored, viscous, saccharine urine.

Lesions. As already noticed the most constant lesion is hypertrophy of the liver, which is swollen unevenly, has thickened borders, is yellow or red, very friable and often fatty, or caseated. The capsule of Glisson is thickened and fibrous. Hypertrophy and fatty degeneration of the thyroid have been found in different cases and in one instance insufficiency of the mitral valve with systemic venous congestion. Ocular troubles are constant.

Treatment. Put the patient on an exclusive diet of skim or butter milk or if this is impossible it may be conjoined with lean meat—raw or cooked—keep warm and dry, but give plenty of open air and sunshine. Avoid fatigue and over exertion. Restricted diet is of great importance. During the

siege of Paris the short rations led to the disappearance of glycosuria from many human patients.

Among medicinal agents, cholagogues come first, sulphate of soda with chloride of sodium, bicarbonate of soda, salicylate of soda, salol, nitro-muriatic acid, may be named. For the febrile condition, antipyrine, acetanilid, or phenacetin may be employed, and iodoform, ergot or codeine may be tried when other measures fail. Bitter tonics and mineral acids may be beneficial, and lastly blisters to the region of the liver may prove of service.

OBESITY AND EMACIATION.

Sugar essential to growth. Ratio of liver to active increase in weight. Obesity and fatty degeneration. Small inactive liver and stunted growth, or loss of weight. Biliary fistula, death in 12 months. Influence of pancreas. Nitrogenous food for obesity, outdoor life, exercise, cholagogues, salines. Saccharine and starchy food for emaciation, hepatic, and pancreatic stimulants. Mild laxatives and cholagogues, pure air, green, succulent food, pure water, bitters, tonics, moderate exercise.

There is reason to believe that both of these occur as results of hepatic disorder. The generation of sugar in connection with rapid cell growth in germinating seeds and growing plants, and also in rapidly growing animal tissues as in the body of the foetus and foetal membranes, and finally in the inflammatory products of pneumonia and in leucocytes, seems to imply that it is essential to such cell growth (Murchison). In keeping with this is the fact that the liver is of relatively much greater size in the foetus and in the young and rapidly growing animal, and also in meat producing animals which have been selected and bred through many generations for early maturity and rapid fattening. The enormous development of adipose tissue and of lipomata in such animals is essentially abnormal, though it is a deviation from the natural that is esteemed as evidence of excellence, and a necessary condition of success in the meat producing industry. While other conditions are necessary to the production of such obesity, such as abundance of rest, slow, shallow breathing, a genial climate, and a generous hydro-carbonaceous food, yet all of these would prove ineffective without a large liver, working under high pressure in producing a large output of sugar. The mere obesity in the meat producing animal is not considered as disease and it is only when the tendency to fat production culminates in an adipose degeneration of the muscles and other tissues that actual disease is conceded.

Emaciation in certain cases is traceable to the opposite condition. A small or inactive liver with a diminished production of sugar and fat will ensure loss of weight, which is still further aggravated by decreased secretion of bile and insufficient absorption of peptones. Experimentally this condition has been repeatedly brought about by making a biliary fistula, and cutting off all bile from the intestine. Death preceded by extreme emaciation ensues in 12 months (Murchison). This being the case with the escape of all the bile secreted, a slower but no less certain emaciation must follow on a structural or functional disorder of the liver which is attended with a greatly lessened production of bile. This, indeed, is the condition met with in advanced glycosuria, when the liver is the seat of general fatty degeneration.

From the experiment of the removal of the pancreas we may infer that diseases of this organ which pervert or destroy its normal functions, will check glycogenesis in the liver by withholding the supposed pancreatic ferment, and by so doing will conduce to emaciation and marasmus.

So far as obesity and emaciation are dependent on diet they may be checked by subjecting the patient to the dietary which will favor a more healthy liver function. For excessive *obesity* a strictly nitrogenous food in restricted amount will tend to lessen the glycolytic action of the liver and secure the formation of muscle rather than fat. An outdoor life, and an active though not exhaustive use of the muscles will greatly favor this result. For the carnivora or omnivora a diet of lean meat or skim milk might be employed, while for the herbivora, wheat bran, cotton seed hulls, beans, peas, vetches, or cotton seed would measureably meet the demand. Cholagogues and saline laxatives, by eliminating from the liver and intestine, will contribute to the same end.

If *emaciation* depends on a deficiency of sugar, that may be freely fed along with richly amylaceous food, and the liver may be stimulated to increased glycogenesis, by stimulants such as chloroform, ammonia, or ether, and by a moderate use of carbonate of soda or other alkalies. Ether has in addition a stimulating effect on the pancreas and will tend to increase that ferment which stimulates the liver to its glycogenic work. Mild laxatives and cholagogues will second this, such as small doses of podophyllin, taraxacum, nitro-muriatic acid, chloride or bromide of ammonium, plenty of pure air, abundance of green or aqueous food, and plenty of pure drinking water. Finally moderate exercise, by increasing the aspiratory action of the chest and thereby accelerating the hepatic circulation is a material stimulant of the glycolytic function. Bitter and other tonics are contributions to the same object and should not be neglected.

SECONDARY OR REMOTE RESULTS OF LIVER DISEASE.

In gout: Arrest of oxidation of proteids into urea. Deposits of biurate of lime on joints, and other disorders. Urinary calculi containing urates, cystine, xanthine, etc., also from imperfect oxidation of albuminoids. Oxalic acid represents a similar arrest. Kidney degenerations from irritating urates and oxalates. Fatty kidney from excessive glycogenesis. Digestive disorders from excess or deficiency of bile or torpid liver. Nervous disorders, dullness, lameness, vertigo, spasms, irritability from hepatic inactivity and resulting poisons. Sore throat and bronchitis from hepatic derangement. Skin eruptions in tardy or imperfect action of the liver. Treatment: Abundant water, succulent vegetables, ensilage, fresh grains, balanced ration, in carnivora and omnivora oat meal, buttermilk, clear meat juice, avoid sweets, gravies, spiced animal food. Dangers for pampered horses, dogs, and old improved meat producing animals. Open air exercise. Laxatives with alkalies, salines, mercurous and mercuric chloride, pilocarpin, chlorides, iodides, bromides, nitro-muriatic acid, ipecacuan, euonymus, bitters.

Among the many secondary results of hepatic disorder, and which are habitually described as affections of other organs a few may be mentioned as indicating the wide range of influence exercised by the liver in disease as well as in health.

Gout as it appears in fowls and omnivora is directly due to the arrest of the transformation of the albuminoids into urea. Circulating in the system in the form of the less perfectly oxidized and less soluble uric acid, it determines deposits of biurate of lime around the joints, with local inflammations, and disorders of circulation and innervation, and altered spirit, temper, etc.

Urinary calculi in the same animals, are composed largely of urate of lime, cystine, xanthine and other nitrogenous products representing various stages of oxidation short of the final transition into urea and ammonia. Recognizing the active rôle which the urinary bacteria fill in this respect we must still acknowledge the great importance, as causative agents, of an excess in the urine of these comparatively insoluble products.

The oxalic acid found in certain calculi points in the same direction, as this acid, both in the body and in the laboratory, is found to result from the oxidation of uric acid (Wohler, Schenck, Hutchinson).

Degenerations of the Kidneys are to be largely traced to the same hepatic source. The uric acid diathesis, and the oxalic acid diathesis, both the result of imperfect liver function, are among the most frequent causes of irritation of the kidneys, by which channel they are eliminated from the body. Hence acute and chronic nephritis, as well as nephritic calculi result from morbid conditions which have their starting point in the imperfect function of the liver. Again fatty degeneration of the kidney is very liable to result from derangement of the glycogenic function of the liver, the tendency to the formation of fat and the constant irritation caused by the passage of the sugar contributing to the tissue degradation. In such cases albuminuria is a not uncommon accompaniment.

Derangements of the Digestive Organs may be said to be a necessary result of hepatic disorder. Excessive secretion of bile stimulates peristalsis and may induce diarrhœa, while diminished secretion tends to constipation, light colored, foetid stools, intestinal fermentations and poisoning by the irritant products. A torpid hepatic circulation means congestion of the whole portal system, indigestions, colics, chronic muco-enteritis, intestinal hemorrhages, hemorrhoids, etc.

Derangements of the Nervous System. In this connection may be named the lameness of the right shoulder which accompanies certain disorders of the liver, the extreme dullness and depression that attends on others, the sluggish pulse that appears in certain types, the unsteadiness of gait (giddiness) in others, the muscular cramps, and irritability in still others. These appear to be due in some instances to the nervous sympathy of one part with another, whilst at other times they as manifestly depend on the circulation in the blood of partially oxidized and other morbid products of hepatic disorder which prove direct poisons to the nervous system.

Derangements of the circulation, like extreme rapidity, or slowness of the pulse, irregularities in rhythm and intermissions, may be charged more directly on the nervous affection, though primarily determined by hepatic disorder.

On the part of the *Respiratory Organs*, affections of a chronic type, like sore throat and bronchitis may often be traced to hepatic torpor or disorder.

Skin Diseases are notoriously liable to come from inactive or disordered liver, the irritant products circulating in the skin or sweating out through it, giving rise to more or less irritation. The result may be a simple pruritus, an urticaria, an eruption of papules, vesicles or even pustules. In any such cases it is proper to look for other indications of liver disease,—pale color and offensive odor of the fæces, muco-enteritis, indigestion, icterus or yellow patches on the mucous membranes, tenderness on percussion over the asternal ribs, muscular neuralgia, nervous disorder, the passage of bile, hæmoglobin, albumen, sugar or other abnormal elements in the urine, etc.

TREATMENT OF SECONDARY AND FUNCTIONAL DISEASES OF THE LIVER.

Diet. Many hepatic disorders, and especially those that are exclusively or mainly functional may be corrected by diet alone. Prominent among dietary influences is the abundant supply of water. The succulent grasses of spring and early summer constitute the ideal diet, hastening and increasing elimination, and lessening the density of the bile, even to the extent of dissolving biliary calculi and concretions. Upon dry winter feeding such calculi are common especially in ruminants, whereas after a month or two at pasture they are extremely rare. In winter the same good may be arrived at by the use of ensilage, brewer's grains, roots, fruits, or even scalded hay or bran. The two extremes of highly albuminous and highly carbonaceous or saccharine food are to be avoided or used only in limited amounts. In the one class are clover, alfalfa, sainfoin, vetches, cowpea, lespedeza, especially in the form of hay, beans, peas, cotton seed, gluten-meal, rape and linseed cake. In the other are wheat, buckwheat, Indian corn, sorghum, sweet-corn and cornstalks. Some agents like beets which are rich in saccharine matter may be actually beneficial by reason of their laxative and cholagogue action. In the carnivora the food should be largely of simple mush of oatmeal, wheat seconds, or barley meal, skimmilk or buttermilk. If it is needful to tempt the appetite in a fleshfed animal this should not be done by rich, fat gravies, highly spiced animal food, or rich saccharine puddings, but rather by the addition of a little pure juice of lean meat, or some well skimmed beef tea.

It is as important to regulate the quantity as the quality of the food as the heavy feeder will overcharge the liver as much by an excess of otherwise wholesome food, as will the ordinary animal by the indigestible and unwholesome articles. As a rule the improved breeds of meat producing animals, have acquired such facility in fat production that much of the surplus is largely and profitably disposed of in this way, and in their short lives little obvious evil comes of the overfeeding, but in cases in which this outlet proves insufficient, as in horses and dogs that are highly fed on stimulating or saccharine diet, and which are kept for the natural term of their lives, with little exercise, the evil tends to reach a point of danger. Nursing mothers and dairy cows find a measure of safety in the free flow of milk and the yield of butter, but breeding cows that have been improved till they have no longer a capacity for milking, but must have their calves raised on the milk of other and milking strains are correspondingly liable to suffer.

Exercise in the Open Air. As enforced idleness, on a full diet and in a warm and moist environment is a main cause of hepatic disorder, so abundant exercise in the open air and especially in a cool season is beneficial in a marked degree. Beside the bracing effect on the digestive organs and the improvement of the general tone of the system, the action of the muscles in hastening the circulation greatly favors the removal and elimination of waste matters. Still more advantageous is the increased activity of the respiration and the aspiratory power of the chest in at once unloading the portal system and the liver by hastening the progress of the hepatic blood into the vena cava and right heart, and in furnishing an abundant supply of oxygen for the disintegration of the albuminoids and amylaceous products. Such exercise must of course be adapted to the condition of the animal and its power of sustaining muscular work, but judiciously employed, it is one of the most effective agencies in correcting and improving hepatic disorder or hepatic torpor. Idle horses, the victims of obstinate habits of constipation, muco-enteric irritation, indigestion, nervous, urinary or cutaneous disorders will often be greatly benefited or entirely restored by systematic exercise. This is one of the great advantages of a run at pasture, as the subject secures at once the laxative cholagogue diet, an abundant supply of oxygen, a better tone of the muscular and general system, and a more perfect disintegration of albuminoids. Sea air with its abundance of ozone is especially advantageous.

In the carnivora while we cannot send them to grass, much can be done in the way of systematic exercise, and in the case of city dogs a change to the country, where they can live out of doors and will be tempted to constant exercise and play, will go far to correct a faulty liver.

Laxatives. Cholagogues. When a free action of bowels and liver cannot be secured by succulent food and exercise, we can fall back on medicinal laxatives. These are advantageous in various ways.

Some laxatives like podophyllin, aloes, colocynth, rhubarb, senna, jalap, and taraxacum act directly on the liver in increasing the secretion of bile. These may be used for a length of time in small doses and in combination with the alkalies. Other aperients act directly on the bowel carrying away the excess of bile, the albuminoids and saccharine matter that would otherwise be absorbed, and by a secretion from the portal veins, abstracting nitrogenous and saccharine elements which would otherwise overtax the liver to transform them. Thus indirectly these also act as cholagogues by withholding the excess of material on which it has to operate, and by rousing its functions sympathetically with those of the bowels. Thus sulphates of magnesia and soda, and tartrates and citrates of the same bases, given in the morning fasting, dissolved in a large quantity of warm water and conjoined with sodium chloride, ammonium chloride, sodium carbonate or other alkaline salts, or with one or more of the vegetable cholagogues above mentioned, may be continued for a length of time until the normal functions have been re-established, and will maintain themselves irrespective of this stimulus.

Calomel (and even mercuric chloride in small doses), though it is not experimentally proved to be a direct cholagogue, is one of the very best correctives of impaired hepatic function. It expels the bile from the duodenum and bowels generally, thereby preventing its reabsorption; it proves antiseptic to the ingesta; it eliminates much of the peptone, saccharine and fatty matter from the intestines and portal system thus relieving the liver materially; and it is supposed further to modify the other liver functions by a direct action on the hepatic cells, and by reducing the cohesion of fibrine, and promoting the disintegration of albumen. Certain it is that calomel gives most substantial relief in many torpid and other disorders of the liver and as it is not in itself an active liver stimulant but has rather a soothing action on that gland it can be safely resorted to in states of hepatic irritation in which the more direct cholagogues would prove more or less hurtful.

In some forms of hepatic disorder where a speedy and abundant secretion is demanded, pilocarpin may be employed, with great caution so as not to reduce the strength unduly by the attendant diaphoresis, diuresis, salivation or diarrhoea.

Alkalies have long been recognized as of great clinical value in hepatic disorders. Though carbonate of soda decreases the secretion of bile, (Nasse, Röhrig), yet the alkalies generally appear to promote oxidation, and to hasten the disintegration of albumen and the albuminoids. They increase the disintegration of sulphur compounds materially adding to the sulphates and urea in the urine. They further tend to increase the hippuric acid, carbonate of soda (2 drs.) even determining the abundant excretion of this acid in man (Nasse). It may be concluded that the acknowledged value of alkalies in these diseases, is largely due to their hastening of the metabolic processes in albuminoids. Small doses of sodium carbonate further stimulate the gastric secretion and may thus benefit by rendering the process of digestion more complete and satisfactory.

Chlorine, Iodine, Bromine and their Salts. These halogens are of great value in many hepatic disorders. The universal craving for sodium chloride indicates the need of its elements in the animal body, and whether this is mainly the supply of chlorine for the hydrochloric acid of the gastric juice, or to fulfill its uses in favoring the oxidation and disintegration of the nitrogenous matters in the blood and tissues, or for other more or less obscure uses, it is well to recognize and act upon the indication. The various mineral waters which are held in high esteem in liver affections contain a large proportion of sodium chloride. As a medicinal agent ammonium chloride maintains an equally high position. Large doses thrice a day, so as to induce diaphoresis and diuresis greatly relieve hepatic congestions. This agent determines a great increase in the urea eliminated so that it is even more effective in the same direction, than sodium chloride. Free chlorine is also effective in hepatic torpor and congestion, and to this in part may be attributed the great value of nitro-muriatic acid.

Bromide and iodide of potassium have been found to be effective in reducing hepatic enlargement and thus in conducing to a more healthy activity of the liver.

Ipecacuanha, Euonymus, etc. These agents are more or less hepatic stimulants and may be found beneficial as combined with the laxative or alkaline agents in securing a better functional activity in cases of torpor or deranged function.

Tonics, Bitters. Tonics are often useful when the health has been undermined by long continued

hepatic disorder. The iron tonics are as a rule contraindicated as tending to check secretion of bile, unless they can be given with alkalies. Iron sulphate or chloride, combined with sodium or potassium carbonate so as to establish a mutual decomposition will obviate this objection. The vegetable bitters (gentian, cascarilla, calumba, salicin, serpentaria, aloes, nux vomica) combined with alkalies are often of great value. Quinia, like opium, checks secretion and is to be avoided or used with judgment and in combination with cholagogues.

HÆMOGLOBINÆMIA. AZOTÆMIA. AZOTURIA. HÆMOGLOBINURIA. TOXÆMIA FROM IMPERFECT HEPATIC FUNCTION.

Definition. Theories, of hysteria, uræmia, spinal myelitis, myelo-renal congestion, rheumatic lumbago, myosito-myelo-nephritis, rheumatic chill with destruction of muscle albuminoids. Yet it occurs in our semi-tropical midsummer with a temperature of 80 or 90, in spring and autumn, and rarely even in the cold, damp stable in midwinter in the absence of exercise. Constant conditions: One or more days absolute rest, preceding steady work, a strongly nitrogenous ration, continued during the rest, sudden active exertion accelerated breathing and unloading of peptones and proteids from portal vein and liver into the general circulation. Sanguineous albuminuria from excess of albuminous food, free ingestion of water, suppressed milk secretion, forced marches. Transfusion of blood. Excess of albumen dangerous, excess of red globules not dangerous. The blood concentration of diuresis or diaphoresis is not dangerous. Continuous muscle decomposition from work bars the disease. Stable miasm untenable. Poison may be drawn suddenly from the enormous mass of blood in the liver, spleen and portal system. The absence of icterus antagonizes the bile theory. Benzoic acid, unaltered peptones, and glycogen are examples of elements destructive to blood. Normal destruction of red globules in liver, spleen and bone marrow. Sudden access of resulting hæmoglobin to the blood. Other products of disintegrated globules. Poisons from food, and antitoxic action of liver in presence of glycogen. Carbon dioxide favors solution of red globules. Theories of hæmoglobinæmia in man. Lesions: Blood black, diffuent, iridescent, has no avidity for oxygen, with excess of urea and extractives, serum of clot red, globules, small, pale, distorted, not sticky, extravasations, liver, enlarged, congested, blood gorged, spleen congested, swollen: Lumbar or gluteal muscles pale, infiltrated, with loss of striation; bone marrow congested, hemorrhagic; kidneys congested infarcted; urine dark brown or red, with excess of urea and hæmoglobin. End of spinal cord has congestion or infiltration. Symptoms: History of high condition, constant work, high feeding, a day's rest, then exercise and attack. To full life, follows flagging, droops, moves one or both hind limbs stiffly, knuckles, drags toes, crouches, trembles, perspires, breathes rapidly, is tender on back, loins, croup or thigh, muscles firm, parietic, and drops unable to rise. Urine retained, brown, red or black, sometimes glairy, later may have casts. Appetite may return. In mild cases, stiffness, lameness, with or without visible muscular lesions or tremors. Urine glairy, dense, with excess of urea and nitrogenous products. Recover under careful feeding and exercise, and relapse under original causes. Progress: May recover under rest. In bad cases accelerated breathing and recumbency forbid rest and recovery. Recovery in a few hours or after a week. Urinary casts with renal epithelium, imply nephritis and grave conditions. In persistent paresis, muscles waste. Modes of death. Mortality 20 per cent. Diagnosis, by history of onset, etc. Prevention: When highly fed and hard worked, give daily exercise, with comparative rest, reduce ration, and give laxative or diuretic. Plenty of water. Treatment: Rest, sling, diffusible stimulants, bleeding, bromides, water ad libitum, fomentations, unload liver and portal vein, purgative, eserine, barium chloride, enemata, diuretics, for remaining paresis, derivatives, strychnia, diet, laxative, non-stimulating, restore to work gradually.

Definition. An acute auto-poisoning occurring in plethoric horse on being subjected to active exertion after a period of idleness, and manifested by great nervous excitement and prostration, paresis commencing with the hind limbs and the passage of hæmoglobin in the urine.

Nature and Causes. The most varied conclusions as to the nature of this disease have been put forward by different authors. In England, Haycock called it hysteria, mistakenly supposing that it was confined to mares, and Williams attributed it to uræmic poisoning, conveniently ignoring the fact that the sudden manifestation of the most extreme symptoms in an animal which just before was in the highest apparent health and spirits contradicted the conclusion. In France (Trasbot) and Southern Europe (Csokor) it has been looked on as a spinal myelitis, a conclusion based on the disturbed innervation of the posterior extremities in the great majority of cases, but which is not always sustained by the pathological anatomy of the cord. In Germany veterinarians have viewed the disease from widely different standpoints. Haubner calls it myelo-renal-congestion (Niëren-Rückenmarks): Weinmann, a rheumatic lumbago; Dieckerhoff defines it as an acute general disease of horses, manifested by a severe parenchymatous inflammation of the skeleton muscles, with a bloody infiltration of the bone marrow, especially of the femur, and with acute nephritis and hæmoglobinuria. He attributes the attack to exposure to cold. If this were the real cause the attack

would be far more common in very cold weather when the horse is suddenly exposed to cold drafts between open doors and windows, than when he is harnessed and driven so as to generate and diffuse animal heat. Yet attacks in the stable are virtually unknown, and in almost every instance the onset occurs during a short drive. Friedberger and Fröhner say that the epithet rheumatismal may be correctly applied to almost all cases that we meet in practice. They quote Goring as having produced the disease experimentally by exposure to cold, and go on to explain that rest in the stable before the attack causes the extreme sensitiveness to cold that is generated by a warm environment. The implication of the lumbar, pelvic and femoral muscles they explain by the stimulation of the nutritive metamorphosis by the action of cold on the sensitive nerves of the skin. The effect of this cutaneous irritation is exaggerated by the heat of the stable to which they have been previously subjected. The products of the destruction of the albuminoids of the muscles, pass into the blood as hæmoglobin, and produce the ulterior phenomena. The muscles of the hind quarters especially suffer because of their greater exposure and because they are subjected to the hardest work in propelling the animal machine. In this connection they quote the experiments of Lassar and Nassaroff in which sudden exposure to cold determines parenchymatous degeneration of muscles; also the cases of paroxysmal or winter hæmoglobinuria in certain susceptible men whenever they are exposed to an extremely low temperature.

There are serious objections to the acceptance of this as the essential cause, among which the following may be named:

1st. The disease is not confined to the cold season but occurs also at midsummer when the outdoor temperature is even higher than it is in the stable.

2d. In our Northern States it appears to be more common in spring and autumn or early winter, when the extreme colds have either already passed, or have not yet set in, but when the abrupt changes of weather (rain-storms, etc.) are liable to shut up the animal indoors for a day or more at a time.

3d. The popular names quoted with approval by these authors—Monday disease, Easter disease, Whitsuntide disease—indicate the prevalence in Europe also, of the malady in the milder, or more temperate seasons rather than during the prevalence of extreme cold.

4th. The fact that the disease rarely or never occurs in the stable, no matter how cold the season, how open the wooden walls or floor, nor how strong the draft between doors or windows, shows that the theory of cold as the sole or main cause must be discarded.

It is not necessary to ignore the action of cold as a concurrent factor in certain cases, or as a stimulant to reflex vaso-motor paresis, to muscular metamorphosis and the increase of hæmoglobin in the blood. It is only necessary that this should be held as subordinate and non-essential to the final result. Several other factors that are accorded a subordinate place by these writers, are so constant and so manifestly essential that they must be allotted a much more important position in the list of causes.

A **period of rest** is a constant precursor of an attack. The more extended the inquiry the more certain we become that a short rest is a prerequisite to equine hæmoglobinæmia. The horse that is kept at daily steady work may be said to be practically exempt. Even the non-professional observer recognizes the fact and names the disease after the weekly or yearly holiday or rest day which was the occasion of it. To him it is the Monday morning disease, the disease of the day following Thanksgiving, Christmas, New Year, or Fourth of July. It is the disease of wet weather, of heavy snowfalls, of the blizzard, or of the owner's absence from home, of any time that entails one or two days of absolute inactivity in the stall.

But again the affection does not appear in the horse that is absolutely idle for a length of time. It is the **short period of rest in an interval of otherwise continuous work** that determines it. In short the subject must be in good muscular condition and with a hearty, vigorous appetite and good digestion. The short unwonted rest interrupts the disposal of the rich products of a vigorous digestion, and tends to overload the portal veins, the liver, the blood and tissues with an excess of proteids. The condition of the animal is so far one of plethora.

Another feature that bears this out is that the attack comes only in the animal that is **heavily fed**

on a strongly nitrogenous ration. It is not the disease of the horse kept on straw, or hay, or which receives a limited amount only of grain. It does not occur in the animal which has its grain suspended or materially reduced during the one or two days of idleness. It does not select the horse that has had a laxative either in the form of food or medicine. This last may increase the sensitiveness to cold, but it certainly lessens the tendency to hæmoglobinæmia. The most rational explanation appears to be that it affords this protection by interfering with the thoroughness of digestion and absorption, by securing elimination from the portal veins and liver, and by reducing the amount of albuminoids in the blood.

A blood abnormally rich in albuminoids, as it is in the transient plethora induced by a short period of rest, in the well-conditioned working horse, without any restriction of his diet, may therefore be set down as one of the most important factors in producing hæmoglobinæmia. Nor is this without approximate examples in human pathology. Von Bamberger has shown that "hæmatogenous albuminuria" will occur in healthy individuals when there is an excess of albumen in the blood-plasma, as after a too free use of albuminous food, or after suppression of the milk secretion (Landois). A similar result comes from increase of blood pressure, as after drinking freely, or when, under emotion or violent exertion, the heart's action is increased in force and the blood is thrown with greater impetus into the large renal arteries. Senator has found albuminous urine to attend and follow, for several days, upon forced marches made by young recruits. Here the muscular work is added to the increased blood tension superinduced by the more active contractions of the heart.

In this connection it is interesting to trace the changes in the blood after transfusion. The dilatability of the capillaries enables the system to accommodate itself to a very great increase in the volume of blood. An increase of 83 per cent. may be borne without serious results, but above this limit there is increasing risk and an increase of 150 per cent. entails immediate danger to life. In the restoration of the blood to its normal condition, the secretion of water sets in promptly leaving an excess of albuminoids and blood globules. The next change is in the albuminoids which in two days are almost entirely transformed into urea. This leaves the blood abnormally rich in globules (Panum, Lesser, Worm-Müller), the red globules break up much more slowly and may still be in excess after the lapse of a month (Tscherjew).

In this light, temporary plethora cannot of itself be accepted as the main or essential cause of the disease. It must be admitted to be a more constant and important factor than the mere exposure to cold, but of itself it is inadequate to the production of hæmoglobinæmia. In the absence of exertion the general plethora fails to produce the specific disease; again, after transfusion a plethora of albumen lasts for one or two days, but hæmoglobinæmia sets in only in the first few minutes after the animal starts out from the stable, (never after an hour or two at work): once more, excess of globules may last for a month, but with steady work there is no danger of this disease, after the first mile or two has been traversed, on the first day of the resumption of labor.

A similar plethora of albuminoids and globules may be induced in a plethoric animal by a profuse diarrhœa, diuresis or perspiration, the blood having been robbed of its watery constituents, and concentrated especially as regards its globules and albuminoids, but hæmoglobinæmia never occurs as the result of such an artificial concentration. On the contrary a free secretion by the bowels or kidneys is of the greatest value in cutting short its progress after it has set in.

The doctrine of poisoning by hæmoglobin produced by excessive work and disintegration of the muscles is equally insufficient to account for an attack. Excess of muscular work and of muscle-decomposition-products, would not reach its maximum within the first few minutes after the animal has started from the stable, but, other things being equal, would increase with the continuance of work and the accumulation in the blood of a constantly increasing amount of these products. The sharp line of restriction by which the attack is limited to the initial period of work, while it is never seen after hard work continued for hours in succession, rules out this from the list of essential causes. It may be that the products of muscular decomposition aggravate the attack, but to set them down as the cause of the attack is to beg the whole question and to contradict the truth that continuous and severe muscular work with its consequent increase of waste products is a direct bar to the development of the disease. It should be noted in this connection that the increase

in the waste of nitrogenous bodies, as shown by the increase of urea, is dependent far more on the amount of nitrogenous matters ingested than on the muscle work or decomposition. In eleven hours just before ascending the Faulhorn, Fick passed 21.686 grs. of urea per hour; in eight hours ascending the hill, 12.43 grs. per hour; and in six hours after the ascent he passed 13.39 grs. per hour.

A general survey of the field shows that it is not the simple increase of any normal waste product in the blood which determines hæmoglobinæmia, and on the other hand the suddenness and severity of the attack bears all the marks of a profound poisoning. The nature of the poison has not yet been definitely ascertained, yet one or two hypothesis may be hazarded, as furnishing a working theory, in anticipation of the actual demonstration which may be expected in the early future.

The action of a *stable miasm* as claimed by some writers is contradicted by the fact that the disease does not develop so long as the animal is left to inhale that *miasm*, and on leaving the stable, the life and vigor are usually remarkable.

The morbid agent must be sought in some source from which it can be supplied with great rapidity under the stimulus of a short but active exertion. The chylopoietic viscera furnish such a source. The healthy liver contains one-fourth of the entire mass of the blood. The torpid congested liver of the vigorous high conditioned horse, after a short period of idleness, on full, rich feeding, must hold much more than this normal ratio. The spleen, the natural store-house or safety valve of the portal veins, is also gorged with this liquid in the high fed, idle animal. This organ which is always turgid after meals, is especially so in the over-fed horse, which for twenty-four hours has been denied the opportunity of working off by exercise, the superfluous products of an active digestion and absorption. Then the whole of the portal veins and the capillaries in which they originate are surcharged with rich blood which cannot make its way with the necessary dispatch through the inactive liver.

In this condition there is incomparably more than a quarter of the entire mass of blood, enriched to the highest degree in proteids, ready to be discharged through the liver and hepatic veins into the general circulation. Under the action of the hurried breathing and circulation, caused by the sudden and active exertion, this whole mass of rich blood is speedily unloaded on the right heart, the lungs and the systemic circulation. One can hardly conceive of a more effective method of inducing a sudden plethora, with an excess of both globules and albuminoids.

The presence of actual poisons in such blood is not so easily certified.

The absorption of *bile* elements and especially of taurocholic acid, which is a solvent of the red blood globules, and would set free their globulin might account for the characteristic condition of the blood. The powerful aspiratory action of the chest, would speedily empty the whole of the liver blood vessels, and lessening their tension below that of the biliary radicals would determine an active absorption of bile or of the more diffusible of the bile elements. A manifest objection to this view is the absence of an icteric tint in the mucous membranes of the affected animals. The visible mucosæ are of a brownish red hue, such as might come from hæmoglobin dissolved in the blood serum, rather than the yellow tint which might be expected from bile pigment. The theory of poisoning by bile acids therefore, would require an explanation of concurrent suppression or decomposition of the bile pigments.

Other sources, however, offer solvents for hæmoglobin, benzoic acid, which is derived from a cellulose in the fodders, and forms the source of hippuric acid, dissolves red globules (Landois). In the over-fed horse with active digestion, but inactive body and liver, this must accumulate in the liver, spleen and portal system, and when suddenly drawn into the blood without time for oxidation in the liver it will contribute to the condition of hæmoglobinæmia.

Peptones, being very diffusible, are very rapidly absorbed, but they are not found, in healthy conditions, in the portal vein (Neumeister). These are manifestly transformed into albumen in the intestinal mucosa (Salvioli), or taken up by the very numerous leucocytes and transformed or carried elsewhere (Hoffmeister). But peptones injected into the blood of the dog render it incoagulable, and in large quantity are fatal (Landois). An excess of glycogen dissolves the red globules, and the conditions of heavy feeding and torpid liver, are calculated to produce this in

great excess and to store it in the liver cells.

Under the extra vigorous aspiratory force of the chest, these highly diffusible agents, present in great excess, are likely to be drawn on through the mucosa, into the portal vein, liver, and cava, without an opportunity for complete transformation by leucocytes or liver cells. These would tend to rob the blood globules of their normal physiological vigor, would unfit them for maintaining the healthy functions of lungs, kidneys, brain or muscle, and would unfit the globules for successful resistance to solvents and other inimical influences.

Again it is an important function of the liver, spleen and red bone marrow to disintegrate worn out or abnormal red globules. These are taken up by the white blood corpuscles of the hepatic capillaries, by the cells of the spleen and the bone marrow and are stored up chiefly in the *capillaries of the liver*, in the *spleen*, and in the marrow of bone. They are transformed, partly into colored and partly into colorless proteids, and are either deposited in the granular form, or are dissolved (Landois). Quincke says: "That the normal red blood globules and other particles suspended in the blood stream are not taken up in this way, may be due to their being smooth and polished. As the corpuscles grow older and become more rigid, they, as it were, are caught by the amœboid cells. As cells containing blood corpuscles are very rarely found in the general circulation, one may assume that the occurrence of these cells within the spleen, liver, and marrow of bone, is favored by the slowness of the circulation in these organs." From this chain of normal processes of blood disintegration, we may reasonably infer, a greatly exaggerated work of blood destruction when, in connection with an increased density of the plasma, and the presence in the portal blood of poisonous products of digestion, the red globules have been altered in density, in outline and in vitality, so that they become ready victims of the amœboid cells of blood and tissues. Then the stagnant condition of this altered blood in the compulsorily idle animal favors the greatest excess of this destruction and the storing up of an increased quantity of hæmoglobin and other products, to be poured suddenly into the general circulation as soon as the movement of the blood is quickened by exercise.

This destruction of the red blood globules by disintegration contributes to the formation of numerous decomposition-products, like succinic, formic, acetic, butyric and lactic acids, inosite, leucin, xanthine, hypoxanthin, and uric acid, some of which are strongly toxic. The tendency will be to lower the vitality of the red globules and thus to render them the easier victims of the leucocytes and of the liver, spleen and marrow cells. Even the freed hæmoglobin appears to exert a solvent action on the red blood globules. These are, of course, most concentrated and effective in the seat of their production, yet when drawn suddenly in large amount, into the general circulation, by the vigorous aspiratory action of the chest, they may prove seriously detrimental to the blood at large.

Again a variety of toxic matters are introduced into the system in the food and others are developed from the food in the stomach and intestine. Brieger found in the gastric peptones a potent alkaloid having the effect of urari, and which in excess would determine muscular paralysis. The alkaloidal and other poisons produced by fermentations in the intestines have to be safely disposed of. The ptomaines, if not too abundant, are arrested or even decomposed in the liver which thus stands as a guardian, at the outlet of the portal system, to protect the body at large. But this antitoxic function of the liver is only exercised in the presence of glycogen (Rogers, Landois), and forced muscular movement soon removes all glycogen from the liver of the dog (Landois). Again glycogenesis in the liver is now believed to be dependent on a ferment produced by the pancreas. If therefore, the sudden active exercise and the aspiratory action of the chest freed the liver of its glycogen, and hurried the alkaloidal and other poisons through its capillaries too rapidly to allow of the protective action of the liver cells, or if the pancreas as well as the liver had become torpid and had failed to produce the requisite amount of glycogen-ferment for the liver, the poisoning of the blood and system at large would be imminent.

Not to mention the other toxic products which come from imperfect metamorphosis in the liver, it may be noted that a venous condition of the blood or an excess of carbon dioxide contributes greatly to the solubility of the red blood globules. It also tends greatly to modify the fibrinogenous elements. Thus the blood of a suffocated animal fails to coagulate or coagulates loosely, and the blood of the portal vein of a suffocated horse is strongly toxic (Sauson). Now the conditions

attendant on the onset of equine hæmoglobinæmia are such as to give free scope to both of these inimical influences. The great mass of blood in the portal vein, spleen and liver is venous blood strongly charged with carbon dioxide, and by the sudden, active exertion this is forced rapidly through the liver and lungs without time for full æration, so that the whole mass of the circulating blood is speedily reduced below par, and laid specially open to the action of blood solvents. By the same action the systemic blood is charged with poisons, direct from the food, and fermenting ingesta, and from the overworked spleen and liver whose functions are profoundly impaired, and later from other important organs, the healthy functional activity of which can no longer be maintained by the deteriorated blood supplied to them.

Hæmoglobinæmia in dogs has been produced experimentally by the injection of water into the veins the mere dilution of the plasma dissolving out the coloring matter from the red globules (Hayem); also by the inhalation of arseniureted hydrogen (Naunyn and Stadelman); by the ingestion of toluylendiamine, or phosphorus (Afanassiew, Stadelman); by snake venom, septicæmia, influenza, contagious pneumonia, petechial fever, anthrax, etc. These cannot be looked on as causes of the acute hæmoglobinæmia in the horse, but they serve as illustrations of changes in the plasma, and poisons in the blood determining the escape of hæmoglobin from the cells.

Ralfe recognizes two forms of hæmoglobinæmia in man:

1st. That in which the hæmoglobin is simply dissolved out of the blood globules, the solution taking place chiefly in parts exposed to cold.

2d. A more severe form in which the dissolution is general and probably attended by some destruction of red globules in the liver, spleen and even in the kidneys. The general opinion appears to be that the attacks are due to some nervous disturbance, which causes vaso-motor disorder and it is supposed that there is an exaggerated sensibility of the reflex nervous system. It has been suggested that peripheral irritation causes irritation of the vaso-motor centre, and in turn this causes local asphyxia in the part stimulated, under which conditions the red globules part with their hæmoglobin (Roberts).

Murri holds that the disease depends on an increased irritability of the vaso-motor reflex centre, and the formation, owing to the disorder of the blood forming organs, of corpuscles unable to withstand exposure to cold or carbon dioxide.

While it is not assumed to point out the actual poisons of hæmoglobinæmia in the horse the above suggestions may offer valuable hints as to the lines of inquiry that may be followed with the best hope of reaching definite results.

Lesions. These are especially found in the blood, liver, spleen, muscles, bone marrow and kidneys. The spinal cord and nerve trunks are occasionally affected.

The *blood* is charged with carbon dioxide and is black, tarry, comparatively incoagulable remaining in the veins and showing an iridescent reflection. It does not absorb oxygen readily though exposed to the air, and thus bears a strong general resemblance to the blood of anthrax. It contains an abnormal proportion of urea and allied extractive matters which greatly increase its density, and interfere with the healthy exercise of the different cell organisms and functions. These are not due to excessive muscular activity as stated by Friedberger and Fröhner, but are derived mainly from the abundant products of digestion. When the shed blood coagulates it forms a soft clot without buff and the expressed serum is reddish from the presence of hæmoglobin, and of hæmatoidin crystals. The uncoagulated blood drawn over a sheet of white paper stains it deeply by reason of the same coloring matters in solution. The red corpuscles may be paler than natural, some even entirely colorless, and they are often notched or broken up in various irregular forms. They have lost the natural tendency of the shed equine blood to stick together, to collect in rouleaux and precipitate to the bottom of the vessel, so that no buffy coat is formed, should the blood coagulate. The white corpuscles are relatively increased. Finally the coloring matters contained in the plasma are imbibed by the different tissues and give a brown or reddish tinge to such as are naturally white. Limited blood extravasations are not uncommon especially in the more vascular organs like the muscles, liver, spleen and kidneys.

The *liver* is more or less congested and enlarged, friable, yellow, or mottled yellow and red and

exudes black blood freely when incised. The bile is thick, viscid and dark green, as in cases of experimental intravenous injection of hæmoglobin.

The *spleen* is also swollen and congested with blood, and the pulp is very high colored from the excess of hæmoglobin and other products of blood destruction. The *muscles of the croup* are usually the seat of visible lesions. There may be pallor, œdema and swelling, but not unfrequently there are blood extravasations varying in size from a pin's head upward and giving a dark red aspect to the affected tissues. Under the microscope the affected fibres are seen to have lost their transverse striation and to have assumed a more or less granular or hyaline appearance. Next to the gluteal muscles, these changes are frequently found, in the muscles of the thigh (especially the rectus femoris, and triceps extensor cruris), and in those of the loins (psoas, ilio-spinalis, and longissimus dorsi). Exceptionally the pectoral muscles are involved or even the abdominal muscles. A considerable straw-colored œdema may be found in the intermuscular connective tissue.

The *red bone marrow* primarily of the large bones of the limbs (femur, tibia, humerus, radius,) and less frequently of other bones, even of the vertebræ, is often the seat of intense vascular congestion and even of hemorrhage. The medullary matter is of a deep red or black color, and there is an abnormal accumulation of red globules in various conditions of growth and destruction (red nucleated corpuscles, fragments of corpuscles, colored granules). Dieckerhoff considers the condition one of osteomyelitis, but it seems to be rather a sudden, extraordinary exaggeration of the processes of blood metamorphosis. Neumann found that when the blood regeneration process is very active even the yellow marrow may be changed into red, and this throughout all the bones of the extremities.

The *kidneys* are usually the seat of congestion, and black spots of infarction, when the disease has lasted for twenty-four hours. In rapidly fatal cases they may appear normal. There may be enlargement of the kidneys with softening and granular degeneration of the renal epithelium in cases that survive for some days.

The *bladder* contains dark brown or red glairy urine of a high density and loaded with urea, hæmoglobin, etc.

The *terminal portion of the spinal cord* and the lumbo-sacral plexus, or some of its branches, are sometimes blood stained, or the seat of an exudate or surrounded by one.

Symptoms. In the regular type of hæmoglobinæmia in the horse the history of the attack is highly significant. The subject is in good working condition, he may be fat, or lean, but in either case the muscles are firm and well developed, diet has been liberal, embracing a large proportion of albuminoids, work has been constant up to within a day or two preceding the attack, when the animal has been left absolutely idle in the stall without any reduction of feed. Then finally it has been suddenly subjected to active exertion which demands vigorous muscular movement, and above all activity of the respiratory muscles and the heart. This exertion usually consists in riding under the saddle or going in harness, but may attend on casting in the stall, lounging in a ring, or in a playful run when suddenly set at liberty.

Severe Cases. The attack comes on early in the course of such exercise. The patient may not have gone more than one hundred yards from the stable or he may have traveled for half an hour or an hour, but the disease rarely shows itself after a longer period of work.

The horse which left the stable full of life and spirit, suddenly flags and hangs on the bit, the ears or head may drop, and one or more limbs usually the hind ones, are moved stiffly and awkwardly, or even stagger. He knuckles over at the fetlocks, drags the toes on the ground, flexes the joints imperfectly, the muscles appearing to be rigid and uncontrollable, or he crouches, the joints remaining semiflexed the animal in vain attempting to extend them. The patient trembles violently, sweats profusely, breathes deeply and rapidly and assumes a pinched, anxious, agonized expression of countenance. The heart beats tumultuously, the pulse (in 84 per cent. Friedberger and Fröhner) is accelerated to a variable degree, and the temperature is still normal (in 80 per cent. Friedberger and Fröhner), or rarely exceeds 101.5°F. There is often tenderness on percussion and sometimes even on manipulation over the loins, short ribs, and the croup, and pinching of the loins may cause wincing. The affected muscle or muscles (lumbar, gluteal, crural) are usually firm, hard

and tender, they may be the seat of spasm or of œdema and paresis. These parts may, however, have their sensitiveness lessened and even punctures or electric currents may have little effect on them.

Soon the increasing muscular weakness is incompatible with the maintenance of the standing position, the bending of the limbs and crouching become extreme, the animal makes vain efforts to control the muscles and extend the joints, and helplessly drops to the ground. When down he moves his legs convulsively, but is unable to coördinate the muscular movements and all efforts to rise are unavailing.

The spasms and paresis may attack other parts of the body such as the pectoral region the shoulders and even the abdomen, but the earliest and most persistent disorder is usually in the divisions of the lumbo-sacral plexus affecting the supra or sublumbar muscles, the gluteals, the patellar (triceps,) the adductors and the abductors. The caudal muscles are exceptionally involved. In a series of ten cases Bouley noticed that the left hind limb was always the first paralyzed (evidently a simple coincidence).

Urine may be passed freely or the bladder may be paretic so that it must be emptied with the catheter. In severe cases the urine is of a high density and of a dirty brownish gray, red or almost black color. It contains no blood clots, nor blood globules, but granular hæmoglobin, tyrosin and other waste products contribute to produce the reddish color. In some instances there is an abundant *metalbumen* which renders the liquid glairy, causing it to fall in fine threads or films. Urea is usually present in great excess. Hippuric and even uric acid are usually present but not in excess. When the disease has advanced to nephritis the albuminuria is complicated by the presence of casts of the uriniferous tubes, renal epithelium, white and even red blood globules.

During the violence of the attack there is no disposition nor leisure to eat, but when the more violent symptoms abate appetite is usually manifested. There may be more or less paresis of both bowels and bladder, so that neither fæces nor urine is passed yet in other cases both are discharged spontaneously.

The senses are preserved, excepting in the case of the affected muscles and the integument which covers them. There may, however, be more or less dullness and stupor in certain cases from poisoning of the cerebral centres by the poisons circulating in the blood.

Mild Cases. In the mildest cases there is stiffness and lameness in one, or less frequently in both hind limbs, coming on when put to work after a period of idleness, and not associated with any appreciable lesion of the limb in question. There may or may not be hardness and swelling of the gluteal or other muscles of the quarter or loins. This has the appearance of rigidity or spasm but may be primarily due to œdema or exudation into the substance of the muscle. In some instances the muscles of the breast, shoulder, or forearm are the seat of the trouble. Muscular trembling and perspiration may be present and if the urine is examined, it is often found to be glairy, or charged with urea, and allied nitrogenous products. These cases are not benefited by local applications, but they recover (temporarily) under rest and above all under active eliminating treatment. Under gentle and progressive exercise too they improve and get well. They recur, however, with great readiness under a rich nitrogenous diet and a temporary rest followed by sudden exertion.

Between the mildest and gravest cases there are infinite gradations of severity, one-third to one-half of the worst cases usually terminating fatally, whereas the mildest are always amenable to treatment.

Progress. The course of the disease depends on the severity of the attack but also, in no small degree, on the good judgment of the driver. Cases that develop with great suddenness, and apparently with extreme severity may subside spontaneously if the animal is placed in a condition of absolute rest. If, however, we can secure rest of the muscles of progression only, while the breathing remains rapid and labored, improvement is unlikely, as the system continues to receive large accessions of the toxic products. When the patient is down and unable to rise, the enforced rest may be beneficial, but too commonly, the greater effort with which breathing is carried on in the recumbent position, and the frequent ineffectual struggles of the limbs prevent the requisite muscular quietude.

In some cases, and especially in the mildest, recovery may seem to have been effected in a few hours, and in others it will be seen in twenty-four or forty-eight hours, while in still others the paresis and helplessness may continue for a week and yet be followed by recovery. In these cases appetite may be retained in greater or less degree, but the intestinal peristalsis is usually weak and imperfect, the fæces small in quantity and dry, and the bladder atonic so that the urine may have to be drawn off with the catheter. It usually retains the deep red color, or improvement may be heralded by a change to a dirty grayish hue. If, however, it shows an excess of albumen, cylindroid casts entangling renal epithelium and white or red globules it will indicate the access of diffuse nephritis and a prolonged or even a fatal illness.

When control of the limbs is not restored at the end of a week, the paretic muscles usually undergo marked and rapid wasting, which may last for months or years. This is especially common in the case of the patellar muscles (muscle of the fascia lata, triceps extensor cruris) in which the atrophy may become so extreme that the skin covering the inner and outer sides of the thigh may be brought virtually in contact in front of the femur. This entails an almost complete inability to sustain the body on the hind limbs. When atrophy is less extreme, there is only a weakness, stiffness, or swaying or staggering on the hind limbs in progression.

In fatal cases death may occur early in connection with the violent struggles, the excited breathing, pulmonary hypostasis and congestion, a cyanotic hue of the visible mucous membranes and a gradual increase of stupor. Though delayed for several days, there is a continuation of the muscular struggles, and the labored breathing; the red or glairy character of the urine persists or is exaggerated; the nervous irritability increases, with muscular trembling; and cyanosis, or stupor increases until death.

The *mortality* is always high in the severe forms of the disease, the deaths ranging from 20 per cent. upward.

After a first attack there is a strong predisposition to a second under similar exciting conditions.

Diagnosis. The peculiar symptoms of this disease and the circumstances attending its onset, are usually sufficient to distinguish it from all others. There may be danger of confounding certain cases with thrombosis of the posterior aorta, or of the iliac arteries or their branches, but the absence, in such cases, of the special history of the attack and of the morbid state of the urine, and the absence of pulsation in the arteries distal to the thrombosis will serve to prevent confusion. Spinal myelitis will be distinguished by the gradual nature of the onset, by the absence of the conditions attending on the attack of hæmoglobinæmia, and usually by the absence of hæmoglobin, urea and other nitrogenous products in excess in the urine.

Prevention. The hard worked or systematically exercised horse, which is at the same time heavily fed must not be left in a state of absolute rest in his stall for twenty-four hours. A fair amount of exercise must be given on every day in the week, and at the same time, the food should be restricted in ratio with the restriction of exercise. Turning for an hour or two daily into a yard may be a sufficient precaution. When from any cause, rest is imperative, the diet must be materially reduced and given in part in a laxative form (bran, roots), or a slight laxative (Glauber salts) or diuretic (saltpeter) may be added. Cleanliness and a free ventilation of the stable, are also of value in obviating at once auto-intoxication and the admission of poison through the lungs. In the same way a free allowance of drinking water is beneficial as favoring a general elimination from the various emunctories, and a dilution of the plethoric blood.

These precautionary measures are especially important in the case of horses which have passed through a first attack and which are in consequence strongly predisposed to a second. Horses fed liberally on highly nitrogenous food (oats, beans, peas, cotton seed meal), will also require specially careful oversight when at rest for a day or two only.

Treatment. The first and perhaps the most important consideration is absolute rest. If the subject is stopped instantly on the appearance of the first symptoms, the disease may be often aborted. It is better to avoid the exercise of walking to a stable until such time as the severity of the attack has somewhat moderated and then to move the subject only in the slowest and quietest possible way. If the patient is already down and unable to rise, he may be carried to the nearest

stable in an ambulance or on a stone-boat, and there helped to his feet and supported in slings. Though he may be unable to continue in the standing position without the sling, yet if he can use his limbs at all for support, and is prevented from lying down, the breathing will be rendered so much more free and quiet, that it may greatly lessen the transfer of the poisonous elements into the general circulation and materially contribute to recovery. If, however, he cannot stand on his limbs at all, but must settle in the slings, the compression of the chest will so excite the breathing that it will induce dyspnoea, pulmonary congestion and a rapidly fatal result. In such a case a good bed must be provided and the patient made as comfortable as possible in the recumbent position.

In some cases in the earliest stages a full dose of sweet spirits of nitre or even half a pint of whiskey has seemed to assist in aborting the disease though the urine was already of a deep red color. It probably acted by supporting the already oppressed heart, and securing a prompt elimination by the kidneys.

Friedberger and Fröhner strongly recommend bleeding in all cases of dyspnoea and excited heart action, and considering the plethoric condition of the animal it would equally commend itself in other cases as well. This is the most prompt sedative of the nervous and vascular excitement, and the most speedy and certain means of removing much of the poisons accumulated in the blood, and of diluting what remains by reason of the absorption of liquids from every available source. This will more than counterbalance any temporary increase of poisons drawn from the portal system to fill up the vacuum in the systemic veins caused by the emission of blood. When the thick tarry condition of the blood seriously hinders a speedy abstraction both jugulars may be opened at once.

In some cases of great nervous excitement bromides may be useful in moderating circulatory and respiratory movement, but on the whole the advantage is greater from an immediate resort to eliminating agents.

One of the most effective agents is water. If the patient is thirsty he should have all he will drink, and if not, it may even be given from a bottle, or thrown into the rectum. A still more effective resort would be to introduce water intravenously in the form of a normal saline solution, or even to pass it into the trachea through a small cannula or large hypodermic needle. This serves to dilute the over dense blood, to stimulate the kidneys and other emunctories to active secretion, and to retain in solution the hæmoglobin, urea and other products which would otherwise cause greater irritation. This would be especially applicable after the blood tension had been diminished by phlebotomy.

Warm fomentations to the loins or croup are not without their influence. They tend to soothe the irritated parts and to solicit the action of the kidneys more particularly. The old resort of a fresh sheep skin, with the fleshy side in, may be used as a substitute.

Perhaps the most important indication is to secure depletion from the overloaded portal system and liver. Where nothing better offers, a pint or quart of castor oil, or a pound of Glauber salts, or a half drachm of podophyllin and four drachms of aloes may be given. If available 1 to 1½ grains of eserine, or 7 grains of barium chloride may be given hypodermically in distilled water or that which has been raised to the boiling point. This may be supplemented by frequent injections of hot soap suds or even of laxative saline solutions. If the bowels can be roused to free secretion the removal of toxic matters from the portal blood and the delay in the progress of similar matters through the liver will go far toward securing a favorable result. When free purgation has been secured recovery can usually be counted on.

The action on the bowels must be followed up by diuretics to eliminate the offensive matters from the general system. Colchicum has been recommended because of its action in increasing the solids of the urine, and this may be combined with saltpeter or other diuretic, or the latter may be used alone and repeated twice a day. If, however, the patient can, by the free use of common salt or otherwise, be induced to drink freely of water, the elimination through the kidneys will be sufficiently secured.

The muscular weakness and paralysis that remain after the acute symptoms have subsided must be met by stimulating liniments and even blisters to the loins or affected muscles, by the internal use of strychnia (2 grs. twice daily) until the jerking of the muscles indicates that its physiological

action has been secured, and by an electric current daily for ten minutes at a time through the affected nerves and muscles. Animals that have been helpless for weeks have, in our hands, recovered under such treatment, and even cases of several months' standing, with the most extensive atrophy of the triceps, and in which the animal could barely stand, have made a satisfactory recovery.

Any remaining nephritis must be treated according to its indications.

During recovery and in the convalescent animal the diet should be laxative and non-stimulating. Bran mashes, turnips, beets, carrots, green fodder, ensilage and scalded hay may be allowed. Oats, corn, beans, peas, vetches, etc., must be carefully avoided. If the food fails to maintain the bowels in a gently relaxed condition one, two or more ounces of sulphate of soda may be added daily.

In the mild cases a good dose of purgative medicine succeeded by a course of diuretics will serve a good purpose.

In all cases alike work must be resumed very gradually. At first the animal may be walked a few hundred yards, and the pace or load and duration of exercise may be increased day by day until full work can be safely endured. In an animal that has once suffered the same gradual inuring to labor should be followed, after any short period of rest on a fairly good ration.

JAUNDICE, ICTERUS, THE YELLOWS.

Symptomatic. Causes: Mechanical obstruction of bile duct, gall-stones, hydatids, distomata, extraneous bodies, inflammation, stricture, obliteration, absence, ulceration, spasm, tumor, enlarged lymph glands, gastric tumors, pancreatic, kidney or omental tumor, aneurism, fæcal accumulation, pregnancy, ovarian tumor: Without mechanical obstruction, ptomaines and toxins, animal venoms, mineral poisons, hepatic atrophy, fear, other emotions, cerebral concussion, imperfect oxidation, excess of bile, hepatic inflammation, constipation and reabsorption of bile, experimental jaundice, balance of tension in gall ducts and blood vessels, duodenitis, compression of aorta, hæmatoidin and bilirubin, destruction of blood globules by hydroæmia, taurocholate of soda, chloroform, ether, freezing, heat, electricity, alkalies, nitrites. Hæmoglobin: Its solubility in horse. Bile acids and blood pigment. Summary of causes. Gravity of icterus. Symptoms: Coloration, yellow, orange, brown, of tissues and secretions: Tests, staining white paper, Gmelin's test, nitric and sulphuric acids, rainbow hues: Pettenkofer's test for bile acids, syrup and sulphuric acid, dark violet: Stranburg's test syrupy paper and sulphuric acid, dark violet; clay colored fœtid stools; gravity.

The terms icterus and jaundice are applied to a yellowness of the mucosæ, urine, skin and tissues caused by the presence in them of the coloring matters of bile. The condition is a symptom of many different affections rather than a disease *per se*, yet the phenomenon is so characteristic that it has been hitherto accorded a special place and article in systematic works.

Jaundice is either associated with *mechanical obstruction of the bile duct* or ducts, or it is *independent of such obstruction*. The following enumeration of its causes slightly modified from Murchison, is equally applicable to the lower animals as to man:

A. Jaundice From Mechanical Obstruction of the Bile Duct.

I. Obstruction by foreign bodies within the duct:

1. Gall stones and inspissated bile.
2. Hydatids and distomata.
3. Foreign bodies from the intestines.

II. Obstruction by inflammatory tumefaction of the duodenum or of the lining membrane of the bile duct with exudation into its interior.

III. Obstruction by stricture or obliteration of the duct.

1. Congenital deficiency of the duct.
2. Stricture from perihepatitis.
3. Closure of the orifice of the duct in consequence of ulcer of the duodenum.
4. Stricture from cicatrization of ulcers in the bile duct.
5. Spasmodic stricture.

IV. Obstruction by tumors closing the orifice of the duct or growing in its interior.

V. Obstruction by pressure on the duct from within, by:

1. Tumors projecting from the liver itself.
2. Enlarged glands in the fissure of the liver.
3. Tumor of the stomach.
4. Tumor of the pancreas.
5. Tumor of the kidney.
6. Post peritoneal or omental tumor.
7. An abdominal aneurism.
8. Accumulation of fæces in the bowels.
9. A pregnant uterus.

10. Ovarian and uterine tumors.

B. Jaundice Independent of Mechanical Obstruction of the Bile Ducts.

I. Poisons in the blood interfering with the normal metamorphosis of bile.

1. The poisons of the various specific fevers (Anthrax, Texas fever, Hog cholera, Swineplague, Petechial fever, Pyæmia, Septicæmia, etc.).

2. Animal poisons: snake poison.

3. Mineral poisons: phosphorus, mercury, copper, antimony, etc.

4. Chloroform, ether, etc.

5. Acute atrophy of the liver.

II. Impaired or deranged innervation interfering with the normal metamorphosis of bile.

1. Severe mental emotions: fright, anxiety, etc.

2. Concussion of the brain.

III. Deficient oxygenation of blood interfering with the normal metamorphosis of bile.

IV. Excessive secretion of bile, more of which is absorbed than can undergo the normal metamorphosis.

Congestion of the liver: *a.* Mechanical, *b.* Active, *c.* Passive.

V. Undue absorption of bile into the blood from habitual or protracted constipation.

Mechanical obstruction, by tying the bile ducts in a dog, caused in two hours yellow coloration of the contents of the hepatic lymphatics and thoracic duct, and also of the blood in the hepatic veins (Saunders). That this jaundice is due to reabsorption and not to suppressed secretion of bile, already present in the blood, may be fairly inferred, from the complete absence of icterus, where, from general disease of the liver, the secretion of bile has been entirely suspended, and in which the gall ducts and bladder contain only a little gray mucus (Haspell, Frerichs, Budd, Murchison), also from the fact that after complete extirpation of the liver in frogs not a trace of biliary acids nor pigment can be detected in the blood, urine, or muscular tissue (Müller, Runde, Lehmann, Moleschott). Bile acids and bile pigment are formed in the liver by disintegration of blood globules, and when present in excess in the blood it is by virtue of reabsorption.

This reabsorption will take place under the slightest favoring influence. The obstructions in the bile duct, above referred to, cause the tension in these ducts to exceed that of the blood in the capillaries of the liver and at once osmosis of bile into the blood vessels sets in. This may occur from so slight a cause as the congestion and swelling of the duodenal mucosa around the opening of the bile duct. Again reabsorption of bile may be determined by a lessening of the normal fullness and tension of the hepatic capillaries as when the aorta is mechanically compressed by abscess, neoplasm, ingesta, or otherwise, just behind the diaphragm (Heidenham, Brunton). The cause is the same in both cases, namely, the want of balance between the fullness and tension of the bile ducts, and the hepatic blood vessels. There is increased fullness of the hepatic biliary ducts, or decreased plenitude of the hepatic capillaries and lymphatics.

It must be added, however, that the coloring matter of the bile is apparently produced, in the liver, from that of the blood, and that the pigment (hæmatoidin), found in old extravasations of blood, is probably identical with bilirubin, and that any agent or condition which causes liberation of the coloring matter of the red blood globules, will cause a staining of the tissues, like that of jaundice. The following agents are known to have this effect on the blood globules: water, in hydroæmic states of the blood (Hermann); taurocholate of soda from absorption of bile (Frerichs, Kuhne, Feltz, Ritter); chloroform (Chaumont); ether (Burdon-Sanderson); freezing (Rollet); a high temperature +60° C. (Schultze); frictional and induction currents of electricity (Burdon-Sanderson); the alkalies (ammonia, potash and soda) and nitrites when present in excess.

The injection of hæmoglobin into the veins of dogs has been followed by the appearance of bile pigment in the urine, but Naumyn, Wolff, Legg and Brunton failed to obtain the same result in rabbits.

It is noticeable that the hæmoglobin of horses' blood is very soluble at all temperatures and that of dogs very slightly so (Burdon-Sanderson). This may serve to explain the great prevalence among

solipeds of diseases, associated with dusky brown or yellow discoloration of the mucosæ, with petechiæ, and with the passage of blood pigments in the urine. It may further explain the usually benignant course of jaundice in the horse and its extreme gravity in the dog.

There is further reason to believe that the bile acids, when in excess, may be transformed into bile pigment in certain conditions of the blood, as occurs under the action of sulphuric acid out of the body (Stœdler, Meukomen, Folwarcyny, Röhrig). Moreover, in the healthy state, the greater part of the bile secreted, including acids and pigment, is re-absorbed from the intestinal canal, but is oxidized and decomposed in the blood so that it cannot be detected, in blood or urine. But let the transformation be interrupted, as in certain diseases of the lungs, with imperfect oxidation, and the bile circulates in the blood, stains tissues and urine, and in short causes jaundice.

To sum up: it may be said that icterus is probably never due to simple inactivity of the liver: it may, however, be caused by excessive secretion of bile which is re-absorbed from obstructed bile ducts or bowels:—it may result from imperfect transformation, in the blood, of the bile which is normally re-absorbed from the intestine: or it may possibly be caused by the formation of pigments in the blood from the abnormal transformation of bile acids, or by solution of the hæmoglobin of the blood corpuscles.

The gravity of jaundice varies as much as its causes. It is well known that the system may be saturated with bile, and the tissues and urine deeply stained without much constitutional disorder. The pigment alone is not an active poison. But there may be much attendant suffering from obstructed biliary ducts or bowels, from diseases of the lungs, or from disintegration of the blood globules and imperfect nutrition, or there may be profound nervous prostration and disorder from uræmia, or from the presence in the blood of an excess of effete and partially oxidized albuminoids (See Azotæmia). According to our present knowledge, constitutional disorder, prostration and suffering in cases of jaundice, are mainly due to the presence in the circulation of these albuminoids, and of taurocholic acid which latter has a most destructive effect on the blood corpuscles.

The *symptoms*, therefore, are not characteristic apart from the yellow coloration of the tissues and urine and the chemical reactions of the bile acids and bile pigments furnished by the latter.

The coloration of the tissues may be a simple tinge of yellow especially noticeable in the eye (conjunctiva), or it may amount to the darkest shades of orange and brown. It may or may not be complicated by the presence of spots or patches of blood-staining (ecchymosis) on the visible mucous membranes but especially in cases complicated by poisoning with taurocholic acid or effete nitrogenous products.

The urine may be similarly colored in all shades of yellow or orange brown, and may leave a correspondingly deep stain on white paper.

The *test for bile pigments* (Gmelin's) is simple and beautiful. Pour a little nitric acid into a test tube held obliquely and then add a few drops of sulphuric acid, and finally a little urine, so slowly, that it will remain on the surface. Soon at the point of junction appear in succession the various colors of the rainbow: yellow, green, blue, violet, red and lastly a dirty yellow. It is open to this objection that the characteristic play of colors may be produced by alcohol in the absence of bile pigments. Indican also will produce the green and yellow with blue between but never the violet nor red, nor all in their regular order.

A *second mode* of applying this test is by spreading a few drops of the urine on a white plate and letting fall a drop of nitric acid in the centre. The play of colors is very characteristic.

The *test for bile acids* (Pettenkofer's) is to place a portion of the urine in a test tube, and after adding a drop of syrup, to add cautiously, drop by drop, two-thirds of the amount of sulphuric acid. Shake the mixture and set aside for some minutes. If sufficient heat is not produced by the mixing of the acid and urine warm slightly. The mixture becomes of a dark violet color which is destroyed by a temperature a little above 140° Fah.

A convenient application of this test (Stranburg) is to add a little cane sugar to the urine, dip a piece of filtering paper in the mixture, dry it thoroughly, pour a drop of sulphuric acid on the paper and allow it to run partially off. In a quarter of a minute a beautiful violet color is produced, best

seen by holding up the paper to the light and looking through it (Brunton).

In cases due to obstruction of the bile ducts the dung is destitute of bile, whitish, often clayey and foetid, while in cases due to reabsorption without obstruction the fæces have their natural color and odor.

It is needless to enumerate all the concomitant symptoms of jaundice which will be better noticed under the different disorders which determine it, for a list of which see the *causes*.

The gravity of the affection will depend on the dangerous nature of these concurrent diseases, and the destructive changes in the liver and blood rather than on the depth of color in the textures.

CATARRHAL ICTERUS (JAUNDICE) OF SOLIPEDS.

Causes: infection from duodenum through biliary duct. Suppression of bile favors. Musty, heated, mow burnt fodder, over feeding, irregular feeding, or watering, over work, worms, fatigue, damp stables, duodenal congestion, gall-stones, concretions, pancreatic tumor, ascaris in bile ducts, distoma, infection through portal vein, toxins. Symptoms: of duodenal catarrh, icterus, yellow, viscous, odorous urine, dullness, weakness, somnolence, tardy pulse and breathing, costiveness, or diarrhoea, pale, foetid stools. Duration: 2 to 3 weeks or longer. Lesions: duodenitis, distended biliary and pancreatic ducts, calculi, enlarged softened liver and kidneys. Diagnosis: icteric symptoms in absence of fever. Prognosis: usually favorable. Treatment: laxative diet, pasture, soiling, ensilage, roots, fruits, water freely, exercise, antisepsis, elimination, laxatives, cholagogues, diuretics, calomel, salines, nitro-muriatic acid, podophyllin, castor oil, aloes, tartar emetic, bitters, sodium bicarbonate.

Causes. This may be said to be an extension of infection from the duodenum through the bile ducts. The microbes of the intestinal canal become acclimatized by living in the bile-charged contents of the duodenum until they acquired the power of survival and multiplication in the biliary ducts themselves. The well known antiseptic qualities of the bile, constitute a powerful barrier to this, yet the power of adaptation on the part of certain germs is greater than the defensive action of the bile. The attack is however mostly in connection with indigestion or muco-enteritis, and a more or less perfect suspension of biliary secretion, so that this defensive action is reduced to its minimum and the germs can ascend the bile ducts in the mucous secretion as a culture medium, and by interference with the resumption of a free hepatic secretion, they succeed in safely colonizing themselves in the mucosa and hepatic parenchyma. Whatever, therefore, interferes with the integrity of the duodenal functions directly contributes to the extension of infection from bowel to liver. Old, heated, musty, cryptogamic, dusty fodder, grains that have been badly harvested in wet seasons, feed that has been damp and fermented, overloading of the stomach, irregular feeding and watering, giving drink after a feed of grain, underfeeding, overwork, worms, excessive fatigue, damp, dark stables, etc., tend to induce indigestions and to lay the bile ducts open to infection. Blocking of the bile duct and stasis of its contents may be a sufficient cause. The swollen mucosa around the orifice of the duct not only blocks the passage but favors the formation of a mucous plug as recorded by Benjamin of an equine patient. Wolff found obstruction of the duodenum in the horse by a mass of ingesta, and blocking of the gall duct, with jaundice.

Gall stones and concretions are very direct causes of biliary obstruction and jaundice. Though less common in horses than cattle, these are not unknown in idle, pampered animals when on dry winter feeding.

Tumors of the pancreas or adjacent organs pressing on the gall duct are recognized as causes of equine icterus, (Megnin, Nocard).

With any obstruction to the bile a disturbance of balance of pressure between the bile ducts and the hepatic veins is brought about by respiratory movements. On the one hand the aspiratory power of the chest empties the hepatic veins, lessening blood pressure, and in expiration the contraction of diaphragm and abdominal muscles compresses the gall ducts increasing their tension and favoring absorption of bile.

The entrance into the bile duct of the ascaris megaloccephala is at once a cause of obstruction and of the transference of duodenal microbes, and the presence of trematodes (*fasciola hepatica*, or *distoma lanceolatum*) will also favor obstruction. Other parasites, like the echinococcus or actinomycosis, may press on the biliary ducts and determine jaundice.

Another mode of infection is by way of the portal vein, the microbes entering from the intestine and becoming arrested and colonized in the liver (Dieckerhoff).

Whether from the presence of the microbes or from the absorption of ptomaines and toxins from the intestines, the radical biliary ducts become inflamed, swollen, and even blocked, and the hepatic cells degenerated or even completely devitalized, so that they fail to take an aniline stain. In such cases the remaining sound hepatic cells go on producing bile, but as this cannot any longer escape through the partially obstructed interlobular biliary radicles, it is largely absorbed and produces icterus. Cadeac mentions a case of this kind in a mare in which the toxic matters had not only led to hepatic disease, but also to structural changes in the eliminating organ (the kidney).

Symptoms. In the horse the disease is mostly attendant on subacute duodenitis, and even when this is associated with infective catarrh of the biliary passages the kidneys remain mostly sound and active, and eliminate alike the bile pigments and the more toxic matters so that the disease is not often grave. Beside the essential feature of yellow mucosæ, and urine, the latter viscous and smelling strongly, there is profound depression, sluggishness, weakness and somnolence. Imperfect muscular control and even slight paresis may be present. Tardy pulse and breathing are at times noticeable. At others these, like the temperature, are normal. The mouth is hot and dry. The urine may be slightly albuminous. The bowels incline to costiveness from lack of their customary stimulus, yet this in turn may give rise to diarrhœa. In either case, as the disease advances, the defecations lose the healthy yellowish brown color, becoming pale and foetid.

Duration. The attack may last one, two or three weeks, and generally ends in recovery. With irremediable structural lesions, it is of course permanent and even fatal.

Lesions. The most common feature is duodenitis with thickening around the orifice of the common bile and pancreatic duct. The biliary ducts may be distended and their contents more than usually viscid and glairy from the presence of pus. Their mucosa may show ramified redness, or concretions as casts or calculi. The liver is enlarged, soft and friable giving way readily under the pressure of the finger. Enlargement of the kidneys is usually present, the cortical substance having a brownish red and the medullary portion a yellowish pink hue.

Diagnosis. The absence of hyperthermia in jaundice, serves to distinguish it from the acute febrile affections (pneumonia, influenza, contagious pneumonia, petechial fever, etc.,) which are marked by yellowness of the mucosæ and skin.

Prognosis. The merely functional forms of icterus in solipeds usually end in recovery.

Treatment. The first consideration is a laxative diet. A run at pasture will usually meet every indication. Fresh cut grass, ensilage, turnips, carrots, potatoes, beet, apples, or other succulent diet may be given as substitute. Bran mashes and hay cut and moistened may be allowed in the absence of the above. Abundance of water and especially cool water will stimulate bowels, liver and kidneys, favor the elimination of the bile by contraction of the biliary ducts, and hasten the expulsion of the poisons through the kidneys. Regular exercise an hour after meals stimulates both bowels and liver to action.

Medicinal treatment is largely directed to antiseptics of the bowels and the arrest of the production of injurious toxins; elimination from the bowels and incidental depletion from the portal vein and liver; antiseptics and stimulation of the liver; and stimulation of the urinary secretion.

The preparations of mercury fill several of these indications. Calomel 2 drs., or blue mass 1½ dr., is not only a soothing laxative and antiseptic, but seems to operate as a calmate and antiseptic to the liver as well. It may be continued in 5 to 10 grain doses two or three times a day, according to the size of the animal and the condition of the bowels, and associated with ½ dr. belladonna extract to each dose together with a bitter (quassia, gentian, nux vomica). Or 4 or 5 ozs. sulphate of soda may be given three times a day, with 2 drs. salicylate of soda as an antiseptic. Or, to increase the hepatic action, nitro-muriatic acid largely diluted may be given in sixty drop doses thrice a day in the drinking water. These are especially valuable for their antiseptic action, cutting off at once the source of nervous irritation from the attendant indigestions, and duodenal congestion, and arresting the flow of the irritant toxins and other products through the portal system. Podophyllin, castor oil, aloes, rhubarb, often act well by depletion from the portal vein, and expulsion of indigestible and irritant matters from the intestines, but there is more danger of resulting swelling of the duodenal mucosa than with the mercurials or aqua regia. Goubaux recommends 2½ drs. of tartar emetic.

Siedamgrotzky has had good results from an electric current sent through the region of the liver, but in the horse this is rarely demanded.

A course of bitters, with bicarbonate of soda in small doses, may be demanded to re-establish the healthy tone of the stomach and intestines, and a run at pasture, or at least an open air life, exercise, and a laxative diet with abundance of good water should be secured. Any undue

costiveness should be counteracted at once by a saline laxative.

CATARRHAL ICTERUS (JAUNDICE) IN DOGS.

Pampered artificial life of dogs as predisposition. Eating carrion. Chills especially when heated and exhausted. Infection from bowels. Obstruction of gall duct by inflammation, calculi, neoplasms. Catarrhal exudate as a protector of microbes. Toxins from intestines, food or water. Youth, lack of acclimation, mental shock, blocking of bowel, blood effusion in gall bladder, incubation. Symptoms: signs of gastro-enteritis, prostration with invasion of the liver and especially of the kidney. Icteric urine without jaundiced mucosa. Gravity of icterus with suppression of urine. Hypochondriac tenderness, arched back, dullness, irregular bowels, excited circulation and breathing, preliminary fever tends to subside, tympany, colic, trembling. Death in one or two days or more. Lesions: congestion, degeneration, ecchymosis, ulceration of gastro-duodenal mucosa, extending into liver ducts and acini, bile inspissated, liver enlarged, yellowish brown, softened, fatty, shrunken, distorted hepatic cells. Kidneys congested, ecchymosed, cortical part with necrotic foci; lymph glands congested. Diagnosis: by icterus of tissues and urine tests. Prognosis: grave in acute cases, more hopeful in tardy ones. Treatment: antiseptics, cholagogues, salol, salicylates, alkalies, carbonates, tartrates, iodides, laxatives, cold enemata, aloes, electricity, water freely, pilocarpin, strychnia, aqua regia, digitalis, bitters, muriatic acid, convalescent diet.

Causes. The dog is much more subject to jaundice than the horse, and the affection is liable to be much more severe, than in solipeds. He leads a more artificial life, especially in cities, where the lack of open air exercise, and of the facility for attending to nature's wants, together with an excessive, varied, stimulating diet predisposes him to constipation, indigestion, and disorders of the stomach, bowels and liver. In other cases the devouring of decomposing food and foul water proves a cause of direct microbial infection, and of poisoning by ptomaines and toxins generated out of the body. Exposure manifestly has something to do with the prevalence of canine icterus, which is more common in spring and especially in autumn than at other seasons. In hunting dogs, out of condition, the suddenly induced over-exertion and fatigue, and the succeeding chill in cold air or water, become accessory factors.

It appears to be most commonly the result of the transference of germs from the intestine, either by way of the bile ducts, or with the blood through the portal vein. The first form is usually the sequel of muco-enteritis affecting the duodenum, with swelling of the walls of the common bile and pancreatic duct at its orifice, or from obstruction by gall-stones, concretions, impacted bowels or neoplasms. With the arrest of the biliary flow the intestinal ferments gain an entrance into the common duct and the sac of Vater, finding protection from the antiseptic bile in the resulting catarrhal exudate, and in this way they reach the gall-bladder, the biliary radicles and the acini. With the entrance of bacteria or toxins by the portal vein on the other hand, there is first a troubled condition of the acini and hepatic cells, an over-secretion of thick bile, and blocking of the passages so that little is passed into the intestine, the greater part being absorbed into the hepatic veins. Fermentation microbes in the stomach and intestines, the germs of suppuration and septicæmia, and saprophytic germs from outside the body are held to be causative of icterus. Cadeac lays much stress on the putrefactive germs in water, and traces different attacks to marshes and foul ponds.

As in other infecting diseases, early age has a predisposing influence. The older subject has presumably been already exposed to the microbe and acquired some measure of immunity. Animals coming new to the locality and poison, are equally susceptible with the young. Trasbot found that 14 out of 17 dogs thus attacked were between three and eighteen months.

Leblanc and Trasbot claim mental shock as a cause of icterus in the dog. The disappointment and weariness caused by the master's absence, the excitement of a fiercely contested fight, and brutal punishment are adduced as cases in point. Abuse of emetics and purgatives, in connection with a pre-existing hepatic or duodenal disorder or as a supposed prophylactic of canine distemper has induced jaundice.

Obstruction of the small intestine has proved a factor, partly by the reflex irritation through the splanchnic nerves, and partly through obstruction to the common bile and pancreatic duct.

Walley records a case of obstruction of the cystic duct by extravasation of blood in the gall bladder.

Icterus not infrequently supervenes during canine distemper in which the early gastric and duodenal irritation becomes an occasion of the extension of the catarrhal infection to the common

bile duct. Even apart from this Trasbot has seen the majority of cases ushered in by a gastro-duodenitis. In this connection it is interesting to quote the remark of Pfuhl that 26 out of 27 persons using the foul baths of a given establishment contracted icterus, while the soldiers bathing in another branch of the Elbe entirely escaped.

The fact that icterus usually sets in several days after such an exposure, whether in man or dog, indicates a period of incubation, and thereby sustains the theory of infection.

Symptoms. In the majority of cases, gastro-enteritis constitutes the first step of this affection and the early symptoms are characteristic of that disease. Frequent vomiting, at first of food, and later of a glairy fluid which may be discolored by bile, redness of the tongue, especially along its margins, bloodshot, watery eyes, lying down with the nose on the right side of the abdomen, or standing with the abdomen tucked up and the back arched, halting movements of the limbs, wincing if manipulation is made of the spine or the epigastrium, elevated temperature, accelerated pulse and breathing, anorexia and perhaps purging, mark the onset. So long as the liver is unaffected prostration is not a marked feature.

Even when the liver is invaded, a fair measure of life and activity may often be retained so long as the kidneys remain sound and active. The bile pigments and salts, and the toxins generated by the invading microbes are alike eliminated in large part in the urine, and the profound poisoning and prostration of the nervous system are in a measure prevented. In many cases therefore the urine is strongly jaundiced without much or any discoloration of the mucosæ and in such cases the prospects are usually good for an early and complete recovery. It must not, however, be inferred that such a happy issue will always follow, as the pigments are by no means as toxic as are other hepatic products and fatal results may ensue with very slight jaundice of the tissues.

As a rule, however, the jaundice of the tissues (eyes, nose, mouth, white portions of the skin) is to be accepted as a grave manifestation, indicating either an excessive production and absorption of bile, or a suppressed secretion through the kidneys, or both. Then the poisoning by biliary salts and bacterial toxins is shown in profound dullness, prostration, muscular weakness, indisposition to rise, moving stiffly and only when dragged by the collar, the limbs trembling and the back arched. The tender back is arched, the right hypochondrium sensitive, the expression dull, stupid and listless, and constipation or a foetid diarrhœa is liable to set in. The heart beats may be strong and irregular, the breathing is easily disturbed and hurried. The temperature at first 104° to 107° may descend to the normal as the system becomes charged with the toxic products, and does not usually exceed 99.5° after two or three days of jaundice of the tissues.

The icterus is shown in the eye or mouth, or on any white portion of the skin, and in the urine it will be detected by the eye or by the tests above given.

In the worst cases the urine is very scanty and of a deep yellowish brown color, prostration is extreme, tympany, colic, obstinate constipation or bloody diarrhœa may set in, the breathing, hurried or not, is trembling, the pulse small, and the temperature at first high may descend to 95° or even much lower before death.

The *course* of the disease varies according to its gravity. If there is complete retention of bile, and abundant production of toxins, the animal dies in one or two days in a state of collapse. If there is general progressive degeneration and destruction of the hepatic tissue without at first absolute suppression of the discharge of bile into the duodenum, the patient may last till the fourth or fifth day, or later.

Lesions. There are usually congestion, tumefaction, friability, ecchymosis and even ulceration of the gastric and duodenal mucosa. The organs are empty, but show a reddish brown exudate of a glairy consistency, and containing red blood globules and pus corpuscles. The same inflammatory lesions are to be traced into the common bile duct, the cystic duct and bladder, the biliary ducts, and the acini. The mouth of the common duct is usually blocked with a plug of tenacious mucus, the gall bladder having been unable to expel this and the inspissated bile into the intestine. The liver is slightly enlarged, yellowish, with patches of brownish yellow more or less deep, and the acini contain an abundance of oily globules, and yellowish brown granules. The acini have no clear line of delimitation, and the contained hepatic cells are shrunken and distorted, standing apart

from each other in a dropsical or watery medium.

The kidneys are congested and ecchymosed; the cortical substance brown, friable, and with numerous areas of necrosis of a bluish white color, and even abscesses. The medullary substance is yellow and the uriniferous tubes contain an abundance of yellowish brown granules.

The lungs have a yellowish red color, with patches of ecchymosis.

The lymph glands generally are congested and many of them gorged with blood, of a dark red color, and lacking in consistency and cohesion.

Diagnosis. The characteristic icterus is lacking in the early stages, and active treatment gives good hope of success. When indigestion, persistent vomiting and tenderness of the epigastrium, and right hypochondrium, are associated with diarrhoea, it is highly important to examine the urine for even slight traces of bile. When the jaundice is due to impaction of a biliary calculus, the symptoms may increase slowly, and yet reach a sudden climax with acute colicky pains and tenderness of the right hypochondrium.

Prognosis. In acute rapidly developing cases a fatal issue is to be expected. In those which develop more slowly, recovery may be hoped for if early treatment is instituted.

Treatment. Cases due to biliary calculus must be treated for that lesion.

In purely infective icterus attempts must be made to arrest the intestinal and hepatic fermentation. As intestinal antiseptics, naphthol, benzo-naphthol, naphthaline, 5 grains four to six times a day. As hepatic antiseptics, salol 5 grains, salicylate of soda 8 grains, or calomel 1 grain four times a day. The salol and salicylate tend to increase biliary secretion and to render it more fluid. The same end is attained by alkalies (carbonates of potassa, or soda or lithia, bitartrate of soda, iodide of potassium). These are further valuable in hastening the elimination of toxic matters by the kidneys. The expulsion of bile, and of intestinal microbes and toxins may be sought by laxative doses of Glauber salts, or by cold enemas of the same. Verheyen recommends aloes in laxative doses for six days. Siedamgrotzky had good results from induction currents of electricity, sent through the region of the liver twice a day for ten minutes on each occasion. To assist in elimination abundance of pure water or of watery fluids may be used. The most effective eliminating agent is pilocarpin in $\frac{1}{6}$ gr. dose hypodermically, repeated daily. In weak conditions frequent small doses of strychnia, ether, aqua regia, or digitalis may prove valuable.

In case of improvement a course of bitters is usually demanded, and these may be combined with hydrochloric acid or small doses of sodium bicarbonate.

Throughout the disease, gruels, beef tea, buttermilk, whey or any simple nutritive aliment which the animal relishes may be given, but both then and during convalescence fatty matters and indigestible materials should be carefully withheld.

JAUNDICE IN CATTLE AND SHEEP.

Usually with gall-stones or concretions, or distomata. In sheep from decomposing vegetation. Symptoms: anæmia, emaciation, pallor, icteric mucosæ. Digestive disorder and bilious stools suggest worms. Treatment: as in horse, or vermifuge.

Though less common than in the dog icterus in ruminants occurs, but most commonly in connection with gall stones and concretions, or with trematodes in the gall ducts. These forms will be noticed under these respective headings. Verheyen describes an icterus of sheep which occurs enzootically in damp low undrained localities, and is attributed by shepherds to the consumption of dead and fermenting leaves. The symptoms are those of anæmia, emaciation, and increasing weakness, with a pallor and more or less dull yellow of the conjunctiva, and, later, of the other mucosæ. The loss of appetite, indigestion, yellow liquid fæces, suggest the possible presence of parasites as a cause, and the prescribed treatment by common salt, juniper berries and tonics strengthens the suspicion. Other forms must be treated according to cause on the lines laid down above.

JAUNDICE OF THE NEW-BORN. ICTERUS NOUVEAUX NÉS.

In mules; less frequently in horses and cattle. Bacteridian. Predisposition from alleged miscegeneration. Offspring of lymphatic mares. Spoiled fodders, foul buildings, exposure, infection by umbilicus. From gastro-enteritis, diarrhoea, dysentery. Congenital. Symptoms: refuses the teat, scours, red urine, palpitation, colics, perspirations, inflammations, or abscesses of navel, arthritis, other internal disorders. Death from exhaustion or in convulsions. Or symptoms subside and pass in 15 days. Diagnosis: prostration, palpitation, icterus, and bloody urine with omphalitis. Streptococci. Prognosis: with hæmaturia nine-tenths fatal. Lesions: icterus, heart soft, flabby, lungs congested, general ecchymosis, blood black, clot diffuent, liver congested, enlarged, spleen, swollen, softened with spots of brownish yellow, meconium bloodstained, kidneys swollen, congested, points of infarction or necrosis, urine bloody, navel lesions. Treatment: for congenital cases improve hygiene for dams; for offspring, antiseptics to navel, shelter, warm box; aloes to dam, or oil to colt, antiseptics internally, demulcents, stimulants, derivatives, opium, water, alkalies.

This has been observed in cattle (Kitt), and in horses (Levrier, Bernadin, Lhomme) but above all in mules (Villa-Roya, Carrere, Levriere, Bernadin, Lhomme, Hartmann, Dieckerhoff).

The affection is a bacteridian disease, in which there is a great destruction of red globules, and liberation of hæmatin, with hæmaturia and yellow coloration of the tissues.

Causes. Carrere attributes much to a supposed congenital weakness induced by the unnatural generation of ass with mare. Cadeac noticed that the offspring of certain mares and horses remained sound, while the progeny of the same animals, and asses suffered largely from icterus. Levrier found it especially in the mules born of mares having a lymphatic temperament or suffering from canker, grease or chronic lymphangiectasis. Bernadin and Lhomme attribute much to bad hygiene, and especially to poor or spoiled fodders. Lhomme, Lafosse and Trasbot find accessory causes in cold and wet weather. Retention of the meconium is another cause. Dieckerhoff, Hartmann, Cadeac and Bournay trace the disease to bacterial infection as the essential cause. Many cases originate in septic infection and inflammation of the navel and umbilical veins. Others commence with mucous gastro-enteritis, attended by diarrhoea or dysentery. In other cases the germs appear to have reached the liver through the circulation as the animal is already affected at birth.

Symptoms. When not congenital, symptoms are usually seen in the first few days of life, usually before the fifth day.

In congenital cases the new born animal is unable to stand at all or for longer than a very short time, it lies listlessly and makes no attempt to suck even when held up to the teat. There is acceleration of pulse and respiration, the heart beats are tumultuous, the mouth is dry, the mucosæ of a straw yellow, the bowels are costive, and the urine, whether discharged in life, or found in the bladder after death, is bloody.

When attacked later, the animal becomes dull, weak, and stupid, refuses the teat, and has the bowels relaxed. The mucosæ become pale yellow, the breathing accelerated, the heart beats violent, and the pulse rapid, small and weak. The urine is passed slowly and with effort and has a red hue more or less deep according to the severity of the attack. Colics are not uncommon, causing uneasy shifting of the limbs and tail, cries, frequent lying down and rising, and partial or general perspiration. Complications on the part of the navel and umbilical veins are to be looked for, in open sores, swellings, abscess, phlebitis, and in arthritis and secondary abscesses in different organs. Death may occur quietly, as from exhaustion, while in other cases it is preceded by convulsions.

In case of recovery, there is an improvement of the general symptoms, the heart and respirations become moderate, appetite is restored, the little animal sucking the teat, there are abundant, yellow, semi-solid defecations, the strength increases day by day, and convalescence may be fully established by the twelfth to the fifteenth day.

Diagnosis. This is mainly based on the extreme weakness and prostration, the violent heart action, the jaundiced hue of the mucous membranes, and the bloody urine. Confirmation may be sought in the presence of streptococci in the fresh urine, kidneys, liver and blood, and in the artificial cultures made from them. The presence of omphalitis is further significant.

Prognosis. Bernardin says that if hæmaturia is present nine out of ten cases are fatal, while in the absence of this feature nine-tenths recover.

Lesions. The jaundiced appearance of the conjunctiva and other tissues is constant. The heart is pale, soft and flaccid, with petechial patches of a deep red extending into the muscular substance; the lungs are congested with similar patches of blood-staining; the blood in the heart and larger vessels is dark, and fluid or only loosely clotted. The liver is congested to twice or thrice its normal size (10 lbs. in place of 3 or 4 lbs.); the spleen is enlarged, softened, and shows spots of brownish yellow. The small intestine contains a yellowish or dark red meconium, and it may contain effusions of dark blood, while its mucosa is inflamed, thickened, easily lacerated, and pigmented, or marked by petechiæ. The large intestines and especially the rectum are packed with hard dry balls.

The kidneys are enlarged, often to double their volume, and deeply congested, with infarcts, and patches of necrosis, of a pale brownish yellow hue. The urine in the bladder is deeply stained with blood coloring matter.

Treatment. For congenital cases it is manifest that treatment must be preventive and applied to the dam, before parturition. An open air life, moderate exercise, sound, easily digestible and nourishing food; grooming; in the stable, cleanliness, dryness and good ventilation; good water, are essential.

For the offspring, antiseptics (tannin, mercuric chloride lotion, copperas, calomel, iodoform), applied to the navel, and protection against cold winds and rains, and damp lairs. For mules and other young animals born in severe, winter weather a dry, warm, foaling box is desirable, and the little animal should be rubbed dry and covered with a warm woolen blanket. When the temperature approaches zero or the barn is cold, the smaller animals, as soon as they are dropped, should be placed under a box with a jar of hot water wrapped in woolen coverings, or with hot bricks similarly wrapped, and should only be let out for food when they are completely dried, or when the weather has moderated.

Therapeutic treatment may be commenced by a dose of aloes given to the dam, or of olive or castor oil or manna given to the offspring. As a substitute sulphate of soda may be used. Antiseptics like salicylate of soda, salol, or the sulphites may be added. To act as a demulcent on the alimentary and urinary tracts, well boiled flaxseed tea is usually recommended. Weakness may be met by warm strong coffee, salicin, quinia, or other bitters, and more stimulating agents like camphor, angelica, assafœtida, or even oil of turpentine may be added. Diarrhœa may be checked by linseed tea, mustard plasters, or in obstinate cases, by opium. Elimination should be sought by administering abundance of pure water or watery demulcents, and even by the use of alkalies like bicarbonates of soda or potash. A moderately free action of the bowels must be constantly maintained.

Antiseptic treatment of the navel and umbilical veins must not be overlooked.

LUPINOSIS, ACUTE TOXÆMIC ICTERUS, ACUTE YELLOW ATROPHY OF THE LIVER.

Attacks sheep, goat, ox, horse, stag; and, experimentally, dog. Causes: consumption of lupins, at a given stage of ripeness, from a given part of a field, or from centre of a stack. Lupinotoxine, conicine, methyl conicine, lupinine. Cryptogamic or bacterial poison. Weak subjects, sheep and even ewes and lambs, suffer most. Symptoms: Acute form: anorexia, fever, excited pulse and breathing, stupor, or hyperæsthesia, vertigo, swellings on head. Poisonous lupins are first rejected. Bloody nasal froth. In two or three days icterus. Urine may be bloody. Fæces at first hard, coated, bloody, later dark brown and often liquid. Emaciation. Death in 1 to 5 days. Chronic form, gastro-enteritis, emaciation, anæmia. Nasal catarrh. Facial swellings and sores. Lesions: hepatitis, nephritis, muco-enteritis, enlarged spleen, icterus, blood extravasations; hepatic tissue, infiltrated, cloudy, granular, fatty, later cirrhosis. Kidneys contain casts: Spleen tumid, blood gorged. Prognosis: grave: acute cases die, chronic may recover. Prevention: feed no lupins, avoid dangerous fields, wash off poison from lupins with a soda solution. Ensilage with acid producing fodder in alternate layers. Treatment: avoid alkalies, give acids, purgative, castor oil, water. In horse, causes anorexia, anæsthesia, dullness, stupor, colic, constipation, urinary irritation, fever, slight jaundice, vertigo, orange nasal discharge, sores on tongue and face, and lower part of limbs. Diagnosis. Prognosis hopeful. Treatment as in sheep. Icterus from other fodders.

This affection has been noticed especially in sheep, but also in the goat, horse, ox and stag as the result of eating lupins. The dog has contracted the disease under experiment. It has been studied especially in Northern Germany where the lupin is largely cultivated as a fodder crop. The yellow lupin (*Lupinus luteus*) is mainly to blame for the disease, but the *Lupinus albus* and *Augustifolius* are also spoken of as factors.

The disease caused by altered seeds and straw of the lupin is mainly characterized by jaundice, fatty degeneration of the hepatic cells and hypertrophy of the connective tissue of the liver causing acute atrophy of the organ.

Causes. The essential cause of the disease appears to be the consumption of lupins. But all lupins are not equally poisonous. Those taken from one portion of a field are harmless, while those from another are toxic. In stacks built in the field and weathered the upper and outer portions are often harmless while the interior remains poisonous. It would seem as if the poison were washed off by the rain, or deprived of its potency by the action of the air. It successfully resists dry heat, for three hours at boiling temperature, but is rendered harmless by steam acting under the pressure of two atmospheres for the same length of time. A poisonous principle (lupino-toxine) has been obtained from the toxic lupins but it is not quite certain that this is the sole toxic ingredient. This agent is extracted from the powdered seeds by macerating them for two hours in a soda solution (in which it is very soluble) at 102° F., and purified by treating the solution successively with acetic acid, lead acetate, hyposulphuric acid and alcohol. This agent produces the symptoms of lupinosis in the acute or chronic form according to the doses of the agent administered. Eichhorn and Baumstarch have isolated from lupins an alkaloid analogous to *conicine*: Stener found an alkaloid which he believed to be *methyiconicine*: Baumert attributed the activity to another alkaloid *lupinine*. It is not definitely known whether the poisoning is usually effected by a simple poison or by a combination of several. Nor is it certain whether the toxic matter is a normal product of lupins grown on particular soils and under given conditions and harvested at a particular stage of growth, or if it is the product of a cryptogamic or bacterial growth. Some leguminous seeds are poisonous at a given stage of ripening but there is as yet no proof of lupinosis being confined to any particular stage. The common moulds often grow on lupins without rendering them poisonous, but it does not follow that some less familiar cryptogam is equally harmless under all circumstances. The soda extract of the poisonous lupins was deadly though it contained no cryptogams, but it is not shown that it was free from soluble chemical products (toxins) of the cryptogams. The same remark applies with equal force to the bacteria which have been invoked as the cause of the poisons. Though not themselves present in a given deadly extract of the lupins this does not exclude from such extract the toxic products of bacterial growth. It is claimed that Arnold has produced lupinosis with lupins that had been first robbed of their alkaloids. But the absence of alkaloids does not prove the absence of nonbasic (neutral) poisons, of vegetable, cryptogamic or bacterial origin.

That certain lupins contain a deadly poison is certified, but the precise source of the poison remains to be demonstrated.

In estimating causes we must take into account the lessened power of resistance of animals lacking in constitutional strength and vigor. Thus sheep suffer far more severely than horses, oxen, or even goats. Ewes and lambs perish in greater numbers than rams, hoggets and wethers.

Symptoms in Sheep. In the acute form the disease appears suddenly, as manifested by anorexia, hyperthermia, rapid and oppressed breathing, accelerated pulse, stupor, vertigo, and not unfrequently swellings of lips, ears or face. Inappetence may be first manifested by the rejection of poisonous lupins, while sound ones and especially other food are still eaten, but soon all are refused alike. Temperature, which may reach 104° to 106° F. on the next day after feeding on the poison, may rise and fall day by day, and finally fall materially as a herald of death. Respiration rises to 100 per minute and becomes labored or panting, with, in some cases, a bloody froth in the nostrils. The pulse rising to 130 and upwards keeps pace with the hyperthermia and general excitement. Vertigo is shown in the staggering gait when moved, and by a tendency to steady by resting the head on the trough, rack, fence or ground. The recumbent position is often preferred, the head being extended on the ground, and the animal remaining oblivious to all efforts to raise him—even to blows. Sometimes there is stupor, and at others hyperæsthesia, or indications of fear. There is grinding of the teeth, and sometimes trismus (Schütz, Kotelman).

In two or three days jaundice is shown, more especially in the conjunctiva and the urine. The latter, however, is not unfrequently colored with blood, and contains albumen, bile acids, and renal epithelium and casts. It is passed frequently in small quantity, so that its condition is easily ascertained.

The fæces are at first scanty and hard with a coating of yellowish mucus, and it may be streaks of blood. Later they are uniformly stained of a dark brown, and diarrhoea may alternate with the constipation. Emaciation advances with rapid strides.

Death may occur as early as one day after the attack but is usually deferred to the fourth or fifth.

A steady amelioration of all the symptoms may be welcomed as a precursor of recovery.

In the *chronic form* jaundice may be entirely absent, and a subacute gastro-enteritis may be attended by emaciation and anæmia. Roloff has frequently found the implication of the other mucosæ especially those of the nose and eyes which become catarrhal, and Zurn notes the implication of the skin of the face with exudations, swellings and the formation of sores and scabs on the eyelids, lips, ears, etc.

Lesions in Sheep. The prominent lesions are parenchymatous inflammation of the liver and kidneys, muco-enteritis, enlargement of the spleen, and icterus, with more or less blood extravasation.

The liver is the seat of acute hepatitis. It is usually swollen and abnormally friable, with a yellowish color often as deep as citron. The parts recently attacked exhibit albuminoid infiltration and cloudy swelling, the older lesions show fatty or granular degeneration. These changes exist especially in the hepatic cells, and fatty metamorphosis bears a direct relation to the obesity of the sheep. If the patient has survived the first few days, the liquefied products are absorbed, there is a relative increase of the interstitial connective tissue and the volume of the organ is materially decreased (acute yellow atrophy of the liver, Schütz). In chronic cases, the hypertrophy of the interstitial connective tissue is the most prominent feature, constituting a distinct cirrhosis (fibroid induration) and the atrophy becomes very pronounced while the surface is very irregular and uneven. The gall bladder is distended, and its mucosa congested.

Jaundice, which may be little marked apart from the conjunctiva, is usually shown in the subcutaneous connective tissue, the skin of the abdomen, the omentum and mesentery, but according to Cadeac is never shown in cartilage, bone nor tendon.

The kidneys show parenchymatous inflammation, congestion, swelling which gives a hardness of touch through the resisting fibrous envelope, and often a pervading shade of yellow. The epithelium of the uriniferous tubes is cloudy, turbid and granular and the tubes themselves contain cylindroid casts. The bladder is catarrhal and has been too irritable to contain urine.

The spleen is tumid, soft, reddish gray, and on section shows excess of blood, pulp, and here and there marbling by fibrinous exudate.

The heart is pale, granular, friable, with a tinge of yellow and with a slight citron colored effusion in the pericardium. The cardiac blood is dark and thick but coagulates and brightens in color on exposure to the air.

There is usually some oedema of the lung, larynx and pia mater.

There are catarrhal lesions of the abomasum and entire intestinal canal with granular degeneration of the epithelium and spots and patches of arborescent congestion and hæmorrhage. The icteric tint is usually distinct.

Prognosis is always grave. Acute cases are almost always fatal. Chronic cases due to eating lupins in which the poison is relatively weak, or a very restricted amount of the more poisonous lupins, may recover. This mortality is very serious in the affected districts 5.8 per cent. of all the sheep of some districts in Pommerania perishing yearly from this affection and entailing the loss of almost the same number of lambs (Von Below-Seleske).

Prevention. Radical prevention can be secured by the exclusion of lupins from the ration. Where this is undesirable the fields that produce poisonous lupins can be ascertained and devoted to the production of other crops. When a crop of lupins has already been produced on a dangerous field the poison may be extracted from the fodder or subjected to the action of an antidote. Fortunately the poison is concentrated on the surface of the lupin and is easily washed off.

Dammann advises to leave the cut forage exposed to the rain so that the poison may be washed off. A more prompt and effective plan would be to put the fodder under a stream of water from a hose, and it could be fed while still fresh and unaltered. Friedberger and Fröhner directs that the forage be steeped for forty-eight hours in a soda solution (1:100) to dissolve off the offensive matter, after which it may be washed and safely fed.

Glaser would put the lupins in a silo, in alternate layers with some material which rapidly undergoes acid fermentation (brewers grains, maize, pulped roots). The acid renders the poison insoluble and is directly antidotal. To develop this acid it is important to put the materials into the silo before they have become too far matured.

The destruction of the poison by steaming the fodder under a pressure of two atmospheres, as recommended by Kuhn and Roloff requires a too elaborate apparatus for ordinary use.

Therapeutic treatment. The first desideratum is to render insoluble any poison which has already entered the alimentary canal. Alkalies, as solvents and distributors of the poison must be carefully avoided and acids (acetic, nitric, hydrochloric, sulphuric) freely used. These may be mixed with the drinking water or given from a bottle. If appetite is still retained, they may be mixed with the food, or acid aliment (sour ensilage, old grains, or fermented swill) may be supplied.

The next resort is a purgative to eliminate the poison from the alimentary canal. But the cathartics usually given to the ruminants are dangerous. Sulphate of soda undergoing decomposition, furnishes an alkaline solvent for the poison. Castor oil is therefore to be preferred.

An abundance of water will favor the action of both bowels and kidneys and hasten the elimination of the poison which may have been already absorbed into the system.

European writers recommend the use of the flesh as human food, unless the animal had reached the last stage of the disease.

Symptoms of Lupinosis in the Horse. There is always the history of the presence of lupins in the oats, or of lupin straw as a food or litter. Though less fatal than in sheep the disease sets in with great severity, the symptoms referring especially to gastro-enteritis, and disorders of innervation. There is complete anorexia, impaired sensibility, dullness and stupor, the head resting on the manger or drooping with the nose near the ground. There is grinding of the teeth, colicky symptoms, and constipation, the fæces being passed as a few small, hard balls covered with mucus and foetid. Urine is passed frequently in small amount and albuminous. There is more or less hyperthermia (rising at times to 102° to 103°), the respiration is hurried (36 to 40 per minute) and pulse is rapid (60 per minute). Jaundice is usually present but less prominent than in sheep. When moved the animal sways unsteadily or staggers. Butzert notices, in addition to the above, a thick orange colored discharge from the nose, and the formation of sores and scabs (mummification) of the lingual mucous membrane, of the lips, of the skin, of the face, and of the pastern, and swelling of

the lower parts of the limbs.

Diagnosis. The development of disease with the above symptoms, in the inmates of a single stable, or in horses having a common ration, in which the lupines are found, will make diagnosis easy and reasonably certain.

Prognosis is hopeful or confident. The disease is not fatal in the horse.

Treatment should follow the same lines as in sheep.

Allied or identical diseases. Friedberger and Fröhner quote a number of outbreaks of hepatic inflammation or disorder with icterus in the absence of lupins, but on rations that were otherwise faulty. Haubner describes a “malignant icterus” in sheep fed on malted potatoes; Sander records a “hepatic typhus” in the horse when fed on inundated pastures; Reinemann and Jansen speak of a similar affection in animals fed on the straw of peas, beans and vetches.

CONGESTION OF THE LIVER.

Physiologically after a meal is hyperæmic, and may increase one-third. Rich feed and temporary idleness may cause morbid congestion. Other causes are: ptomaines and toxins in spoiled food, hepatic irritants, damp, hot, tropical climates, warm, damp, buildings, overfeeding, dilated right heart, insufficiency of tricuspid valve, pulmonary emphysema or congestion. Absence of valve between the right heart and hepatic veins, slow hepatic circulation, dilatability of hepatic vascular system, compression of lung by false membranes, œdema, hepatization, infarction, hydrothorax. In horse: special causes: spoiled fodders, carminatives, volatile oils, work in hot sun, vaso-dilatation in heat and atony, falls, kicks, goring, blows; passive forms from obstacles to the circulation. Lesions: Hepatic enlargement to 20 or 30 pounds, and dark red color, darker spots of extravasation, hepatic vein congestion in centre, and portal vein congestion in periphery of acinus, softening, granular, fatty, nuclei disappear from hepatic cells, nutmeg liver, cirrhosis. Symptoms: general febrile symptoms, great prostration, drowsiness, slight colics, arched loins, trembling or jerking, lies on right side, percussion on last ribs painful, slight icterus and increased urination and urea. In passive cases, less icterus and kidney trouble; tends to intestinal catarrh and ascites. Prevention: good hygiene, moderate laxative ration, cool air, exercise. Treatment: green or laxative foods, salines, derivatives, water at will, always before morning feed. In dog: overfeeding, indolence: pulmonary and circulatory troubles. Lesions: nutmeg liver, degeneration, cirrhosis. Symptoms: pampered idleness, obesity, pulmonary or cardiac disease, intestinal catarrh, piles, ascites, slight icterus, percussion tenderness of right hypochondrium, lying on right side, increase of percussion flatness. Treatment: saline purgatives or laxatives, antiseptics, restricted laxative diet, open air exercise, derivatives, etc.

Physiologically the liver undergoes hyperæmia in connection with an abundant meal and active digestion. Within the limits of health it may increase one-third in bulk and weight. When this hyperæmia attends on temporary idleness and rich feeding it may overstep the limits of health and become pathological. Other causes of pathological congestion are, tainted food rich in ptomaines and toxins, food injured by cryptogams, and food that contains principles irritating to the liver. It is especially common in swampy regions in tropical or semi-tropical latitudes, and where the warm air is surcharged with moisture. Hot, close, damp stables, with overfeeding and lack of exercise in the pampered horse or dog, or under the forcing system of feeding for exhibition, or for the butcher, acts in a similar manner. Dilatation of the right heart, or imperfection of the tricuspid valve is a direct cause of mechanical congestion and emphysema, congestion or other obstruction in the lungs has a similar effect. In short the absence of valves between the right heart and the hepatic veins determines a passive congestion of the liver whenever there is any serious hindrance to the passage of blood through the right heart and lungs. The tardy circulation through the liver (5 mm. per second) also predisposes to congestion. The great development of the circulatory system in the liver, and its dilatability predisposes it to such congestions in a pre-eminent degree. For the same reason the liver suffers in the horse that is attacked with pulmonary congestion, whether as the result of overwork or of heat congestion. It will also suffer from compression of the lungs by false membranes or hydrothorax, or obstruction, by œdema, hepatization or infarction. The toxic matters produced in infectious diseases, and especially those affecting the intestinal canal and its connections, are arrested in the liver and contribute to hyperæmia.

CONGESTION OF THE LIVER IN THE HORSE.

Causes. Beside the general causes above mentioned, may be specially named, musty, decomposed, and irritant fodders: those which like green legumes, are easily fermented; and those which contain stimulating volatile oils or carminative principles. They are also especially exposed to such causes as severe and prolonged work under a hot sun, the nervous atony which causes vasodilatation in a hot climate, and such traumatism as come from falls, kicks, goring, and blows by shafts, poles and clubs. These especially induce *active congestion*. The *passive* forms come mainly from obstruction in the lungs, or heart (dilatation, right valvular insufficiency, pericarditis, hydropericardium, myocarditis, fatty degeneration, endocarditis), or in the posterior vena cava.

Lesions. The congested liver is enlarged and deeply colored with blood. The weight of twenty to thirty pounds is often attained. While the color is of a deep red throughout, there are spots of a still darker hue indicating the seat of subcapsular or deeper seated hemorrhages. The color varies according as the congestion is passive or active. In the former the coloration is deeper in the centre of the acinus (nutmeg liver) indicating congestion of the hepatic veins, while in the latter the periphery of the acinus may be most deeply stained implying congestion of the portal vein. The consistency of the organ is diminished, and the more acute the attack the greater the friability. In such cases there is a parboiled appearance indicating granular and commencing fatty degeneration. Under the microscope the relative distension of the intralobular, and interlobular veins and the hepatic capillaries becomes more distinctly marked and the presence of pigment and fatty granules and the lack of protoplasm and nuclei in the hepatic cells indicate their progressive changes. When the peripheral cells are pale from fatty granules the contrast between the light margin and dark centre of the acinus, makes the mottled or nutmeg aspect of the liver much more pronounced.

In old standing cases of passive congestion the liver may be the seat of fibroid degeneration, extending from the capsule inward in bands or trabeculae, and giving to the organ a firm resistant character (sclerosis, cirrhosis).

Symptoms. The symptoms are general and suggestive rather than pathognomonic. There are dullness, prostration, unsteady walk, pendent head, with occasional jerking, semi-closed eyes, redness of the conjunctiva, slight colicky pains, arching of the loins, muscular tremblings and decubitus on the left side rather than the right. The more definite symptoms are tenderness on percussion with the closed fist over the last ribs (the liver) especially on the right side, increase of the area of hepatic percussion dullness (which may be rendered valueless by a loaded colon), the presence of a slight icterus in the conjunctiva and urine, and an increase of the urine secreted and an excess of the contained urea.

In *passive* cases however the obstruction to the escape of blood from the liver prevents the development of icteric symptoms, of uræmia and of polyuria. In all such cases however there follows a general congestion of the portal system and if it persists for any length of time gastrointestinal congestion and catarrh and even ascites may develop.

In all cases alike the history of the attack will help towards a satisfactory diagnosis.

Prevention. A rational hygiene embracing daily work or exercise, moderate laxative diet, green food in its season, pure cool air are important precautions.

Treatment. A moderate supply of green or laxative food, the withholding for the time of grain, and especially of maize, wheat or buckwheat, saline laxatives daily, and a stimulating embrocation or blister to the tender hypochondrium are the most important measures. Exercise in a box stall, or still better in a yard or paddock in the intervals between more systematic work forms an important adjunct to medicine. As a laxative sulphate of soda is to be preferred at first in a full cathartic dose and later in a daily amount sufficient to relax the bowels. Given in a bucket of water every morning before the first meal a very small dose will be effective.

CONGESTION OF THE LIVER IN THE DOG.

Active congestion is very rare excepting in over-fed and indolent family pets. *Passive* congestion induced by diseases of the lungs and heart is however far from uncommon.

Lesions. True to their origin these usually appear as the spotted nutmeg liver with the deep congestion in the centre of the acini. For the same reason the fibroid degenerations shown in chronic cases, show the firm fibroid neoplasm chiefly around the hepatic veins. Granular, fatty and pigmentary degeneration of the cells are found as in the solipeds.

Symptoms. These are as obscure as in the horse. There is always a history of a sluggish, gourmandizing life, and in the early stages, a manifestation of embonpoint which suggests a torpid liver. Further suggestions may also be obtained from coexisting diseases of the lungs, or heart, from gastro-intestinal catarrh, from piles, or ascites. Then there is at times a slight icterus of the conjunctiva and urine. Finally tenderness on percussion on the right hypochondrium, decubitus on the left side, and an increased area of dullness on percussion may afford useful hints for diagnosis.

Treatment. In the rare cases due to infection from the intestine, an active saline purgative followed by antiseptics (salol, naphthalin, naphthol, etc.,) daily will be of value. It is also desirable to keep up the action of the bowels by morning doses of salines. In cases consequent on chest disease attention must be given to such primary trouble. In all cases a restricted laxative diet, and graduated but increasing exercise in the open air are demanded.

HEPATIC HÆMORRHAGE OR RUPTURE.

Causes: Mechanical injuries, falls, blows, kicks, degenerations, amyloid, fatty, granular, congestion, neoplasms, glanders, tuberculous, myomatous, microbial infection. In the horse, disease of liver, heart, lungs, hepatic artery, portal vein, degenerations following overfeeding, idleness, foreign bodies, arsenic, phosphorus, parasites, violent movements in colic, running, draught, leaping. In the dog, pampering and traumatism. In cattle forced feeding, emaciation, microbial infection. In birds, tubercle, tæniasis, microbial infection. **Lesions:** extravasation, intracapsular, or through capsule into the peritoneal cavity. The extravasation bulges of a deep black, covering a dark softened, pulpy, hepatic tissue, with light colored fatty tissue around. Clots may be stratified from successive bleedings. Liver usually enlarged. **Symptoms:** onset sudden, or preceded by stiffness, soreness and other signs of hepatic trouble. Extensive rupture, entails weakness, unsteady gait, perspiration, pallor of mucosæ, small weak rapid pulse, palpitations, dilated pupils, rolling eyes, amaurosis, tremors, convulsion in case of survival, coldness, œdemas. Death in five hours to five days. Risk of relapse in recovering cases. **Treatment:** rather hopeless, rest, laxative, ergot, ferric chloride, tannic acid, witch hazel, cold water, snow or ice to right side. In meat producing animals fatten.

Causes. Hemorrhage and rupture of the liver are closely correlated to each other, the accumulation of extravasated blood in the parenchyma in the one case leading to over distension of the capsule, and the laceration of this capsule and of the adjacent substance of the liver occurring in the other as a mere extension of the first. They usually occur as the direct result of mechanical injury (falls, blows, kicks) acting on a liver already softened and friable through disease. These predisposing degenerations may be amyloid (Caparini, Johne, Rabe), fatty (Julien, Gowing, Adam, Siedamgrotzky), granular softening, hepatitis or congestion (Zundel), glander neoplasms (Mathis), tubercles, angiomas (Trasbot), microbial infection (Stubbe), tumors (Brückmüller).

In the horse predisposing conditions may be found in diseases of the liver, heart or lungs, in embolism of the hepatic artery (Wright), in obstruction of the portal vein (Pierre), in infarction of the liver, in degeneration with softening, in sarcomatous, melanotic, glanderous or cancerous deposits in its substance, in degenerations consequent on over feeding, idleness, congestions, on the penetration of husks of grains into the liver substance, on arsenical or phosphorus poisoning. The presence of flukes, echinococci and other parasites may also cause congestion and softening. To the *immediate* or *traumatic* causes above named may be added the violent movements attendant on a severe attack of colic, and violent exertions in running, draught, leaping, etc. (Friend).

In the **dog** we must recognize all the pampering conditions which predispose to congestion and degeneration, together with more direct operation of kicks, blows, falls, fights, over exertion, etc.

In **cattle** a forcing regimen is especially predisposing, and yet the loss of vigor resulting from a diametrically opposite treatment, must be accepted as an occasional cause. Stubbe found in emaciated cows miliary hemorrhagic infarcts of a dark red color which gradually extended to an inch or more in diameter. These he traced to microbial infection coming by way of the chronic intestinal lesions which are common in old cows. The final result of such infarctions was loss of hepatic substance and the formation of cicatricial tissue with a marked depression on the surface of the organ.

In **birds** fatal hepatic hemorrhages occur in connection with local tubercle (Cadiot), tæniasis of the liver, or microbial infection.

Lesions. The hemorrhage may take place into the substance of the liver only, or the capsule may be lacerated so that the blood escapes into the peritoneal cavity in considerable quantity.

In the *horse* it usually occurs in the right or middle lobe, rarely in the left. There may be one or more hemorrhagic effusions varying in size from a cherry to a duck's egg, or even an infants' head (Lorge). This projects from the surface of the organ and its deep black contrasts strongly with the white of the adjacent capsule. When laid open the hepatic tissue is seen to be softened and pulpy, and its dark color forms a striking contrast with any surrounding fatty liver. Any form of degeneration may be revealed on microscopic or chemical examination. Not unfrequently small clots of blood form under the capsule raising it in the form of little sacs. Such clots are usually stratified indicating a succession of small hemorrhages.

When the capsule is torn, the lesion may extend from one surface of the organ to the other, and

the edges, smooth, uneven or fringed, are united together by a blood clot.

In case of hemorrhagic infarcts the lesion usually has a distinctly conical outline corresponding to the vascular distribution. These are especially characteristic of cases supervening on heart disease.

The volume of the liver is usually increased and the weight may reach 30 lbs. (Schmeltz), 34 lbs. (Lorge), or even 66 lbs. (Trasbot).

In *other domestic animals* analogous lesions are found modified largely according to the size of the subject.

Symptoms. These may develop instantaneously without any marked premonitory indication. In other cases tenderness on percussion over the liver, stiffness or groaning under sudden movements or turning, arching of the back, hanging of the head, slowness in rising, costiveness, slight transient colics, and even icterus may have been detected on close observation. The symptoms of actual rupture are essentially those of internal hemorrhage. The animal becomes weak, or unsteady upon its limbs, perspires, arches the back, and shows a marked pallor of the visible mucosæ. The pulse is small, thready, weak and accelerated, and the heart beats violent or palpitating. The percussion dullness over the liver is extended (Weber), the loins become insensible to pinching, and there may be some distension of the abdomen. Dilatation of the pupils, retraction or rolling of the eyes, amaurosis, tremors of the muscles of the neck, lying down, or falling, and general convulsions may precede death. This may occur in a few hours or it may be delayed if the lesions are restricted. In case of survival, coldness and œdema of the extremities and sheath have been observed. The lesser hemorrhages may terminate in recovery if there is no attendant incurable disease. In anthrax, glanders, cancer, tuberculosis, septicæmia, etc., a favorable issue is not to be looked for.

Duration. Termination. In severe cases a fatal issue may be expected in from five hours to five days. In the milder cases which make a temporary recovery there is great danger of a second hemorrhage from the new vessels in the tissue undergoing organization or from the adjacent degenerate liver tissue. The course of the affection may be altered by such complications as arthritis (Dieckerhoff), pneumonia, pulmonary thrombosis (Leblanc), enteritis or peritonitis (Cadeac).

Treatment is usually of no avail. Rest, and the administration of laxatives and hæmostatics, have been especially recommended. Of the latter, ergot by the mouth or ergotin subcutem, tends to contraction of the blood-vessels and to check the flow. Ferric chloride is also used, though apt to interfere with hepatic function. Tannic acid, hamamelis, and other astringents may be used instead. Cold water, snow or ice applied to the right hypochondrium may act as a check to the hemorrhage. Unless in purely traumatic cases in an otherwise healthy liver, a recovery is at best temporary, and the already degenerate liver is liable to relapse at any moment. In horses and dogs, therefore, recovery is by no means an unmixed good. Meat producing animals that recover should be prepared for the butcher.

HEPATITIS.

Forms of hepatitis: Parenchymatous hepatitis. Definition: Degeneration of hepatic cells. Relation to enteritis and nephritis. In horse—causes:—as in congestion, pampering, spoiled fodder, malt, inundated meadows, chill, overfeeding, hot moist climate, hæmoglobinæmia, infection. In cattle—causes:—forcing ration, hot weather, overwork, infection. In dog—causes:—infection from alimentary canal. Lesions: Enlarged, softened liver, round edges, a week later yellow atrophy, granular on section, bloodless. Acini with indefinite margins, cells granular, nuclei lost. In dog centres of softening. Symptoms:—in horse: Attack sudden, rigor, fever, dullness, prostration, yellowish red mucosæ, unsteady gait, slight colic, anorexia, urine decreased, glairy, brownish red, groaning in defecation, excited circulation and breathing, increased icterus by third day, fœtid, colorless diarrhœa. Diagnosis: Coincidence of fever, prostration, icterus, painful defecation, fœtid diarrhœa, light color of stools, tenderness and flatness on percussing hepatic area. From influenza by absence of watering eyes and contagion. Prognosis in horse: Very grave unless urine is free. Treatment in horse: Portal depletion, calomel, ipecacuan, salines, diuretics, fomentation of loins, antiseptics, derivatives, mineral acids, bitters. Careful laxative diet in convalescence. Symptoms in cattle: Slower onset, anorexia, dullness, depression, drivelling saliva, grinding teeth, icterus, constipation, later fœtid diarrhœa, pale colored stools, recumbency, groans on rising, arching back, tender right hypochondrium, fever. Prognosis grave. Death in five to six days. Treatment as in horse: Only saline laxatives. Symptoms in dog: Muscular tremors, staring coat, hyperthermia, icterus, fœtid breath, ventral decubitus, extreme prostration, anorexia, tender right hypochondrium, diminished urine, death in two or three days. Treatment in dog: Calomel and jalap, diuretics, laxatives, derivatives, germicides, in convalescence, mineral acids, bitters, careful diet.

The different forms of inflammation of the liver are distinguished according as they affect, especially the hepatic cells and tissue of the acini (parenchymatous), as they result in suppuration (suppurative, catarrhal, abscess), as they cause necrobiosis in nodular masses (infectious or necrotic), as they lead to fibroid thickening under the peritoneum and proper capsule (perihepatitis); or as they cause general fibroid induration of the organ by increase of its connective tissue (cirrhosis).

PARENCHYMATOUS HEPATITIS, ACUTE YELLOW ATROPHY OF THE LIVER.

The characteristic morbid lesion in this disease is the degeneration of the liver cells, loss of their protoplasm and nuclei and of their normal functions. It may be circumscribed to limited areas, or may affect the liver, generally. As the hepatic functions, are so intimately related to those of the bowels and kidney, the affection is usually accompanied by inflammations of these organs as well.

Causes in horses. The same general causes which produce congestion, may also determine the further morbid stage of inflammation. Cadeac mentions a case which developed in a horse kept alone and idle in the stable. He makes no mention of condition, food, cleanliness nor ventilation. Haubner and Franzen have traced it to a diet of malt or of hay harvested from inundated meadows. Zundel records a case following exposure to extreme cold. More commonly the disease is secondary to the overtaxing of the liver, by heavy feeding in warm moist climates, or in hæmoglobinæmia, or to the arrest of the micro-organisms of the food, or of infectious diseases.

Causes in Cattle. These suffer rarely, but from essentially the same conditions. It has followed apthous fever (Eletti), and arisen under a forcing ration, in hot weather (Callot, Cruzel), or under overwork (Cruzel).

Causes in Dogs. Most cases result from infection by way of the stomach and intestines, or by the transfer to the liver of the ptomaines and toxins of such infections. It is thus related in its origin to catarrhal jaundice and hyperæmia.

Lesions. In the earliest stage with albuminoid exudation into its substance the liver may be greatly enlarged, its sharp edges rounded, and its consistency softened. After a week's illness atrophy may have set in and the organ appears shrunk and of ochreous yellow. In the early stages there may be sanguineous engorgement, the cut surface may bleed freely, and small extravasations may show throughout the liver substance, later the clay yellow hue, the granular aspect and the absence of blood on the cut surface are characteristic. The margins of the adjacent acini are indefinite or lost, and under the microscope the hepatic cells are charged with granules (albuminoid, fatty and pigmentary), while the nuclei are no longer demonstrable.

In *cattle* the liver may be double the normal size and at first of a deep purple red, which may change later to the earthy yellow.

In *dogs* the liver is tumid and yellow, and marked by small pea-like centres of softening. There is marked softening and the microscope reveals the characteristic degeneration of the hepatic cells.

Symptoms in the Horse. These resemble those of congestion rendered more intense and therefore somewhat less obscure. The attack is usually sudden, there may be rigor followed by hyperthermia, dullness, pendent head, drooping eyelids, injected conjunctiva with a yellowish tinge, unsteady gait and slight indications of colic. There is anorexia, partial suppression of urine, and what is passed is thick, glairy and brownish red, fæces are passed with pain, and groaning, probably from compression of the liver, the heart beats violently, while the pulse is small, breathing accelerated and perspiration abundant. The temperature rises (101° to 106°) and remains high throughout unless lowered through biliary intoxication. Percussion over the liver and especially on the right side shows increased area of dullness and marked tenderness. On the second or third day the icterus usually increases, and a slight fœtid diarrhœa may set in with marked fœtor of the pale or colorless discharges. The jaundice is not, however, a criterion of the danger, as it may become less marked or entirely disappear because of the extensive degeneration of the hepatic cells and the arrest of the formation of bile.

Diagnosis in the horse. The disease is recognized by the coincidence of fever, with great depression, icterus, painful defecation, constipation followed by a fœtid diarrhœa with lack of color in the stools and by increased area of dullness and tenderness in the region of the liver and especially on the right side. From influenza which it resembles in many respects, it is distinguished by the absence of watery discharge from the eyes, and by the entire absence of all indication of contagion. The cases occur one at a time.

Prognosis in the horse. The disease is exceedingly fatal. When the kidneys remain active, the

poisons are eliminated and there may be hope of recovery, but when urine is suppressed an early death by poisoning is to be expected.

Treatment in the horse. A most important indication is to secure depletion from the portal system. Calomel 1 dr., aloes 4 drs., ipecacuan 1 dr. may be given in bolus, and followed by small daily doses of sulphate and nitrate of soda with bitters, with or without the ipecacuan. Action on the kidneys is essential to secure elimination of the poisons which threaten a fatal poisoning if retained. To favor the same action fomentations may be applied to the loins. The frequent presence of pathogenic microorganisms either in the bowels or liver suggests the use of germicides (salol, salicylic acid, salicylate of soda, naphthalin, naphthol, beta-naphthol, etc.) as in catarrhal jaundice. Sinapisms or blisters applied to the right side of the chest and over the short ribs may be useful, and after the subsidence of the more violent symptoms, dilute mineral acids and especially nitro-muriatic acid may be resorted to in combination with diuretics and bitters.

When appetite returns succulent, laxative, non-stimulating food in small quantity should be given. Wheat bran mashes, carrots, turnips, potatoes, apples, fresh grass, ensilage may be adduced as examples. Throughout the disease the ingestion of an abundance of pure water should be encouraged.

Symptoms in the ox. These may appear more tardily than in the horse, loss of appetite, staring coat, dullness, pendent head and ears, unsteady movements, rigors, drivelling of saliva from the mouth and grinding the teeth are usually noted. To these are added the more diagnostic symptoms of slight (or severe) jaundice, constipation followed by a foetid light colored diarrhoea, a strong disposition to remain recumbent, marked suffering attendant on rising, arching of the back when up, and tenderness on percussion over the right hypochondrium. The temperature gradually rises, though more slowly than in the horse, and may again descend under a profound poisoning.

Course. The disease reaches its acme in four to six days, and generally has a fatal issue.

Treatment, is on the same lines as for the horse only as a purgative, sulphate of soda may advantageously replace the aloes.

Symptoms in the dog. The symptoms are those of congestion in an exaggerated form. There are muscular tremors, erection of the hair, followed by rising temperature up to 105° or 106°, an icteric hue of the mucosæ, the pulse is accelerated, strong, irregular, respiration rapid, panting, foetid breath, ventral decubitus, and prostration extreme. Appetite is completely lost, the bowels become relaxed, the stools foetid, the right hypochondrium painful on pressure or percussion, and the urine greatly reduced and icteric or suppressed. This feature of urinary suppression, determines a rapid poisoning and death in two or three days.

Treatment must follow the same lines as in other animals, a purgative of calomel and jalap, followed by diuretics, laxatives, derivatives, and above all germicides. In case of survival mineral acids, aqua regia, bitters, and a carefully regulated diet will be in order.

SUPPURATIVE HEPATITIS. HEPATIC ABSCESS.

Causes in horse: pyæmia, omphalitis, thrombosis, infection, biliary calculi, concretions or parasites, foreign bodies, hot, damp climates, strangles, brustseuche, glanders, endocarditis. Lesions in horse: from parasites and mechanical irritants, pea-like or hazelnut; embolic abscess, pin head to hen's egg; infection from strangles, foreign bodies, etc., may be of large size, and burst into adjacent organs, the peritoneum or externally. Symptoms in horse: of pre-existent malady, remitting fever, successive chills, intermittent icterus, hypochondriac tenderness. Spontaneous recovery, aspiration, opening, antiseptics locally and generally. Lesions in ruminants; secondary multiple abscesses, bean-like or (with foreign body) very large, may extend into adjacent parts. Symptoms in cattle: fever, chills, jaundice, tympany, diarrhoea, dysentery, wasting, tender right hypochondrium. Treatment: as in horse. Causes in dog: foreign bodies, tumors, infections, blows, traumas. Lesions: traumatic abscesses, single, large, infectious abscesses multiple, small. Former fœtid. Symptoms in dog: hepatic congestion or colic, then chills, prostration, irritability, tenderness of right hypochondrium, nausea, vomiting. Treatment in dog: antiseptic aspiration, laparotomy.

Causes in the Horse. Hepatic abscess arises from a great many primary morbid conditions. As a secondary abscess it is seen in the different forms of pyæmia and especially in suppurative omphalitis in young animals. It may start in thrombosis determined by clots or septic matters carried from a distance through the portal vein or hepatic artery, in biliary calculi or concretions, in parasites introduced from the duodenum, in barbs or husks of the cereals that have penetrated through the biliary ducts, or in bacteria or their toxins which have been carried from the bowels, spleen or pancreas. The government veterinarians have found it a comparatively common lesion in the hot damp climate of Hindoostan, and a similar frequency has been noticed in west Africa. Among general affections it is liable to occur in strangles, contagious pneumonia, glanders, endocarditis of the left heart and phlebitis with the formation of thrombi in the lungs. In the two last named disorders, the affection takes place by the simple transference of detached clots to the liver to block its arteries or capillaries. Or it may be that micro-organisms are transferred in the same way. With modern views of suppuration the presence of the pyogenic organisms must be conceded.

Lesions in the horse. Cadeac distinguishes the different types of hepatic abscess as: *1st biliary abscess* in which suppuration commences in the interior of the biliary ducts and usually from parasites or mechanical irritants introduced or from calculi or concretions formed within them: these rare abscesses contain biliary salts, pigments, and epithelium and acquire the size of a pea or hazelnut: *2d Metastatic abscesses* which start in the arterial, portal, or capillary vessels, by the arrest of infecting clots, which determine a further clotting, the obstruction of the vessel, the accumulation of leucocytes and the formation of abscess of the size of a pin head or larger up to a hen's egg, surrounded by a hæmorrhagic infarct softening in the centre: these are numerous disseminated through the liver: *3d Mechanical Abscess* due to the penetration of foreign bodies or parasites: *4th Infection* as in strangles. These may attain a large size, cause adhesion to adjacent organs, and rupture into the chest, the colon, stomach or peritoneum. The pus may even escape externally through the right hypochondrium.

Symptoms in the horse. These are always obscure and vary much with the source of the malady. If there has been a pre-existing hepatic malady the symptoms of that will be in evidence; if an omphalitis its existence may still be recognizable; if pulmonary or cardiac disease, that may be detected; if parasites, evidence of their existence may perchance be found; if gall stone, a previous violent hepatic colic with icterus may have occurred; and if intestinal septic disorder, there may be the testimony of intestinal troubles. The more diagnostic symptoms are a fever of a remittent type, one or several violent shivering fits, a marked jaundice which like the fever shows exacerbations, and a similar irregularity of the condition of the urine which may be successively of a dark brown, a deep yellow, and a transparent amber color. Tenderness and grunting on percussion of the right hypochondrium would be an additional aid in diagnosis.

Treatment. Death has been hitherto considered as the inevitable result, yet recoveries may ensue after rupture into the colon or through the abdominal walls. If the seat of the abscess can be ascertained its evacuation through an aspirator and the subsequent injection of an antiseptic would be appropriate. The concurrent use of antisympurants like hyposulphite of soda, or sulphide of calcium would also be in order.

Causes in Cattle. Hepatic abscess is much more frequent in cattle, and is commonly a result of perforation by sharp pointed bodies (needles, pins, nails, wires, etc.) from the reticulum and rumen, or of parasites, or biliary calculi. Other cases are occasioned by the presence of tubercles, actinomycosis, or omphalitis.

Lesions in Cattle and Sheep. Secondary abscesses are usually multiple and disseminated through the organ, though Cadeac says they are more common in the left half. They vary in size from a bean to a pigeon's egg, project often from the surface, and contain a viscid, creamy, yellowish or greenish pus. Abscesses dependent on foreign bodies often attain a great size, so as to contain a pint or quart of pus (Landel). They may make their way through the diaphragm, rumen, or abdominal wall leaving a thick cicatrix in the liver, or they may become slowly absorbed and dry up into a putty-like or cretaceous mass. Brusaferro found hepatic abscesses in lambs twenty to thirty days old—probably of omphalic origin.

Symptoms in Cattle are usually very obscure. Fever, shivering fits, jaundice, indigestion, diarrhoea or dysentery, emaciation, colics, tender right hypochondrium, and peritonitis may all be in evidence but the diagnosis is little better than a guess.

Treatment when possible at all would be on the same lines as for the horse.

Causes in the dog. According to Cadeac these are mostly foreign bodies (needles, pins, etc.) which have been swallowed, tumors of the liver or adjacent organs, phlebitis and thrombosis of the portal vein, pyæmia, septicæmia, and external injuries (kicks, blows, contusions, falls, etc.)

Lesions in the dog. As in the other animals *traumatic* abscess is usually solitary and large, *secondary* abscess multiple and small. The pus developed around a foreign body is reddish, greenish and fœtid, that of the metastatic abscess is usually whitish or yellowish and with a sweet odor.

Symptoms in the dog are those of hepatic congestion, or violent gall stone colic, followed by severe rigor, great depression, or irritability, and tenderness over the right hypochondrium. Nausea and vomiting is a marked symptom though not a diagnostic one.

Treatment. If the flaccid abdominal walls will allow of the locating of the abscess it should be treated by aspiration and antiseptic injections. It would even be admissible to perform laparotomy, stitch the wall of the abscess to the external wound, and empty it under due antiseptic precautions.

INFECTED HEPATITIS. NODULAR NECROBIOSIS OF THE LIVER.

In ox, sheep, pig, dog, horse. Necrotic areas projecting on surface of liver. Causes: bacteria, toxins, from bowels, womb, navel. Lesions: In cattle dirty gray nodules in brownish red liver, nodules firm, granular, necrotic, elements do not stain, later leucocytes and fibro-plastic growth in periphery. In lambs the nodules are white, common to the lungs and pleura, pathogenic to rabbit. In pigs nutmeg liver, cells without nuclei, fatty, granular, pathogenic to rabbits, guinea pigs, rats and young pigs. In dog, nutmeg liver, with violet areas, and white spots, 1–2 lines, having granular, fatty cells without nuclei. Symptoms: fever, constant lying, tarry fæces, icterus, tender right hypochondrium, and those of the primary disease. Treatment: antiseptics of primary seat, and bowels, elimination by kidneys, general antiseptics, stimulants, etc. Case usually hopeless. Prevention.

This has been observed particularly in cattle, but also in sheep, pig, dog and horse. It is characterized by the formation of circumscribed areas of gangrene, becoming hard, dry, yellowish and usually slightly projecting beyond the adjacent surface. Its infected character is shown by the presence in the lesion and adjacent parts of the hepatic tissue of an abundance of bacteria, which, from the varied description, appear to differ in different cases. The cause may however be safely stated as one of the bacteria of gangrene. It is alleged with some show of reason, that the lesion may be determined by the action of toxins and ptomaines produced by bacteria in the alimentary canal and carried to the liver with the portal blood (Cadeac). The bacteria themselves commonly come from the same source, (Stubbe), but also from the uterus (Berndt), the mammæ (LeBlanc), and above all from the suppurating or septic umbilicus. McFadyean in five cases found a long slender bacillus, Hamilton in a single case in the horse found cocci, Rivolta in an infectious hepatitis in sheep found *bacterium subtilis agnorum*, and Semmer found the same condition in young pigs from micrococci introduced through the diseased umbilicus.

Lesions. In cattle the liver has a general brownish red, or greenish white color, and shows projecting, hard nodules of a dirty gray color more or less tinged with yellowish brown. The margins of these hard nodules are very sharply defined, and on section show a homogeneous granular surface, devoid of areas of softening or of connective tissue, and formed of the hepatic parenchyma in a state of necrobiosis. The granules and nuclear elements do not stain like those of healthy liver. As the disease advances the periphery of the nodule may be invaded by leucocytes and become the seat of a fibro-plastic hypertrophy (McFadyean) with the ultimate formation of cicatricial tissue (Stubbe).

In lambs Rivolta found the necrosed nodules standing out as white patches under the capsule of the liver, but similar lesions were met with in the lungs and pleuræ, an observation which has been confirmed by Hanbold. The affection was conveyed by inoculation to the rabbit.

In pigs Semmer found nutmeg liver, deep red or grayish yellow, hypertrophied, the hepatic cells swollen and divested of nuclei but containing fatty and pigmentary granules. It was inoculable on rabbits, guinea pigs, white rats and on young pigs.

In the dog, Courmont and Doyon found congested liver (portal congestion) with projecting patches of a deep violet color and sharply defined borders, and one to two lines in diameter, also salient white spots with distinct outlines. In the white spots the hepatic cells had lost their nuclei and were charged with fatty granules.

Symptoms. These are indications of hepatic disease. In *parturient cows*, Berndt noted fever (102° to 104°), anorexia, stiffness, cough, labored breathing, intense thirst, constant decubitus, and constipation followed by lowering temperature, tarry fæces and icterus. The region of the liver was very sensitive to pressure or percussion. In the other animals the symptoms appear to be largely over-shadowed by those of the primary disease, but the same general indications of jaundice, hepatic tenderness and digestive disorder are superadded.

Treatment when it can be intelligently adopted, consists largely in evacuation and antiseptics of the seat of primary infection, and of the *prima viæ*, and in maintaining elimination by the kidneys. In this way, as in congestion and hepatitis, the concentration of the poison is as far as possible counteracted, and an opportunity may sometimes be furnished for the recuperation of the liver cells. As a rule, however, the case is hopeless, and thus preventive measures, by cleanliness, disinfection and antiseptics of the ascertained sources of the infection are indicated.

PERIHEPATITIS.

Inflammation of capsule of liver (external and Glisson's). Causes: Traumas, infective diseases, phlebitis of the portal vein, chill, distomatosis. Lesions: Peritonitis and inflammation of the capsule in patches, yellowish gray exudate, fibroid thickening or pus. Adhesions to adjacent objects. Thickening of trabeculæ. Symptoms, tardy respiration and circulation, tender hypochondrium, colics, diarrhœa, painful defecation, moan with expiration. Slight cases recover. Sequelæ: compression of portal vein or bile duct, gastric catarrh, piles, etc. Treatment: Salines, alkaline diuretics, mineral tonics, bitters.

This is inflammation of the external capsule of the liver and Glisson's capsule. It may arise from direct mechanical injury, or by extension of inflammation from adjacent structures, such as the peritoneum. It may also complicate contagious pneumonia in the horse, tuberculosis in the ox, pneumoenteritis in pigs, and also phlebitis of the vena portæ (Cadeac, Morot). It may follow a chill, or distomatosis.

Lesions. These are essentially peritonitis circumscribed by the liver, and extending to the proper capsule, and its vaginal investments of the hepatic vessels. It is usually limited to certain spots which become the seats of a yellowish gray exudation, with a tendency to fibroid development and thickening, but sometimes degenerating into pus. The deposits on the outer side of the hepatic peritoneum may develop false membranes and fibrous adhesions to surrounding objects, the diaphragm, omentum, stomach or intestine. The deposits under the peritoneum lead to similar fibrous development with hypertrophy or thickening of the capsule, the trabeculæ extending thence into the liver and the vaginal sheaths of the vessels. Such areas of thickening are revealed as depressed spots or patches of a white color, and showing a firm fibrous, pearly appearance when incised. Such lesions are not uncommon in the livers of horses, cattle and swine. In the pig they may have a violet, or brownish red color, but with spots of other colors—grayish or brownish (Kitt).

Symptoms. Dopheïde, who has studied the disease in cows and to a less extent in horses, found a reduction in pulsations (26 per minute) and respirations (6 per minute) in connection with a mild peritonitis, intestinal catarrh, colicky pains and diarrhœa. The conjunctiva is pale, the pulse compressible, the respirations unequal and accompanied by a moan, and the appetite impaired or lost.

If confined to mere spots on the liver, a restoration to apparently vigorous health may take place, but if extensive it may lead to compression and obstruction of the portal vein or bile duct, or to compression and atrophy of the liver, with corresponding symptoms.

Treatment. As in other congestions of the liver, the use of salines to deplete the portal system, and of alkaline diuretics are especially indicated, to be followed by bitters and mineral acids. Sinapisms and other counter-irritants to the region of the liver are of great service. If not complicated with abscess, or microbial infection, cases of this kind will often do well.

CIRRHOSIS OF THE LIVER. FIBROID DEGENERATION.

Definition. Increase of connective tissue, decrease of gland parenchyma. Causes: in man, alcoholism; in animals, chronic heart disease, chronic recurrent perihepatitis, biliary obstruction, toxins. In horses: age, emphysema, unwholesome fodders, vegetable alkaloids, infection. Symptoms: prostration, hebetude, impaired appetite, colics, constipation, later diarrhoea, unthriftiness, emaciation, dropsy, icterus, ascites, intestinal catarrh, tender hypochondrium, early fatigue. Lesions: increase of connective tissue, compression and absorption of parenchymatous tissue, greatest around portal vessels, thickening of fibrous stroma between capillaries of acini, shrunken, granular, pigmented liver cells. Treatment: salines, Glauber salts, diuretics, sodium carbonate, or iodide, or salicylate, derivatives, mineral acids, bitters, open air, laxative food, pure water. In cattle: obstruction to circulation or the flow of bile; advances from the vessels, causes absorption, caseated foci, adhesions, enlarged liver. Symptoms: jaundice, yellow, red, albuminous urine, chronic indigestion, tends to fatal though slow advance. Treatment: green food, open air life, saline laxatives, alkalies. In dog: common following heart disease, parasites, bacteria. Lesions: Congested brownish red liver, fibroid increase from Glisson's capsule, compression of acini, their elevation above surface, fatty and pigmentary degeneration of hepatic cells, increasing sclerosis. Symptoms: as in parenchymatous hepatitis with slower advance, in time tender loins, brownish or reddish urine, ascites, intestinal catarrh, it may be icterus. Treatment: Correct cardiac troubles, digitalis, strophanthus, and intestinal, careful diet, mineral acids, bitters, pure water, saline laxatives, antiseptics, alkaline diuretics. Potassium iodide. Derivatives. Draw off liquid. Laxative non-stimulating diet.

Definition. An interstitial inflammation of the liver characterized by a great increase of the connective tissue and compression, atrophy and degeneration of the glandular elements.

The same final result may undoubtedly originate in various different primary morbid processes.

In man cirrhosis is looked upon as almost always the result of abuse of alcohol. In animals this cannot be the case, apart from a few kept in connection with breweries or distilleries.

In heart disease a long continued mechanical congestion of the liver causes compression and degeneration of the secreting cells in the centre of the acini (around the intralobular veins), while the peripheral portions undergo cell proliferation and increase of connective tissue.

In chronic or recurrent perihepatitis, a whole lobe may be compressed by the hyperplasia of the investing connective tissue, and the hepatic cells are degenerated and absorbed.

Overdistension of the biliary ducts from obstruction to the flow of bile (gall stone, catarrhal inflammation, constipation), leads to proliferation and hyperplasia in the walls of the biliary radicals throughout the entire liver.

The presence in the liver of toxic agents, ingested, or generated from microbial fermentation in the intestinal canal or liver is another recognized cause of connective tissue hyperplasia.

CIRRHOSIS IN THE HORSE.

Cirrhosis of venous origin has been observed mainly in old horses, while hypertrophic cirrhosis from biliary obstruction occurs rather in the young (Cadeac). Bruckmüller records a case of the first kind in a horse with extreme pulmonary emphysema. Walley gives a bad condition of fodders as the main cause, virtually implying, in many cases, infective catarrh and obstruction of the biliary ducts.

A form of the disease prevails at Schweinsberg in Hesse, and has been variously attributed to spoiled fodders (Nicklas), to vegetable alkaloids and other poisons in the food (Friedberger and Fröhner), to clover, to telluric poisons (Redner), to infection (Meminger), and to heredity (Neidhardt). It is a suggestive fact that it is confined to the valleys of the Ohm, Glon, and Zusam where the land is peaty or swampy and subject to inundations, while it is unknown on the dry table lands (Friedberger and Fröhner). This strongly suggests intoxication with microbes or their deleterious products. The gastric catarrh that frequently attends the disease may point in the same direction.

Symptoms. These are too often general rather than diagnostic. Dullness, prostration, hebetude, yawning, hot, sticky mouth, lost, irregular or depraved appetite, colics, constipation or diarrhoea, dry, harsh coat, emaciation, weakness, oedema of the limbs, vertigo and drowsiness may be among the symptoms. More characteristic are icterus, abdominal distension from ascites, or congestion of the liver, yellow or high colored urine, intestinal catarrh, indigestion, and tenderness in the region of the liver. The mucosæ are usually pale at first and not always icteric later. On exertion the horse shows early fatigue, tumultuous heart beats and oppressed breathing.

The Schweinsberg disease often lasts for months, with alternate improvements and exacerbations, but almost invariably ends in death, and sometimes completely depopulates a stable.

Lesions. These consist primarily in the great increase of the connective tissue and the relative decrease of the hepatic tissue. This is usually mostly around the divisions of the portal vein and the periphery of the acini, but also in the end around the hepatic veins as well. When it has formed around the biliary canals there is a great increase of the liver (often doubled) and its edges have become rounded. Within the acini the increase of the fibrous stroma is seen between the radiating capillaries, and the hepatic cells are contracted, granular, pigmented, and comparatively destitute of protoplasm around the still persistent nucleus.

Treatment. Glauber salts to clear the bowels of offensive matter, and deplete from liver and portal vein, bicarbonate of soda or iodide of potassium to eliminate the poisons through the kidneys and to lessen the induration, and finally salicylate of soda as a liver stimulant and intestinal antiseptic are suggestive of the line of treatment that may be pursued. The saline laxatives and diuretics, and antiseptics may be changed for others according to special indications, and bitters and mineral acids may be resorted to. Counter-irritants to the right hypochondrium should not be neglected in case of local tenderness. In the otherwise fatal Schweinsberg disease, Imminger, Künke and Stenert had a remarkable success from the free use of potassium iodide, which suggests a cryptogamic origin, as this agent is so valuable in polyuria which results from musty fodder. In all cases, gentle exercise in the open air and a moderate ration of laxative food (green) are of great value. Above all the old suspected diet should be carefully avoided, also any impure water supply.

CIRRHOSIS IN CATTLE.

This has been recorded by different observers and usually as the result of some obstacle to the circulation, or of catarrh and obstruction of the biliary passages. Morot saw it in young calves, which showed greatly enlarged liver (in one case 24 lbs.) and kidneys, the former containing numerous cysts and marked sclerous thickening around the vessels. This advancing thickening of the connective tissue, causes increasing firmness of the liver and absorption, distortion and diminution of the lobules. Albrecht describes a chronic interstitial hepatitis with caseated centres (nontuberculous) many of them an inch in diameter. The liver is brown or grayish with whiter callosities which extend into its substance and make points of attachment to the diaphragm or other adjacent organ. The contrast between the fibrous layers and the hepatic tissue has been likened to a checker board (Höhmnn). The enlarged liver may weigh 30 lbs.; in one remarkable case it weighed 300 lbs. (Adam). The bile is of a light color and mixed with mucus.

Symptoms. The symptoms are indefinite: a gradually increasing jaundice, the passage of yellowish red urine becoming more and more red and albuminous, and finally coagulating on the walls of the urethra or on the litter, chronic indigestion, salivation (Schäffer), weakness, breathlessness and more or less fever may give indications of the disorder. Höhmnn failed to find tenderness of the right hypochondrium. The disease is liable to go on to a fatal issue, so that it is often sought to prepare the animal for the butcher.

Treatment will follow the same line as in the horse. Green food, pasturage, open air life, saline laxatives, and alkalies with a free use of potassium iodide to check the sclerosis will be indicated.

CIRRHOSIS IN THE DOG.

In the dog, cirrhosis is much more common than in the larger animals, in connection with idle pampered habits, the frequency of diseased heart and consequent disturbance of the circulation, and the presence of parasites in the liver or biliary ducts. Bacteria intoxication and infection are also common.

Lesions. The liver is at first tumefied, with hard consistency and rounded edges, and a deep brownish red color, but this is modified by the grayish fibroid hyperplasia which is especially abundant in and around the vaginal sheaths of the capsule of Glisson. In cases arising from diseased right heart or lungs the induration is rather concentrated around the hepatic veins. The contraction and shrinking of the fibroid hyperplasia as the disease advances causes the projection of the hepatic tissue in minute rounded elevations which give a peculiar uneven appearance to the surface of the organ. The fibroid growth gives a remarkable hardness to the liver which resists even the edge of a knife. The hepatic cells are the seat of fatty and pigmentary degeneration. Inflammation and tumefaction of the kidneys, and ascites are common features of the malady.

Symptoms. The general symptoms are as in parenchymatous hepatitis with a more tardy development. There are impaired or irregular appetite, dullness, sluggishness, in an obese animal short-windedness or palpitations on slight exertion, symptoms of disease of the heart, lungs or digestive organs, a spasmodic cough, constipation followed by relaxation of the bowels, nausea and vomiting. As the disease advances tenderness of the loins, the passage of brownish or reddish, albuminous urine, the formation of ascites and of gastro-intestinal catarrh may be noticed. Icterus may be entirely absent, but, with a flaccid abdomen, enlarged liver and spleen may be detected.

Treatment. The indications are to first combat the causes. Irregularities in the heart's action may be met by digitalis or strophanthus; gastro-intestinal catarrh by a carefully regulated diet, with mineral acids and bitters; portal congestion by a free use of water and other diluents and by saline laxatives; intestinal fermentations by antiferments (salol, naphthol) and toxic matters in the blood by alkaline diuretics. For the liver hyperplasia, potassium iodide may be freely used. Blisters to the right side will occasionally prove useful. The ascitic fluid must be drawn off when it accumulates. A diet of milk, bread and milk, buttermilk and mush, or one in which albuminoid elements are in minimum amount and the action of which is laxative is to be preferred. Out door exercise is desirable.

CHRONIC ATROPHY OF THE LIVER.

Chronic Atrophy: In *old horses*: in right and spigelian lobes; others show hypertrophy. In *ruminants*, *omnivora* and *carnivora*: in areas compressed by tumors or parasites. Perihepatitis. Sclerosis. Remedy causes if possible. *Fatty Degeneration:* Oil globules in liver cells, pathological when they destroy the protoplasm. In ducks and geese on forced feeding. Causes: poisoning by phosphorus, arsenic, antimony, lead, phenol, iodoform, alcohol; excess of fat in food, spoiled fodders, colchicum autumnale, yellow lupins, bacteria, hemorrhages, inflammations, tumors, parasites; improved meat producing breeds, old animals, hot stables. Lesions: liver enlarged, pale, yellow, bloodless, knife in cutting is smeared with fat, oily stain on paper, liver cells enlarged, protoplasm replaced by fat or oil; may be circumscribed. Symptoms: obesity, over-fed in fats and starches, of fattening breed, kept in confinement, in hot moist environment, if fed certain poisons, with costiveness and indigestion, no endurance, short winded, slight icterus, scanty urine, little urea, later, emaciation, palpation of enlarged liver. Treatment: send to butcher, pampered horses, cows from swill stable, a run at grass, with shade trees, a poor pasture, salines, cholagogues, mineral acids, bitters, iron with alkalies, currying, massage, douches.

Acute yellow atrophy has been referred to under parenchymatous hepatitis but a chronic atrophy is also met with in all domestic animals.

In *old horses* it affects, by preference the right and spigelian lobes, the portal circulation of which is less direct because of the veins of supply leaving the parent trunk at right angles (Leblanc), and because these lobes are more exposed to compression by solid accumulations in the double colon (Kitt). In such cases a compensatory hypertrophy of the left and middle lobes is often observed.

In *ruminants* the lesion is often circumscribed to the areas that have undergone compression by tumors or parasites (echinococcus, actinomycosis), and there may be compensatory increase elsewhere in the organ.

In *swine*, *dogs* and *cats* the same conditions are operative. In all alike perihepatitis may be a causative factor, and sclerosis (cirrhosis), with contraction of the fibrous hyperplasia may also operate.

Symptoms are very obscure and *treatment* unsatisfactory unless the active causes can be recognized and arrested.

HEPATIC STEATOSIS. FATTY LIVER. FATTY DEGENERATION.

The presence of oil globules in the liver cells is normal and physiological, the liver acting to a certain extent as a store-house for fat. This is always a marked feature, in healthy animals on high rations, and taking little or no work, but so long as the protoplasm and nuclei of the cells retain the normal characters and functions the condition is not a morbid one. It may, however, become excessive, with great enlargement of the liver, and with the substitution of fatty granules for the protoplasm of the cells as in ducks and geese subjected to forced feeding, and the condition becomes a distinctly pathological one.

In true fatty degeneration the protoplasm of the hepatic cells is destroyed and replaced by fatty granules, the resulting condition being a permanent destruction of the cell for physiological uses.

Causes. The liver cells undergo fatty degeneration under the action of certain poisons like phosphorus, arsenic, antimony, lead, phenol, iodoform and alcohol. According to Neyraud oxide of antimony is given daily to fattening geese to hasten the development of fatty liver.

An excess of fatty elements in the food leads to the same result as shown first by Majendie in dogs, in which not only did the liver undergo this degeneration but the sebaceous glands of the skin secreted an excess of volatile fatty acids.

The cryptogams and their products on musty fodders determine a gastro-enteritis in herbivora, accompanied by fatty degeneration of the liver.

Colchicum Autumnale, and poisonous yellow lupin both determine this degeneration.

The products of a number of pathogenic bacteria have a similar effect. This has been noticed in the cat with bacillus pyocyaneus (Charrin), the cholera spirillum, pyæmic and septicæmic infection, contagious pneumonia of the horse, strangles, and ulcerative endocarditis. It has been long noticed to be a complication of pulmonary tuberculosis, the result in this as in other affections of the lungs having been attributed to lessened oxidation in the tissues. It occurs also in hæmorrhages, ruptures and inflammations of the liver and in passive congestions of the organ, the impairment of the normal functions (in the altered conditions of nutrition, or under the influence of poisons,) proving an important factor in the process. The same remark may apply to the fatty degeneration which complicates most other liver diseases, cirrhosis, catarrh of the bile ducts, distomatosis, echinococcus, carcinoma, and epithelioma.

Certain other factors must be taken into account. The inherited disposition to the production of fat which characterizes the improved breeds of butcher animals, and particular individuals of all breeds, mature age which predisposes to the deposit of fat in internal organs, old age which lessens the vitality of the cells, and hot, damp climates or stables, all operate more or less in determining the fatty change.

Lesions. In fatty degeneration the liver is enlarged, pale, bloodless, yellowish, its cut surface exudes an oily fluid which smears the knife, and it is so light that it floats on water. If scraped and the material drawn across a sheet of paper it forms a transparent oily stain. Under the microscope the liver cells are seen to be enlarged and to have their protoplasm and nuclei replaced by fat or oil. If due to obstruction in the heart or lungs the degeneration is greatest toward the centre of the acinus, if due to an infectious disease it is usually greatest towards its periphery. In infectious diseases too the liver is not pale yellow, but usually of a deep brownish or yellowish red. The degeneration may be local or general. McFadyean found a circumscribed lesion in an ox's liver, of a bright ochreous color, and the cells completely transformed into fat cells, while the rest of the liver was sound. In the dog fatty areas, up to an inch in diameter, are not uncommon. The swollen cells pressing on the adjacent vessels, account for the bloodless condition, and favor the degenerative process.

Neyraud records a fatty liver of 28 lbs. weight from the horse, and Kitt one of 10 lbs. from the pig.

Symptoms. Like as in most chronic liver diseases the indications are uncertain. The conditions may, however, suggest fatty degeneration; if the patient is very obese; if it has had an abundant food, rich in hydrocarbons and carbohydrates, and little exercise; if it has received in food or water continuous doses of phosphorus, arsenic or antimony; if it has lived in a hot moist climate or

stable; if there has been a tendency to costiveness and indigestion; if the patient is weak, easily fatigued and short-winded; if there is a slightly yellowish red tinge of the conjunctiva and if the urine is scanty and contains little urea. If the disease is more advanced and the animal emaciated, it may be possible in the smaller animals at least to manipulate the liver to make out its increase, its smooth surface, and its absence of tenderness.

Treatment. When met with in meat producing animals the best resort is to turn these over to the butcher. When in an animal which is mainly valuable for breeding purposes, or in horses or carnivora, something may be done to check the progress of the malady, and maintain at least the present condition. The value of this will of course depend on how far the disease has already progressed. Cows that have spent a winter in a hot swill stable are of little use afterward for breeding or dairy uses and advanced cases of fatty degeneration in the horse or dog hold out little hope of a satisfactory issue. For cases in the earlier stages, nothing can be better than a run at grass, where there is opportunity for shelter from the noonday sun. If the pasture is short and the animal has to exercise to secure a living, so much the better. If kept indoors the patient should have a clean, roomy airy box stall, with a moderate allowance of easily digested food, and laxatives and cholagogues daily such as Glauber salts, aloes, calomel, podophyllin or cream of tartar. Mineral acids, especially nitro-muriatic acid, and bitters may also be given. The preparations of iron are sometimes useful in maintaining the tone of the digestive organs and counteracting anæmia but they must be conjoined with diuretic doses of bicarbonate of soda.

There is great advantage in stimulating the skin, and active brushing, currying, hand-rubbing, and even cold douches may be resorted to.

AMYLOID DEGENERATION OF THE LIVER.

Degeneration of basement substance of connective tissue, swollen, transparent, homogeneous, colored mahogany brown by iodide. In wasting diseases, tubercle, cancer, malaria, dysentery, leukæmia, suppuration, ulceration, pleurisy, pericarditis, peritonitis, chronic catarrh, broncho-pneumonia, orchitis, biliary calculi, nephritis. Chronic. Lesions: Affected part swollen, sinks in water, bloodless, clear, smooth, homogeneous, yellowish or reddish gray, under compound solution of iodine becomes mahogany brown, under sulphuric acid dark violet. Extends from vessel walls to adjacent connective tissue. Symptoms: Of wasting diseases, but not diagnostic. Treatment: Unsatisfactory, directed to causative disease.

This is a condition in which the basement substance of the connective tissue, and especially of the walls of the vessels, becomes swollen and composed of a transparent, homogeneous substance, albuminous in character, and which stains of a deep mahogany brown on the application of a solution of iodine. The degeneration is usually associated with severe wasting diseases, in the human being with tuberculosis, syphilis, malignant tumors, malarial infection, dysentery, leukæmia, and chronic suppuration or ulceration, especially of the bones.

In the *lower animals* (horse, dog, ox, sheep, rabbit, poultry) it has been seen to attend or follow on similar cachectic conditions. In the *horse* it has been seen in connection with the effusions of pleurisy, pericarditis and peritonitis (Rabe), in chronic bronchial catarrh (Fischkin), in chronic broncho-pneumonia, and dilated right heart (Trasbot), in orchitis, phlebitis and cachectic states (Caparini), and in calculous obstruction of the biliary duct (Burgoin). In *cattle* it has accompanied chronic nephritis (Brückmüller), tuberculosis, leukæmia, etc. In *lambs* kept in confined stables, though well fed on oats (Werner). In long standing suppurations and in animals fed on distillery still it has been observed.

It may last for months or years, and predispose to other disorders, functional and structural. It does not, however, interrupt secretion as bile continues to be formed.

Lesions. The affected part of the liver is enlarged, the entire organ in the horse may amount to 32 lbs. It is smooth and even, though thick and rounded at its inferior border, yet occasionally on the posterior aspect there may be hyperplasia and a rough irregular surface. The diseased liver is heavy and sinks in water, unlike the fatty liver. In the horse it is soft and friable or even pasty whereas in man it is firm and resistant. The cut surface is bloodless, smooth, clear, homogeneous and grayish, yellowish or reddish gray. When treated with a solution of iodine and potassium iodide it changes to a deep mahogany brown; if dilute sulphuric acid is then used it changes to a deep violet, almost black color. If the iodine solution is brushed over the smooth cut surface the mahogany color of the amyloid stands out in marked contrast with the bright yellow of the healthy hepatic tissue. The amyloid commences in the walls of the smallest arteries, in the media and intermediary layers of the intima, and thickens the walls so as to obstruct their lumen more or less completely and render the part comparatively exsanguine. It may extend to the connective tissue of the organ, but it is not certain that the hepatic cells are involved in the process. The cells are, however, pressed upon by the diseased vessels and stroma and undergo consequent fatty degeneration. The amyloid may be confined to but a small part of the liver or to its smaller blood-vessels or it may extend to the whole. In fowls it is always in multiple centres (Leisering). It may be found in other important organs, kidneys, spleen, lymphatic glands, intestinal mucosa, etc.

Symptoms are not diagnostic. If with an old standing, exhausting disease, paresis, weakness, emaciation and unfitness for work, there is loss of appetite, dryness of the mouth, congestion of the rectal mucosa, yellowish, whitish, or dark tarry fæces, and a slightly brownish or yellowish tinge of the visible mucous membranes (Rexante) it may be suspected. In fowls Leisering noticed, weakness, lameness, ruffling of the feathers and attacks of vertigo. Icterus, ascites and tenderness over the region of the liver may all be absent. In the absence of ascites, tympany, or an excess of fat in the smaller animals, manipulation may detect the considerable enlargement of the liver, and the characteristic smoothness, of its surface. In other cases some indication may, at times, be had from the increased area of dullness on percussion.

Treatment is essentially unsatisfactory even if a correct diagnosis can be made. The most hopeful course would be to correct the debilitating disease in which the amyloid seems to have originated. Diseased bones, ulcers, chronic suppurations, and catarrhs may be done away with, and at least

any further advance of the degeneration arrested. Open air exercise and a green or otherwise laxative diet would be indicated. The amyloid in lambs fed on oats was corrected by a change of diet (Werner). As medication the alteratives, potassium iodide and potassium arseniate have been mainly resorted to. Bitters and iron may also be of use to build up the strength. The latter should be given with potassium bicarbonate.

BLACK PIGMENTATION OF THE HEPATIC CELLS. BROWN ATROPHY.

In horse. With melanoma and atrophy, or without, pigment granules fill hepatic cells, liver becomes brown or black. In calves. In sheep. Apart from melanosis, the real cause unknown.

The accumulation of granules of black pigment in the hepatic cells has been noticed in old and worn out horses (Louis Blanc, Cadeac, Bruckmüller), in calves (Degive, Cadeac), and in sheep (Siedamgrotzky, Barrier). In *horses* it has been found in connection with atrophy, or in other cases, with melanotic tumors in other parts. In atrophic cases the liver is small, puckered, brown and dull, with a leathery appearance on section, and with the hepatic cells charged with pigment granules so that each acinus has a stellate appearance from the radiating lines of cells. This constitutes *brown atrophy*.

The second form which may be called *melanotic liver*, is not associated with atrophy, but is characterized by the crowding of the hepatic cells with black pigment granules, which fill up the protoplasm and crowd the still pale nucleus to one side. The affected portions become of a deep black.

In *calves* the pigmentation may be confined to the superficial portion of the liver (Degive).

In *sheep* pigmentation may be in the peripheral cells only of the acinus (Cadeac) but is about equally distributed on the surface, and throughout the interior of the liver, and may extend to the stroma of the gland (Siedamgrotzky).

Apart from the general causes of melanosis, benignant or malignant, no definite reason for this pigmentation has been assigned. The development of melanæmia and tissue pigmentation in man from malarious microorganisms suggests that other germs and their products may have a similar effect in the lower animals but nothing certain is known as to the true cause.

Apart from melanosis, it is not known that this pigmentation of the hepatic cells is of any essential pathological importance. It is important however for the veterinarian to be acquainted with the condition, that he may intelligently deal with such lesions whether seen in ordinary post mortem examinations, or in the course of meat inspection.

DILATATION OF THE GALL BLADDER AND BILE DUCTS.

Causes: obstruction of common bile duct, distoma, round worms, tæmiæ, gall stones, encrustations, inflammations, tumors, cicatrices, hydatids. Congenital absence. Ducts stand out on liver. Symptoms of colic, icterus, bile poisoning, marasmus. Treat the causative conditions.

This may occur in all our domestic animals except solipeds in which latter there is no gall bladder.

Causes. Any serious obstruction to the discharge of the bile into the duodenum may cause it. The presence of trematodes, nematodes, or even tæniæ in the ducts, gall stones, incrustations, occlusion of the ducts by inflammatory swelling, tumors of the liver or adjacent parts, echinococcus, cysticercus, or cicatrices may be cited. Cadeac mentions a case of congenital atresia of the bile duct in the calf. Vigney records a case in the cow in which the greatly dilated gall bladder formed a hernial mass in the epigastric region which was, however, easily reduced by manipulation.

In all such cases the distended bile ducts stand out as white branching lines on the back of the liver converging toward the portal fissure. The walls of the ducts may be attenuated or thickened and it is alleged calcified. They are usually lined by a deposit of cretaceous consistency precipitated from the retained bile. The contents of the distended ducts and bladder are variable. They may have the color (yellow, green) and consistency of bile; they may be thick, dense and albuminous; they may be thin and serous from inflammatory or dropsical exudation; they may be granular, or purulent.

Though there is no gall bladder, in the soliped, a similar condition of the biliary ducts may be produced in the same way.

According to the degree of obstruction there may be more or less acute symptoms of biliary colic, icterus, marasmus, poisoning by bile acids, etc.

Treatment must be directed toward the removal of the special cause of dilatation.

DOUBLE GALL-BLADDER.

As a congenital formation the gall bladder is sometimes divided into two at its fundus, and in other cases the division extends throughout, forming two complete sacs. This has been found in the sheep, cat, ox (Gurlt, Goubaux) and pig (Goubaux). Such a redundancy does not interfere with normal functions.

CHOLELITHIASIS. BILIARY CALCULI. GALL STONES.

In ruminants, omnivora, and carnivora, less often in horse, 1 to 1000, or more, biliary sand to marble or more, casts; nucleus, bile pigment, blood mucus, etc., with concentric layers of cholesterine, pigment, salts and lime. Causes: idleness, overfeeding, dry feeding, thickening of bile, colloids, bacteria, wintering in stable. Colloids induce globular deposits. Parasites. Inflamed biliary epithelium secretes excess of cholesterine and lime. Age. Acid bile, constipation. Diseased liver. Lack of bile salts. In *solipeds*: all sizes and numbers, green, brown, yellow or white. Liver disease. Symptoms: colic with depression, prostration, yellow or red urine, icterus, sudden recovery. Treatment: sulphate of soda, olive oil, antispasmodics, warm drinks, fomentations, salicylate of soda, chloroform, pasture, or succulent food, and open air exercise. In *cattle*: musk odor; calculi dark green, yellowish green, whitish or orange, dry red, all sizes and numbers. Causes: dry winter feeding, etc. Lesions: dilated biliary ducts and bladder. catarrh, thickening, rupture, septic peritonitis. Symptoms: relapsing colic, with icterus, prostration, and tender right hypochondrium, in stalled animal on dry feeding. Suggestive: not pathognomonic. Treatment: as in horse: succulent spring pasturage. In *sheep*: rare, musky odor; concretions and casts common in distomatosis. Hypertrophied ducts. Symptoms of distomatosis. Treatment: for distomatosis and calculi. In *swine*: rare: circular. In *carnivora*: round, dark green, pin's head to hazel nut. Symptoms: colics, constipation, emesis, icterus, tender right hypochondrium, concurrent catarrh of bowels, heart disease, dyspnoea, sudden relief. Mode of relief. Treatment: olive oil, bile, sodium sulphate, or salicylate, antispasmodics, alkalies, enemata, fomentations. Laxative food, exercise, open air.

Gall stones are most frequent in animals having a gall bladder. Some medical writers say they are formed in the gall bladder only, but the soliped which has no gall bladder has in particular instances furnished hundreds of gall stones. Yet the ox, dog, sheep and pig are the common victims of biliary calculi among our domestic animals. In these the calculi appear to be mostly deposited from the stagnant bile in the gall bladder, yet concretions on the biliary ducts and hollow casts inside the ducts are by no means uncommon.

A gall stone may be single, or they may be multiple up to hundreds or even thousands, and when very numerous they are individually small, perhaps no larger than a pin's head. They may, however, attain the size of a marble or more, and by mutual pressure and wear they assume various polygonal forms. If they lie apart in the gall ducts or bladder they are regularly rounded. They are sometimes mulberry shaped as if conglomerate. In other cases the solid masses are so small as to have secured them the name of biliary sand. Casts and incrustations in the ducts are not necessarily made up of smaller globular masses.

On section a calculus shows a nucleus, composed of bile pigment, blood, mucus, with the debris of parasites or bacteria. Around this nucleus the calculus is deposited in concentric layers, of a hard material consisting largely of cholesterine, but containing also bile coloring matter, bile salts, and lime, in short all the constituents of bile.

Causes. Various conditions contribute to the precipitation of biliary solids in the form of calculi or encrustations. The most prominent causes are: lack of exercise, overfeeding, dry feeding, concentration of the bile, the presence of colloids and bacterian infection.

Idleness is especially operative in cattle, which are quite subject to biliary calculi and concretions, when shut up in the stall on *abundant, dry feeding* for a long winter. They are not noticed in stalled animals, that are fed watery or succulent rations, such as green fodder, distiller's or brewer's swill, ensilage, brewer's grains, mashes, roots, potatoes, apples, pumpkins, and in case a tendency to their formation is developed on the dry feeding of winter, the concretions may be re-dissolved and entirely removed by the succulent spring grass. A similar influence is noticed in the human family, as the female sex living mostly indoors, and males pursuing sedentary occupations furnish the greatest number of gall stones.

Concentration of bile results in part from muscular inactivity and hepatic torpor, but also from overfeeding which loads the portal blood and indirectly the bile with an excess of solids, and from dry feeding which lessening the secretion of water leaves the bile more dense and predisposed to precipitate its solids. The density of the liquid, however, developed from a rich and dry ration and a

prolonged inactivity, may continue for a length of time, without the occurrence of actual precipitation. It usually requires some additional factor to make this predisposition a direct cause.

Presence of Colloids. This may be found in the presence of solid or semi-solid particles. Just as the introduction of a thread into a concentrated solution of sugar or salt will induce an instant crystallization on the filament so the presence of solid bodies determines a similar condensation in solid form of the solids of the bile. But this tendency is increased materially if the solid body is itself of a colloid or non-crystallizable material. Rainey and Ord have shown experimentally that colloid bodies like mucus, albumen, pus, blood, epithelial cells, not only determine the precipitation of crystallizable salts from a strong solution, but that they cause the precipitate to assume the form of globular or spherical particles, which by gradual accretions on their surfaces tend to grow into calculi. They found that salts which are deposited by mere chemical reaction, without the intervention of colloids, appear in the form of sharply defined angular crystals. The very fact that a precipitate assumes a spherical form suggests the presence of colloids as an active factor in the precipitation. Heat appears to intensify this action, though probably the normal body temperature operates mainly through the more active proliferation of bacteria.

Bacteridian infection. In connection with the action of colloids it has been observed that when such bodies are in a condition of fermentation they are much more potent as precipitants than if inactive and sterile. But as all fermentations are the work of microorganisms we are at once brought to the conclusion that bacterial infection is one of the most potent causes of calculous formations. The invading microbes operate upon the dissolved solids, causing changes in their condition which reduce their solubility, and thus determine the separation of calculi and concretions in a manner allied to the precipitation of nitrates in the soils.

But the same microbes operate in producing the colloids which coöperate so effectively in the formation of calculi. The catarrhal biliary ducts, or bladder, shed their epithelium, and transude white and red globules, and form pus and an excess of mucus, all tending to the separation of the biliary solids or forming nuclei on which these solids may condense. The calculi and concretions tend in their turn to maintain and advance the inflammation.

The access of the microbes to the biliary duct or bladder may be effected through the blood of the portal vein or hepatic artery, or in the new-born, through the umbilical vein from an infected navel. As other modes of access may be named, a gradual advance from the duodenum through the common bile duct, or more speedily on or in the bodies of parasites (*ascaris*, *strongylus*, *stephanurus*, *tænia*, *echinococcus*, *distoma*, *fasciola*, *coccidia*), etc.

Changes in the chemical composition of the bile have been invoked as a cause of gall-stones, and Naunyn has found that the inflamed biliary epithelium secretes an excess of cholesterine and salts of lime. Thomas has also observed a great increase of cholesterine in connection with a catarrhal angiocholitis in the dog.

Among other alleged causes of biliary calculi are *advanced age* (Rigot, Hering), *acidity of the bile* (Zundel), *constipation*, and any *organic disease of the liver and bile ducts which interferes with excretion of bile*.

Age is supposed to act by inactivity, lessened secretion, hepatic torpor, and the greater presumption of liver disease, acidity by the precipitation of cholesterine and the dissolving of lime present in the tissues, and constipation through hepatic inactivity, obstruction of the flow of bile, and the tendency to infection through intestinal fermentations.

It may be added that any diminution of glycocholate or taurocholate of soda or potash, decreases the solubility of cholesterine and bile acids and favors their precipitation.

GALL STONES IN SOLIPEDS.

Characters. The biliary calculi of solipeds are of all sizes and shapes. When numerous they are mostly the size of a pin's head (Lucet). Birnbaum found in one animal 400 like peas. Dieckerhoff has repeatedly found four or five of the size of a hazelnut. Verheyen found one as large as an apple and says one exists at the Berlin Veterinary College which weighs several pounds. Rigot found 90 in the biliary ducts of an old horse, and Zundel records the death of a stallion of twenty-six years from multiple gall stones.

The calculi may be little larger than grains of coarse sand. When larger and solitary they are mostly globular or mulberry shaped; if many are together they have become polygonal by friction. In other cases notably with distomata they form hollow tubular incrustations on the bile ducts, and contain a thick grumous bile. They are usually of a green color, but may be yellowish brown, yellow, or whitish. When cut across they present a nucleus enclosed in successive layers, each successive one often differing from the last in color. Their specific gravity is low, some will even float in water when taken from the ducts, and all float when dried. Their composition is variable but chiefly cholesterine, bile acids, resin and pigments, an albumoid matter, with lime salts, etc. The nucleus may be the remains of a dead parasite, epithelial cells, blood, pus, mucus, etc. The outer layers are usually the hardest.

There may be attendant hypertrophy of the liver, cirrhosis, amyloid and other degenerations, catarrh of the biliary ducts, and distension or (according to Birnbaum) rupture of the portal vein.

Symptoms. There are no reliable diagnostic symptoms apart from the colics which accompany the obstruction of a bile duct by a passing calculus. These in the main resemble the colic of ordinary indigestion, but they may be complicated by unusual depression and nervous prostration. There may be drooping of the head, ears and eyelids, watery eyes, resting the head on the manger or pushing it against the wall. The urine is liable to be red or reddish (Jobelot), and if it or the mucosæ show a yellowish tinge it is strongly suggestive. There may be constipation or diarrhœa. The colics are severe and may last for several days (Seaman, Lucet, Burgoin) without fever and recover abruptly when the stone passes into the duodenum. They recur, however, with the impaction of another stone, and this intermittent feature, with the marked prostration, and the access of slight jaundice with each colic furnishes the best means of diagnosis.

Treatment. During the access of colic give a full dose (1 to 2 lbs.) sulphate of soda in warm water along with some active antispasmodic (belladonna, lobelia, chloroform, ether), and foment the loins and hypochondriac regions. Olive oil in large doses (1 to 2 quarts) has been found effective. Salicylate of soda in full doses is beneficial in stimulating the biliary secretion, diluting the bile, and securing some measure of antiseptis in both bowels and liver.

In the intervals between the colics, sodium salicylate, sodium or potassium carbonate, or olive oil in continuous doses may assist in disintegrating the calculi or passing them on. Chloroform tends to break them up by dissolving the cholesterin. But any such treatment must be accompanied by the abundant ingestion of water, and this is often best secured by a run in a rich green pasture. In the absence of pasturage, succulent fruits, and roots, ensilage, mash, and gruels may be advantageously substituted, and conjoined with systematic exercise in the open air.

GALL STONES IN CATTLE.

Characters. The biliary calculi of cattle are characterized by a faint odor of musk, which becomes stronger on the addition of potassa and the consequent disengagement of ammonia.

They are distinguished according to their color as dark green, yellowish green, and orange, brownish or white. There are also the sedimentary deposits (biliary sand).

The *dark green calculi* are the most frequent, and being found in the gall bladder and larger bile ducts, they attain a larger size than the others. Those in the gall bladder may be pear-shaped, and those in the ducts, round, ovoid or cylindroid. They are often rough and uneven on the surface with deep cracks and holes penetrating deeply into their substance and often filled with cholesterin. The pigments may change to a blood red when dried. The consistency of these calculi varies, some being hard, resistant and heavy, while others are soft and friable. All are composed of concentric layers around a central nucleus as in those of the horse. They vary in weight up to seven ounces in exceptional cases. They contain cholesterin, fat, resin, pigments, and lime and magnesia salts.

The *yellowish green calculi* are usually spherical unless moulded into polygonal shapes by mutual contact. In the last case they have flattened surfaces. These have a firm consistency and are composed of successive layers of nearly equal color and density surrounding the central nucleus. They are on an average smaller than the dark green variety but individual calculi have been found of three ounces.

The *whitish or orange calculi* are usually in the form of hollow casts of the bile ducts having a dull white color externally and a yellowish brown internally. They are usually thin, fragile and crystalline and contain relatively more earthy salts than the two first named varieties. From this cause also they have a higher specific gravity. One specimen weighed 8 ounces.

The *biliary sand or pulp*, is made up of granules of a yellowish, dark green or black color, forming with the bile a pultaceous mass but drying into a consistent mass. It may be firmly adherent to the mucosa of the gall bladder and require to be scraped off. These granules may be looked on as the first step in the formation of calculi or encrustations.

The *causes* of biliary calculi in cattle are mainly close confinement, dry feeding, abundant rations, the presence of trematodes in the bile ducts and finally microbial infection.

Lesions vary. With obstruction of the common bile duct or cystic duct, there are usually dilatation of the bile ducts with fibrous thickening of their walls so that they stand out as white branching lines on the back of the liver. In extreme cases the common duct may acquire the calibre of the small intestine. The gall bladder may participate in this thickening (Chassaing) or may even rupture (Proger, Shaw).

In connection with obstruction microbial infection extends upward into the liver, and in rupture of the bladder an acute, diffuse septic peritonitis follows (Chassaing). This only follows on infective inflammation of the gall bladder. Aseptic bile causes little or no irritation.

Symptoms. As in the horse, general symptoms of ill health or hepatic disorder are not pathognomonic. The presence of intermittent attacks of constipation, and colic, with icterus, tympanies and violent efforts at expulsion are the diagnostic symptoms of an acute attack. Pulsation and respiration are accelerated, and the urine dense, high colored, oily and slightly yellow. Reboul has noticed that symptoms are aggravated on exposure to cold; there are great prostration and dullness, frequent moaning and marked indications of tenderness when the right hypochondrium is percussed. Charlot has observed that the only symptoms may be persistent jaundice with scanty, high colored urine, containing some sediment.

Treatment is essentially the same as in the horse. Vanswieten and Verheyen draw special attention to the fact that whereas biliary calculi are very common in cattle during winter, they are rarely found in animals that have been for even a short period on the spring grass. Spring pasture is therefore the best therapeutic agent. During paroxysms of colic, Glauber salts, or olive oil, antispasmodics and fomentations over the liver are to be tried. In the intervals salicylate of soda, sodium and potassium carbonate, olive oil, chloroform, and ether may be used. Abundance of water and aqueous rations are essential.

GALL STONES IN SHEEP.

Calculi are very rare. One described by Morton had a brownish yellow color on its surface, and a white color spotted with green internally; it had a bitter taste, colored saliva yellow, and melted when heated, diffusing the odor of musk. It weighed twelve grains and contained 70 per cent. of cholesterin, calcic phosphate and carbonate and the usual biliary elements.

But if spherical calculi are rare, concretions and casts of the bile ducts are common, especially in distomatosis. These are of a yellowish, reddish, greenish or blackish brown, and form granular plates, or veritable cylindroid casts often firmly adherent to the mucous membrane of the duct.

In such cases the walls of the encrusted ducts are hypertrophied and stand out on the back of the liver as white bands diverging from the portal fissure.

Apart from the usual *symptoms* of distomatosis no special indications have been observed.

Treatment is primarily that for distomatosis, to which the general measures advised for calculi may be added.

GALL STONES IN SWINE.

Characters. The calculi are spherical, rough or on their opposed surfaces flat, clear and glistening where they have become polished by friction. They are found as a fine sand or as calculi the largest of which have been 75 grains, and of a high density (1303 to 1484). Bruckmüller found that they contained carbonate of lime and biliary mucus. Verheyen found biliary resin, mucus, pigment, and a little fat. They are rare in fat hogs in America. No diagnostic *symptoms* have been observed.

GALL STONES IN DOG AND CAT.

These are more or less spherical, dark brownish green, and usually found in the gall bladder or larger bile ducts. They may vary in size from a pea to a hazel nut. Their chemical analysis is wanting.

Symptoms. There may be evidence of biliary obstruction and if this occurs intermittently and is associated with colic, it becomes somewhat characteristic. Constipation, emesis, icterus, and sometimes tenderness of the right hypochondrium would indicate the source of the colic. A pre-existing and concurrent catarrh of the bowels corroborates these indications.

Cadeac explains that the obstructing calculus is called on to resist the impulse of the bile forced upon it by the spasmodic contraction of the bile ducts, which distends the bile duct immediately back of the stone to perhaps ten times its normal size. Then under a suspension of the spasm or even an antiperistaltic contraction of the duct, the calculus is forced back into the dilated portion or even into the gall-bladder, and the attack is relieved. Under repeated irritations of this kind the inflammation of the bile ducts extends into the liver and determines cirrhosis. The irritation further through the sympathetic produces a reflex constriction of the pulmonary capillaries, with the natural results of increasing tension of the pulmonary artery and right heart, and dilatation and degeneration of the walls of the latter even in the best nourished animals. Thus dyspnœa and modified heart sounds (murmurs) may be symptoms of biliary calculi.

Treatment. Three or four ounces of olive oil were found to greatly increase the quantity and fluidity of the bile in from thirty to forty-five minutes. Bile, sulphate of soda and salicylate of soda are excellent cholagogues, and the latter at the same time an antiseptic. Anti-spasmodics are especially indicated to relieve the colics, but they must be used in relatively smaller doses than in the herbivora. Potassic and sodic carbonates or tartrates (Vichy) may be used as enemas if they cannot be administered by the mouth. Fomentations may be resorted to. The food must be laxative and aqueous, and exercise must be imposed as far as the animal can bear it.

FOREIGN BODIES IN THE LIVER.

In *horse*: spikes of leguminosæ, barley awns. Symptoms: of internal hemorrhage, pallor, weakness, vertigo, death; jaundice, prostration, stupor, weakness, crossing of fore limbs, tender right hypochondrium. In *cattle*: bodies passing from rumen. In *swine*, sand. In *dog*, sharp bodies from stomach. Treatment: laparotomy.

Foreign bodies are rare in the liver in our domestic animals. *Horse*. St. Cyr has found the spikes of leguminosæ and Megnin the beards of barley. St. Cyr believed that he traced the passage followed by the stalk through the walls of the duodenum, and portal vein where it divided to be distributed through the liver. At the point of supposed entrance the walls of the vena portæ were thickened and its lumen filled with clots. The further course of the portal vein and its branches showed similar thickening and clots, and on the branch leading to the right lobe was a large abscess containing 4 decilitres of pus. Clots extended into the splenic, omental and mesenteric veins, and between the folds of the mesentery of the small intestine were a number of minute ruptures and blood extravasations.

Megnin found traces of the passage of the barley beards through the gastric walls and into the substance of the liver close to the portal fissure. Around the centre where the barbs were implanted there was an irregular hæmorrhagic extravasation in the liver, and in the abdominal cavity an effusion of 8 or 10 quarts of blood.

Symptoms. In such a case the only definite symptoms are those of internal hemorrhage, pallor of the mucous membranes, gradually increasing weakness, vertigo, unsteady gait, and an early death. In more protracted cases slight jaundice, dullness, prostration, stupor, drooping of head, ears and eyelids, resting it on the manger or walls, muscular weakness, crossing of the front limbs, and it may be tenderness on percussion on the right side of the chest posteriorly. It resembles the *coma* or *immobility* of the horse but the patient backs more easily.

Cattle. In ruminants sharp pointed bodies passing from the rumen will occasionally penetrate the liver, and give rise to symptoms of hepatic disorder. Augenheister found in a cow dilatation of the larger bile ducts, which contained about 10 quarts of sand, that had apparently entered from the duodenum by the common bile duct which had an orifice of an inch in diameter.

Pig. The gall ducts of a pig's liver, in the Veterinary College of Berlin contains a large amount of sand (Gurlt).

Dog. The liver is exceptionally perforated by sharp pointed bodies coming from the stomach. Cadeac and Blanc report three cases of needle in the liver. Blanc's case had been killed because of old age; one of Cadeac's showed symptoms resembling rabies.

Treatment of these cases would be very hopeless as nothing short of laparotomy and the removal of the foreign body would promise success.

TUMORS OF THE LIVER. NEW GROWTH.

Largely secondary, from stomach, intestine, lymph glands, spleen, pancreas; the hepatic tumor may be disproportionately large. In *horse*: sarcoma rapidly growing soft, succulent, slow-growing, fibrous, tough, stroma with round or spindle shaped cells and nuclei. Symptoms: emaciation, icterus, enlarged liver, rounded tumors on rectal examination. Melanoma, in old gray or white horses, with similar formations elsewhere; not always malignant. Lymphadenoma. Angioma. Carcinoma. Epithelioma, lesions, nodular masses, white or grayish on section, and having firm stroma with alveoli filled with varied cells with refrangent, deeply staining, large, multiple nuclei, cancerous cachexia and variable hepatic disorder. In *cattle*: sarcoma, adenoma, angioma, cystoma, carcinoma, epithelioma. In *sheep*: adenoma, carcinoma. In *dog*: lipoma, sarcoma, encephaloid, carcinoma, epithelioma. Wasting and emaciation, yellowish pallor, temporal atrophy, ascites, liver enlargement, tender right hypochondrium, dyspepsia, symptoms of primary deposits elsewhere.

The great quantity of blood which passes through the liver lays it open, in a very decided way, to the implantation of germs and biological morbid products. Hence tumors of the liver are largely secondary, the primary ones being found mostly in the stomach, intestine, abdominal lymph glands, spleen and pancreas. The primary neoplasm is often comparatively small, while the hepatic one supplied with a great excess of blood may be by far the most striking morbid lesion. The hepatic tumors are mostly of the nature of angioma, sarcoma, melanoma, adenoma, lipoma, cystoma, carcinoma, and epithelioma.

NEOPLASMS IN HORSES LIVER.

Sarcoma. This is usually a secondary formation from the primary tumors in the spleen and peritoneum, and it occurs as multiple masses throughout the substance of the gland. The liver is greatly increased in size, extending far beyond the last rib on the right side, and weighing when removed as high as 70 lbs. (Mason), or even 88 lbs. (Cadeac), in extreme cases.

The whole surface of the liver may show bulging, rounded masses, and the morbid growth may have involved the capsule and caused adhesion to the back of the diaphragm (Bächstädt). The cut surface of the neoplasm is smooth, elastic, yellowish and circular or oval in outline. It may have a variable consistency—friable or tough, according to the activity of growth and the relative abundance of cells and stroma. The portal glands are hypertrophied and thrombosis of the portal vein is not uncommon.

Microscopic examination of the dark red scrapings shows numerous blood globules, intermixed with the round or spindle shaped cells and nuclei of the tumor. Sections of the tumor show these cells surrounded by a comparatively sparse fibrillated stroma. The round cells may vary from .005 to .05 m.m. They contain one or more rather large nuclei and a number of refrangent nucleoli. The nuclei are often set free by the bursting of the cells in the scrapings. They become much more clearly defined when treated with a weak solution of acetic acid. Small grayish areas in the mass of the tumor represent the original structure of the liver, the cells of which have become swollen and fatty.

A liquid effusion more or less deeply tinged with red is usually found in the abdominal cavity.

Symptoms are those of a wasting disease, with some icterus, sometimes digestive disorder, and a marked enlargement of the liver. The last feature can be easily diagnosed by palpation and percussion. If an examination through the rectum detects the enlargement and irregular rounded swellings of the surface of the liver or spleen, or the existence of rounded tumors in the mesentery or sublumbar region, this will be corroborative. The precise nature of the neoplasms can only be ascertained after death.

Melanoma. Melanosis of the liver is comparatively frequent, especially in gray horses, and above all when they are aging and passing from dark gray to white. In many cases a more certain diagnosis can be made than in sarcoma for the reason that primary melanotic neoplasms are especially likely to occur on or near the naturally dark portions of the skin, as beneath the tail, around the anus or vulva, in the perineum, sheath, eyelids, axilla, etc. The extent of the disease is likely to be striking, the liver, next to the spleen, being the greatest internal centre for melanosis. The whole organ may be infiltrated so that in the end its outer surface is completely hidden by melanotic deposit. The surface deposits tend to project in more or less rounded, smooth masses of varying size according to the age of the deposit and the rapidity of its growth. Individual deposits may vary in size from a pea to a mass of 40 or 50 lbs. They are moderately firm, and resistant, and maintain a globular or ovoid outline. The color of the melanotic deposits is a deep black with a violet or bluish tint. If the pigmentary deposit is in its early stage it may be of a dark gray. The deposits are firmer than the intervening liver tissue and rarely soften or suppurate.

Melanosis in the horse is not always the malignant disease that it shows itself to be in man, and extensive deposits may take place externally and considerable formations in the liver and other internal organs without serious impairment of the general health. It is only in very advanced conditions of melanosis of the liver that appreciable hepatic disorder is observed. If, however, there is marked enlargement of the liver, in a white or gray horse, which shows melanotic tumors on the surface, hepatic melanosis may be inferred.

Lymphadenoma. Adenoid Tumor. Lienaux describes cases of this kind in which the liver was mottled by white points which presented the microscopical character of adenoid tissue, cells enclosing a follicle and a rich investing network of capillaries.

Angioma. These are rare in the horse's liver, but have been described by Blanc and Trasbot as multiple, spongy tumors on the anterior of the middle lobe, and to a less extent in the right and left, of a blackish brown color, soft and fluctuating. The largest mass was the size of an apple, and on

section they were found to be composed of vascular or erectile tissue. The tendency is to rupture and extensive extravasation of blood (30 to 40 lbs.) into the peritoneum.

Carcinoma. Epithelioma. These forms of malignant disease are not uncommon in the liver as secondary deposits, the primary lesions being found in the spleen, stomach, intestine, or pancreas, or more distant still, in the lungs. The grafting or colonization of the cancer in the liver depends on the transmission of its elements through the vena portæ in the one case, and through the pulmonary veins, the left heart and hepatic artery in the other.

Lesions. The liver may be greatly enlarged, weighing twenty-seven pounds (Benjamin) to forty-three pounds (Chauveau), hard, firm, and studded with firm nodules of varying sizes. These stand out from the surface, giving an irregular nodular appearance, and are scattered through its substance where, on section, they appear as gray or white fibrous, resistant, spheroidal masses shading off to a reddish tinge in their outer layers. Microscopically these consist of a more or less abundant fibrous stroma, enclosing, communicating alveoli filled with cells of various shapes and sizes, with large nuclei (often multiple) which stain deeply in pigments. The relative amount of fibrous stroma and cells determines the consistency of the mass, and whether it approximates to the hard cancer or the soft. In the horse's liver they are usually hard, and, on scraping off the cut surface, yield only a limited quantity of cancer juice. In the epithelial form, which embraces nearly all that have originated from primary malignant growth in the walls of the intestine, the epithelioid cells, flattened, cubical, polyhedral, etc., are arranged in spheroidal masses or cylindrical extensions, which infiltrate the tissues more or less. These seem in some cases to commence in the radical bile ducts (Martin), and in others in the minor coats of the larger biliary ducts.

As the disease advances a brownish liquid effusion is found in the abdomen, and nodular masses formed on the surface of the peritoneum.

Symptoms. As in other tumors of the liver these are obscure. As the disease advances there may be œdema of the legs and sheath, indications of ascites, stiff movements, icterus, occasional colics, tympanies, and diarrhœa. Nervous symptoms may also appear, such as dullness, stupor, coma, vertigo and spasms. Emaciation goes on rapidly and death soon supervenes.

TUMORS OF THE LIVER IN CATTLE.

Sarcoma. Round or spindle shaped celled sarcomata have been described by Sodero and Cadeac, leading in one case to perforation of the vena portæ and death by hæmorrhage into the peritoneum.

Adenoma. Martin records a case of a hepatic tumor formed of adenoid tissue which had extended into the vena portæ, and microscopically presented a cylindroid character. In the advanced stages it caused some jaundice, digestive disorder, obstinate constipation, progressive and extreme emaciation, and weakness which kept the animal constantly in a recumbent position.

Angioma. These are rarely seen in the young but are comparatively common in old cows as they are in aged men. They form masses of a dark red color and very variable size, and have a limiting sac of connective tissue or merge into the adjacent hepatic structure. Microscopically they consist of a series of irregular lacunæ filled with liquid blood, blood clots, or leucocytes, and communicating with small blood vessels in the walls and partitions. They are believed to be formed by dilatation of the liver capillaries with subsequent thickening of their distended walls, and atrophy of the nearest liver cells. Cases of the kind have been recorded by Kitt, Martin, McFadyean, Saake, Van der Sluys, Korevaar and others. Though often seen in abattoirs, they seem to have little effect on the general health, and no special symptoms have been noted as indicating their existence.

Congenital Cysts. These are found on the anterior surface or lower border of the liver in young calves. They have no connection with the blood vessels, nor biliary canals, contain no head of larval tænia, and do not constantly show the presence of any particular bacterium. These walls are thin and their contents alkaline, with sometimes slight blood extravasation, or a yellowish deposit in which cocci have been found. Unless connected with cirrhosis or other serious disease of the hepatic tissue, their presence seems to have no pathological significance.

Carcinoma. This has been recorded in the liver of cattle by Gurlt, Brückmüller, Kitt and others. From the walls of the gall bladder it grows in pyriform masses, and on the surface and in the interior of the liver, it may appear as hard, cancerous masses of all sizes.

Epithelioma. This has been described by Kitt, Martin, Blanc, Leblanc, Morot, Cadeac, and Besnoit. It appears in masses varying in size from a millet seed up, bulging from the surface of the organ or deeply hidden in its substance, and stained yellow or green with bile. The liver is usually enlarged, amounting to even 34 pounds (Cadeac). The formation commencing in the acini invades all surrounding parts causing compression and atrophy of the liver cells, and the formation of nests of epithelioid cells often with multiple nuclei and nucleoli. Cirrhosis is not uncommon, and fatty and other degenerations. Microbic invasion and necrobiosis are also common.

NEOPLASMS IN THE SHEEP'S LIVER.

Adenoma has been met with by McFadyean, Johne, Kitt and Bollinger. They hung as pediculated tumors from the surface of the liver, and were in part wedged into its substance displacing the hepatic tissue and vessels. In general they consisted of a dense fibrous stroma with cylindroid and biliary cells in great abundance, sometimes arranged in tubular form. Specimens described by Kitt and Bollinger attained to the size of a man's head and were stained of a deep green color.

Carcinoma. Casper reports a case of hepatic cancer in the sheep secondary to cancer of the mesentery.

NEOPLASMS IN THE DOG'S LIVER.

Lipoma. Trasbot describes two fatty tumors in the liver of a bitch, one of them as large as an infant's head. It had a yellowish white color, and had taken the place of the proper hepatic tissue.

Malignant Tumors. These are rather common. *Sarcomatous* masses with round and fusiform cells in a fibrous stroma; *encephaloid* with a delicate stroma and large alveoli filled with cells, and having a soft brainlike consistency; *carcinoma* with dense and thick fibrous stroma and nests of cells in comparatively small numbers; and *epithelioma* with flattened, cylindroid or other epithelial cells in masses often affecting a tubular aggregation, are seen in different cases. Sometimes apparently primary, they can more commonly be traced to pre-existing centres of the same formation on the course of the portal vein or elsewhere.

Symptoms. A gradual wasting and emaciation with a yellowish pallor of the mucous membranes are characteristic. Trasbot gives the excessive atrophy of the temporal and masseter muscles as pathognomonic. Ascites is a usual complication. Enlargement of the liver, as shown by percussion of the right hypochondrium, and, in case of flaccid abdomen, by manipulation, and attendant signs of tenderness are corroborative. Variability or loss of appetite, and vomiting is not uncommon, and in case of primary or secondary deposits in other organs in the abdomen, thorax or elsewhere, the symptoms resulting from functional derangement of such organ may be found. Treatment is hopeless.

CALCAREOUS NODULES AND DEGENERATIONS OF THE LIVER.

Calcified roundish nodules, in groups, under capsule. Mostly in solipeds. Theories of origin: parasites, microbes, emboli, omphalitis, intestinal disease, biliary obstructions. Calcification of liver with large abdominal aneurisms. Cszoker's case, Diagnosis and specific treatment impossible.

In the domestic animals in general the liver may become the seat of imperfectly spherical nodules of a white, yellow or brownish white color, varying in size from a millet seed to a pea or hazel nut, and of a gritty consistency and feeling, from the deposition of earthy salts. These may be seen in groups under the proper capsule, the adjacent hepatic tissue being healthy, or atrophied, sclerosed or pigmented. These lesions have been found most abundantly in solipeds.

Pathogenesis. The most varied doctrines have been advanced as to the origin of these lesions. They have been attributed to the previous presence in the liver of linguatula, echinococcus, coenurus, oxyurus, distoma, and other parasites (Cadeac, Mazanti, Olt, Ostertag, Gripp, Leuckart, Ratz), to glanders, to microbial attacks (Dieckerhoff), to minute embolic infarcts in omphalitis in the foal, or intestinal disease in the adult (Kitt), and to obstructions by the eggs of distomata in the biliary ducts (Galli-Vallerio). It is not improbable that the lesion may be due to any one of these in a specific case, and this may be ascertained by the existence of certain definite features and conditions. Linguatula, echinococcus and coenurus can only be suspected in districts where these prevail, and a careful examination of the central mass of the nodule should reveal the presence of the indestructible hooklets, as certified for given cases by Olt, Ostertag and Gripp. In case of nematoid worms or distomata, the eggs may possibly be found as in the cases of Villach and Ratz, or the embryos (Mazanti). Or there may be traces of channels formerly hollowed out by the worms in the vicinity of the nodules, as seen by Leuckart. Coincident tumors of the intestinal mucosa from larval nematodes, or aneurism or emboli in the anterior mesenteric artery would corroborate this conclusion. If distomata had started the lesions, the distension of the gall ducts and the thickening of their walls would be likely to indicate their former presence. Glander nodules might be suspected from the absence of a distinct rounded or oval outline, from the lack of a distinct, clear line of demarcation between the nodule and the adjacent liver tissue, and by the manifestation in the periphery of the nodule and around it of free cell proliferation, showing the mode of progression by the invasion of new tissue. If still active, the bacilli should be discoverable in stained scrapings or sections. There should also be distinct indications of the lesions of glanders in the lymph glands of the portal fissure, of the mediastinum, of the submaxillary region and of other parts.

Heiss records an interesting case of general calcification of the horse's liver, with large aneurism of the abdominal aorta, mesenteric and renal arteries. The liver was thirty-two pounds, puckered on the surface and showed calcic degeneration of the walls of the vessels and hepatic tissue, to such an extent that when the organ was dried it did not add materially to its hardness. Microscopically the diseased centres indicated minute blood clots (thrombi), with fibrinous development and cretification. The lesions in this case were attributed to multiple emboli in connection with the aneurism. It might suggest further, microbial infection of both the aneurismal and hepatic vessels. In another case of extensive cretification of the horse's liver reported by Cszoker, the calcified masses tended to assume rounded forms like tubercle, and had a clear glistening surface.

These lesions are mainly interesting in a pathological sense, and unless they are very extensive do not give rise to appreciable symptoms.

Treatment could only be prophylactic and directed to the removal of the special conditions, in which the calcification originated in a given locality.

ACTINOMYCOSIS OF THE LIVER.

On damp infested soil, in cattle and swine. Round tumors, hard surface, soft centre, fibrous sac, club-shaped cells in tufts. Symptoms of liver disorder. Coincident external actinomycosis. Treatment: potassium iodide.

In damp soils where actinomyces are present in the soil and vegetation, it is not uncommon to find the characteristic growths in the liver of cattle and swine. Rasmussen saw twenty-two cases of hepatic actinomycosis in one year (1890) and in a number of cases he has found the liver, spleen, peritoneum and intestine simultaneously affected. Jensen who has also recorded hepatic cases, found tumors extending from the liver to the diaphragm. He describes them as rounded masses, of different sizes, enclosed in a fibrous envelope of variable thickness, hard and resistant at the surface and somewhat softened toward the centre. Microscopic examination detects the club-shaped cells arranged in tufts and radiating from a common centre.

Symptoms are only the general indications of hepatic disease differing according to the size, and position of the morbid product and its interference with normal functions. When, however, superficial actinomycosis is found these symptoms may be fairly attributed to the existence of similar products in the liver.

Treatment consists in the administration of potassium iodide in full doses, daily for a week, followed by a laxative, and then, after an interval of two days, repeat the treatment for a second week, and so for a third, fourth and fifth until the microbe has been destroyed.

PARASITES OF THE LIVER.

Lying as it does in the channel of the blood charged with the products of absorption from the intestine, the liver is especially liable to parasites. Among protozoa are: *Monocercomonas hepatica* (pigeon), *saccharomyces guttulatus* (rabbit), *eimeria falciformis* (rabbit), *coccidium oviforme* (rabbit, pig, dog). Among the lower cryptogams are *actinomyces* (ox, pig). Of the tapeworm family are: *Cysticercus tenuicollis* (ruminants, pig), *c. pisiformis* (rabbit), *c. cellulosa* (dog, pig), *echinococcus veterinorum* (animals, man), *tænia fimbriata* (sheep, deer), and an undetermined *cœnurus* (cat). Of trematodes are: *Distoma hepatica* (herbivora, man), *distoma lanceolatum* (herbivora, man), *distoma giganteum*, or *Americanum* (cattle), *d. truncatum*, *d. conjunctum*, *d. campanulatum* (dog), *amphistoma explanatum* (ox). Of nematodes are: *Stephanurus dentatus*, *ascaris suis*, *oesophagostoma dentatum* (pig), *sclerostoma equinum*, *ascaris megalcephala* (horse), *ascaris bovis* (ox), *oesophagostoma columbiana* (in ruminants), *filaria hepatica*, *enstrongylus gigas*, *ascaris marginata* (dog), *ollulanus tricuspiis*, *ascaris mystax* (cat). (See Parasites).

DISEASES OF THE PANCREAS.

Obscure. Shown only by digestion or hepatic disorder. Excess of fat in stools suggests suppression of secretion. Intestinal fermentations. Suspension of glycogenesis and consequent emaciation, stunting or poisoning. Pancreatic calculus and icterus.

Diseases of the pancreas are even more obscure than those of the liver. Situated on the course of the duodenum, beneath the lumbar vertebræ and their right transverse processes, and separated from the lateral walls and floor of the abdomen by the great mass of the intestines, it is not open to manipulation or satisfactory percussion, and its secretions being used up in the function of digestion, so that they cannot be perceived and tested externally like the secretions of the kidneys. Beside the general constitutional disorder therefore, we must look rather to the derangements of the digestive functions, to the abnormal condition of the fæces, and to the alterations in subordinate functions like the glycogenic action of the liver, for indications of an unhealthy state of the pancreas. The suppression of the pancreatic secretion has long been associated with the occurrence of fat in the stools, yet this may result from the lack of bile which has important functions to fulfill in emulsifying fat, and in securing its endosmosis. On the other hand the lack of pancreatic juice may hinder the complete digestion of the albuminoids, and favor their fermentation and the occurrence of tympanies, congestions, abnormal secretions, etc., which may be easily attributed to another origin. Then again the dependence of the liver on the pancreas for its stimulus to glycogenesis, would suggest a series of disturbances from the abundance of the unused food principles, from the hindrance to nutrition and growth, and perhaps from the toxic action of the hepatic products. Once more, through the common excretory duct, infection of the pancreas may extend to and involve the liver, and blocking of the common duct by pancreatic parasites, or calculi, may stop the flow of bile and cause jaundice or other icteric disorder. And yet, it is rarely the case that pancreatic disorder is successfully diagnosed, and it is too often only at the post mortem examination that the actual lesions are revealed.

CATARRHAL PANCREATITIS.

Probable causation by parasites, calculi, irritants, microbes. Lesions: mucosa reddened, thickened, ducts dilated, epithelium fatty, granular, desquamating, pus, connective tissue indurated. Interdependence of pancreatitis and hepatitis in horse, sheep and goat. Liver lesions. Symptoms: loss of vigor, endurance, appetite, and condition. Icterus, costiveness, foetid, fatty stools, percussion tenderness—right side. Treatment: antithermics, eliminants, antiseptics, derivatives, alkalies, salicylates, ether.

Causes. We know little of the causes of this affection, but it may be inferred that parasites, calculi and other irritants, will produce in this as in other mucosæ a mucopurulent inflammation. Then again the presence of pus suggests the coöperation of pus microbes as in the infective catarrhal icterus. The blocking of the common gall and pancreatic duct, by gall stones or biliary products, will entail arrest of the discharge of pancreatic juice, and a consequent pancreatitis, just as blocking with pancreatic products will cause hepatitis and icterus.

Lesions. The mucosa of the pancreatic ducts is reddened, congested and thickened and their lumen blocked by a white, granular matter, containing pus globules, fibrine filaments, and granular, ciliated epithelium. The blocked ducts become dilated, and their walls thickened, the epithelium is desquamated to a greater or less extent, and the raw exposed surface may present ulcers or granulations. The pancreatic cells undergo fatty degeneration and the connective tissue becomes steadily indurated (sclerosis). These lesions were especially noted by Megnin and Nocard in a case of pancreatitis in the horse.

In the horse, sheep and goat, which have a common outlet for the bile and pancreatic juice, the blocking of the latter and the arrest of the bile almost of necessity causes hepatitis, and infection in the one gland is directly transferred to the other so that pancreatitis and hepatitis are mutually causative of each other. In the ox, pig, dog and cat, in which the bile and pancreatic juice are poured into the duodenum through separate ducts and orifices, this mutual pathogenic action is not so certain.

When the liver is implicated, there is catarrh and dilatation of the bile ducts, fatty degeneration commencing in the centre of the acini, pigmentation appearing at their periphery, and sclerosis of the organ follows.

Symptoms. In Nocard's equine case there was progressive loss of spirit, energy, and endurance; appetite was poor and eating listless; after two weeks jaundice set in, the visible mucosæ and skin showing a yellow tinge, and the scanty urine becoming brownish yellow; the bowels became costive the fæces being formed of small hard discolored balls, but no excess of fatty matter is recorded. Emaciation advanced rapidly, the most marked wasting being in the muscles of the back, loins and croup. Death ensued at the end of two months from the commencement of the illness. In man sudden, violent colic, with nausea, tympany and collapse are prominent symptoms.

Diagnosis is more satisfactory when with digestive disorder, tardily developing icterus, and rapid emaciation, there is an excess of fat in the ill-smelling fæces. Pain on percussion of the right hypochondrium would be an additional feature.

Treatment can rarely be adopted because of the uncertainty of the diagnosis. It would proceed on general principles, antithermics, eliminants, antiseptics, and counter-irritants being resorted to as the conditions seem to demand. Alkaline laxatives and diuretics, salicylates of soda or potash, and guarded doses of sulphuric ether to solicit the action of the pancreas, might be resorted to. The disorder of the liver would require attention along the lines indicated under catarrh of that organ.

INTERSTITIAL PANCREATITIS.

Causes: paresis, marasmus, septic infection, blood diseases. Lesions: connective tissue in excess—pancreas and liver; catarrhal complications; calcic points; congestion and petechiæ in septic infection. Areas of fat necrosis in the pancreas and abdominal adipose tissue. Stearates of lime. Calcic foci in animals. Symptoms: obscure. Treatment.

This is especially liable to accompany paretic and wasting diseases, septic infection, and diseases of the blood. Radionow examined the pancreas in animals that had suffered from chronic paralysis, gastro-intestinal catarrh, hepatic catarrh, chronic anæmia and marasmus, and found fatty degeneration of the epithelium, with atrophy and pigmentary degeneration of the glandular epithelium. The fibrous tissue of the gland was in excess in the pancreas and in the liver (sclerosis), and mucous cysts were found.

Siedamgrotzky found a chronic interstitial pancreatitis connected with alopecia, œdema and leucocythæmia. The pancreas was indurated, fibrous, resisting the edge of the knife and sprinkled with gritty particles. Much of the glandular tissue had been destroyed, and the ducts were filled with a dense, grayish, grumous mucus.

Kirilow and Stalnikow have found interstitial pancreatitis marked by congestion and ecchymosis, with intervening anæmic areas, in animals injected with septic matter. There was increased secretion in the early stages.

A marked feature of pancreatitis in man is the occurrence in the interlobular tissue of the gland, the omentum, mesentery and abdominal fatty tissue generally, of circumscribed areas of fat necrosis, each varying in size from a pin's head upward even to a hen's egg. On section these show a soft tallowy consistency and Langerhans has shown that they are composed of lime and fatty acids in combination. When lime is in excess they become gritty. According to Osler they may be dependent on some other primary affection (Bright's disease). The partially calcified concretions found in the pancreatic ducts, and the yellowish white, gritty areas, which represent the degenerate lobules in animals (Seidamgrotzky) are suggestive of a similar morbid condition of the pancreas or it may be of some distant organ. Of late years a number of cases have been recorded in man and a very high mortality noted.

The *symptoms* in the lower animals are very obscure, and an accurate diagnosis is looked upon as almost impossible. They are essentially the same as given above under catarrhal pancreatitis.

Treatment too has the same narrow limitations.

PANCREATIC ABSCESS, SUPPURATIVE PANCREATITIS.

A complication of strangles or purulent infection. Symptoms: Colics, chill, tender right hypochondrium, emaciation, fatty stools. Treatment: Constitutional.

Reimers has reported several cases of pancreatic abscess, as a phase of irregular strangles (rhinoadenitis). In one case multiple abscesses with an aggregate capacity of 2½ quarts were found, and some of the pus had escaped by rupture into the peritoneum and produced infective inflammation. The abscesses had destroyed the greater portion of the gland, only a few isolated lobules being left.

Galland found an abscess as big as a walnut in the pancreas of a horse which had multiple tumors in the abdomen.

Symptoms. Colics occur from the local phlegmon, and it may be from its pressure on the duodenum so as to obstruct it, and this appearing in the course of strangles would indicate a forming abdominal abscess. Staring coat or shivering may coincide. Tenderness of the abdominal walls has been noticed by Reimers, together with a partial loss of appetite and a characteristically rapid emaciation. Fatty stools, if present, would be almost the only pathognomonic symptom.

Prognosis is that the abscess will open into the abdomen, and cause fatal infective peritonitis. It is only as an exceptional occurrence that its rupture into the duodenum or colon can be hoped for, yet in such a case recovery is possible.

Treatment. Little can be done. It would be well to treat the constitutional symptoms, and await results.

FOREIGN BODIES IN THE PANCREAS.

Brückmüller has noticed needles and other sharp objects in the pancreas of the dog, determining abscess and the formation of a thick, greenish pus in the adjacent glandular follicles. Goubaux once found a fragment of straw in the pancreatic duct of the horse. Such conditions are not likely to be diagnosed, but if this could be done laparotomy might be permissible in the dog for the removal of the foreign body.

PANCREATIC CALCULI.

Mostly in cattle from over feeding, dry feeding, inactivity. Small. Multiple. Round, angular, lobulated. Nucleus. Composition. Dilated ducts. Atrophied or sclerosed glandular tissue. Prevention: succulent food, water at will, open air life, correction of local catarrh.

Pancreatic like biliary calculi have been found especially in cattle. They appear to be predisposed by their stimulating, forcing feeding, by their quiet life apart from all causes of excitement and especially by the combined effect of dry feeding and prolonged confinement in the stall through the long winter.

The calculi are usually small but numerous, Jungers having found 36 weighing 38 grammes. Bär has found a mass with an aggregate weight of 23 grammes.

The form of the calculus varies; many are angular from mutual attrition in the large ducts; others from the smaller ducts are rounded; those from the glandular follicles may be even lobulated, in keeping with the divisions of the cavity. The color is white and each shows a distinct central nucleus of epithelial, mucus, or other origin. Their specific gravity is 2.397 (Fürstenberg), and their composition 92 per cent. calcium carbonate, 4 per cent. magnesia, and traces of calcium phosphate (Gurlt).

The pancreatic ducts are as a rule greatly dilated and thickened (in man they form enormous cysts, Senn, Osler), and the glandular tissue is atrophied, indurated (sclerosed), and of a brownish yellow color.

Treatment of such cases would be unsatisfactory. By way of prevention succulent food, abundance of pure water, and the correction of any infective catarrhal affection of the duodenum, or of the bile or pancreatic ducts would be specially indicated. Free exercise in the open air would be desirable.

PANCREATIC NEOPLASMS. TUMORS.

Often malignant, and secondary. Melanoma in white horse. Carcinoma in mare and dog. Epithelioma. Debility, icterus, abdominal swelling, emaciation. Treatment: laparotomy, or potassium iodide.

Tumors of the pancreas are quite frequently malignant, and show a preference for the head of the organ. They may be primary but are more frequently secondary.

In gray horses melanotic tumors are found, in connection with similar formations externally, and especially as age advances. Brückmüller found them of varying size, from a pea to a hazel nut, scattered through the pancreas and adjacent tissues.

Gamgee records a carcinoma of the pancreas of a mare.

Carcinoma is more frequent in this organ in dogs, the neoplasm having an irregular form, an imperfect line of delimitation from surrounding parts and a hard, fibrous stroma enclosing caseous centers, undergoing fatty degeneration.

Nocard reports an epithelial tumor of the head of the pancreas in a bitch. The animal which had been ill for six weeks was debilitated, emaciated, and icteric with a marked abdominal swelling. It died two weeks later, and necropsy revealed a whitish sublumbar tumor, the size of a large apple, with irregular rounded projections. This pressed on the posterior vena cava, surrounded the vena portæ and gall duct and completely closed the latter. Microscopic examination showed it to be an epithelioma. The liver was undergoing cirrhosis.

Treatment, usually hopeless, would be by laparotomy. If actinomycosis were present give potassium iodide.

DISEASES OF THE SPLEEN.

No guidance through palpation or secretion. Leukæmia. Lymphadenoma. Spleen a favorite culture ground for microbes. Congestions, engorgements, ruptures. Safety valve to portal system and liver. Rhythmic splenic contractions under reflex action.

The spleen even more than the pancreas is so deeply seated and so surrounded by other organs, that its diseases are not readily appreciable by physical examination, while the absence of any special secretion excludes the possibility of diagnostic deductions through this channel. Even the relation of the condition of the organ to the number of the leucocytes and red globules fails to afford trustworthy indications of disease, since leucocytes originate in other tissues as well as the spleen, and the destruction of red globules may take place elsewhere. Yet an excess of eosinophile leucocytes in the blood suggests hypertrophy or disease of the spleen, and an excess of leucocytes in general is somewhat less suggestive of disease of this organ (see Leucocythemia). If adenoma is further shown, in enlargement of lymphatic glands elsewhere there is the stronger reason to infer disease of the spleen.

The physiological relation of the spleen to the blood especially predisposes it to diseases in which the blood is involved. The termination of splenic capillaries, in the pulp cavities, so that the blood is poured into these spaces and delayed there, opens the way, not only for the increase of the leucocytes, and the disintegration of red globules, but for the multiplication of microorganisms which may be present in the blood, and for a poisoning (local and general) with their toxins. Hence we explain the congestions, sanguineous engorgements and ruptures of the spleen in certain microbial diseases (anthrax, Southern cattle fever, septicæmia, etc.)

We should further bear in mind that the spleen is in a sense a safety valve for the blood of the portal vein, when supplied in excess during digestion. In this way it protects the liver against sudden and dangerous engorgements, but it is itself subjected to extreme alternations of vascular plenitude and relative deficiency. This may be held to take place largely under the influence of the varying force of the blood pressure in the portal vein, but according to the observations of Roy on dogs and cats, it is also powerfully influenced by muscular and nervous action. He found rhythmic contractions of the organ due to the muscles contained in the capsule and trabeculæ, repeating themselves sixty times per hour, and which might be compared to tardy pulsations. He further found that electric stimulation of the central end of a cut sensory nerve, of the medulla oblongata, or of the peripheral ends of both splanchnics and both vagi caused a rapid contraction of the spleen. The spleen may thus be looked on not only as a temporary store-house for the rich and abundant blood of the portal system of veins during active digestion, but also as a pulsating organ acting under the control of nerve centres in the medulla. That the various ascertained normal functions of this viscus may be vicariously performed by others, as shown in animals from which it has been completely extirpated, does not contradict the occurrence of actual disease in the organ, nor the baleful influence of certain of its diseases on the system at large.

ANÆMIA OF THE SPLEEN.

General anæmia, debility, wasting diseases, starvation, hæmorrhage, stimulus to formation of red globules, asphyxia, electricity, cold, quinine, eucalyptus, ergot. Symptoms: lack of eosinophile leucocytes in the blood of a debilitated subject may lead to suspicion. Treatment: tonic, light, sunshine, pure air, exercise, nutritive food, iron, bitters.

In cases of general anæmia the spleen is liable to be small, shrunken, wrinkled, and when cut the surface is drier and lighter colored than in the normal condition. This condition may be seen after old standing debilitating diseases, but is common in animals that have been reduced by starvation, just as the opposite condition of hyperæmia and enlargement comes of abundance of rich food and an active digestion. It may shrink temporarily as the result of profuse hemorrhage, but Bizzozero and Salvioli found that several days after such loss of blood it became enlarged and its parenchyma contained many red nucleated hæmatoblasts. The result of hemorrhage is therefore to stimulate the organ to enlargement and to the resumption of its embryonic function of producing red blood globules. Contraction of the spleen further occurs under asphyxia, the deoxidized blood being supposed to operate through the medulla oblongata. As already noted the spleen shrinks under stimulation of the central end of a sensory nerve (vagus, sciatic). An induced current of electricity applied to the skin over the spleen causes marked contraction (Botkin). Cold, quinine, eucalyptus, ergot and other agents also induce contraction. In the normal condition there is an inverse ratio between the bulk of the spleen and the liver, the enlargement of the one entailing a diminution of the other, but in certain diseased states, such as anthrax, ague, etc., both are liable to enlargement at the same time.

Symptoms of splenic anæmia are wanting, through a lack of eosinophile leucocytes, in the blood of a starved or otherwise debilitated animal, may lead to suspicion of the condition.

The *treatment* of such a case would be addressed rather to the general debility which induced the splenic contraction than to the contraction itself. Light, sunshine, pure air, exercise, grooming, nourishing food and the avoidance of all debilitating morbid conditions would indicate the principles of therapeutic management.

HYPERÆMIA—CONGESTION OF THE SPLEEN.

Four hours after full meal in splenic diastole. In well fed, high conditioned. From obstruction of splenic or portal vein or vena cava, heart, liver, or pulmonary disease, inhibition from encephalon acting through splanchnics or vagi, microbes, ptomaines, toxins, paresis, albuminoid diet. Spleen may be seven times its normal weight. Lesions: simple blood engorgement: proliferation of pulp cells: increased friability; rupture; dark color; hyperplasia of trabeculæ—hypertrophy. Symptoms: none; or colic; palpation in ruminants; tenderness. Treatment: directed against the causative disease; quinine, cinchonine, eucalyptus, ergot, cold douche, electricity, puncture.

Considerable hyperæmia of this organ takes place physiologically in connection with active digestion in the first four or five hours after an abundant meal, and especially at intervals of a minute, during what may be called the diastole of the viscus. The supply of blood is also much greater in the well fed animal, than in the emaciated and impoverished one.

Pathological hyperæmias of a *passive* kind may occur as the result of obstructions in the veins leading from the spleen, such as the splenic veins, the posterior vena cava, or that part of the portal vein comprised between its junction with the splenic and the liver. Diseases of the right heart or its valves, of the lungs (emphysema), or of the liver which hinder the onward flow of blood and increase the blood tension in the vena cava or portal vein have a similar action. Perhaps we should include inhibition of the nerves (splanchnic, vagi) and nerve centres (medulla oblongata, cerebral cortex) which preside over the contraction of the splenic vascular walls, and of the capsular and trabecular muscles. There is reason to believe that the ptomaines and toxins of several microbial diseases, operate through these centres, while other such microbes and toxins operate directly on the spleen itself.

Active congestions of the spleen are most commonly associated with microbial diseases and may be attributed partly as above stated to the action of the toxic products on the contraction nerve centres, and on the splenic vessels and parenchyma, but also in no small degree on the active proliferation of the germs themselves in the splenic pulp, and of the splenic cells. Among the most notable instances of this kind are, *in man*, malarious, yellow and typhoid fevers, and, *in animals*, anthrax, and Southern cattle fever. In most febrile diseases, however, there is a tendency in this direction, which may be fairly attributed to the paresis of the organ and the delay of the blood in its pulp channels and spaces with the consequent local increase of microbes and toxins. The microorganisms can usually be found abundantly in such cases, in the liquid of the pulp, and in the interior of the leucocytes and other cells that go to make up its solid constituents.

It has been long recognized by veterinarians that acute congestion often arises in connection with a sudden transition from a poor or insufficient diet to an abundant and nutritious one and especially to one that is rich in albuminoids (beans, peas, vetches, lucerne, sainfoin, clover, trefoil, in the fresh or preserved condition). If these are not in themselves the direct causes of acute and fatal engorgements of the spleen, they at least contribute in no small degree to the overdilation of the pulp spaces, the paresis of the organ and its successful invasion by pathogenic microbes.

The acute congestion attendant on specific microbial infection may be estimated by the increase in weight of the spleen. In the Southern Cattle fever this organ, which is normally 1.45 lb., is habitually 2 to 5 lbs., and may reach 8 or 10 lbs. and in anthrax an equal increase may be noted.

Lesions. In such cases the organ may appear as if there were a simple blood engorgement, and this is largely the case in the early stages, but with the persistence of the disease there occurs an active proliferation of the splenic cells and especially those of the pulp. With the hyperæmia the consistency of the organ is diminished, and still more so with the cell hyperplasia, so much so that in extreme cases rupture may ensue. The color is always darker (purple or blue), but this is only in part due to the abundance of blood and in part to the thinness of the splenic capsule. If the condition persists a hyperplasia of the capsule and trabeculæ ensues, and the condition becomes essentially one of hypertrophy.

Symptoms. In the slighter congestions there are no appreciable symptoms. In the more severe there may be more or less violent colic, but this is usually marked to some extent by the profound depression attendant on the specific fever which is the cause of the congestion. Palpation of the

spleen is impossible in the horse. In ruminants it may sometimes be felt along the upper border of the rumen just behind the last rib on the left side. It is soft and yielding retaining the indentation of the finger. If manipulation produces signs of pain it is all the more significant.

Treatment. As a rule this is the treatment of the fever which determines the hyperæmia. Apart from this, laxatives, quinia other alkaloids of cinchona bark, eucalyptus, a current of cold water directed to the region of the spleen, or induction currents of electricity to the same region are also decided stimulants to contraction. Ergot has been used with alleged advantage. In cattle acupuncture of the spleen has been put in practice in anthrax.

CHRONIC CONGESTION OF THE SPLEEN. HYPERTROPHY.

Hypertrophy from chronic congestion, over feeding, hepatic cirrhosis. In *horse*: from mechanical obstruction in heart, lungs, posterior cava, splenic veins, angioma, from glanders or tubercle in lungs, chronic splenic congestion, disease of splenic plexus. Lesions: increase enormous; mainly of pulp, or largely of fibrous framework. Special neoplasms. Symptoms: excess of leucocytes in blood, eosinophile cells, weakness, anæmia, emaciation, bleeding from mucosæ, stretching, right hypochondriac tenderness, stiff gait, ascites, colic, disorder of the bowels, rectal exploration. Treatment: is that of primary disease; not encouraging; quinia, eucalyptus, saline laxatives, open air, sunshine, electricity. In *cattle* is habitually enlarged in Texas fever area. In lymphadenoma increase mainly of fibrous framework and Paccinian bodies, and of adjacent lymph glands. Symptoms: leukæmia, employ palpation, percussion, rectal exploration. Treatment as in the horse. In *swine*: from high feeding, leukæmia, lymphadenoma, tuberculosis, neoplasms, liver, heart and lung disease. Lesions: great increase of Paccinian bodies, fibrous capsule and trabeculæ. In *dog*: from traumas, leukæmia and lymphadenoma. Enlarged Paccinian bodies and adjacent lymph glands. Symptoms: leukæmia, many eosinophile cells, abdominal enlargement, palpation, icterus. Treatment: as for large animals.

A continuation of passive congestion from the causes enumerated above, leads to permanent increase of the fibrous reticulum and connective tissue and increase of the splenic pulp. Even the stimulus of a rich and abundant alimentation increases the size of the whole organ, the amount of pulp and the number and development of the Paccinian bodies. Apart from disease the spleens of well fed cattle or horses are always decidedly heavier than those of the starved or debilitated. Of mechanical causes the most potent is cirrhosis of the liver or some other obstacle to the free passage of blood through that organ. The most common causes are, however, the continuous operation of those specific poisons which determine the acute hyperæmias.

SPLENIC HYPERTROPHY IN HORSES.

Causes. It occurs as the result of mechanical obstruction of the posterior vena cava as noticed by Varnell, from obstruction in the splenic artery or veins by Ellenberger and Schütz, as the result of an angioma by Martin, as the result of the morbid hyperplasias in specific diseases—glanders, tuberculosis—taking place in the spleen or lungs and thus directly or indirectly causing chronic congestion of the spleen (Morot, Leisering, Nocard, Varnell) and again as the result of innervation, in disease of the splenic plexus of nerves (Varnell).

Lesions. The increase in size may be enormous (42 lbs. (Bouret and Druille), 92 lbs. (Cunningham), and over 100 lbs. (Girard)). The consistency is varied. There may be such a redundancy of blood and splenic pulp that the capsule is distended to its utmost or even ruptured (Peuch). In other cases the splenic veins have given way and the blood has poured out into the abdomen with fatal result (Crafts, Cunningham, Reis). In other cases the spleen is enlarged, unevenly swollen and indurated by the formation of angioma (Jacob), lymphadenoma, glander or tuberculous nodules. In still others the capsule and fibrous framework are greatly thickened and the substance of the organ has assumed the consistency of the hepatized lung (Rodet).

Symptoms. These are suggestive rather than diagnostic. Most prominent is the condition of the blood with excess of leucocytes and especially of the eosinophile cells. Weakness, emaciation, feebleness of pulse, bloodlessness, bleeding from the nose or other natural passages, are attendant symptoms. In cases of extreme hypertrophy distension of the abdomen is marked and even the enlarged spleen may be made out by palpation, there may be special tenderness and dullness on percussion. Even partial sweats over the region of the spleen (Cadeac), and stretching with the fore feet far in advance (Welsby) have been noted as symptoms. In such conditions the animal walks stiffly, groans in turning, or when suddenly started and is with difficulty urged beyond a walk. There may be ascites, signs of colic, or irregularity of the bowels. Rectal exploration may reveal the hypertrophy.

Treatment is usually the treatment of the primary disease. In glanders, tuberculosis, lymphadenoma, or leucocythemia there is little to hope for. Nor is there much in hepatic cirrhosis, obstruction of the vena cava or valvular disease of the heart. In simple hypertrophy we may resort to quinia or other bitters, eucalyptus, saline laxatives, exercise in the open air and sunshine, and local currents of electricity.

SPLENIC HYPERTROPHY IN RUMINANTS.

A moderate hypertrophy is the rule in the case of cattle which have passed through the Southern cattle fever, but have continued to live within the area of its prevalence. Gamgee's observations in 1868 were very conclusive on this point. In over 1,000 western cattle the average weight of the spleen was 1.45 lb., in 441 Cherokee (Indian Territory) cattle the average was 2.34 lbs., and in 262 Texas cattle the average was 2.66 lbs. All these animals were killed for beef, in what was considered to be perfect health. The difference relative to the weight of the entire animal is even greater than is indicated above, for at that date even more than at present, the Texas steer was a small and thin animal in comparison with the portly western bullock.

In lymphadenoma the organ may weigh 24 lbs. (Tannenhauser); in simple hypertrophy it has been found to weigh 37 lbs. (Koch). There was usually a marked increase in the size and number of the Paccinian bodies, and hyperplasia of the fibrous reticulum, while the pulp might be deficient and the cut surface rather dry. The adjacent lymph glands are usually enlarged.

Symptoms. Unless in the case of excessive increase, no symptom is usually observable, apart from leucocythæmia. With enormous hypertrophy the enlarged organ may be recognized by palpation, percussion, and perhaps rectal exploration.

Treatment is unsatisfactory apart from the control and arrest of the primary diseases. For simple hypertrophy, bitters, laxatives and electricity may be tried.

SPLENIC HYPERTROPHY IN SWINE.

Causes. This disease appears to be rather frequent in pigs, in connection with high feeding, and more particularly with leucocythæmia and lymphadenoma. It is further a complication of tuberculosis and of neoplasms located in the spleen, and of hepatic, cardiac and pulmonary disorder.

Lesions. In leucocythæmia there is general enlargement of the spleen, and especially of the Paccinian bodies which may attain the size of a pea (Leisering, Fürstenberg, Bollinger, Siedamgrotzky, Röhl, Ellinger). The total weight of the organ may attain to 5 lbs. (Mathieu), or 13 lbs. (Goubaux). In a remarkable case recorded by Zell, the organ measured 30 inches in its longest circumference and 20 inches in its shortest. It had an enormous thickening of the capsule and trabeculæ which enclosed softened contents in a state of fatty degeneration.

Symptoms are wanting, as most of the observed cases were only discovered after the animal had been killed for pork.

SPLENIC HYPERTROPHY IN THE DOG.

This condition has been less frequently seen in dogs, the recognizable causes having been traumatism (Notz), and leucocythæmia (Zahn, Forestier, La Forgue, Nocard). Lymphadenoma is another complication (Nocard, Leblanc, Siedamgrotzky, Bruckmüller). The spleen has been found to weigh 2 lbs., (Bollinger, Siedamgrotzky). As in other animals the enlargement of the Paccinian bodies has been a marked feature. In other cases the splenic lymph glands are enlarged.

The *symptoms* are obscure as in other animals. Yet the presence of white cell blood, with a predominance of eosinophile cells, enlargement of the abdomen, and the detection of a large solid body in the left hypochondrium which proves tender to the touch may prove more satisfactory than in other animals. In certain cases it has obstructed the biliary duct by pressure and entailed hepatic disorder and jaundice.

The *treatment* would not differ from that of the larger animals.

Siedamgrotzky has also observed splenic hypertrophy in the cat in connection with leucocythæmia.

SPLENITIS. PERISPLENITIS.

Causes: extension from adjacent inflammations, penetrating bodies, contusions, lacerations, infections, over exertion, cold, damp, over feeding. Symptoms: those of primary disease, visible traumas, chill, fever, swelling, flatness of percussion sound, absence of crepitation, anorexia, vomiting, constipation, diarrhoea. Prognosis usually good. Treatment: castor oil, enemata, cold douche, electricity, phlebotomy, in infective cases quinine, salol, salicylates, iodides.

No accurate border line can be drawn between splenic hyperæmia and hypertrophy on the one hand and inflammation of the spleen on the other. It is, however, not difficult to assign to inflammatory action all cases that tend to suppuration and abscess. Also in perisplenitis with adhesions to adjacent parts like the liver, stomach, intestine, kidney or abdominal wall inflammation cannot be doubted.

Causes. Extension from the disease of adjacent parts—perihepatitis, perinephritis, peritonitis, enteritis—is a distinctly appreciable cause, as are also penetration of the spleen by foreign bodies, contusions, lacerations and infections of the organ. Cruzel, who claims an extensive acquaintance with the disease in working oxen, attributes many cases to violent exertions, overdriving, cold and damp weather, and an overstimulating alimentation. As inflammation may supervene on hyperæmia and hypertrophy we must accept the various causes of these conditions as factors in producing inflammation.

Symptoms. Most observations of inflammation of the spleen and its results have been made only post mortem, so that we must allow that the simple forms occur and undergo resolution without obvious symptoms. In the perisplenitis supervening on another disease also in infective cases there will be the antecedent symptoms of such primary diseases. In those resulting from traumatic injury, bruises, swellings or wounds, cutaneous or subcutaneous, there will often be suggestive features. In the more purely idiopathic cases symptoms are only shown when the lesions are extensive and acute. In oxen, Cruzel has noted the initial chill, followed by disturbance of the respiration, more or less hyperthermia, and a swelling of the left flank and hypochondrium in the absence of tympany of the rumen. The nature of this swelling is the most characteristic feature, as it gives a flat instead of a drumlike sound on percussion, and does not bulge outward and downward over the whole left side of the abdomen, pit on pressure, nor crepitate uniformly all over from fermentation, as in overloading of the stomach.

If abscess should form, chills and high febrile reaction are marked symptoms. In vomiting animals, anorexia, nausea, vomiting, constipation, and even diarrhoea may appear.

Prognosis. Unless in extreme cases and those due to traumatism or infection, the result of splenitis is usually favorable.

Treatment would consist in depletion from the portal system and spleen by rectal injections, and laxatives which like castor oil, will operate without extensive absorption. Cold water or ice applied to the left flank and induction currents of electricity may also be resorted to. General blood-letting is strongly advised by Cruzel, and Friedberger and Fröhner. In infective cases quinia, salicylates, salol, and the sulphites, or iodides would be indicated.

HÆMORRHAGIC INFARCTION OF THE SPLEEN.

In congestive conditions. Absence of free capillary anastomosis and contraction, absence of valves in splenic veins. Embolism of splenic artery. Clots in pulp spaces. Wedge shaped infarcts, first black, later yellow, later caseated, or cicatrized. Abscess. Prognosis good in non-infective forms. Treatment as for hyperæmia, or infection, or both.

This condition appears in hyperæmia, hypertrophy, splenitis, and splenic infection and largely because the structure and circulation in the organ conduce to such trouble. The splenic arteries terminate in open vascular spaces filled with splenic pulp and where all trace of a freely anastomosing capillary network is lost. The splenic veins in the same manner originate from these open vascular spaces. There is, therefore, an absence of the free communication of capillary network, which virtually acts as a safety valve in other vascular tissues, and the vascular cavities connected with each terminal artery are independent of those belonging to another, and find no way of ready relief when they become over distended, or when there occurs obstruction (thrombosis) of their afferent or efferent vessels. From blocking of arteries or veins there is at once produced a wedge shaped area of stagnation which cannot be relieved through any collateral circulation. Again the splenic veins, being destitute of valves, offer no obstacle to the reflux of blood into such vascular spaces whenever the further access of blood has been arrested by the blocking of the artery. The blocking may occur in the afferent artery through embolism by clots carried from the lungs or left heart, or formed within the vessel by the colonization of microbes on its walls. Even more likely is the formation of coagula in the vascular spaces themselves as the result of the introduction of pus, or septicæmic microbes, which are long detained and have ample time for multiplication in these cavities. In either case the result is obstruction to the sanguineous current, the filtering of blood backward from the veins and the engorgement of the cavity with blood. The plugs consist of fibrinous matter enclosing colonies of micrococci, and the result is not only black infarction of the spleen, but a subsequent general infection of the system at large.

The wedged shaped infarcts are usually situated at the surface of the organ, the base turned outward and forming a dark projection on the surface, and the apex turned inward. The aggregation of two or three in one group may considerably alter the outline. If recent they are of a dark red color. Later from absorption of the coloring matter and fatty degeneration of the mass they assume a pale yellow hue and the swelling flattens or disappears. Later still through complete fatty degeneration they may be transformed into caseated masses, or through organization into fibrous tissue they may form thick white cicatrices. If pus cocci are present suppuration and abscess may be the outcome.

The simpler forms recover like cases of simple hyperæmia while the severe infecting forms may become the point of departure for the formation of multiple abscesses in other organs, and of more or less fatal general infections.

These conditions can only be discovered post mortem, and any symptoms directing attention to the spleen could only suggest such treatment as would be indicated in hyperæmia. Any purulent or septic disease which might coexist would of course serve to indicate a germicide line of treatment.

ABSCESS OF THE SPLEEN.

In *Solipeds*: in infectious diseases, pyæmia, embolism. Symptoms: of primary disease or ill health. Involving other organs. In *cattle*: foreign bodies from reticulum, distomata, embolism, microbes. Enlargement: involving other organs: seen in left hypochondrium, fever, albuminuria. Treatment: aspiration, antiseptic injections, internal antiseptics.

Soliped. Abscess of the spleen in this animal is unusual and has only been discovered post mortem. It has been found as the result of the local colonization of pyogenic microbes, in connection with strangles, contagious pneumonia and other infectious diseases and can then often be traced to an infected embolus in the splenic blood vessels. The peculiar vascular structure of the spleen is very conducive to abscess as it is to infarction, as has been already noticed and hence this complication of a pre-existing infection in another part is a natural pathological sequence. Symptoms are rather the general ones of a rigor followed by hyperthermia than any diagnostic ones of splenic disease. Bourges found a splenic abscess in a cachectic, melanic mule but no definite splenic symptom was observed even on rectal examination. Nottel found an abscess as large as an infant's head, in the base of the spleen, closely adherent by its sac to the left kidney and containing a floating mass of splenic tissue as large as the closed fist. Rutherford found a neoplasm connecting the great curvature of the stomach, to the diaphragm, and hollowed out into a series of pus cavities. Fetzner and Cadeac report cases of extensive abscesses in the head of the spleen and intimately connected to both stomach and diaphragm. Hahn found abscesses in connection with the penetration of the spleen by foreign bodies. In other cases the substance of the spleen was studded with abscesses varying in size from a pea upward and containing necrotic tissue or adjoining such dead tissue.

Ruminants. In cattle the penetration of the spleen by sharp pointed bodies coming from the reticulum appears to be the most common cause of abscess. Other cases depend on the penetration of distomata carrying the pyogenic microbes, and still others are due, as in the horse, to local infection with embolism. External traumatism is unusual causes. There is usually considerable enlargement of the spleen as a whole, rounded swellings indicating the seat of the abscess, and adhesions to surrounding parts, such as the rumen, the left kidney or the diaphragm. When the abscess is chronic, there is emaciation, unusual flatness on percussion of the left hypochondrium, and, at times, of the flank, swelling and tenderness of the flank, above all, according to Imminger, a persistent elevation of temperature (104° to 106° F.), which is not lowered by antithermics, and albuminuria. In cattle it is sometimes possible to diagnose the disease, and if the abscess can be definitely located, aspiration and antiseptic injections into the sac would be indicated, conjoined with calcium sulphide, or sodium sulphite internally.

FOREIGN BODIES IN THE SPLEEN.

In *horse*: body from intestine. In *ruminants* bodies from reticulum. Laparotomy.

One such case in the **Horse** is reported by Hahn. A mare had loss of appetite, slight colics, frequent efforts to urinate, dullness, prostration, profuse perspirations, and tremors of the muscular walls of the abdomen. Rectal examination detected a staff-shaped body extended from behind forward in the direction of the stomach. The mare survived twenty days, when it was carried off by a more violent access of colic. At the necropsy, the spleen was found to measure 28 inches by 8; its base was adherent to a loop of intestine, and presented a large cavity filled with a grayish brown foetid liquid, and a piece of oak measuring 17 inches by $\frac{1}{2}$ inch.

Ruminants. In cattle and especially in those that are stabled, needles, pins, nails, wires and other sharp pointed bodies, that have been swallowed with the food, and have become entangled in the reticulated walls of the second stomach, have been found to penetrate the spleen and determine local abscess and fistulæ. The offending body in such cases is found in the interior of the abscess or in its walls. If such cases can be diagnosed the superficial position of the spleen would seem to warrant surgical interference for the removal of the foreign body.

RUPTURE OF THE SPLEEN.

Solipeds: Causes: Blows, kicks, goring, leukæmia, compression of splenic or gastric veins, anthrax infection. Lesions: Blood may escape into peritoneum or remain confined under serosa. Splenic degeneration or pulpy condition. Fractured ribs, ecchymosis, surface swellings. Spontaneous arrest. Symptoms: Of internal hemorrhage. Vomiting. Trembling. Vertigo. Coma. Treatment: Rest, quiet, locally ice, snow, cold, internally iron chloride, matico, astringents, anodynes. *Cattle*: Blows, crowding, leukæmia, youth, anthrax, Texas fever, microbes. Symptoms: Persistent lying down, advancing bloodlessness, surface coldness, stiffness, local tenderness, fluctuation. Treatment as in horse.

Horse. This is not a frequent lesion in solipeds, yet the number of cicatrices of the spleen which are found post mortem in old horses would indicate a considerable number of slight and non-fatal cases. The most common cause appears to be external violence and especially kicks or blows with horns on the left hypochondriac region. Horses running at large in pastures, or in yards, or standing side by side in short stalls or tied with too long halters are the usual victims. Cadeac refers to cases reported by Tausch, Millot, Berndt, Humbert and Pont, and one case occurring in a three year old colt came under the notice of the author. The subject stood in a stall to the right of an irritable mare, and though the kick left no noticeable skin lesion the colt died in three hours with symptoms of internal hemorrhage. At the necropsy a laceration of the spleen of about five inches long was disclosed, and a large quantity of blood had accumulated in the peritoneum.

Brandis mentions a case consequent on a violent fall on the left hypochondrium.

In other cases pre-existing disease of the spleen or its blood-vessels have proved active factors. In the friable degenerated spleen of leucocythæmia multiple small lacerations have been found (Peuch, Laulanie); in engorgement of the spleen consequent on thrombosis of the splenic vein (Wiert); in chronic indigestion with habitually loaded stomach compressing the gastric and hepatic arteries and determining a reflux of blood through the cæliac axis into the spleen (Mongin).

Finally, though less frequently than in the ox, the engorgement of the spleen with blood in cases of anthrax may be a cause of rupture.

Lesions. The rupture may be on any part of the spleen and it may be complete or incomplete; in the latter event the capsule may have ruptured while the more elastic peritoneal covering has remained intact enclosing a coagulum of variable size bulging above the level of the spleen. When the peritoneal coat has given way, its laceration is usually smaller than that in the spleen and its proper envelope. Any degeneration of the spleen will affect the appearance of the lesion. In one case caused by external violence the adjacent portions of the spleen were reduced to a soft pulp. In such a case there is a slow but continuous flow of blood in a small stream which may, however, prove fatal (Humbert and Pont).

Again in cases caused by external violence there may be fractures of the ribs, ecchymosis, local swellings or even wounds of the skin, but all these may be absent. The blood effused into the peritoneum is usually clotted. If the effusion has taken place slowly it is more or less coagulated around the edges of the wound or even in its depth and in this way the hemorrhage may be arrested. When the peritoneum is still intact the pressure of the clot beneath it has served to arrest the flow. In such cases the clot may be in part liquefied and absorbed and in part organized into fibrous tissue, constituting the cicatrices of the spleen found in old horses.

Symptoms appear to have been varied. Colic pains are generally noted. Tausch has observed vomiting, Millot vertigo, and Wiert coma and trembling. In the author's case the animal was found down, unable to rise, almost unconscious, pulseless, with great pallor of the visible mucous membranes, dilated pupils, and cold extremities. A diagnosis was made of internal hemorrhage, but its actual seat was only revealed post mortem.

Treatment. The early mortality usually forbids treatment. When opportunity is furnished keep the animal absolutely still and quiet, apply snow, ice or other refrigerant to the left hypochondrium, give internally tincture of muriate of iron, matico, or other astringent, and relieve any severe suffering by anodynes (hyoscyamus, belladonna, opium). External wounds may be treated antiseptically.

Cattle. The *causes* of laceration and hemorrhage of the spleen are similar to those acting in the

horse. Blows with the horns on the left side, crowding through a doorway or gateway, and direct blows of other kinds are charged with its pathogenesis. The friability which attends on leucocythæmia has been noted as a predisposing cause. Calves by reason of their small size and the relative bulk of the spleen are especially liable to rupture by kicks from animals or men.

Much more commonly than in solipeds, rupture of the spleen occurs as a complication of specific microbial diseases like anthrax and Southern cattle fever.

Symptoms. The mature animal assumes the recumbent position, refusing to rise, and dies in a few hours. In calves, life may be prolonged for a few hours longer, and there have been noticed, anorexia, watering of the eyes, accelerated pulse and respiration, arrest of intestinal peristalsis, cold ears, rigid limbs, and moderately full belly (Notz). There should also be tenderness on manipulation or percussion of the left hypochondrium, and until coagulation occurs, fluctuation in the lower part of the abdomen, with pallid mucous membranes and other signs of profuse internal hæmorrhage.

Treatment is useless in the majority of cases. In the slighter forms it would be the same as in the horse.

TUMORS OF THE SPLEEN.

Secondary. Delay of blood favors. *Sarcoma*: horse, cattle, dog. *Carcinoma*: horse, dog. *Melanoma*: common and large in gray and white horses, rupture, external melanosis. *Angioma*: horse, ox. *Lymphadenoma*: horse, ox, external adenoma.

The different tumors of the spleen are usually secondary. The intimate structure of the organ, the peculiarity of the circulation through the pulp cavities, and the delay of the blood in the pulp spaces, predispose it in a very especial manner to the growth of neoplasms, the germs or bioplasts of which are carried in the blood.

Sarcoma. In the *horse* sarcomata have been found in the spleen secondary to similar tumors in the other parts of the abdominal and thoracic cavities. They may attain to any size, from a pea to the closed fist and, in exceptional cases, of a mass which practically fills and distends the abdominal cavity.

In the *cow* an encephaloid sarcoma in the spleen, weighed nine pounds and was associated with similar formations in the lymph glands generally of the abdomen and chest.

In the *dog* also sarcomata are common in the spleen.

Carcinoma. These are found in the *horse* in connection with similar primary tumors, as in the case of the sarcomata. They are at times extremely vascular and soft, and at other times they are hard and fibrous (scirrhus).

In the *dog* secondary cancer of the spleen is comparatively common.

Melanoma. Black pigment tumors are especially common in gray and white horses. Their common seat is on the black, hairless portions of the skin (anus, vulva, perineum, tail, sheath, mamma, eyelids, lips, etc.), and secondarily in the lymph glands and spleen. In the latter they may grow to an extreme size, Wehenkel having mentioned one specimen of 60 pounds. Its surface is marked by uneven, rounded black swellings, the entire organ, indeed, seeming to be a conglomerate of these masses. The intimate structure is that of a sarcoma, so abundantly charged with black pigment granules that these appear to make up the greater part of the mass.

Rupture of these neoplasms with the escape into the abdomen of blood highly charged with the melanic matter is not uncommon.

The *symptoms* of the splenic deposits are not usually recognizable, but indications of chronic abdominal disease in connection with external melanotic formations may well lead to a reasonable suspicion.

Angioma. In a *horse's* spleen weighing 30 lbs., there were numerous soft nodules of a deep cherry color. These were cavernous masses with connective tissue walls and the meshes filled with blood.

Similar vascular cavernous tumors have been found in the cow.

Lymphadenoma has been found in the spleen of horses and cattle in connection with the same disease of the lymph glands.

Like the other splenic tumors this is obscure and usually only found after death. The existence of adenoid swellings elsewhere conjoined with excess of white globules and indications of abdominal pain would be suggestive of splenic disease.

AMYLOID DEGENERATION OF THE SPLEEN.

Amyloid: horse: with exhausting diseases. Waxy secretion staining mahogany brown with iodine.
Gangrene: Swine. *Tubercle and glander nodules.*

This has been occasionally detected in the spleen of the horse. It is usually connected with longstanding suppuration especially of bones, with advanced tuberculosis or other exhausting disease. The organ is usually greatly enlarged and the affected parts are firm, resistant and swollen. On section it has not the soft friable or pulpy appearance of the spleen, but an uniform waxy looking consistency, grayish or sometimes stained with blood. On the application of a solution of iodine and iodide of potassium the healthy splenic tissue is colored yellow, while the amyloid portion becomes of a deep mahogany brown.

GANGRENE OF THE SPLEEN IN SWINE.

Hertzen records the case of a pig in which the spleen had become gangrenous and lay free in a surrounding fibrous capsule.

TUBERCLES AND GLANDERS NODULES OF THE SPLEEN.

Tubercles in the spleen are common in cattle, swine, guinea-pigs, rabbits and cats, in the last largely as the result of ingestion of tuberculous meat. In the larger mammals individual tubercles are usually of the size of a walnut and upward, while in the smaller they show as miliary deposits. The products are often caseated or calcified.

Glander nodules are found in the spleen of the horse and other solipeds and as the result of inoculation in that of rabbits and guinea-pigs. In solipeds they may be of considerable size whereas in the inoculated rodents they are usually small and numerous—like millet seed or pins' heads.

PARASITES OF THE SPLEEN.

Parasites are less common in the spleen than might be expected yet the encysted parasites of the liver and pancreas, are also to be found in the spleen. Thus *echinococcus* is found in the spleen of cattle, and headless hydatids in that of the horse; *cysticercus tenuicollis* in the spleen of sheep; *cysticercus cellulosa* in that of pigs; *distomata*, and *pentastoma denticulata* in the spleen of cattle; *coccidia* in the spleen of rabbits; and *actinomyces* in that of horses and cattle.

In addition to these the spleen is a general *rendezvous* for the different pathogenic organisms that can survive in the blood stream, such as the bacilli of tubercle, glanders, septicæmia, anthrax, black quarter, swine plague and hog cholera, and for the cocci of suppuration, strangles, contagious pneumonia, etc. (See Parasites and Contagious Diseases).

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