ception or invaginated portion. This may be primary, or secondary to an intussusception of the small bowel, enteric, which pushes the valve in front of it. In no other sense should the term be used, and care should be taken that its use is based on positive and not negative evidence.

Finally, I would like again to draw attention to the agonal intussusceptions that occur during the "death agony," which are characterised by their multiplicity, being frequently of the ascending variety, easily reduced, accompanied by no signs of inflammation, etc. These are undescribed in animals.

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ON CERTAIN SEPTICÆMIAS AND SOME OTHER INFECTIONS OF YOUNG ANIMALS.¹

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FOR many years, in different parts of the world, raisers of stock have had to contend with losses of young animals which die a short time—a few days or weeks—after birth.

Deaths in young stock may be attributed to a variety of causes, but there is one cause now almost universally recognised, and which is included under the general name of septicæmia. That septicæmia, a very elastic term in this connection, is indicated on *post-mortem* examination most people who have had opportunities of a large number of *post-mortem* examinations will readily allow, but the question arises, what is the cause of the septicæmia, is there more than one cause, and how does infection occur? Then, again, do all animals that are infected die? or does the primary infection at times pass away, leaving the body deprived of its protection, open to the inroads of numerous saprophytes and parasites which otherwise would remain outside the body and harmless? Does the entrance of these organisms lead to serious lesions and perchance death, a secondary lesion, or may be lesions, developing as the result of their entrance into the body? I think it well, though it may appear to extend beyond the presumed scope of the paper as indicated by the original title, to consider these secondary lesions, as I hold that their value and importance is not inferior to the true cases of septicæmia which are more or less rapidly fatal.

But first, what is septicæmia? I consider septicæmia may be due to the presence and development of an organism in the body, it may be due to a toxin, or it may be due to both combined.

In a great number of septicæmias the causal agent may be more or less readily found in the blood, in other fluids or in organs, and inoculation of a minute quantity of material taken from such a case is capable of producing the same disease in suitable animals. To make my point clear, I consider anthrax in this sense a septicæmia.

In a second class we have an intoxication; the affected animal being poisoned by the toxin poured into the body. In this case

¹ Contributed to the proceedings of the National Veterinary Association, Sept. 1903.

inoculation of the fluids or juices of the body will not produce the original disease in animals of the same weight and size as the primarily affected animal, the amount of toxin in the fluid used for injection is too small, and will only produce any reaction in the smallest experimental animals where the dose of injection is relatively high when compared to the weight of the experimental animal. In this latter case there is no new formation of virulent material in the experimental animal such as in the first case, where the virulent material is capable of rapidly proliferating and giving rise to an immense new stock of virulent organisms. It is possible in this second form of septicæmia to produce an infection by injections of toxin produced by micro-organisms that have never been in the animal body.

If these two types of septicæmia are admitted, then the third is not improbable—a few micro-organisms producing a virulent toxin capable of producing death, an extension of the process to the body which occurs frequently enough in different, it may be isolated organs of the body.

It is perhaps to septicæmia of the first type—septic infection—that we must look as the chief form of septicæmia in young animals. Septic infection occurs through a wound either of skin or of mucous membrane, through any portal that will allow of the entrance of a micro-organism. But can infection occur through an uninjured membrane where there is no solution of continuity? This is not so certain, though many experiments, feeding experiments particularly, seem to show that ingestion of virulent material may set up disease through the alimentary tract. Swine fever, tubercle, anthrax, possibly black-quarter, may be cited. It is difficult to assert that there is no wound of such a membrane as that lining the alimentary tract, because it may be microscopic, merely a denudation of a few epithelial cells, and yet be sufficient to admit the organisms to the body. It is probable that where animal parasites are present in the gut the condition of the mucous membrane is rendered favourable to the entrance of organisms. Young animals, a few days old, however, are not commonly the hosts of intestinal animal parasites.

The skin when intact offers practically an insuperable barrier to the passage of organisms, though the rubbing in of a pure culture of the staphylococcus pyogenes aureus gave rise to a crop of abscesses upon the apparently intact skin of the experimenter. The application, if violent, may have produced a wound, but in this case the cocci are said to have been arrested in the hair follicles and glands of the skin. Even if this is the case they were still outside the body, but it is probable that the toxin associated with them may have killed the epithelial cells, allowed the entrance of the organisms, and as a consequence the formation of an abscess. In most cases, however, I think we may consider that our septicæmias are not due to entrance of organisms through an intact epidermis, but that in most, if not in all, there is a wound. In the newly born there is such a wound, the umbilicus, and the position of the wound is favourable to soiling—to infection. As a rule young animals are born where hygienic surroundings are altogether absent. Frequently enough they are born on a floor soiled by excretions, and their

very helplessness favours the infection by the vain attempts to rise made by the newly born. Until quite recently no attempts were made to cleanse the umbilical cord nor to seal the umbilical wound, and that infection does occur through the wound and cord may be readily demonstrated in cases of omphalo-phlebitis, the umbilical vein being distended with pus carrying an immense variety of micro-organisms. The presence of pus in this vein and nowhere else discoverable in the body shows it is the primary centre.

I think therefore we are justified in concluding that septic infection occurs through a wound; that the wound may be in the mucous membrane of the intestines, or in the skin, or on the superficies of the body; and that most probably the umbilical wound is the main port of entrance. In some cases the above remarks apparently do not apply, as in septic pleuro-pneumonia in calves, a disease said to be contagious, but we shall refer to this again.

Of recent years the different forms of septicæmia, those grouped in the class of septicæmia hæmorrhagica, have multiplied or possibly, owing to better technique and advance of science generally, observers have recognised maladies which previously were passed over, or included under other forms, and hence it is difficult in a paper such as this to mention all the types recognised by authors. I hope, however, to touch upon the most important and better established.

WHITE SCOUR IN CALVES.

A disease very prevalent in the south-western parts of Ireland and investigated in 1901 by Prof. Nocard and myself. The mortality from this disease was very serious, indeed many farmers were practically unable to rear any of their calves, and the yearly loss to the country became appalling. It has probably been prevalent for many years on isolated farms, but recently, in the decade 1890-1900, it became a veritable scourge. The disease manifests itself by diarrhœa, which usually appears on the second or third day following birth; rarely it makes its appearance on the first day, sometimes as late as the sixth; the farmer, if the calf shows no signs after the eighth day, thinks his calf will escape. The discharges from the bowels are thin, yellowish-white, frothy, and stinking. There is considerable tenesmus. The diarrhœa and straining soon render the animal so weak as to be unable to stand, and it lies constantly, soiled with its own dejections, moaning and grinding its teeth. Frequently there is a thin mucopurulent discharge from the nose which the calf makes no effort to clear away. The diarrhœa continuing becomes streaked with blood and bloody, the eyes sink in the head, the animal lies continually, groaning all the time. It refuses its food. It coughs, and sooner or later dies—a miserable object indeed. The temperature, at first high, may remain normal, or about normal for some little time, to fall and become markedly subnormal. I have observed a temperature as low as 92° F. just prior to death.

In some cases of acute white scour there is an intense arthritis of one or more joints. The joints particularly involved are the elbow, knee, stifle, and hock. The joint is hot and swollen and no weight is put upon the limb at all, the animal persistently lying, or, when up, standing with the limb flexed. Sometimes the limb between the joints

stifle, and hock, is also involved as well as the two joints. The muscles, intermuscular fascia, and fasciæ of the leg generally are infiltrated with a yellowish serum which gives rise to the swelling.

Post-mortem Examination.—The umbilical cord is thickened, soddened with a blood-stained fluid. The umbilical vessels are thrombosed, swollen, with the thrombus breaking down. The peritoneum joining the arteries is puce colour. Numerous petechiæ and ecchymoses found upon the peritoneum, especially on the serous covering of the stomachs. The liver is swollen but shows no gross lesions, microscopically there are minute areas of necrosis. The lymphatic glands are swollen, juicy, sometimes hæmorrhagic. The kidneys often congested, sometimes showing catarrhal nephritis. The urinary bladder contains a highly albuminous urine, and the mucous membrane is very vascular, swollen, and ecchymosed. In later cases there is a fibrinous peritonitis. The mucous membrane lining the first three compartments of the stomach is often diffusely hæmorrhagic; in the abomasum minute capillary punctiform hæmorrhages are common, the appearance on the washed mucous membrane is like a sprinkling of coffee grounds. The small intestines are congested, and the lymphoid tissue is infiltrated with blood and often breaking down. This condition is sometimes well seen in the large intestine, and especially when the fæces are blood-stained.

In the chest the pleura shows petechiæ and ecchymoses; the lungs areas of collapse, often small lesions of a hæmorrhagic pneumonia. The epicardium and the endocardium show beneath them minute hæmorrhagic extravasations. Similarly minute hæmorrhages into the substance of the valves may be found.

The lesions in acute cases are distinctly those of septicæmia. The sequelæ observed in animals that have recovered from an acute infection—white scour, are lung lesions, pyæmia, and necrosis.

The cause of the infection, as demonstrated by Nocard in Ireland, by Lesage and Delmer in France, and by Lignières in France and the Argentine, is an organism which belongs to the same family as that causing fowl cholera. This family is notorious for the septicæmic lesions produced, and is classified as that giving rise to septicæmia hæmorrhagica. Lignières has proposed the name *pasteurella* for the group, and names the diseases pasteurelloses. As showing the importance of these micro-organisms we may mention in passing that one or other of the family is the causal agent of fowl cholera, some forms of swine-fever, septicæmia of sheep, the disease of wild or semi-domesticated ruminants (wild seuche and rinderseuche), barbone des buffles, various forms of so-called influenza of the horse, and distemper in the dog. "The organism is a cocco-bacillus, non-motile, not taking Gram, polymorphous, not liquefying gelatine, not coagulating milk, which remains normal, does not grow on normal acid potato, does not produce indol in pancreatic bouillon, does not form acid, aerobic, but may be grown as an anærobe, produces an odour *sui generis*. No spores, no cilia. Of variable virulence; usually, however, it is high. When injected into the blood stream, the organisms show a predilection for serous and synovial membranes."¹

The organism of white scour, according to Lesage and Delmer,

¹ "Contribution à l'Etude et à la classification des Septicémies Hémorragiques," par J. Lignières, Buenos Aires, 1900.

may be found with many others in the intestinal discharges of suffering calves, and they say also in the bronchial mucus. Feeding experiments to healthy calves have failed to give the disease (Nocard, Lesage, and Delmer). Intravenous injection of a culture of the organism will kill a calf within thirty hours, and an adult cow which received 5 cc. of a culture died in twenty-six hours with most pronounced lesions of septicæmia. Application of a culture to the navel, either by rubbing on the culture or by tying on a pad of cotton wool soaked with culture, produced the disease (Moussu). Subcutaneous injections of the culture were not so successful. It appears, therefore, that entrance of the organism into the blood stream is essential to produce white scour, and that the port of entry in practice is the umbilical wound.

Thomassen has also described a septicæmia in calves associated with nephritis and urocystitis.¹ The disease affected calves in the neighbourhood of Utrecht, and proved to be very fatal. As the symptoms and lesions are different from those mentioned as observed in white scour, it may be as well to cull largely from the original paper and give them in some detail.

Symptoms.—The first symptoms are observed, not soon after birth, but towards the fifth to eighth day and even as late as four or five weeks. The animals have lost their spirits and lie constantly, either stretched out on the ground or with head turned back on the thoracic wall. Made to get up *they stretch themselves and bend downwards* (not arch upwards) *the back and loins*. (Particular stress is laid upon the symptom italicised.) The muzzle is dry and the respirations hurried. Pulse 100 to 150. The temperature is 104° F. and even higher. Sometimes there is a dry cough. Though the appetite is not good still the animal will take milk. Generally the droppings are normal in colour and consistence; they may be streaked with blood; diarrhœa has been noticed, but nothing approaching dysentery. The urine was often passed in small quantities; it was clouded, but red blood corpuscles were not at first suspected. Boiling with potash lye, however, gave a red colouration. It contained a large quantity of albumen, epithelial casts, and micro-organisms. The thoracic organs were sound. In some patients there were cerebral symptoms (somewhat similar symptoms were observed in calves suffering and dying from white scour. Toxæmia?).

The disease generally lasts some five or six days, and terminates fatally.

Lesions.—The lungs were found collapsed, the pleuræ were sound. No inflammation of the lungs. The blood in the heart was not coagulated, numerous ecchymoses upon endocardium and especially involving the valves. The bronchial lymphatic glands were swollen, softened, and showed capillary hæmorrhages. From the peritoneal cavity a clear fluid escaped on opening the abdomen, and the serous covering of the gut showed hæmorrhagic areas. The spleen was very much enlarged, even five or six times greater than normal; the capsule tense, violet, and glistening. The organ was soft, filled with blood.

There was, in all animals, a hæmorrhagic parenchymatous nephritis. The kidney on section was red, sometimes the redness being confined

¹ "Annales de l'Institut Pasteur," Tome onzième, 1897. Page 523.

to the medulla of the organ. The urinary bladder contained a highly albuminous urine with casts and epithelial cells and organisms. The mucous membrane was brownish-red in colour, uniformly or in streaks or patches. The ureters were also coloured.

The mucous membrane of the abomasum and small intestine showed ecchymoses, and the patches of lymphoid tissue were often swollen. The liver showed nothing pathognomonic. A slight meningitis with effusion was found in those cases where brain symptoms had been shown.

Thomassen shows that this septicæmia is different from that described by Jensen or Poels, and that it was not the septic pleuro-pneumonia of calves was evident, as the lungs and pleuræ were free from lesions.

It is also different from the malady we recognise as white scour, not only as to period of incidence of the disease, but also as to symptoms and lesions. Thomassen shows that this form described by him is due to an organism not taking Gram. It is allied to the colon bacillus (*B. coli communis*). It grows readily upon the usual media. Does not liquefy gelatine. It grows upon potato—which separates it from the class of organisms of which that causing white scour is a member; the culture, however, is an invisible one, which differentiates it from the colon bacillus and allies it with the organism causing typhoid fever of man. It is a motile organism. I have merely mentioned these particulars to show that Thomassen's organism is not that causing white scour. Further, it is more highly pathogenic than the usual coli organisms. Injected into calves, it produced death with all the symptoms and lesions in the experimental calves of the original disease. The organism is not pathogenic for the dog or horse.

Attempts at treatment failed. Carbolic acid in 2 per cent. solution was injected subcutaneously. Eucalyptol in olive oil was employed in like manner without result. Spirits of camphor per os, preparations of iodine injected into the veins, were all equally useless.

Another comparatively common septicæmia of calves is that known as septic pleuro-pneumonia, a disease first recognised in Holland, then in Belgium, Denmark, Prussia, France, Italy, and Russia. The organism found in animals dead or killed when suffering from the disease is similar in appearance, tinctorial behaviour, and in culture media to others of the septicæmic group. It affects calves, pigs, and kids. The symptoms of serious ill health are pronounced, and often the subject dies in a few hours after it is noticed to be ill. The young animals rapidly become weak, are unable to rise, refuse to suck or take food. In this malady the skeletal muscles are affected. The limbs become stiff, those in front are bowed, the hind limbs thrust far under the trunk. The animal moves with difficulty, and is evidently in great pain. Lung symptoms make their appearance, as noted by the respirations and cough, and by the soreness of the chest to palpation and manipulation generally. Auscultation shows dullness, râles, and tubular breathing. A fetid diarrhœa then sets in. Sweating, congestion of the visible membranes, frothing at the mouth, are also noticed. In young pigs the symptoms are allied to those of swine fever.

Lesions.—The lesions in the acute forms are found in the thoracic cavity. They are those of pleurisy—the effusion coagulating on exposure to the air, and ecchymoses are found. The lung is increased in size, hard and friable, and in appearance is likened to the lesions of contagious pleuro-pneumonia. The heart muscle has a parboiled appearance, it is paler than normal and friable. Lesions are found also in the abdominal cavity, but are less evident—petechiæ and ecchymoses upon serous and mucous membranes, and engorgement of the solid viscera with blood. The skeletal muscles also show small hæmorrhagic centres scattered through the muscles, blackish or a black violet tint. These centres may be a state of degeneration. The muscles may be infiltrated with serum. The causal organisms may be demonstrated in the lesions.

It is believed that this disease is spread by infection and by contagion. Poels thinks the organism is a facultative parasite, and that it is present in certain soils and upon forage, and that it obtains entrance into the body by the respiratory tract as well as by the alimentary route.

In the United States a form of septicæmia hæmorrhagica has been noticed which perhaps should be mentioned, because young animals die from it as well as older animals; indeed, neither age, nor sex, nor condition is proof against it. The evolution is rapid, death may occur in six hours, generally in twenty-four hours, rarely the animal lingers three or four days. The symptoms are interesting. There is loss of spirits, suppression of milk, loss of appetite, difficulty of movement, lameness and pain when moving. The pain is sometimes acute, the animals cry out and there is contraction of the muscles. If the animal lives more than twenty-four hours there is rapid loss of condition. The temperature, at first high, rapidly falls. Œdematous swellings appear in various parts of the limbs, and throat especially. The fæces, streaked with blood, and black, become watery and hæmorrhagic. Sometimes hæmaturia and blood-stained discharges are noticed. Lung lesions are not observed. [The symptoms are those of Wild and Rinderseuche, the clinical manifestations of which are little known.—Leclainche.] The lesions observed are those characteristic of septicæmia, blood extravasations of a varying degree being found upon the surface of membranes and in the interior of organs. The spleen is generally normal in appearance and consistence, as are the kidneys. In some cases the joint cavities contained a blood-stained synovia.

Jensen, who published the result of an experimental enquiry into the cause of diarrhœa in calves¹ in 1894, came to the conclusion that diarrhœa in calves was the result of a gastro-enteritis complicated with septicæmia, and that it was due to a pathogenic variety of a normal inhabitant of the gut closely related to the bacterium coli commune. His results were confirmed by others—Piana, Mazzanti and Vigezzi, Monti and Veratti, Piana and Galli-Valerio, and Romano, Italian workers. Nocard and Leclainche, however, are dubious as to the pathogenicity of the organism in question. It must be granted that the bacterium coli commune can very frequently be found in the tissues prior to natural death, the invasion occurring

¹ Ueber die Kalberruhr und deren Actiologie *Monatschrift für Thierheilkunde* IV.

when the animal is *in extremis*, and that its presence may be associated with the cause of the disease, though in reality it had nothing to do with it; and, on the other hand, the effects of the invasion of the bacterium *coli commune* are so various that one need not be surprised that a variety of the organism may produce most fatal results. As regards Jensen's form of the disease it would perhaps be as well to keep an open mind, especially as his own results differ from those of workers who have recently investigated diarrhœa of calves.

Jensen's organism is motile, it produces gas in gelatine stab cultures, it coagulates milk, it grows upon potato, and the cultures have a fetid odour, characteristics which at once differentiate it from the organism of white scour.

The symptoms of the natural disease are like those of white scour, though in some cases the discharges from the rectum were greenish or green. Often in these cases of Jensen the materials first passed by the newly-born calf were liquid.

The lesions noted are as follows.¹ If the animal is opened immediately after death, the intestine is anæmic in appearance, but later it becomes dark red. The mucous membrane of the abomasum and small intestine is infiltrated, congested, studded with ecchymoses and desquamated patches. Peyer's patches are enlarged, the colon and rectum contain gas and a greyish viscid material mixed with little yellowish grains. The rectum is thick, red, inflamed, as is also the skin around the anus. Mesenteric glands are swollen and contain hæmorrhagic centres. The spleen and *urinary bladder* are not altered, (italics mine), the liver and kidneys are hyperæmic. The lungs are sound. Lesions of broncho-pneumonia sometimes found are due to foreign bodies. The myocardium is intact, the endocardium is ecchymosed." The condition of the urinary bladder here, should be compared with Thomassen's cases and with those seen in Ireland. The absence of lung lesions is significant. The lung lesions in the Irish calves were certainly not due to foreign bodies in the bronchi.

In this, as in all others, a knowledge of how the organism gets into the body of the calf is desirable. Jensen believes that the organism is always present in the alimentary canal of cows, and that it is expelled along with the fæces, soiling the parts with which they come in contact. The udder is contaminated when the cow lies down, and the calf takes in a dose of the organisms when it sucks the teat. Poels believes, on the other hand, that the infection is produced by the umbilical wound being soiled with the organism, which is a particularly virulent type of the colon bacterium. The umbilicus is infected during parturition or afterwards; virulent organisms have been found in the vaginal mucus. Jensen obtained infection in seven calves, by feeding them on milk mixed with intestinal discharges of animals suffering from the disease. These calves were two days old; older animals were not infected.

A particularly interesting experiment of Jensen's is worth mentioning. He gave newly born calves such substances as creolin, pyoctanine or trichloride of iodine, and saw them die within 48 hours with diarrhœa. The preparation given had injured the mucous membrane of the gut, and allowed intestinal organisms to cross the otherwise

¹ Nocard and Leclainche. "Les Maladies Microbiennes des Animaux," 3rd Edition, 1903.

efficient barrier and invade the body. These organisms recovered and grown in suitable media produced the disease when ingested by another calf.

It appears, then, that anything which will injure the natural defences of the body will allow of entrance of organisms which may produce death, and that these organisms are generally harmless when in the lumen of the bowel. This is probably the cause of death in many young animals—foals, calves, and lambs, which become the hosts of numerous animal parasites which wound the mucous membrane of the stomach and gut generally, and allow the access of micro-organisms which, under ordinary conditions remain outside the body in the alimentary canal. A similar explanation may be given for death as the result of retention of fæces or obstruction of the bowel. It is not so much absorption of the contents of the bowel—auto-intoxication—as it is injury to the bowel wall, which is rendered permeable to members of the intestinal flora. There is also considerable evidence in support of the contention that secondary lesions to a septicæmia from which the animals recover, are due to the entrance into the body of organisms which are normal inhabitants of the intestine, and which under ordinary circumstances thrive there without doing harm to the host. Indeed, it is probable that many members of the intestinal flora are absolutely essential to thorough and efficient digestion in the intestinal tube and to the normal development of the host, as witness Nuttall's experiments upon guinea-pigs fed upon bacteria-free food and kept in aseptic compartments, compared with others kept under ordinary conditions. Still, from the knowledge we have already gained as to the life history of organisms, we are driven to conclude that from some cause or condition or other, parasites or saprophytes which are ordinarily harmless may produce varieties which may give rise to serious lesions. The various coli-bacillary infections are examples of such. Perhaps better technique will demonstrate to us that what we now call varieties are quite distinct species.

Although it is somewhat beyond the scope of the original title of this paper, I think it well that mention should be made of several lesions that may be found in young animals, dying a short time—a few weeks, or may be two months or so—after birth. I mention these lesions because I am convinced that in many cases they arise as sequelæ to an antecedent septicæmic infection. Such secondary infections are the lung disease in calves, necrosis of the liver in calves and lambs, and pyæmia and pyæmic arthritis in calves, lambs, and foals.

LUNG DISEASE.

I have already mentioned that in calves dying from white scour, or in those killed in the later stages of the infection, lesions were found in the lungs, which varied from collapse to a hæmorrhagic pneumonia. The lesions are observed more particularly in the anterior lobes of the lungs. In some cases the lesions observed are those of bronchitis and catarrhal pneumonia, and it is to these last that I wish to direct attention. Calves that have apparently recovered from the "white scour" sooner or later begin to cough, and from the accelerated respirations, and from other clinical evidence, there is little doubt that the lungs are affected. The animals die in a month or six weeks

with consolidation of the anterior lobes of the lungs, the consolidation extending backwards, and formation of abscesses. I cannot call to mind a case where the posterior lobes have alone been affected, though there are cases where the posterior lobes to a greater or less extent are diseased with the anterior lobes. The lung on section is grey and solid, and studded with innumerable minute yellowish centres which are miliary abscesses. A pleurisy frequently is present, and adhesions, which may be so firm as to require severance with the knife, develop. The abscess formation in the lung continues, by the colliquation of the septa of adjacent centres which have become necrosed, and larger abscesses result. By an extension of the process the lobe may be replaced by an accumulation of abscesses, little if any of the lung tissue being left. At the periphery of the lesion a reaction occurs on the part of the still more or less normal lung, and attempts made to encapsule the lesion, and such is the condition observed in those animals which have apparently recovered from the lung affection. In some cases the lung involved becomes converted into a tough greyish mass, resembling in feel and in appearance morocco leather. The lung lesion is believed to result from an invasion of organisms found normally upon the respiratory mucous membrane, which during the septicæmic infection loses its power of preventing access of saprophytes into the tissues. The organisms found in the pus of the diseased centres are cocci, both staphylococci and streptococci, streptothrix, the bacillus of necrosis, the bacillus of caseous lymphadenitis. Nocard blamed particularly the last organism, and after he had injected the toxin formed by the organism of white scour into a calf he caused the calf to breathe an atmosphere charged with the organism of caseous lymphadenitis. In eight days the lesions had developed, and were similar to those observed in cases of the naturally acquired disease. But, though it is very probable that numerous cases of lung disease are due to entrance of the causal germs through the impaired bronchial mucous membrane, there are, I am convinced, other routes whereby the cause may gain access, and I think we cannot altogether exclude the umbilicus or other wound.

In this connection I may mention a number of cases I saw where there were lung lesions in calves that certainly never had white scour, calves that were collected from a very wide area, indeed I may say from the whole of Ireland. The calves in question were born about the end of March and the beginning of April. Some, to which we may particularly refer, were born in the most favourable surroundings, and the navel was treated in the approved fashion. There were never any symptoms of white scour. The calves thrived and were in admirable condition until the end of May. About the 26th May the males were castrated, and in about ten days from this date, on an average, the calves began to cough. Lung disease, clinically speaking, had put in appearance. I was now asked with another member of the profession to see the calves. We chose the worst for observation, and they were sent into the College. They had not long been in the College when the scrotum was found swollen, and in two cases a discharge appeared. The calves were then all carefully examined, and in every case a thickened or suppurating cord was found. In one instance the cord was excised and a piece of twine which had been used as a ligature was found in the centre. Two calves were killed

and found to have lung lesion; the same organism was found in the lung lesion as in the cord, and the organism was not the bacillus of caseous lymphadenitis.

I next made enquiries as to the relative numbers of the two sexes showing the lung lesions, and learnt that they were all males. The good health of the calves up to the time of castration, the appearance of the cough at or about the tenth day following the operation, the condition of the scrotum and cords, the presence of the same organism in both cord and lung, were suggestive as to the method of infection.

Another case which was brought to my notice by Mr R. Sargent, M.R.C.V.S., of Mullingar, is instructive. The patient was the last but one of a herd of twenty-seven calves; most of the others had perished. The calves were pedigree Polled Angus. The calf was sent into College living, and was kept six days for observation. It was in good condition, though coughing, and had all the clinical symptoms of lung disease. The temperature whilst in College never went above 39° C. (102·2° F.). The calf was killed by opening the carotids.

The *post-mortem* record is as follows: Umbilicus healed but thickened, no pus, umbilical vein sound and normal. Liver showed a few white specks under capsule, but otherwise normal. Spleen normal. Lymphatic glands throughout the body enlarged and juicy. Kidneys loosely attached (floating), fat dropsical, kidneys otherwise normal. In thorax both lungs involved nearly throughout, only sound part in posterior lobes. Anterior lobes showed large abscesses, and there was a pleurisy of some age. Adhesion to chest wall. The left anterior lobe united firmly to pericardium and filled with large abscesses. Smaller abscesses along sternal edge of lung. Where no abscesses save in posterior lobes, the lungs were broncho-pneumonic. In the mouth was an opening leading into a blind sac filled with caseous pus; the swelling produced by the sac had been noticed during life. Pus in the lungs contains the bacillus of necrosis and an organism taking Gram like the Preisz-Nocard organism (bacillus of caseous lymphadenitis). There was an indication of suppurative middle ear disease of left side. (Discharge from the ears had been noticed in this animal and others of the same herd.)

This calf had in the lungs typical lesions of the so-called lung disease, and yet white scour is unknown in the district. Clearly, then, in this case some other explanation than the primary infection by the organism of white scour must be offered to explain this invasion.

But there are other cases, and to my mind most instructive. Towards the end of March of the present year I received from Mr J. F. Healy, M.R.C.V.S., Midleton, Cork, livers from young lambs from one to two weeks old. An unusual number of lambs had died, and the livers were sent to me for opinion. I had little difficulty in recognising necrosis of liver, such as M'Fadyean described years ago as occurring in the livers of oxen. The areas were, however, larger than is usually the case, some wedge-shaped, others spherical, and embedded in the liver; one lesion was quite two inches in diameter. They were mostly firm, of a putty colour; others had a soft breaking-centre. The bacillus of necrosis was found in apparently pure culture. I inoculated an emulsion of a necrotic area made with

bouillon into the auricular vein of a rabbit (1). This was on 20th March. On 26th March the rabbit was found dead in the morning. The thoracic organs were sound. There was a peritonitis, with a thick glairy albuminous fluid containing masses of coagulated fibrin. Necrosis of parietal peritoneum. Epiploon thick and infiltrated. Liver covered with exudate and united to diaphragm. Liver was necrotic, one lobe almost entirely so, and there were specks of necrosis through the organ, which was enlarged. The necrosis also had attacked the serous covering of the colon, and the wall of the bowel was necrosed from the serous membranes inwards towards lumen of bowel.

An emulsion was made from the liver of the above rabbit and injected into the auricular vein of another rabbit (6). It was killed twenty-two days later. The scapulo-humeral articulation was affected; a large abscess involving this joint, extending down the humerus to the elbow joint, contained in pure culture the organism of necrosis. The lesions were precisely those of joint evil. The humerus, head and shaft, was necrosed.

Another rabbit (13) received an emulsion in bouillon of the pus of last animal into the thigh. An abscess developed, and a necrotic area had developed in the liver; it contained only a few bacilli. A second rabbit (12) received into the auricular vein pus in bouillon obtained from rabbit with necrosis of the shoulder joint. The animal was killed four days later. The liver contained a lesion as large as a hazel nut with the bacillus of necrosis in pure culture. The urinary bladder was enormously distended with fat-containing urine—chyluria.

The pus from the abscess developing in the thigh of experimental rabbit 13 was inoculated after dilution in bouillon (1 cc. of dilution) into jugular vein of a red and white calf eight days old. The animal lost considerably in condition and the fæces were streaked with blood. The calf died on 14th May with intussusception of the cæcum, the mucous membrane of which was extensively necrosed. A large white rabbit was inoculated at the same time into the auricular vein with .5 cc. of same dilution as this calf. It was found dead on 6th May. The ear of injection became erysipeloid, but this passed off; the lymphatic glands behind the inferior maxilla became enlarged and purulent. On *post-mortem* an intense pericarditis, left-sided pleurisy, and pneumonia had developed. There was firm adhesion to the chest wall and to the diaphragm. There was a pleurisy on the right side, but not so severe. Small necrotic areas were found in the liver and on the visceral peritoneum. In this case the organism had attained an enormous length, and showed in a remarkable fashion the alternate presence and absence of coloration when stained.

I think these few experimental cases show the many-sided rôle the organism of necrosis may play, and that in these experimental animals we have epitomised the sequelæ observed in practice. It is interesting to observe that necrosis of the liver is merely a local manifestation in the liver of lesions occurring in the joints, in the connective tissues, in the serous membranes, and lungs. It is, further, to be observed that the lesion in the liver may extend through the diaphragm into the pleural cavities and involve the pleuræ and lungs; and I have had an opportunity, through the kindness of Mr Healy, of observing a necrotic centre extending through the diaphragm and

involving both liver and lung. The centre had broken down, and contained the bacillus of necrosis.

Similarly, through the courtesy of Mr John Holland, M.R.C.V.S., of Athy, I received the liver of a calf that with others had died from white scour. It was a typical case of disseminated necrosis, such as one sees in adult animals, and which is so well illustrated in M'Fadyean's paper on this disease published in 1891.

Lastly, whilst upon this question of necrosis, I should like to refer to the disease described by Damman, and in which Löffler isolated an organism like to that of necrosis.

Animals of from one to four weeks old are affected. The patient loses its spirits and refuses to suck. The temperature is high. There is abundant salivation and difficulty in deglutition. Opening the mouth is painful; the tongue is swollen. There is a cough, and a muco-purulent discharge from the nose. Greyish patches of a variable size, isolated or confluent, cover the mucous membrane of the gums, tongue, palate, and fauces, formed of a fibrinous exudate, easy to remove, and covering an inflamed mucous membrane. The false membranes increase rapidly in thickness, form a thick, rough grey layer adherent to the tumefied and infiltrated subjacent structures. The jaws, throat, and intermaxillary space, etc., are the site of a diffuse inflammatory swelling. Sometimes the nasal chambers are affected, and the lesions may extend to pharynx, larynx, and trachea. In some cases the feet are affected, the hoofs being lost. Death occurs in from four or five days to three weeks. Damman described the disease as diphtheria of calves; and Löffler, who investigated it when working at diphtheria in man, had no difficulty in showing that there was no connection whatsoever between the two infections.

The lesions of this "diphtheria" of calves are interesting. On the mucous membranes the exudations mentioned above are met with, the mucous membrane itself is progressively necrosed, and the necrosis may extend to the subjacent bones and cartilages. The mucous membrane of the intestine is infiltrated and vascular, and diphtheritic patches of different sizes are to be met with in the cæcum, floating colon, and rectum especially. The lung also frequently shows lesions; probably these are of a nature of a foreign-body pneumonia. Ecchymoses on the serous membranes may also be observed. The disease has been transmitted experimentally to the lamb and calf.

I have mentioned at some length the symptoms and lesions of "diphtheria" in calves because I am convinced that a similar, if not the same affection may be observed in the United Kingdom. During the investigations into the cause of white scour, etc., in Ireland, I had occasion to visit many farms in the province of Munster. On three farms, one particularly, I found a number of calves suffering from what I described as necrosis of the jaws. The calves were all young and were still fed on whole milk. On examination of the worst cases I found that abundance of saliva was escaping from the mouth and that the animal was smacking the lips in a remarkable fashion. Peculiar elongated swellings were noticed along the line of the grinding teeth, even before manipulation. On opening the mouth the mucous membrane covering the gums carrying the incisor teeth was found covered with a greyish granular deposit which on removal exposed an eroded bleeding mucous membrane; some of the incisor

teeth had fallen, others were so loose as to be easily removed. The necrosis extended from the labial aspect of the gum, between the teeth, to the lingual surface. The elongated swelling noticed along the line of the molar teeth was found to be due to an accumulation of deposit, grey, granular, and easily broken down, like to that upon the gums of the incisor teeth. The mucous membrane was necrosed, and necrosis extended from the buccal aspect of the gum between the grinders on to the hard palate. Several calves showed the same condition, and I secured two heads for further examination, which showed me that the organism of necrosis was there in abundance. A thorough search through the viscera failed to discover any other lesion save one minute area in a lung, the size of a pin's head, which unfortunately was lost. All the calves, however, had large navels, and a careful examination of all the calves showed that those with necrosis had suppurating navels; the worst mouth case had a navel with a cavity in it capable of accommodating a large hen's egg. On enquiry the farmer informed me that the disease was well known, and that the calves recovered even when the mouth lesions were advanced, and he showed great reluctance to allow me to kill any of his calves, and I had to purchase.

On another farm two animals died exhibiting the same lesions as noted above. At a third farm I found what I take to be the earliest phase in the necrotic process. It was whilst making a *post-mortem* of four calves said to have died from white scour. The lesion was a minute excoriation, in this particular case upon the bare skin of the muzzle. Surrounding the pit was an areola of intense congestion. It is probable that in acute cases of septicæmia there is an exanthematous eruption—which is generally lost sight of in our patients owing to their hairy covering—an eruption similar to that observed in septicæmia in swine. Infection may then occur by the organism of necrosis through the abraded surface. I recently purchased in Dublin, among other calves for this and other work upon which I am engaged, a roan calf. From my notes I read as follows: "The calf when purchased had a diseased navel which was suppurating, hard, cord like, passing up into the body. During the time he was in the observation house he showed red spots on the bare skin of the muzzle. One healed leaving a pitted depression. Others succeeded upon the muzzle, upon the internal wing and inferior commissure of the anterior nares. The size that of a three-penny-piece and smaller." This particular calf died on 17th May 1903, and was found to have a purulent umbilical vein, as thick as the wrist, extending from the abdominal wall into the liver. The pus in the vein contained a large number of different varieties of organisms, including a *cocco-bacillus* not taking Gram.

Although my cases are not on all fours with those described by Damman and others, still I think I may claim they have a family resemblance, and are worthy of mention as an infection of newly-born calves.

Of pyæmia I do not think I need say much. It is already well known that joint-evil in foals and calves is largely the result of umbilical infection—a purulent omphalo-phlebitis. Lesions are frequently disseminated through the body, involving, it may be, an organ or many organs simultaneously. The clinical manifestations

most common and most striking is the pyæmic arthritis, and from this fact the common name of the infection. Pyæmia, even without joint lesions, is not unknown as a sequel to white scour, pyæmic lesions being recognised in the kidneys, heart muscle, liver, etc. The causal agent in these cases is most often a staphylococcus, but I have also seen a small streptococcus producing the disease in calves.

PROPHYLAXIS.

From what has already been written it may be inferred that in my opinion prevention is to be attained by careful attention to the umbilicus. But no matter how careful one may be in the umbilical treatment, this must be supported by cleanliness in and about the houses inhabited by the young and their dams. Treatment of the umbilicus may save animals; there is no doubt that thorough hygienic surroundings will cut down the mortality; both should theoretically remove altogether the cause of death. This has been our experience in Ireland. Sealing up the navel even in bad surroundings has undoubtedly saved a number of lives, but when to this is added as scrupulous cleanliness as practicable then the loss has become almost negligible. Count Moore at a conference held in Limerick gave his own experience in Tipperary, that whilst he had lost 75 calves out of 115 in 1900, last year he had only 2 deaths out of about the same number of calves born. Another gentleman, the vice-chairman of the Kerry County Council, though he had lost 120 during the past six years, last year did not lose one. Others spoke to the same effect. I know also of a celebrated breeding stud of thoroughbred horses that yearly lost a number of foals from diarrhœa, joint-evil, etc., last year after the navel treatment only lost one foal out of 59 born, and the single death was due to another cause than umbilical infection. I have therefore nothing more to say than that antiseptic treatment of the navel as speedily as possible after birth, preservation of the wound in an aseptic condition, and thorough cleanliness, will save many young animals at present lost. I am also convinced that this treatment will in great part, if not entirely, ward off the serious secondary infections which occur, and which I have endeavoured, I am afraid very imperfectly, to indicate at any rate in part.

That there may be other routes by which infection is brought about I am prepared at present to admit, but I hope that the time is not far distant when we shall be able to block every port of entrance, every vulnerable point, and thus save the lives of many young animals which previously have been doomed to perish.

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The following publications among others, home and foreign, were consulted :—

Les Maladies Microbiennes des Ammaux (to which I owe much),

Nocard and Leclainche, 3rd Edition, 1903. *Contribution à l'étude de la diarrhœa des Jeunes Bovidés et de l'entéqué (Pasteurellose Bovine)* by J. Lignières, Buenos Aires, 1898. *Revue Générale de Médecine Vétérinaire*, Toulouse. *Fortschritte der Veterinär-Hygiene*. *Journal of Comparative Pathology and Therapeutics*. *The Veterinarian*. *The Veterinary Journal*. *The Veterinary Record*. *Annales de l'Institut Pasteur*. *Contribution à l'étude à la classification des Septicémies Hémorragiques*, Par J. Lignières, Buenos Aires, 1900. *Bakterienkunde und Pathologische Mikroskopie*, von Dr Kitt, 4th edit., 1903. *Contribution à l'étude de la Maladie des moutons*, Lignières, 1898. *Mortality among calves in Munster*, Dublin, 1901.

SIX CASES OF CARCINOMA OF THE OX.

By A. M. TROTTER, M.R.C.V.S., Glasgow.

CARCINOMATA are regarded by many pathologists as occurring but rarely in the bovine species. The experience of the writer, however, has led him to believe that such is not the case, and that neoplasms having a carcinomatous structure are frequently met with in the usual routine of a large abattoir. The term "frequently" is used advisedly, but the writer does not wish to be misunderstood; all he desires to convey is that cancerous growths are more prevalent in the bovine species than is usually admitted.

It is interesting to note that all the cases here recorded occurred in Irish cows. The majority of cows exported from Ireland to this country have attained an extreme age, and are frequently in an emaciated condition.

The writer does not intend to review the many theories that have been promulgated from time to time as to the etiology of this disease, and he is content merely to record the results of a macroscopical and microscopical examination of six of his cases. These reports are given somewhat in detail, as he considers them of value in showing the manner in which the disease was disseminated throughout the system of the affected animal.

Case I.—Squamous-Cell Carcinoma of the Orbit of a Cow.

The subject was an aged Irish cow in good condition.

This animal, although evidently suffering acute pain, did not show any disturbance of pulse, respiration, or temperature.

The eyelids in the vicinity of the nasal canthus of the left eye were destroyed and replaced by a fungoid growth, which was irregularly circular, and measured about 10 cm. in diameter. The interstices were filled with coagulated blood and a glairy muco-purulent material. It emitted an offensive and penetrating odour.

On dissection of the parts it appeared that the neoplasm had had its origin in the vicinity of the lachrymal sac. An arm had penetrated the lachrymal duct and entered the middle meatus of the nose. The greater bulk of the tumour, however, had invaded the orbital cavity, and in its progress had pressed the eyeball upwards and outwards. As a result, the normal contour of the eyeball had been destroyed. The cornea was thick and opaque. The eye, so far as