TABLE SHOWING THE ANNUAL LOSSES FROM CONTAGIOUS PLEURO-PNEUMONIA BETWEEN 1871 AND 1887 INCLUSIVE IN THE WHOLE OF HOLLAND *minus* THE SPOELING DISTRICT.¹

<i>Year.</i>	<i>Animals slaughtered attacked with Pleuro-pneu- monia.</i>	<i>Animals Slaughtered Suspected.</i>	<i>Value in Francs.</i>	<i>Amount Ob- tained by Sale of Car- cases.</i>	<i>Indemnities Paid by the State.</i>
1871	6,078	—	—	—	—
1872	4,008	—	—	—	—
1873	3,500	—	—	—	—
1874	2,414	52	—	—	—
1875	2,227	191	—	—	—
1876	1,723	822	—	—	—
1877	970	3,318	1,846,472	486,384	1,360,088
1878	701	2,200	1,016,232	352,136	660,096
1879	157	532	228,228	66,670	161,228
1880	44	451	114,638	39,240	75,398
1881	12	189	67,496	23,686	43,810
1882	11	664	274,490	149,732	124,490
1883	18	986	487,438	282,544	204,894
1884	234	3,050 ²	1,429,198	735,418	694,780
1885	83	324	134,900	65,938	68,962
1886	2 ³	1	240	70	170
1887	1 ⁴	3	1,030	224	806
Total for the 17 years.	22,183	12,783	5,600,362	2,202,042	3,394,722

AMYLOID DISEASE IN THE LOWER ANIMALS.

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THE occurrence of amyloid disease among the domesticated animals has been noted by a considerable number of continental veterinary

¹ [The figures for the last four years of the table refer to the whole of Holland, *including* the Spoeling district.—Ed.]

² Of these 3050 animals slaughtered as suspected 2932 belong to the Spoeling district.

³ & ⁴ The two cases observed in 1886 and the single case of 1887 were in Limbourg, four kilomètres from the Belgian frontier.

surgeons, the species attacked being the horse, the ox, the sheep, the dog, and the fowl. I am induced to devote some space to it here, in the first place, because of the scanty references to it which are found in English veterinary literature, and, in the second place, because my own observations are at variance with the descriptions of the condition which have been given by some of those who have previously recorded cases.

Amyloid or waxy disease is a condition in which a peculiar substance, not existing anywhere in the healthy body, makes its appearance in one or more organs. This substance received the name amyloid from the mistaken view that in its chemical composition it was nearly related to starch (animal cellulose of Virchow), while the term waxy refers to the physical resemblance of the material to wax. Chemical analysis by Kekule, Schmidt, and others, have shown that the amyloid material is a proteid body allied to albumin, from which, however, it is clearly distinguished by certain colour reactions, by its resistance to the action of pepsin, and by its slight tendency to putrefactive decomposition.

In the human subject, in which the disease is undoubtedly much more common than in any of the lower species, several organs are, as a rule, attacked, the liver, spleen, kidneys, lymphatic glands, and intestinal mucous membrane being the favourite seats of the deposit. When amyloid degeneration is not complicated with other changes, the affected organs are increased in volume and in density, the amount of blood in them is diminished, and their cut surface presents a peculiar smooth waxy aspect. The degeneration appears never to arise as an idiopathic or primary disease. The morbid processes in whose course it most frequently makes its appearance in the human subject are pulmonary phthisis, chronic destructive bone disease, syphilis, and traumatic suppuration.

It has already been mentioned that the amyloid substance is sharply distinguished by certain colour reactions. Thus, when a waxy organ is treated with watery solution of iodine, the amyloid substance assumes a dark or mahogany brown colour, while the non-waxy tissues are stained yellowish. After-treatment with a dilute watery solution of sulphuric acid sometimes changes the mahogany brown into a light or greenish-blue tint. A much more delicate and reliable test for the amyloid substance is afforded by staining sections of the suspected organ with a two per cent. watery solution of methyl violet. With this stain the waxy substance takes on a beautiful rose-pink colour, while the non-waxy tissues are stained of a shade which has been variously described as slaty-blue or bluish-purple. The introduction of this micro-chemical test has almost put an end to the earlier divergence of opinion regarding the precise site of the amyloid substance in the affected organs or tissues of the human subject. In the early stage, at any rate, the new material is deposited in connection with the walls of the capillaries and small arteries, and it appears doubtful whether gland cells ever undergo the waxy change.

By way of comparison with amyloid disease as it occurs in man, I shall now describe the results of my examination of two cases of the same condition in the lower animals.

CASE I. *Amyloid liver of a horse.* A large portion of this liver came into my possession during the past year, having been sent by

Mr Campbell, F.R.C.V.S., Kirkcudbright, along with parts of other organs from the same animal, for exhibition at one of the meetings of the Royal Scottish Veterinary Association. It is a point of considerable interest that the primary disease in this case was tuberculosis, affecting chiefly the abdominal lymphatic glands. The mare had been under treatment for upwards of four months, and during the greater part of that time there had been a purulent discharge from a tumour of the neck, which had been irrigated with a strong solution of iodine. Some doubt may therefore exist as to whether the tuberculosis or the suppuration ought in this case to be regarded as standing in etiological connection with the amyloid degeneration. The lungs, kidneys, and lymphatic glands did not participate in the waxy change.

Although I had the opportunity to examine only a portion of the liver, it was evident that the organ had been considerably enlarged. It was thicker than normal, and its consistence was notably increased. I desire to emphasise this last point, inasmuch as it is directly at variance with a number of previously recorded cases. Thus Dieckerhoff says, "I have several times had the opportunity to examine the liver of horses that had died from rupture of that organ, and in these I could always obtain the amyloid reaction, in correspondence with the experience of Rabe. . . . Amyloid degeneration of the liver as an element in amyloid disease is not rare in horses that have suffered from chronic emaciation. It represents a secondary disturbance of nutrition, and, according to Rabe's observations, it occurs frequently as a sequel to chronic pleuritis and peritonitis. In three old and emaciated horses I found the liver of lardaceous consistence, and its surface gave the amyloid reaction. Macroscopically this degeneration is different from that present in liver rupture."¹

Johne says, "It appears a remarkable circumstance, that whereas in man in the advanced stage of amyloid degeneration the consistence of the affected organ is always increased, directly the opposite is observed in at least the case of the horse's liver. In such circumstances the latter is at first friable (*bröcklich-brüchig*), and subsequently softer—a change of consistence which in several cases has occasioned rupture of the liver."²

If it is the general rule that amyloid infiltration leads to softening of the horse's liver, the fact is very remarkable, and it calls for further investigation. It seems *à priori* extremely unlikely that a deposition of amyloid material, which invariably adds to the firmness and elasticity in the human liver, would have the effect of rendering the horse's liver either friable or soft. It is possible, however, that in the cases referred to by Johnes the amyloid infiltration was complicated with fatty degeneration or some other change to which the softening was really due. However that may be, the fact remains that in this case the consistence of the organ was increased exactly as it is in the human liver. Contrary to what is the case in the human subject, the organ contained a considerable amount of blood, but that appeared to have been dependent upon a dilatation of the right ventricle of the heart, which had been noted at the autopsy.

The histological characters of the lesion in this liver are depicted

¹ Lehrbuch der Speciellen Pathologie und Therapie.

² Birch-Hirschfeld's Lehrbuch der Pathologischen Anatomie.

PLATE III.

FIG. 1.—($\times$ about 40.) Section of liver of a turkey stained with methyl violet: waxy substance pink, non-waxy elements slaty-blue. 1. An area of liver tissue composed almost entirely of waxy capillaries; 2. An area where the waxy substance is still yielding the caseous centre; 3. Dark stained caseous material, tubercular nodule; 4. Spots in the remains of waxy vessels; 5. Calcareous granules; 6. Cellular non-caseated zone of nodule; 7. Waxy streaks (partially destroyed capillaries) in this zone; 8. Waxy arterioles in outer fibrous zone of nodule.

FIG. 2.—($\times$ about 60.) Section of horse's liver stained with methyl violet: waxy capillaries pink, non-waxy elements slaty-blue. 1. Central vein of a transversely cut lobule; 2. Area showing waxy substance of non-waxy (slaty-blue) liver cells; 3. Section of a branch of the hepatic artery; 4. Section of a branch of the hepatic bile duct and portal vein are also free from the waxy substance.

AMYLOID LIVER IN THE LOWER ANIMALS.

in Plate III., Fig. 2. The section from which the drawing was made was stained with methyl violet, washed in dilute solution of acetic acid in water, and then mounted in Farrant's medium. To those who are familiar with amyloid disease of the human liver it will be apparent at a glance that the distribution of the rose-pink waxy material is somewhat uncommon, in that it affects almost uniformly the entire network of intra-lobular capillaries. In man the intermediate zone of the lobule appears to be almost always affected earlier and to a greater degree than the peripheral or central zones, but an exception to that rule, and one which closely resembles the present case, is depicted in Professor Hamilton's recently published Text-book of Pathology.

In lobules which are cut at right angles to the central vein there is seen a tolerably regular radial pattern, determined by alternating rose-pink and slaty-blue lines or narrow bands, which run from the periphery of the lobule towards the central vein. The slaty-blue lines are the columns of liver cells, and the rose-pink lines are the waxy intra-lobular capillaries. The latter are much broader than the normal capillaries, and the columns of liver cells are diminished in breadth in the same proportion. The thickening of the capillary walls is not quite uniform, and where it is most marked the column of liver cells lying between two thickened capillaries is reduced to a very narrow line. The liver cells are thus evidently in a condition of atrophy, determined by the pressure exerted on them by the thickened capillaries.

The finer details of the alteration of structure in this liver may be followed by examining under high powers of the microscope sections stained with methyl violet and others stained with picro-lithia-carmin. By the former method I have been able to satisfy myself that the liver cells are entirely free from the amyloid substance. In this liver the hepatic cells are also quite free from any fatty change, and with picro-lithia-carmin their nuclei stain most brilliantly. Strange to say, this is the case even in the narrowest columns, where the cell body has almost entirely disappeared, as a result of the compression exercised by the thickened capillary on each side.

In cases recorded by Johne and Bruckmüller it is expressly stated that the hepatic cells participated in the amyloid change. Thus Johne says (*loc. cit.*), in speaking of a case of amyloid liver of the horse observed by him, "the liver cells participated in a very prominent way in the amyloid degeneration. Not only were a great many of these enlarged, and swollen into homogeneous, shining, denucleated lumps, but, besides, the usual chemical reagents completely corroborated the diagnosis."

When closer attention is turned to the altered capillaries in a methyl-violet stained section, and particularly where a lobule is cut tangentially, it can be made out that the wall of the vessel is greatly thickened and in such a way as to narrow the lumen. Here and there, where a capillary is cut transversely, there lies within the rose-pink circle a nucleus which does not give the waxy reaction—that is to say, which is stained slaty-blue. The nuclei of the capillary wall are, like the liver cells, free from the amyloid substance. The healthy character of these nuclei is further indicated by the fact that they stain deeply with picro-lithia-carmin. The amyloid material takes with this stain

the faintest yellow tint, and it is further rendered distinct by its peculiar translucency. The transversely cut capillaries thus appear as almost colourless translucent discs or thick rings, with a bright carmine nucleus at or near their centre. When in a radial section the waxy capillary runs between two rows of liver cells, carmine-stained nuclei are observable in it quite as numerous as in the normal vessel. That these nuclei belong to the capillary endothelium and not to connective-tissue cells is evident from the fact that they lie towards the axis of the homogeneous thickened capillary. In the sections stained with methyl-violet, the entire capillary, with the exception of its nuclei, gives the rose-pink waxy reaction. As a last point connected with the distribution of the amyloid material within the lobule, it may be mentioned that it is in some places present in the wall of the central vein.

In the human subject the small branches of the hepatic artery are almost invariably affected, the middle coat being infiltrated with the amyloid substance. In this case, however, out of numerous sections examined, I have not found a single branch of the hepatic artery which yielded the amyloid reaction. On the other hand, the rose-pink stain is yielded by some of the small branches of the portal vein, the elements affected being mainly or exclusively muscle cells of the middle coat.

This horse's liver was in the early stage of a miliary tuberculosis, every section showing numerous very small areas of round cells in the connective tissue of the portal canals; and in the case of the larger foci these extend for a little distance into the outer zone of the adjacent lobules. Staining by the usual methods reveals tubercle bacilli in some of these groups of round cells, but never more than two or three can be detected in one focus. Under a low power the larger of these microscopic tubercles, where they have invaded part of a lobule, occasionally show traces of the rose-pink stain. On examination with a higher magnification the larger rose-pink elements are plainly seen to be remains of intra-lobular waxy capillaries, which have offered a greater resistance to destruction than the hepatic cells, and the smaller irregular rose-pink fragments are probably of the same nature.

CASE II. *Amyloid liver of a turkey.* This liver was sent to me during the summer vacation of 1888 by Mr Christie, M.R.C.V.S., of Limerick, as an example of avian tuberculosis. The organ was enlarged to nearly twice the normal, and it presented in a typical manner the physical characters of advanced amyloid degeneration as it occurs in the liver of the human subject. That is to say, it was paler, but denser, tougher, and more elastic than the normal liver; and its cut surface had a peculiar, streaky, smooth, waxy appearance. Throughout the liver substance there were numerous yellow nodules, varying in size from the volume of a pea downwards. These nodules were in large measure converted into the firm caseous material commonly encountered in tubercular nodules in the fowl, and in part they were gritty from calcification.

To illustrate the histological changes that had been effected on this liver Fig. 1, Plate III. has been drawn from a section which included a tubercular nodule of the size of a small pea. At some places in sections stained with methyl-violet very little of the hepatic substance

shows the slaty-blue colour, and under a low power of the microscope it appears as if the normal elements had been completely replaced by the rose-pink amyloid material. In these places the liver structure is totally obliterated, and it is quite impossible to say whether the amyloid masses are composed exclusively of altered capillaries, or partly of waxy liver cells. But in such parts as still show recognisable liver cells, these, as in the horse's liver, are free from the amyloid substance, but are generally deformed, fatty, or atrophied. The middle coat of the smaller arteries is waxy throughout, but the wall of the large branches appears healthy. Occasional streaks of rose-pink are discernible in the wall of the small and middle-sized branches of the portal vein.

The peripheral non-caseated zone of each tubercle nodule is composed of cells of various kinds, with numerous capillaries and occasional small arteries with a distinct middle coat. These vessels are invariably waxy, but in the arterioles the endothelium is obviously free from the amyloid substance. For the most part the cells adjoining the caseous material are spindle-shaped fibroblasts with a few multinucleated giant-cells, and farther outwards the former have advanced to the condition of young fibrous bundles. At some places there are clumps of small round or lymphoid elements, and in the case of some of these it almost seems as if they were infiltrated with the amyloid substance. By careful examination, however, with a high power I have convinced myself that such is not the case. Where a group of rose-pink cells is discernible with a low magnification, a higher power shows that it is in reality a fragment of a waxy capillary with small round cells which have clustered round it, or which in some cases appear to have actually penetrated it.

Lastly it is to be observed that diffuse rose-pink streaks and patches lie in the part which is completely caseous, showing the greater resistance of the amyloid substance, as compared with the non-amyloid elements, to the process of caseation.

There can be little doubt that in this instance the amyloid disease arose out of the tuberculosis. The turkey, which was four years old, had never exhibited any symptom of disease, but during life the liver could be felt through the abdominal wall, and by the poultry-keeper it was thought to be an egg which for some reason or another the bird was unable to lay. The turkey was killed because it had not laid any eggs during the previous year. The only other organ that showed tubercular lesions was the heart.

ON PLEURO-PNEUMONIA AND ITS PREVENTION.¹

By JAMES M'CALL, F.R.C.V.S., Principal of the Glasgow
Veterinary College.

THE subject on which I have been desired by the directors of this Society to address you this evening is the disease—pleuro-pneumonia in cattle, and as a discussion has been suggested to follow thereafter, I

¹ An address delivered to the Renfrewshire Agricultural Society, 14th November 1889.