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Further Observations on the Pathology of Bluetongue in Sheep.

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Previous Experience.

The original descriptions of the symptoms and lesions of bluetongue in sheep by Spreull (1905), Theiler (1905), and Dixon (1915) summarized the extent of our knowledge of the pathology of this disease until quite recently. The observations of Curasson (1925) in West Africa, and of Bekker, de Kock and Quinlan (1934) confirmed the earlier findings. The latter workers, while more concerned with the manifestations of bluetongue in the bovine also undertook the histological study of the stratified epithelium (mucous membranes and skin) in affected sheep, and directed attention to the frequency of vascular effusions as well as to the deterioration which takes place in the superficial epithelium of the mouth and skin leading to excoriations and similar lesions.

The symptomatology and pathogenesis of bluetongue in sheep as described by these authors may be summed up in three phases briefly as follows.—

1. With the onset of fever there is reddening (hyperaemia) of the buccal and nasal mucosa with salivation and frothing. There is slight lachrymation and watery discharge from the nostrils, the latter becoming muco-catarrhal with time, and drying to form crusts. There is swelling of the lips, tongue, face and intermandibular spaces. Reddening deepens and petechiae appear on mucous membrane of mouth and nostrils. The epithelium thickens and is shed leaving excoriations and bleeding points when handled roughly. Anorexia is present and sometimes acute enteritis with diarrhoea. Petechiae and excoriation of the mucous membrane of the forestomachs and hyperaemia of the abomasum are commonly seen.
2. With the subsidence of fever, flushing of the skin and feet becomes evident. The coronets are warm and tender, and show at first a pink periople which later becomes deep red and streaky. Sheep show lameness and stiff gait, and lose condition. Anaemia may be present.
3. Severe emaciation, prostration, muscular weakness, and sometimes wry-neck lasting for periods up to three weeks or more. There may be pulmonary oedema and occasionally pneumonia as a result of aspiration of food. A break in the wool becomes evident and the fleece may fall off.

Material Studied.

Recent observations during the course of extensive experimental immunization of Merino sheep against this disease, and also during natural outbreaks in the field, have enabled us to confirm in the main the above changes and have brought to light a few more interesting facts which are described below.

In many of the experimental animals and in several other sheep, which had contracted the disease naturally, it was found that multiple haemorrhages occurred, not only in organs in which these lesions had been recorded previously, but also in the skeletal musculature. Closer examination of this hitherto overlooked tissue revealed fairly characteristic and extensive histological changes. The lesions in the feet or claws were also studied more fully.

This study is based on material collected from sixty sheep. Of these 42 were suffering from or had died of bluetongue, 8 were more or less healthy control animals, 5 had died of heartwater and 5 had died of various other condtions. During the course of the investigations on the nature and the duration of the immunity in bluetongue, Neitz (1944) established the existence of several antigenically different virus strains, which possess a wide variation in virulence. The recognition of this fact naturally prompted us to gain information on the incidence and the nature of the intramuscular haemorrhages in sheep suffering from the different strains of bluetongue. With this object in view specimens were collected from sheep which had reacted either to the "Theiler", "Bekker", "C. 43", "Nelspoort" or the "Cyprus" strains. It was impracticable to examine histologically very many different muscles, but from the majority of cases the following muscles were taken: trapezius, diaphragm, longissimus dorsi, psoas, biceps femoris, serratus thoracis. Lung was also included and where other organs or muscles showed changes at post-mortem these were examined. Material from additional cases of bluetongue was also studied especially to elucidate points of interest, e.g., torticollis. All material was fixed in formalin, cut frozen or embedded in wax and stained by the usual haemalum-eosin method.

To the casual observer the symptoms and lesions of bluetongue at first may appear ill-defined, vague or inconsistent until experience has been gained in recognising the various types and stages of the disease. It has been shown (Neitz and Riemerschmid, 1944) how various factors can and do modify the course of the disease. It is thus necessary to emphasize that lesions in bluetongue vary greatly in degree according to:—

(*a*) the strain of virus infecting the animal,

(*b*) the individual and race susceptibility of sheep,

(*c*) environmental conditions such as atmospheric temperature, exposure to direct sun's rays, whether wool has been shorn or not, exercise, condition, etc.

Another feature of this disease apparently is that the severity of the lesions bears little if any relation to the height of the febrile reaction.

Post-mortem Appearances.

A. *Skin and mucous membranes.*

The more obvious changes which characterise bluetongue in sheep and from which the disease derives its name, are seen in and around the mouth of the animal, they are hyperaemia, oedema, cyanosis, multiple haemorr-

hages, swelling and shedding of surface epithelium, leading to erosions and even ulcers. Bekker (1934) and his co-workers have described these lesions in detail. One might add, however, that there is an intense general flushing of the skin (it appears a bright pink colour) which may lead to shallow, irregular exanthematous, later crusty patches and excoriations on legs and other parts exposed to abrasion.

B. *Skeletal muscles.*

On removing the skin the subcutis may show a few suffused red areas or spots. It is difficult in all cases to exclude " bruising " from rough handling of the sheep, as a cause of these blood " splashes ". But certainly in many of the sick animals the handling was not severe enough to cause such bruising, and if it did, one must assume that there was a predisposition to easy extravasation through, say, vascular fragility. These dark red and gelatinous areas may extend from the subcutis into the muscles and may be seen along the back, on shoulders, and on the neck. More frequently these haemorrhages are small (half a centimetre or less) and are scattered at random, sometimes isolated and rare, sometimes numerous in one muscle or muscle group. The intermuscular connective tissue and fasciae are usually infiltrated with clear or reddish serous fluid which gives the area a gelatinous, moist appearance. The smaller haemorrhages in the form of spots or streaks, one mm. or less in size, may be missed unless specially looked for. They are best seen in a thin muscle like the trapezius of the shoulder or latissimus dorsi when it is held up to the light. Many more of these capillary haemorrhages are discovered in microscopic sections.

At times a diffuse opacity or cloudiness of the muscle may be seen. This is usually quite localised. In bad cases it resembles a greyish mottling or streakiness, which in one case at least was mistaken for a massive infestation with sarcosporidia. Usually, however, these areas which represent more or less severe degenerative processes are obscured by haemorrhagic effusions. Gross haemorrhages, however, are not always present. Some sheep in the later post febrile stages of the disease, may continue to fall off in condition, their musculature wasting away rapidly, even though they may have started to feed again. Recovery in these cases is extremely slow and protracted.

Torticollis, or wry-neck of earlier writers, is a symptom which definitely can be associated with bluetongue, but which is seen only in a relatively low percentage of cases. It may occur as early as the 6th day of reaction but more usually about the 12th. It sets in suddenly. The head and neck stays bent or even twisted to one side. Spreull (1905) states that " Forcible reduction of the twist shows that there is neither pain nor stiffness of the part but sheer muscular atony alone ". In our experience manual straightening of the neck can be effected without much difficulty or resistance. The head and neck, however, are not limp, but constantly swing back into the twisted position.

There is nothing constant in the extent, distribution and location of these muscular lesions. Sometimes there is nothing to be seen with the naked eye. In general, however, if lesions are present they are found more frequently or easily in the longissimus dorsi and trapezius muscles.

It is remarklable, however, that the majority of muscular lesions whether macroscopic or microscopic, single or multiple, are usually focal in nature, i.e. affect a localised portion of a muscle or a small group of muscle fibres. Of course, when lesions are very numerous and close together, confluence tends to take place.

Histological Findings in Muscle.

For the sake of convenience the histological changes in the musculature will be considered in steps. Generally speaking the natural progression is in the order given but this does not mean that all or some of the changes can not occur side by side. Actually they often do.

1. *Differential Staining.*—The earliest evidence of degeneration of single or groups of muscle is a deeper reddish pink colour contrasting sharply with the pale mauvish pink of normal fibres. The striation and shape of the fibre may be preserved, although distortion and swelling may have set in.

2. *Swelling and Distortion.*—Normal muscle cut lengthwise shows parallel, straight or wavy regular fibres with cross striation. Coagulation of the sarcoplasm apart from different colouration mentioned above, shows up as irregular swelling or bulging of fibres. These stand out rigid, retracted, or distorted among the regularly wavy normal fibres. The sarcoplasm may be gathered together to form a bulge leaving a length of empty tubular membrane (sarcolemma) nearby. The cross striation may be lost early (hyalinisation) or persist until the sarcoplasm distintegrates and becomes phagocytosed. Early necrobiotic changes (pyknosis) of sarcolemma is noted but rarely proceeds to complete necrosis.

3. *Breaking up and Resorption.*—The whole coagulated fibre (sarcolemma and sarcoplasm) may break up or become torn and shredded. (Pieces of fibres were actually seen once in the lumen of larger veins.) It is more usual, however, for the sarcolemma to remain intact, although it may be empty, stretched or wrinkled, or filled and bulging with swollen contents. The coagulated sarcoplasm frequently shows cavities and rents much as perished rubber does when stretched. There may also be rounded vacuoles. In cross section the muscle fibres show a great variation in size—some large, others small and many with spaces in the substance of the fibre. Later on the distorted sarcoplasm starts to disintegrate or it may become stippled with basophil (lime?) granules or streaks. At this stage proliferation of sarcolemma nuclei is well advanced and numerous histiocytes or macrophages invade the affected fibres and liquidate most of the sarcoplasm. Occasionally portions of such sarcoplasm calcify and remain longer. It was noted also that *Sarcosporidia* when present in affected fibres seemed to die and in so doing set up a small local inflammatory reaction.

4. *Haemorrhages.*—Extravasation of blood occurs by actual rupture of capillaries along torn muscular fibres, but probably also by diapedesis. It may vary in size from microscopic specks to pea-sized haematomata and larger (by confluence). A variable amount of fibrin is usually present as also neutrophil polymorphs. Haemorrhages occur frequently in or near foci of muscle degeneration, but they also occur elsewhere in any organ or tissue. No change in the vessel wall could be seen to explain the frequency of extravasation.

5. *Regeneration.*—It was mentioned above that the membranous sheath (sarcolemma)—unlike its sarcoplasmic contents rarely suffered serious damage. On the contrary its nuclei very soon start to multiply, accompanied by proliferation or infiltration of fibrous elements, histiocytes, lymphocytes, etc., which, as has been mentioned, clean up and remove the disintegrated sarcoplasm. Gradually the protoplasm of these multiplying sarcolemma cells increases, and elongates into band-like flashes. These fuse together and form a somewhat confused mass of cells, from which parallel muscle fibres start to reappear, and grow into properly striated and functional units.

Although this process has been called " fibrous replacement " of muscle by some, there is no doubt that true regeneration can and does take place to an astonishing degree. The amount of fibrous tissue deposited with such regeneration must, of course, vary a lot depending on the extent of actual damage to muscle fibres, to blood vessels and to the interstitial tissue and the inflammation which follows thereon. Muscular regeneration was seen to have started within a few days after the height of the fever reaction. It goes on, however, for a long time afterwards as the process is apparently slow and gradual.

6. *Necrosis.*—An outright necrosis of muscular tissue was seen in one case. It involved only scattered and very restricted microscopic centres. The dead tissue was partly resorbed and partly calcified, and evidently would have healed—by what one could more appropriately have called " fibrous replacement ", than by regeneration of muscle.

7. *Wasting of Musculature.*—The mechanism whereby the great muscular weakness, stiffness, and rapid loss of flesh seen in bluetongue, are brought about seems amply explained under 3 above, in which the breaking up and resorption of muscular fibres are described.

It is probable that ordinary atrophy accounts for a part at least of this loss of flesh, but undoubtedly the most important factor is the aggregate effect of the destruction and removal of the contents of muscle fibres and their subsequent slow regeneration. Visual evidence of this may be seen in exaggerated localised form as a depression or constriction in the fleshy belly of a muscle, e.g., the spinalis.

It is necessary to point out that wasting of the musculature goes on apparently for a long time after the passing of the acute febrile reaction. The earliest coagulative and hyaline changes in the sarcoplasm are seen just after the peak of the fever curve has been passed. It is also during the few days after this that the surge of blood in the vascular system becomes most intense. The process of muscular degeneration, however, is not completed apparently during this period but may go on often side by side with regenerative processes for an indefinite length of time.

8. *Torticollis.*—All the cases we have seen were associated with localised more or less severe degeneration of the cervical muscles, especially the anterior extremity of the longissimus dorsi, the spinalis, the semispinalis and the multifidus dorsi. The obliquus capitis anterior and posterior are frequently affected as also most of the lesser cervical muscles. The most striking and extensive degeneration of muscle was seen in the first-named muscles in cases of wry-neck. It is difficult with the naked eyes and even with microscopic sections to compare the degree of damage in muscles from one side of the neck, with that on the other. One gained the impression, however, that the damage was more extensive on the side toward which the head was bent.

How exactly the muscular damage produces a twist to one side is, however, not well understood. Since the bending is towards the more affected muscle one must assume contraction during, or after degeneration takes place. Evidence of such contraction has not been seen in equally affected muscles or muscle groups in other parts of the body.

The permanent fixation of the twist as seen in one case after 2½ months is explained more readily as due to fibrous replacement of injured muscle and consequent immobilisation. Such fixation apparently does not take place in all wry-neck cases, as in many the affected muscle probably regenerates sufficiently to function again.

C. *Claws.*

Bekker *et al* (1934) have noted that the reddening of the coronet and bulb of feet occurred in about 50 per cent. of cases a few days after the fever subsides. Although they use the term " coronitis " to denote this extremely visible reddening, they do mention that sheep so affected walked with a distinct " laminitic gait ". Spreull also likened this walk to that of a " foundered horse ". Our own observations corroborate these findings and show that a state of *acute aseptic pododermatitis* exists which no doubt is comparable to laminitis in the horse.

In those cases in which the feet lesions are not pronounced and lameness is negligible, all that can be seen is a pinkish zone around the coronet accompanied by increased warmth. There is thus only a mild and transient hyperaemia of the corium.

When more pronounced, the symptoms of lameness and reddening of the coronet are quite obvious. In such cases one may see an extension of the reddened coronet (intense hyperaemia) in the form of a zone of small vertical red streaks within the horny periople extending all round the upper edge of the claw and bulb. These streaks are actual haemorrhages into the fine medullary canals of the growing horny substance. When present this " streaky zone " constitutes valuable additional clinical evidence of bluetongue especially during the recovery period when other symptoms may have subsided. In order to see it plainly it is worth while washing the bulb (heel) and claw with a brush and water, as the fine capillary streaks are easily obscured by dirt and moistening tends to render the horn more transparent. This zone is usually more pronounced in the hind feet, and persists for some weeks after the hyperaemia has disappeared. The red streaks change to brown and gradually wear off as new horn grows. It is of interest to mention that a few of the experimental sheep, which had shown a typical bluetongue reaction with the presence of pododermatitis, developed this lesion again when they were artificially reinfected with another virulent heterologous strain of virus. In these cases it was possible to note a second band superimposed above, but distinct from the first.

In the dead animal the marked congestion of the horny corium (sensitive lamina), can be clearly seen either by splitting the claw in half or by paring down the horny wall and sole of the claw. It has been recorded (Bekker) that exungulation may occur rarely, and that a " break " or ring along the growth of the claw is not infrequent.

Microscopic examination reveals an intense hyperaemia of the vascular corium of the claw continuous with that of the skin. The papillary bodies and especially their tips are the seat of intense vascular engorgement, which inevitably leads to serous exudation, extravasation, and infiltration with neutrophiles. There is thus a true inflammatory process (of varying severity) of the whole sensitive lamina of the claw.

The streaky red zone in the horny wall is produced in the following way. During the height of engorgement some of the serous exudate is forced from the tip of the papillary body into its continuation, namely the hollow medullary canals of the horny wall or sole. These canals are mere capillaries but their first portion in the soft periople are capable of distension. It is the filling of this part with extravasated blood which gives rise to the zone of red vertical streaks externally visible. These horny canals, of course, contain serous fluid, and also masses of inflammatory cells, e.g., neutrophiles, besides the extravasated blood. The change of colour after a few days from red to brown is merely the effect of haemoglobin disintegrating and changing to bilirubin.

D. *Lungs.*

Of the 42 experimentally produced cases of bluetongue eight actually died and of these six had developed pneumonia. Among the other 34 which were all killed in different stages of the disease early congestion of the lung was evident in eleven.

While investigating mortality in several localities in the Union of South Africa following bluetongue vaccination in 1934 and during the ensuing years, a superimposed pneumonia, often with fatal termination, was a frequent occurrence. It should be mentioned, however, that inclement weather and exposure of the reacting sheep was probably responsible for the high incidence of pneumonia in these instances.

There is nothing characteristic about the type of pneumonia seen in our experimental cases. It starts with a severe congestion of the lung and passes over to a catarrhal broncho-pneumonia, with a tendency to fibrinous admixture in the worst cases. If capillary embolism of the lung by particles of muscle tissue did take place, we were unable to demonstrate it.

Under our experimental conditions and also in natural outbreaks we did not see cases of aspiration pneumonia such as Spreull mentions and ascribes to paralysis of the oesophagus.

E. *Spleen.*

In many of the bluetongue cases we examined, the spleen showed a moderate accumulation of neutrophil polymorphs in the red pulpa which appeared in the form of a leucostasis rather than an inflammatory process. There was also a distinct increase of reticular cells which sometimes gave the impression of islands or bands in the pulpa. This would account probably for the light tumor splenis usually seen in bluetongue.

F. *Other Organs.*

In the absence of obvious abnormalities in other organs and tissues, no further histological examination was undertaken.

Discussion.

From the findings reviewed in this paper it seems clear that the pathological changes in bluetongue concern mainly—

(*a*) the vascular system,

(*b*) the skeletal musculature.

(*a*) *The Vascular System.*

Beyond noting the occurrence of widespread vascular and circulatory disturbances (hyperaemia, oedema and haemorrhages) we have not undertaken any special and detailed study of this system and are not, therefore, in a position to discuss the pathogenesis of these very extensive changes.

No special attempt was made to demonstrate the presence or effects of the virus in tissue (e.g., inclusion bodies, etc.), and until that is possible, progress is likely to be slow, since we do not know what cells are attacked by or contain the virus. Evidence of hyalinisation of vessel walls could not be seen, but it would be desirable to determine whether the fragility of vascular intima is increased or not. It seems, however, permissible to

suggest that the anaemia which has been seen in some cases of bluetongue, when not caused by verminosis or some other independent agent, could be due to the loss of blood taking place when haemorrhages occur in such widely dispersed manner within so many different tissues of the whole body.

It is believed that the break in the wool which often accompanies bluetongue—and, of course, seriously detracts from its commercial value—is traceable to the severe congestion of the cutis. Perioplic " rings " or " breaks " in the horny claw would be produced in a similar way. No direct evidence has ever been advanced as far as we know to explain how this break is effected. One merely assumes that it is due to temporary malnutrition of the hair root, during *blood stasis* or other circulatory disturbance attendant upon the congestion.

(*b*) *The Skeletal Musculature.*

The changes which have been described in striated muscle in bluetongue vary enormously both in intensity and in distribution,—from simple coagulation and hyalinisation of scattered and isolated muscle fibres down to complete disruption, resorption or even necrosis. We do not know whether the changes seen are the result of direct virus action or merely some secondary effect, e.g., due to excessive lactic acid formation.

According to Ziegler (1929) the degenerative processes in various organs are usually the sequelae of infectious diseases and intoxications. The cross-striated muscle fibres are extremely sensitive to pathogenic organisms and toxins. Pathological changes are frequently observed in the skeletal musculature long before any abnormality is visible in the parenchymatous organs. In the case of bluetongue the macroscopical and microscopical lesions were present in the experimental Merino sheep irrespective of the strain to which the sheep were reacting. Generally speaking it can be stated that the severity of the lesions varied according to the virulence of the strain of virus employed. It should, however, be remembered that the susceptibility of individual sheep varies within wide limits (Neitz, 1944) and that it was sometimes noticed that the lesions in sheep artificially infected with a mild strain of bluetongue virus, e.g., the " Theiler " strain, were as severe as in sheep suffering from a virulent strain such as the " Bekker " virus. The development of these muscular lesions even in sheep which reacted to the " Theiler " strain of virus, which has been passaged in sheep for more than thirty generations, is further evidence in support of the contention advanced by Neitz (1944) that no true attenuation is brought about by the serial passage of bluetongue strains in sheep.

The veterinary literature abounds with descriptions of very similar muscular changes produced apparently under widely different circumstances, e.g., the so-called " Stiff lamb disease ", Metzger and Hagan (1927) and Udall (1933); myodegeneratio hyalinosa in the horse, young pigs and lambs, Hobmaier (1924 and 1926); muscular dystrophy in biliary fistula dogs, Brinkhous and Warner (1941); paralysis of the young of vitamin E deficient female rats, Olcott (1938). Hall and Wakefield (1927), working on dogs showed that by raising the body temperature artificially, lactic acid increased enormously in the skeletal muscles although muscular lesions are not mentioned. It is generally accepted also that in heat stroke the early and strong muscular rigor set up is associated with abnormal quantities of lactic acid in the muscles. In certain virus diseases of man, e.g., influenza, Dengue, etc., muscular pains and stiffness are well-known symptoms and it is suspected that some muscular lesions are present. In

our present study which included for comparative purposes ten sheep which died of diseases other than bluetongue, we found light, but nevertheless distinct, changes of a similar character in muscles from the five cases of heartwater (rickettsiosis) and in one of enzootic icterus.

Wester (1935) in his discussion of the pathogenesis of paralytic myoglobinuria (azoturia) of horses, states that a collapse or compression of the muscle capillaries has been demonstrated by the injection intravenously of certain pigment suspension (ink). In cases of azoturia the pigment failed to circulate through the finer capillaries of affected muscle while in healthy animals there was no such difficulty. He goes on to say that this partial or total obstruction of capillary blood flow would so retard or prevent the elimination of lactic acid formed in muscle fibres that its accumulation undoubtedly would lead to coagulation and other damage to the sarcoplasm.

Investigations along these lines as far as muscular changes in bluetongue are concerned might possibly be fruitful. A transient obstruction to capillary blood flow, whether by compression, constriction, or simply by stasis (i.e., choking of capillary by say highly viscous blood) could explain, as Wester does, the necrobiosis and coagulative degeneration of muscle fibre.

Incidentally it would also fit in with our suggested explanation (see vascular changes) of the break in the wool. If such a mechanism were established it would also be easier to reconcile the occurrence of similar muscular changes in a variety of non-related diseases.

As regards the possible relationship of vitamin E deficiency to such circulatory disturbance as a link in the causation of muscular degeneration we have been able to find no reference at all. We can only state that the descriptions and pictures of muscular lesion given by Telford (1938), Brinkhous (1941), Knowlton (1939) and others, as occurring in vitamin E deficient rats and dogs resemble very closely that described by us in sheep.

If it is impossible as yet to give a full account of the development of these lesions, at least these observations do shed some light on some hitherto obscure phases of the disease; e.g., firstly the part played by the intense vascular engorgement in producing haemorrhages, lameness (pododermatitis), break in wool, etc., and secondly the reason for muscular weakness, torticollis, and wasting of musculature leading to so-called " bad doers " with the necessarily slow muscular regeneration when recovery does take place.

Since the recognition of the above described muscular lesions, it has become apparent that certain clinical symptoms and the very long convalescent periods, which were inexplicable up to the present, can now be accounted for.

This discussion would be incomplete if nothing was mentioned about the economic aspect of this phenomenon. The losses from bluetongue due to mortality may sometimes be as high as 30 per cent., but as a rule they do not exceed 10 per cent. of the flock. The financial losses experienced by sheep-breeders, as the result of emaciation and poor wool production are even more important especially since they are not so readily measured in terms of money. It may sometimes take months before sheep become fit for slaughter. In a country like South Africa, where exogenous breeds of sheep are reared on a very large scale for wool as well as for mutton indirect losses due to bluetongue assume a different and greater significance when viewed in the light of these findings.

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Fig. 1.—Shoulder muscle showing multiple haemorrhages.

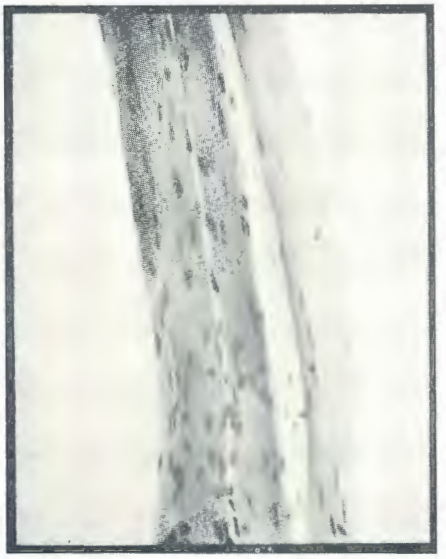

Fig. 2.—Normal muscle fibre (sheep) with cross-striation. Magnification 180×

Fig. 3.—Early hyalinisation in bluetongue. Note loss of striation and pyknosis of sarcolemma nuclei. Magnification 180×.

Fig. 4.—Hyalinisation of muscle fibres in bluetongue. Note irregular outlines and " perished rubber " tearing of the sarcoplasm. Magnification 80×.

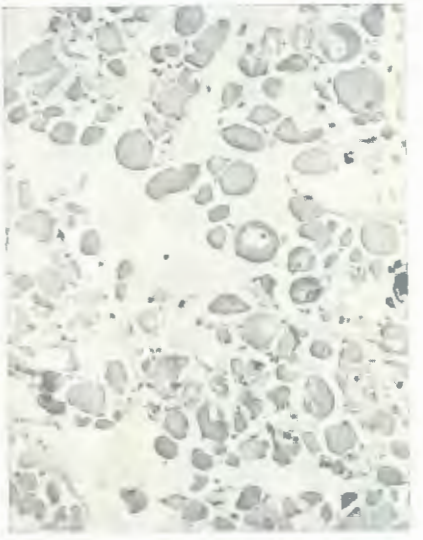

Fig. 5.—Cross-section through a degenerated muscle in bluetongue. The size of the fibres is variable and many show tears in the sarcoplasm. Magnification 120×.

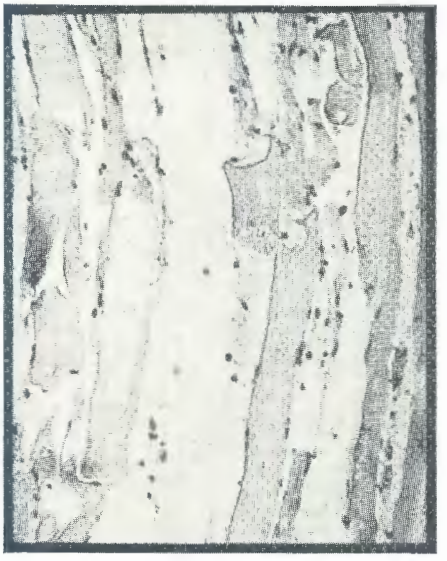

Fig. 6.—Irregularities in coagulation and tearing of the sarcoplasm. Empty portion of the sarcolemma can be seen. Magnification 140×.

Fig. 7.—Regeneration of muscle tissue in bluetongue.

Above pale hyalinised fibres and then a band of extravasated blood. In the middle pale and basophilic punctated swollen fibre in course of liquidation by macrophages. Below proliferation of sarcolemma nuclei, some forming short bits of sarcoplasm, which later fuse to make a fibre.

Fig. 8.—Hind feet of a bluetongue case. Arrow points at the " streaky zone ".

Fig. 9.—Fore foot with distinct " streaky zone " just below the periople of claw.

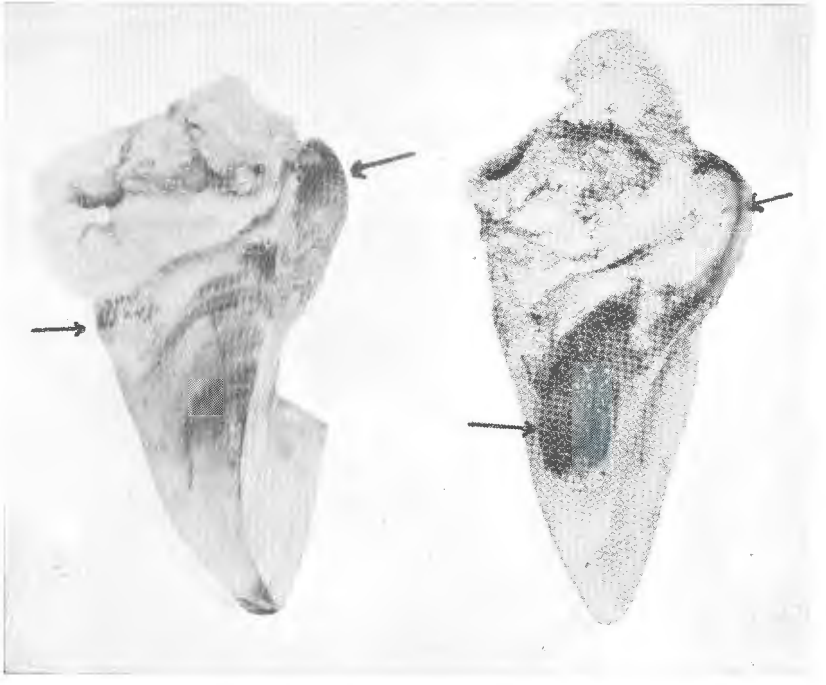

Fig. 10.—Claws fixed in formol. Arrows on the left claw point to the haemorrhagic, " streaky zone ", here prominent at the bulb. From the claw on the right, the horny wall has been removed and the arrows indicate the congested corium.