FEMORAL NERVE PARALYSIS IN CATTLE

by

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INTRODUCTION

Little is known of femoral nerve paralysis in calves. The major reports consist of a description of experimentally-created paralysis in two calves by Hunting (1964) and clinical and pathological findings in field cases (Tryphonas et al., 1974).

The report by Frauchiger and Hofmann (1941) described femoral nerve paralysis as a rare occurrence because the nerve was well protected by being buried deep in muscular and facial tissue. However, because of the large number of cases described by Tryphonas et al. (1974) and the number seen recently at Dykstra Veterinary Hospital, it would appear to be a more common condition than previously thought. This coupled with increasing reports from veterinary practitioners suggested that this condition required further investigation.

This research project was designed to study the pathogenesis of femoral nerve paralysis in young calves. The specific objectives were to study the sequential clinical signs and gross and microscopic changes of femoral nerve paralysis following neurectomy.

REVIEW OF LITERATURE

Clinical Signs

Clinical signs of femoral nerve paralysis in the bovine were first reported in 1904 by Grunth. He reported a cow that became suddenly lame and could bear no weight on the left hind leg. On attempting to bear weight, the stifle flexed because of paralysis of the quadriceps femoris muscle. He also reported shortened anterior phase of the stride and skin analgesia over the medial aspect of the tibia. The cow recovered in 14 days.

Combining all reported cases gave the following clinical picture of a calf attempting to bear weight on the affected leg. Sudden flexion of the stifle joint occurred resulting in a collapse of the hind limb and a slow and unsteady gait. Hypometria of the affected leg was also consistently reported (Grunth, 1902; Frauchiger and Hofmann, 1941; Frank, 1959; Gibbons, 1963; Vaughan, 1964; Rosenberger, 1970; Greenough et al., 1972; Tryphonas et al., 1974). The resting stance was also affected with the affected leg being flexed and the hip on the affected side held slightly lower (Rosenberger, 1970; Greenough et al., 1972). Atrophy of the quadriceps femoris muscle was apparent by 10 days, and by 30 days, a marked sunken appearance cranial to the femur was evident and the femur was easily palable (Frank, 1959; Vaughan, 1964; Greenough et al., 1972; Tryphonas et al., 1974). As atrophy progressed, the patella became freely moveable and easily luxated laterally or medially (Vaughan, 1964; Rosenberger,

1970; Tryphonas et al., 1974). Vaughan (1964) reported that patellar luxation did not occur spontaneously. Tryphonas et al. (1974) reported patellar luxation in 13 of 19 calves but did not make it clear whether or not it was spontaneous. Johnk (1949), however, reported that patellar luxation was spontaneous with femoral nerve paralysis. Gibbons (1963) stated that the foot was dragged, but Vaughan (1964) and Greenough et al. (1972) disagreed. There was no tendency for the foot to knuckle over (Vaughan, 1964). Absence of skin sensation over the medial aspect of the hock was variable (Frauchiger and Hofmann, 1941). Grunth (1902) reported it in a single case, but Tryphonas et al. (1974) found that it did not occur in dystocic calves since the injury was below the level where the saphenous nerve originated. Prognosis was reported as fair to grave (Frank, 1959; Rosenberger, 1970; Greenough et al., 1972; Adams, 1974).

Etiology

Several causes of femoral nerve paralysis were reported; the most common being overstretching of the nerve (Gibbons, 1963; Rosenberger, 1970; Greenough et al., 1972; Tryphonas et al., 1974). Other reported causes were pressure from an abscess, hematoma, or tumor (Rosenberger, 1970), milk fever (Greenough et al., 1972), and azoturia in horses (Frank, 1959; Adams, 1974). In the calf dystocia or getting the foot trapped were common causes for over-stretching the nerve and leading to paralysis (Gibbons, 1963; Rosenberger, 1970;

Tryphonas et al., 1974). Tryphonas et al. (1974) also proposed a second type of lesion arising from dystocia in which prolonged hypoxia from ischemia due to stretching and compression may result in nerve damage.

Differential Diagnoses

The most important differential diagnosis reported was lateral luxation of the patella (Johnk, 1949; Frank, 1959; Rosenberger, 1970; Greenough et al., 1972). Frank (1959) also mentioned rupture of the quadriceps as a differential diagnosis that could be distinguished by palpation. Signs of patellar luxation are similar to those of femoral nerve paralysis. When an affected animal attempted to bear weight, the limb collapsed resulting in flexion of the hock and stifle (Johnk, 1949; Frank, 1959; Greenough et al., 1972). The leg tended to be held back and stiff (Johnk, 1949) or with all joints flexed and barely touching the ground (Frank, 1959). The patella could easily be palpated on the lateral aspect of the stifle (Frank, 1959; Greenough et al., 1972). Meagher (1974) reported two cases of congenital bilateral patellar luxation. In these cases the calves adopted a crouching stance. Luxation of the patella was a major problem in working cattle in India and the causes reported as a genetic predisposition (Tyagi et al., 1974), excessive strain on young ligaments with heavy work (Pillai, 1944), or nerve damage (Bhatia et al., 1962).

Characteristics for differentiating femoral nerve paralysis from patellar luxations include lack of a patellar

reflex with femoral nerve paralysis and its presence with lateral patellar luxation (Frauchiger and Hofmann, 1941). Meagher (1974) reported that the marked atrophy seen with femoral nerve paralysis did not occur with lateral patellar luxation although the affected musculature was not as well developed as normal. Frank (1959) and Greenough et al. (1972) reported that palpation of the patella lateral to the stifle was important in differentiation as was radiographic confirmation. However, since Johnk (1949) observed spontaneous patellar luxation with femoral nerve paralysis, this is probably not an accurate mean of differentiation. Rosenberger (1970) stated that analgesia over the medial aspect of the hock was important for differentiation, but the findings of Tryphonas et al. (1974) that the saphenous nerve was unaffected indicates that this differential characteristic is not dependable.

Pathological Findings

The only report relating clinical femoral nerve paralysis in the cattle to the underlying pathology was that by Tryphonas et al. (1974). Gross findings in calves necropsied shortly after birth included hemorrhages at the point of entry of the femoral nerve into the quadriceps femoris muscles, rupture of the femoral nerve extramuscularly close to its entry into the muscle, and epineural hemorrhages along the entire extravertebral length. They found the saphenous nerve unaffected in all cases. The soft tissue lesions in

calves 7 to 112 days of age included unilateral atrophy of the quadriceps femoris muscle which progressively shrunk with age and had pale discoloration, loss of texture, and gelatinous transformation. The affected nerve was thickened segmentally and/or diffusely up to twice normal, dull white, firm, and fibrous, and adhesions were formed in some cases. If the affliction existed for more than 40 days, the femur and the whole affected limb were atrophied.

Microscopically there were muscle degeneration and atrophy in calves affected for more than seven days. The degree of atrophy varied between calves and between different fascicles of the same muscle. The affected fascicles were reduced in size and contained fibers in different stages of atrophy. The cross striations persisted, and the nuclei were rounded, shorter than normal, and vesicular. The nuclei occupied up to 90 percent of the cross-sectional area of the severely affected fibers. Other findings included perimysial edema and fat infiltration, myodegeneration, and focal infiltration of inflammatory cells and connective tissue cells (Tryphonas et al., 1974). In describing femoral nerve paralysis in the horse, Frank (1959) described the later stages of degeneration in which the contractile substance completely disappeared, the fibrils degenerated into granules of fat which were absorbed, and the affected muscle was converted into fibrous connective tissue.

The affected femoral nerves had thickened epineurium and perineurium containing islands of degenerating adipose

tissue and several thick walled arteries. There was an extreme loss of myelin and axons. On cross section, the affected nerves contained a variety of proliferating connective tissue cells, endothelial cells, Schwann cells, and macrophages (Tryphonas et al., 1974).

Wallerian Degeneration

In reviewing the literature, the term Wallerian degeneration was used to describe the nerve degeneration observed (Tryphonas et al., 1974). However, no reports were found describing the actual sequential occurrence of this degenerative process in cattle. Therefore, a limited literature review describing this process in other animals is presented.

As early as 1787, Arnemann reported changes in the distal part of transected nerves. Nasse (1839) described fairly accurately the changes in the distal stump of the severed nerve; however, others of that time denied such changes (Bots, 1970). Waller (1850, 1852) resolved this controversy by his classical studies on degeneration of severed hypoglossal and glossopharyngeal nerves in the frog. He stated that the portion of the nerve separated from its trophic centers underwent complete degeneration and that regeneration took place through an outgrowth of new fibers from the central stump.

Wallerian degeneration is believed to be a universal process occurring in any lesion leading to interruption of axonal continuity (Howell and Huber, 1892; Cajal, 1928;

Nageotte, 1932; Bots, 1970).

Cajal (1928) divided axonal degeneration into three phases. The first phase consisted of shrinkage in diameter of the axon with irregular swellings giving a moniliform or varicose appearance; this change occurred as early as 12 hours post nerve section. The second phase was granular degeneration of the neurofibrils beginning 30-48 hours after section. The third phase included axonorrhexis and slipping of the fragments. The first breaks occurred opposite the nodes of Ranvier, the nuclei of Schwann, and then elsewhere (Cajal, 1928; Blackwood et al., 1963; Bots, 1970). The fragments were coiled into spirals or hooked (Cajal, 1928). Rhexis continued until it eventually yielded numerous small segments which eventually became oval (Blackwood et al., 1963; Bots, 1970).

Concurrently with axonal degeneration, the myelin underwent degenerative changes and separated from the nodes of Ranvier. This occurred at times before any axonal breaks and as early as 16 hours post section. This process occurred in different nerve fibers at different rates which progressed more rapidly in the larger fibers (Cajal, 1928; Bots, 1970). The myelin then broke into progressively smaller ovoid fragments with the next breaks coinciding with the incisures of Schmidt-Lantermann (Nasse, 1839; Waller, 1850, 1852; Howell and Huber, 1892; Cajal, 1928; Nageotte, 1932). When the myelin breaks coincided with the axon breaks, the segments were closed by surface tension so that the axonal elements

were contained within a cavity in the myelin. Here they were broken down and dissolved hence the term "digestive chambers" (Cajal, 1928; Nageotte, 1932), a process that was usually completed by 10 days. Nathaniel and Pease (1963) demonstrated that these myelin ovoids were enclosed in membrane-bound vacuoles resembling phagosomes within the Schwann cells. The myelin underwent degenerative changes: breakdown of the lipids into water-soluble components which were reabsorbed and fatty acid sidechains which were esterfied with cholestrol, itself a degenerative product of myelin (Blackwood et al., 1963; Bots, 1970). The cholestrol and cholestrol esters diminished and disappeared, and the lipids progressively assumed the characteristics of neutral fat (Blackwood et al., 1963). The breakdown products were eventually phagocytosed and removed (Cajal, 1928; Nageotte, 1932; Bots, 1970). Whether the phagocytic cells originated hematogenously or directly from the Schwann cells is currently a subject of controversy (Nathaniel and Pease, 1963; Ashbury, 1970; Bots, 1970). Whether Wallerian degeneration progresses somatofugally or occurs along the entire length of the axon simultaneously is undertermined (Nathaniel and Pease, 1963; Simon et al., 1969; Donat and Wisniewski, 1973; Joseph, 1973).

Cells around nerve fibers react quickly to axon severance with Schwann cells enlarging and undergoing mitosis as early as the fourth day (Cajal, 1928; Nageotte, 1932). Schwann cells proliferate markedly and become filled with myelin and axonal breakdown products. This was formerly considered to

form a syncytium but this was not confirmed by electron microscopy (Bots, 1970). Fibroblasts in the endoneurium began to proliferate as early as the second day, and fat absorbed by the endoneurium by the fourth day. Infiltration of leucocytes occurred by the eighth to tenth day (Cajal, 1928). With the final absorption of the breakdown products of myelin and the axon, Schwann sheaths remained and formed so-called band fibers or Bugners bands. These shrink and remain idle until reinnervation occurs (Nageotte, 1932; Bots, 1970).

Neurogenic Atrophy of Muscle

Concurrently with Wallerian degeneration, a process known as neurogenic atrophy occurs in muscle that has lost its nerve supply. The first noticeable change was loss in muscle bulk. Weight loss became apparent as early as the third day (Tower, 1939) and progressed to an end stage of 60 to 80 percent atrophy approximately 4 months post-denervation (Sunderland and Ray, 1950).

The first histological changes occurred approximately one week after denervation and consisted of sarcolemma nuclear swelling (Tower, 1939; Sunderland and Ray, 1950; Adams, 1962). Nuclei of the endomysial tissue underwent similar changes (Tower, 1935). By two weeks, the small blood vessels had thickened and congested, and mononuclear cells appeared (Tower, 1935; Adams et al., 1962). The changes following this consisted of a rapid decrease in the diameter of the muscle fibers with preservation of the cross striations.

The fibrous tissue of the perimysium and endomysium proliferated and thickened (Tower, 1935; Adams et al., 1962). With fibrosis, the amount of fat increased (Chor et al., 1937). At some time during this process, degeneration began in scattered but progressively numerous muscle fibers (Adams et al., 1962). The histological features of this process included vacuolization and swelling of the fibers with loss of cross striations (Tower, 1935). The ultimate fate of the denervated muscle was replacement by adipocytes with variable increase in the amount of reticulin fibrils in the endomysium and collagen in the perimysium and aponeuroses (Adams et al., 1962).

MATERIALS AND METHODS

Experimental Animals

Nine calves from three to eight weeks of age were used in this study. The breeds used were four Jerseys, three Holstein-Friesians, and two Herefords.

There were housed either indoors at Animal Resource Facility of Kansas State University or in outdoor pens if they remained with their dams. The indoor calves were fed milk replacer and free choice grain and were weaned on a ration of grass and alfalfa hay and grain. The dams were fed grass and alfalfa hay.

Femoral Neurectomy

The calves were anesthetized with intravenous xylazine* (0.15 mg/lb. body weight) and surgically prepared in dorsal recumbency (Fig. 1). The right femoral nerve was approached by an incision over the medial aspect of the thigh (Fig. 2) and by sharp dissection of the fascia at the anterior edge of the sartoris muscle (Fig. 3). The nerve was then isolated from the anterior femoral vessels by careful blunt dissection and severed by scalpel or scissors (Fig. 4). A small segment of nerve measuring 0.5 cm was removed. A muscle biopsy of the quadriceps femorus muscle (2 x 1 x 0.3 cm) was taken by blunt disection of muscle (Fig. 5). The skin was then closed by interrupted horizontal mattress sutures (Fig. 6). The left

*Rompun^{R}, Haver-Lockhart

Fig. 1. Calf. Femoral neurectomy. Procedure. Preparation of leg.

Fig. 2. Calf. Femoral neurectomy. Procedure. Incision.

Fig. 3. Calf. Femoral neurectomy. Procedure. Dissection along anterior edge of sartorius muscle.

Fig. 4. Calf. Femoral neurectomy. Procedure. Isolation of femoral nerve.

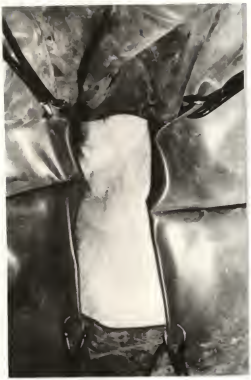

Fig. 1

Fig. 2

Fig. 3

Fig. 4

Fig. 5. Calf. Femoral neurectomy. Procedure. Muscle biopsy.

Fig. 6. Calf. Femoral neurectomy. Procedure. Sutured incision.

Fig. 5

Fig. 6

leg was preserved as a control.

Clinical Signs

The clinical signs of stance, gait, and appearance were observed and recorded on all calves. Photographs and 8 mm movies were taken. Palpation of the affected limb and patellar laxity was performed.

Necropsy

The calves were euthanatized with intravenous magnesium sulfate solution and necropsied according to the following schedule: two at two days post surgery (PS), one at six days PS, two at 10 days PS, one at 14 days PS, two at 60 days PS, and one at six months PS.

The quadriceps femoris muscles were compared right to left, and tissue sections were taken from the right and left rectus femoris, vastus lateralis, vastus intermedius, vastus medialis, and sartorius muscles. The muscle tissue was stretched and stapled to wooden tongue depressors to prevent contractions and artefacts. Tissue was also taken from the proximal and distal stumps of the right femoral nerve, from the left femoral nerve, and from the lumbar spinal cord. The tissues were fixed in 10% buffered-neutral formalin, embedded in paraffin, cut at 6 mm thickness, and stained by hemotoxylin and eosin. Frozen sections were cut at 17 mm thickness and stained for fat by oil-red-o. Special stains were also employed for the muscle sections consisting of toluidine blue,

trichrome, and phosphotungsic acid hematoxylin (PTAH). Special stains for the nerves were Bielchowsky's silver stain and luxol fast blue (Luna, 1968).

Muscle Biopsy

Muscle biopsies were also taken at 2 weeks and one month from one of the calves that lived for 2 months and at 2 weeks, one month, 2 months, and 4 months from the calf that lived for 6 months. The biopsy technique was similar to that used in the femoral neurectomy procedure. The muscle tissue was handled and fixed using the same techniques as used at necropsy.

RESULTS

The clinical signs following neurectomy were those of a severe unilateral, rear limb lameness.

Stance

The resting stance was altered so that no weight was borne on the affected limb. Considerable variation existed in the stance of the different affected calves, although some degree of flexion of the stifle and all distal joints was a fairly consistent feature of the affected leg. The unaffected rear leg was positioned medially to its normal position and oftentimes, but not consistently, posterior to its normal position (Fig. 8). The calves adopted a wide base on their front legs with the front leg opposite to the affected hind leg usually being anterior to its normal position, and the front leg on the affected, side consistently being placed posterior to its normal position (Fig. 7). There was no consistent placement of the affected leg. If the leg was placed far anterior to its normal position, the foot rested flat on the ground (Fig. 8). At any other position, the flexion of the digit caused the foot to rest on its toe (Fig. 7). Many of the calves also positioned the affected limb so that the hock was rotated medially, and the digit was rotated laterally (Fig. 7).

Gait

The lameness was apparent immediately after recovery

from anesthesia. A period of time, several hours to over a day, was required for the calves to learn to attain a standing position. The gait of the affected calves was characterized by a sudden flexion of the stifle and all distal joints when the calf attempted to bear weight on the affected limb. This caused collapse of the limb and dropping of the hip (Fig. 10). In turn, the calf was forced to take a very quick and shortened stride with the opposite hind limb. The placement of the affected limb was accurate with a full anterior phase of the stride being completed normally (Fig. 9). No tendency for knuckling over was observed, and the foot rarely dragged. Variations between calves were noticed. The heavier muscled calves such as the Herefords and Holstein-Friesians had a severe lameness with marked collapse of the leg and dropping of the hip. The lighter muscled Jersey calves, on the other hand, did not demonstrate the clinical signs to nearly as great a degree. The leg did not collapse as markedly as in the heavier calves, but hypometra was more pronounced. In one Jersey calf, lameness essentially disappeared within four days PS, and on clinical and gross postmortem evaluation, it appeared that the tensor fascia lata muscle was able to compensate for the deficient quadriceps femoris muscle. Temperament also apparently played a role in the demonstration of clinical signs. One Hereford calf remained with its mother, and both were excitable. While being observed, the calf would not walk slowly, but only run or stand. The lameness was not readily apparent when the calf was running, but it did

Fig. 7. Calf. Femoral nerve paralysis. Affected stance.

Fig. 8. Calf. Femoral nerve paralysis. Affected stance.

Fig. 9. Calf. Femoral nerve paralysis. Placement of affected foot.

Fig. 10. Calf. Femoral nerve paralysis. Collapse of limb when attempting to bear weight.

Fig. 7

Fig. 8

Fig. 9

Fig. 10

tend to stumble when turning a corner. Careful observation revealed the characteristic stance described above.

Atrophy

A hollowed or shrunken appearance just anterior to the femur was noticeable soon after neurectomy (Fig. 11 and 12). As early as two to three days PS a difference was evident between the affected and control sides. This was largely due to loss of muscle tone since postmortem examination failed to demonstrate any appreciable atrophy at this time. By day six to seven PS, the hollowed appearance was more evident, and postmortem examination confirmed neurogenic atrophy. The atrophy progressed until by two months PS the femur was easily palpable, and a large muscular deficit was visible.

Patellar Laxity

Loss of tone in the quadriceps femoris muscle caused the patella to be loosened and readily luxated either medially or laterally. None of the calves developed spontaneously luxated patellas. Their ability to manually luxate the patellas existed from the first day PS, except in the Jersey calf that had no appreciable lameness after the fourth day PS.

Gross Pathology

At two days PS, very little gross change was evident except for a reddening of the quadriceps femoris muscle. By six days PS gross decrease in size of the quadriceps was

Fig. 11. Calf. Femoral nerve paralysis. Arrow points to area of atrophy of quadriceps femoris muscle.

Fig. 12. Calf. Femoral nerve paralysis. Arrow points to area showing atrophy of quadriceps femoris muscle.

Fig. 11

Fig. 12

easily detectable by comparison of right and left hind legs. The cut nerve was slightly swollen with perineural hemorrhage, and adhesions had formed at the cut ends.

By 10 days PS, the atrophy had further progressed and was readily visible. The affected muscle began to become paler. Comparative weights of the quadriceps femoris muscles were taken at this time; the right weighed 760 g, as compared to 920 g for the unaffected left.

The calf necropsied at 14 days PS had even more advanced atrophy. The right quadriceps femoris muscle weighed 760 g compared to 1090 g of the left. The affected muscle was paler and more tan than the normal red color (Fig. 13 and 14).

At 60 days PS, the affected muscles were smaller and more pale. In one calf with little lameness, the tensor fascia lata muscle of the right leg was greatly hypertrophied.

Six months PS, the quadriceps femoris muscle was atrophied to about 3/4 its normal size (Fig. 15 and 16). This degree of atrophy really differed little from that at 14 days PS and was less extensive than that at 2 months PS. However, the paleness was more marked, and the fibrosis was grossly evident. The muscle had a greasy feel.

Histopathology

No change was detectable histologically at two days PS.

Atrophy of the muscle fibers was detectable by the sixth day PS. This change could only be seen by direct side-by-side comparison of the affected and control muscle. The fibers had

Fig. 13. Calf. 77-2075. Femoral nerve paralysis. Atrophy of quadriceps femoris muscle. Two weeks duration.

Fig. 14. Calf. 77-2075. Femoral nerve paralysis. Atrophy of quadriceps femoris muscle. Two weeks duration.

Fig. 15. Calf. 77-53. Femoral nerve paralysis. Atrophy of quadriceps femoris muscle. Arrow points to affected muscle. Six months duration.

Fig. 16. Calf. 77-53. Femoral nerve paralysis. Atrophy of quadriceps femoris muscle. Arrow points to affected muscle. Six months duration.

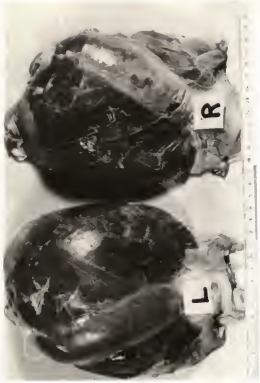

Fig. 13

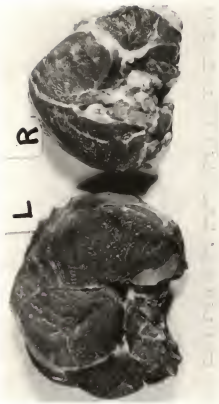

Fig. 14

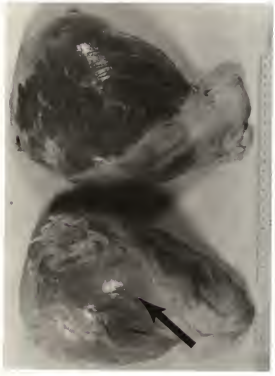

Fig. 15

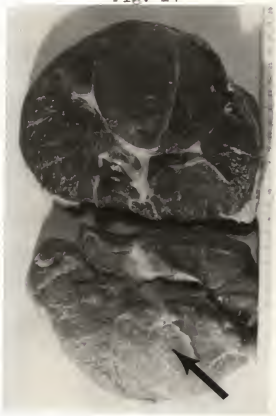

Fig. 16

a smaller diameter on cross section, and the nuclei appeared more concentrated in longitudinally cut sections (Fig. 18). Some axonal swelling had occurred in the nerve at this time.

The histological changes were marked by day 10 PS. The neurogenic atrophy of the muscle was detectable even without a control muscle for comparison. On longitudinal section the muscle nuclei were densely concentrated and slightly swollen giving them a slightly more rounded outline (Fig. 19). The cross-sectional appearance clearly demonstrated a decrease in the size of myofiber (Fig. 28). The perimysium was prominent and thickened. The fibroblasts of the perimysium were proliferating, and their nuclei were swollen and rounded. The changes in the degenerating nerve were quite distinctive at this time. The "digestive chambers" were readily visible with routine stain, but especially so with oil-red-o staining (Fig. 21). Some variability in staining was evident; in one calf the "digestive chambers" were staining positively for neutral fat, and not in the other calf.

Changes in the muscle fibers at 14 days PS were nearly identical to those changes at 10 days (Fig. 24). However, the connective tissue proliferation was more advanced and clearly affected the perimysium and endomysium, and there was more collagen. Fibroblasts were increased in number, and many had swollen, vesicular nuclei of an oval, rather than spindle, shape. Neutral fat was infiltrating the affected muscle between the muscle fibers and along the endomysium in small and large globules. Many of the larger globules were

Fig. 17. Muscle control slide. Longitudinal section: 200x. Hematoxylin and eosin stain.

Fig. 18. Calf. 76-1500. Affected muscle. Six day duration. Longitudinal section. 200x. Hematoxylin and eosin stain.

Fig. 19. Calf. 76-1565. Affected muscle. Ten day duration. Longitudinal section 200x. Hematoxylin and eosin stain.

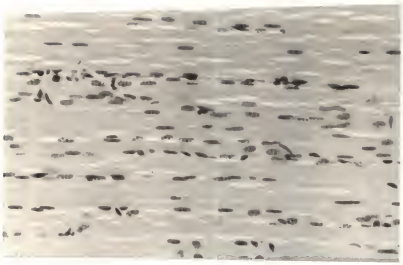

Fig. 17

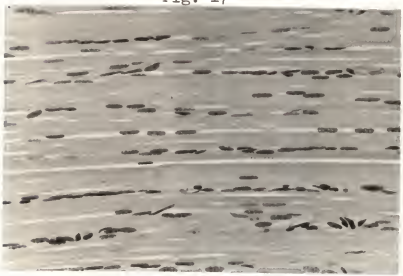

Fig. 18

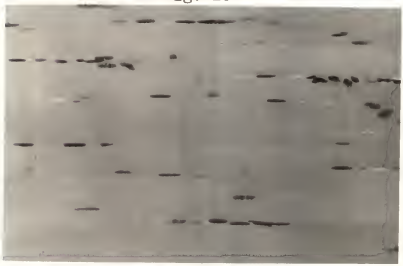

Fig. 19

clearly contained within adipocytes, and the number of adipocytes along the endomysium was increased. The cross striations were preserved. The "digestive chambers" of the nerve were extensively converted to neutral fat (Fig. 22). Many lipid filled macrophages were found around and invading the nerve fibers and phagocytizing some of the axonal remnants. The fibroblasts were greatly increased in number within the endoneurium and perineurium.

Femoral nerve paralysis of two months duration presented the most marked changes of denervation atrophy in the quadriceps muscle. The muscle fibers were decreased to an extremely small diameter with many of them appearing to be nuclei in rows connected by small bands of cytoplasm (Fig. 25). Even in this extremely atrophied state, the cross striations still persisted. Adipose and fibrous tissue had further increased in amount. However, the fibroblasts were more mature appearing with more elongated spindle-shaped nuclei, and collagen was more prominent. The adipose tissue consisted of large, mature adipocytes (Fig. 29). Few muscle nuclei remained rounded and swollen, but most had returned to a more normal elongate shape. The nerve was composed largely of fibrous tissue with empty nerve sheaths. Neutral fat was absent from the fasciculi of nerve fibers but increased in amount in the epineurium. A few large vacuolated macrophages remained in the nerve fasciculi. These did not stain for neutral fat but did stain for myelin. The epineurium and perineurium were thickened with collagenous connective

Fig. 20. Nerve control slide. Longitudinal section. 400x. Hematoxylin and eosin stain.

Fig. 21. Calf. 76-1542. Affected nerve. Ten day duration. Longitudinal section. 400x. Oil-red-o stain.

Fig. 22. Calf. 77-2075. Affected nerve. 14 day duration. Longitudinal section. 400x. Oil-red-o stain.

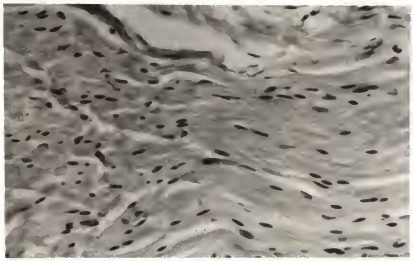

Fig. 20

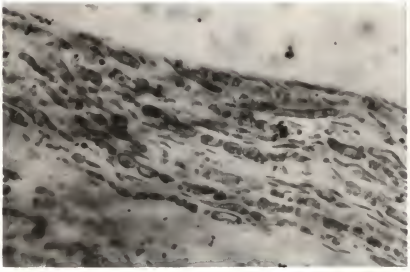

Fig. 21

Fig. 22

tissue and adipose tissue.

By six months PS, the processes of atrophy and nerve degeneration had reversed, and regeneration was the prominent feature. Fat was present in the endomysium and perimysium in increased amounts (Fig. 26). The collagenous connective tissue was present in approximately the same amount as at two months PS. However, many of the muscle cells were of normal size and appearance although a minority remained completely atrophied, and various other muscle cells were intermediate in size. Apparently a small percentage of the muscle fibers had undergone complete degeneration and replacement by adipocytes. This observation was based on the fact that on cross section the affected muscle had relatively less muscle fibers per fascicle than the normal muscle, and the spaces were occupied by adipocytes (Fig. 27). The nerve was also undergoing regeneration with massive budding occurring around the level of the transection (Fig. 31-33). A number of the budding nerves were successful at bridging the gap between the cut nerve ends as evidenced by axons and myelinated nerve sheaths in the distal nerve segments.

Fig. 23. Muscle control slide. Longitudinal section. 200x. Hematoxylin and eosin stain.

Fig. 24. Calf. 77-2075. Affected muscle. 14 day duration. Longitudinal section. 200x. Hematoxylin and eosin stain.

Fig. 25. Calf. 76-2037. Affected muscle. Two month duration. Longitudinal section. 200x. Hematoxylin and eosin stain.

Fig. 26. Calf. 77-53. Affected muscle. Six month duration. Longitudinal section. 200x. Hematoxylin and eosin stain.

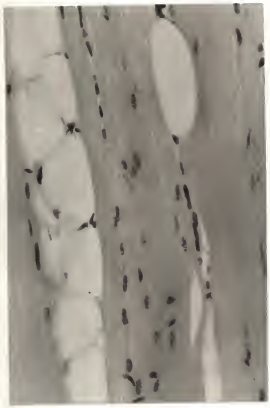

Fig. 23

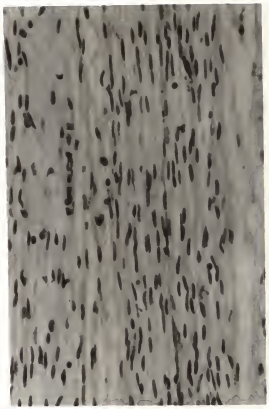

Fig. 24

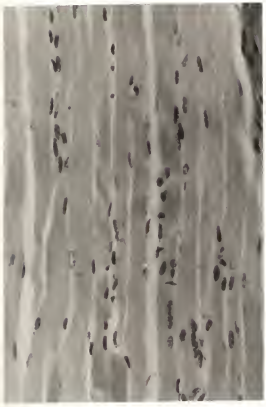

Fig. 25

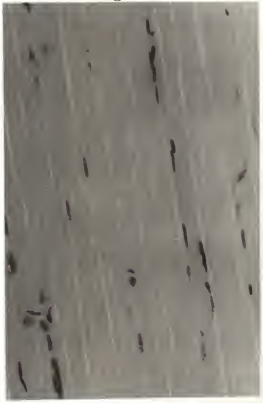

Fig. 26

Fig. 27. Muscle control slide. Cross section. 200x. Hematoxylin and eosin stain.

Fig. 28. Calf. 76-1542. Affected muscle. Ten day duration. Cross section. 200x. Hematoxylin and eosin stain.

Fig. 29. Calf. 76-2037. Affected muscle. Two month duration. Cross section. 200x. Hematoxylin and eosin stain.

Fig. 30. Calf. 77-53. Affected muscle. Six month duration. Cross section. 200x. Hematoxylin and eosin stain.

Fig. 27

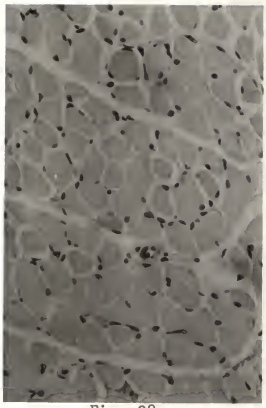

Fig. 28

Fig. 29

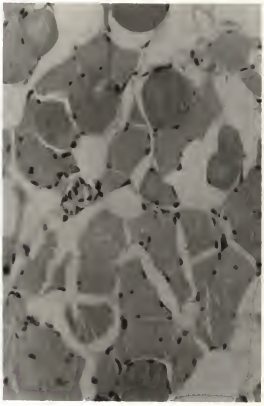

Fig. 30

Fig. 31. Calf. 77-53. Affected nerve, two mm. proximal to transection. Six month duration. Cross section. 400x. Hematoxylin and eosin stain.

Fig. 32. Calf. 77-53. Affected nerve, at transection site. Six month duration. Cross section: 400x. Hematoxylin and eosin stain.

Fig. 33. Calf. 77-53. Affected nerve, two mm, distal to transection. Six month duration. Cross section. 400x. Hematoxylin and eosin stain.

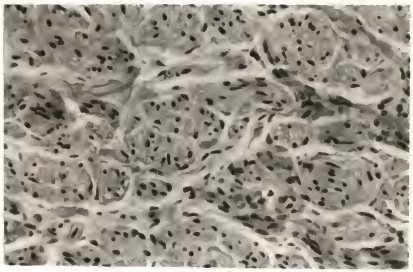

Fig. 31

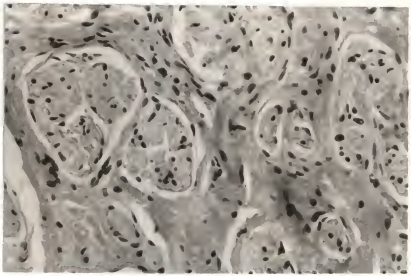

Fig. 32

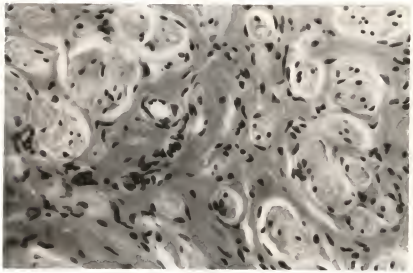

Fig. 33

DISCUSSION

Clinical signs produced by femoral neurectomy were consistent with those reported in naturally occurring cases and with clinical cases observed during this study. The reported shortened anterior phase of the stride (Tryphonas et al., 1974; Grunth, 1902; Greenough et al., 1972), however, was observed in only one case. Hypometria was consistently observed due to inability to complete the stride as a result of leg collapse rather than a shortened anterior phase of the stride. The inability to advance the affected limb and tendency to drag the foot as reported by Gibbons (1963) was not observed. His report dealt with calves following roping injuries, and possibly more factors were involved than simple femoral nerve paralysis. Spontaneous patellar luxation reported by Johnk (1949) was not seen in this study. However, the patella was lax and easily manually luxated medially or laterally within two days after neurectomy. This contrasted with the report by Tryphonas et al. (1974) who indicated that the ability to luxate the patella developed with advanced atrophy.

The most important condition that could be misdiagnosed for femoral nerve paralysis is luxation of the patella. The fact that patellar luxation also occurs congenitally is confusing. In this study spontaneous patellar luxation did not occur; therefore, palpation or radiographic visualization of the patella in a luxated position is considered to be presumptive evidence for primary patellar luxation. However,

the limited scope of this study does not rule out the possibility that femoral nerve paralysis may be an important predisposing factor for patellar luxation. Laxity of the patella and abnormal stresses placed on the stifle joint by complete collapse of the leg with each step could predispose to luxation. Differential diagnosis also includes rupture of the quadriceps femoris muscle but should easily be differentiated from this by palpation.

This study reports for the first time a sequential microscope study of Wallerian degeneration and denervation atrophy in cattle. Wallerian degeneration proceeded with axonal swelling being evident by day 6 PS and digestive chambers clearly visible by day 10. The digestive chambers were split and esterfied to neutral fat by day 10-14 PS and phagocytosis by macrophages began at this time. By two months, the nerves were composed mainly of empty neural tubes. Nerve regeneration occurred by budding of the severed ends and regrowth and remyelination of the existing nerve tubes. Denervation atrophy was apparent within six days by a decrease in nerve fiber size. The atrophy progressed to a maximum by two months PS. Fibroblastic proliferation was observed by the 10th day and fat infiltration by the 16th day. Muscle regeneration was evident clinically and by biopsy by the fourth month, the latter by increasing muscle fiber size with fat and collagen remaining. Routine hematoxylin and eosin staining was moderately useful for the nerve sections and oil-red-o staining was useful for visualizing the digestive chambers and neutral

fat. The luxol-fast blue stain was moderately useful for myelin sheath changes, but Bielchowsky's stain was useless for demonstrating axonal changes. Phosphotungsic acid hematoxylin staining was useful for demonstrating cross striations and collagen in muscles, and trichrome staining was also useful for the latter.

Results from this study suggest that a calf affected with femoral nerve paralysis may recover given sufficient time. With a valuable calf, confinement with protection from environmental factors is recommended. Regeneration will take at least six months.

It may be inferred from the results of this work that if a young calf is presented with a near limb lameness and is found to have a lax or even luxated patella, then femoral nerve paralysis should be ruled out before attempting any reparative procedures of the patellar ligaments.

In conclusion, the incidence of femoral nerve paralysis is increasing with increased cross-breeding with large exotic breeds. The incidence in traditional breeds may increase with the current trend towards larger framed, rapidly growing cattle.

SUMMARY

A study was undertaken to determine the clinical signs and gross and microscopic changes of femoral nerve paralysis following neurectomy. The femoral nerve was transected adjacent to its entrance into the quadriceps femoris muscle. The affected calves had a stance characterized by slight flexion of all joints distal to the stifle. The affected hip was slightly lower than normal. A slight hollowed appearance between the femur and tensor fascia lata muscle appeared early and became more obvious with time. The gait was characterized by flexion of the stifle and all distal joints when weight was borne resulting in a collapse of the affected leg. The most significant gross finding was a progressive atrophy of the quadriceps femoris muscle which became apparent in six days PS and progressed to a maximum by two to four months. The muscle also became progressively paler and more greasy in consistency. The most significant microscopic findings included Wallerian degeneration of the severed nerve and neurogenic muscular atrophy. Axonal swelling was evident by the sixth day PS; the digestive chambers were apparent by the 10th day and were converted to neutral fat by day 10-14; macrophages began engulfing the digestive chamber contents by the 14th day, and by two months, empty neural tubes remained. The muscle atrophy was evident by the sixth day and became progressively more marked until two months PS. Fat and connective tissue increased progressively in amount. The calf held for six months started a reversal of clinical signs and

gross and microscopic changes at about the fourth month. Regeneration was evident in nerve and muscle tissue by the sixth month.

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FEMORAL NERVE PARALYSIS IN CATTLE

by

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AN ABSTRACT OF A THESIS

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PATHOLOGY

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An increasing number of reports of femoral nerve paralysis suggested further clarification of the condition in young calves. This project was designed to sequentially study the clinical signs and gross and microscopic changes occurring in femoral nerve paralysis induced by neurectomy.

Nine calves underwent femoral neurectomy. In all calves the right femoral nerve was severed near its entrance into the quadriceps femoris muscle. The clinical signs were observed and recorded. The calves were euthanized at intervals of 2, 6, 10, 14, 60 days and six months post surgery and the gross and microscopic changes were determined.

Clinically, the calves adopted a resting stance characterized by slight flexion of the stifle and all distal joints and a slight lowering of the hip on the affected side. Atrophy of the quadriceps muscle was evident grossly by a hollowed appearance between the femur and tensor fascia lata muscle which appeared early and became progressively more marked until the second to fourth month. The patella was easily luxated manually. The gait was characterized by a sudden flexion of the stifle and the distal joints causing collapse of the leg when weight was attempted to be borne.

The gross findings were those of atrophy of the quadriceps muscle. A decrease in muscle size was seen by the sixth day, and became progressively more marked until two months duration. The muscle also became progressively paler and greasy in consistency.

Microscopically, the nerve underwent Wallerian

degeneration, and the muscle neurogenic atrophy. Axonal swelling was evident by the sixth day; the digestive chambers were quite apparent by the 10th day and were esterfied to neutral fat by day 10-14; and macrophages began phagocytizing the digestive chambers by the 14th day. By two months, only empty nerve tubes remained. Muscle atrophy was evident as decreased muscle fiber size by the sixth day and progressed until two to four months. Fat and connective tissue increased progressively in amount.

The calf held for six months began showing a reversal of clinical signs by the fourth month. At necropsy, atrophy was not as marked as at two months. Microscopically, nerve regeneration and reversal of the muscle fiber atrophy was observed. This led to the conclusion that given sufficient time, naturally affected calves may undergo regeneration and return to normal function.