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# Myopathy of the "Gracilis – semitendinosus muscle complex" in the dog

Vittorio Capello D.V.M. <sup>(1)</sup>, Carlo Maria Mortellaro D.V.M. <sup>(1)</sup>, Diego Fonda D.V.M. <sup>(1)</sup>.

S U M M A R Y

In the hindlimb of dogs a typical, chronic lameness, is easily recognized by walking the animal and by palpating the medial region of the thigh. Yet it is seldomly diagnosed.

This syndrome, named "contracture" or "fibrotic myopathy", has been indistinctly related to gracilis muscle, semitendinosus muscle, and even semimembranosus muscle lesions.

The difficulties in clinical interpretation led the Authors to suggest the name: "gracilis – semitendinosus muscle complex". The mechanism by which these muscles are affected is not clear, and one cannot exclude that the eventual cause may prove to be neurogenic. This may explain the ineffectiveness of surgery.

13 dogs affected by these lesions were referred to the Surgery Department of the University of Milan. 10 of the 13 dogs underwent surgery.

In this review the Authors outline the anatomic, aetiopathogenetic, clinical, diagnostic, therapeutic and prognostic aspects and proposed electromyography for earlier diagnosis.

*Key-words:* Dog, gracilis muscle, semitendinosus muscle, myopathy, electromyography.

## INTRODUCTION

In the hindlimb of dogs a characteristic, chronic lameness is readily recognized by walking the animal and palpating the medial aspect of the thigh. This syndrome is named in clinical practice and the literature as "contracture" or "fibrotic myopathy" and has been related to gracilis, semitendinosus and even semimembranosus muscle lesions.

Such difficulty in pathological interpretation led the present authors to use the term "gracilis – semitendinosus muscle complex". The mechanism by which these muscles are affected is not clear and the possibility that the origin is neurogenic cannot be excluded. This may explain the ineffectiveness of surgery.

13 dogs affected by this condition were referred to the Surgery Department of the University of Milan and 10 of them underwent surgery.

In this review the authors outline the anatomy, aetiopathogenesis and clinical features of this disorder and emphasise it's therapy and prognosis. The use of electromyography for early diagnosis is proposed.

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The term myopathy which has been chosen to define the pathology has to be explained. Any anatomical or functional change of muscle or its tendinous insertions may be termed a "myopathy", so that primary (myogenic) myopathy may be distinguished from secondary (neurogenic or some other cause) myopathy (1). In fact this difference is more theoretical than real. In veterinary medicine at the moment, it is difficult to say if the muscular fibres are affected primarily or indirectly. According to an histological classification, Kornegay (13) divided myopathies of dogs into inflammatory or degenerative types. On the basis of electromyographical features Chrisman (7) and Sims (20) differentiated among the whole group of muscular lesions, neurogenic from myogenic pathologies calling only the latter "myopathy" (Table 1).

The presence in practice of terms like "contracture" (21,22) or "fibrotic myopathy" (16) to indicate muscular diseases clinically similar to the lesion presently under discussion, the difficulty of classifying it according to the views of other authors and last but not least, the inability to say if it has a myogenic or neurogenic origin are all reasons to prefer the term "myopathy" to define the lesion involving the gracilis and semitendinosus muscles.

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\* Presented by S.C.I.V.A.C.



Table 1: Classification of dog myopathies

| HISTOLOGICAL CLASSIFICATION (KORNEGAY)              |               |                     |          |
|-----------------------------------------------------|---------------|---------------------|----------|
| Inflammatory                                        |               | Degenerative        |          |
| Infective                                           | Non infective | Congenital          | Acquired |
| ELECTROMYOGRAPHICAL CLASSIFICATION (CHRISMAN: SIMS) |               |                     |          |
| Muscular (primary)                                  |               | Nervous (secondary) |          |
| Muscle atrophy                                      |               | Myelopathies        |          |
| Myopathy                                            |               | Radiculopathies     |          |
| Myositis and polymyositis                           |               | Polyneuropathies    |          |
| Myotonia and pseudomyotonia                         |               |                     |          |
| Myastenia gravis                                    |               |                     |          |
| Muscular hypertonicity                              |               |                     |          |
| Muscular dystrophy                                  |               |                     |          |

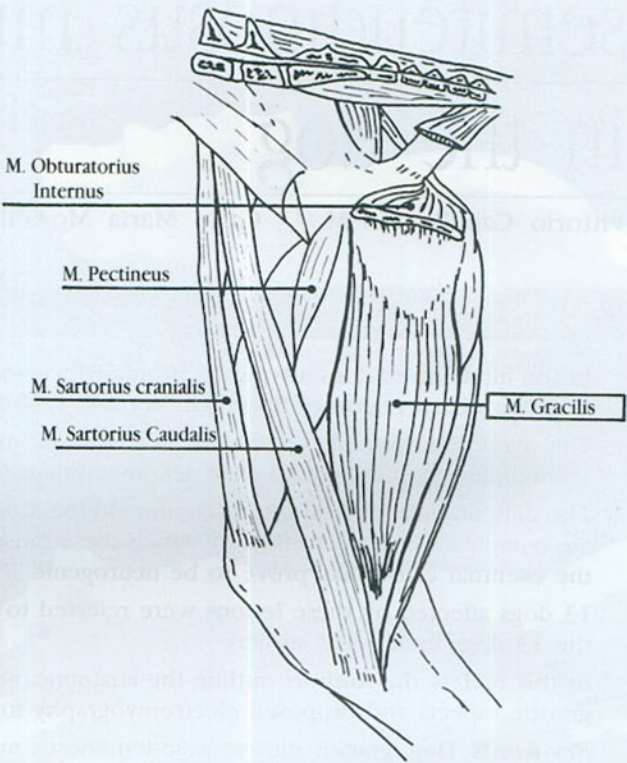


Fig. 1A: Thigh muscles of the dog: medial view (From Barone (3), modified).

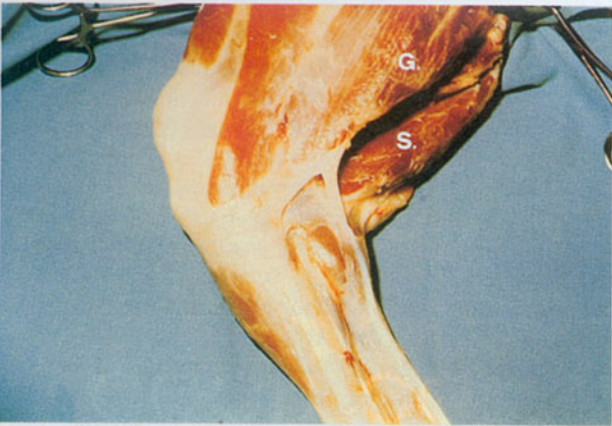


Fig. 1C: Gross post-mortem appearance of hindlimb. Medial view: German Shepberd dog. Note m. gracilis (G.) and m. semitendinosus (S.)

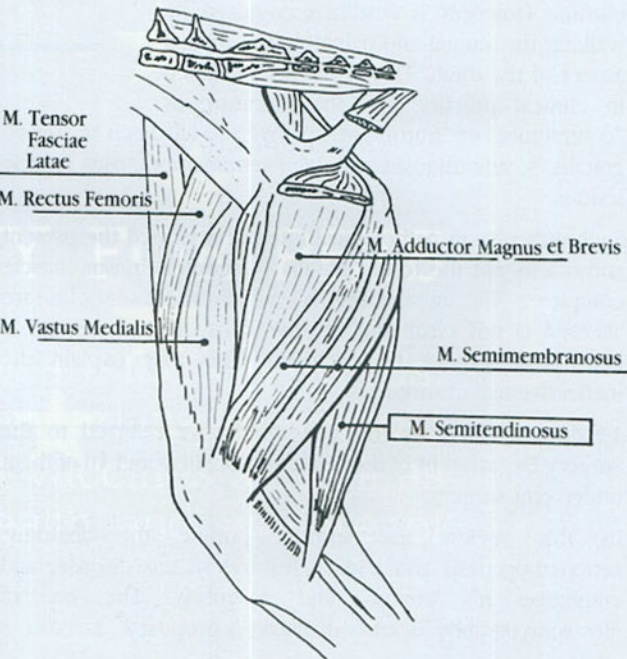


Fig. 1B: Thigh muscles of the dog: medial view after removal of m. gracilis and m. sartorius (From Barone (3), modified).



## ANATOMY

*M. gracilis* (3,5,15,18).

It belongs to the group of adductors of the thigh. It forms a broad muscular sheet which is situated superficially on the caudal portion of the medial surface of the thigh and covers the adductor, semimembranosus and semitendinosus muscles (Figs. 1A,1C).

The gracilis muscle arises from the pelvic symphysis and distally has two insertions; the anterior one ends along the entire length of the cranial border of the tibia within the anterior insertion of the semitendinosus muscle. This aponeurosis is made of two different laminae, the superficial one belonging to the gracilis and the deeper to the semitendinosus muscle (Fig. 2). The aponeurosis also spreads out into the crural fascia in which there is a very distinct bundle inserted into the middle of the medial tibia (Fig. 3). The latter anatomic structure is not mentioned in the literature (3,5,15,18).

From the caudal border of the crural fascia, a well developed reinforcing band (Fig. 4) along with the semitendinosus, gastrocnemius, biceps femoris and flexor digitorum

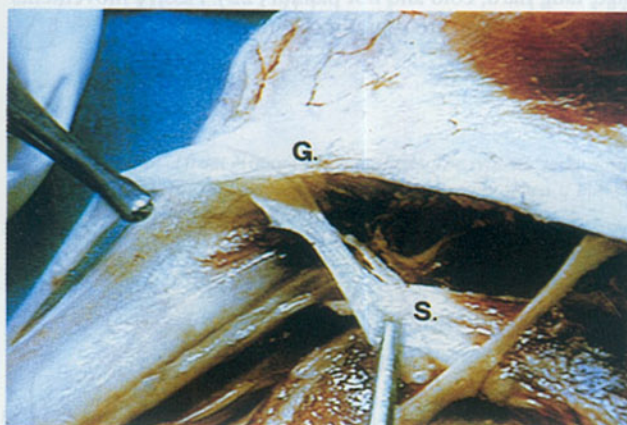


Fig. 2: Close-up appearance of the cranial insertion of *m. gracilis* (G.) and *m. semitendinosus* (S.). Forceps hold the two ends of the aponeuroses.

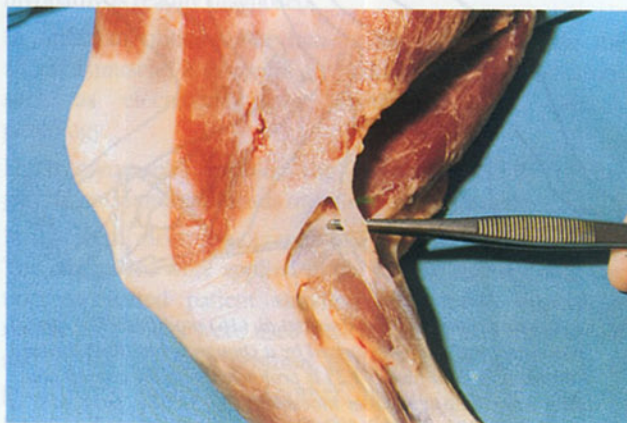


Fig. 4: The caudal insertion of *m. gracilis* is shown.

superficialis muscles forms the chorda magna (6) which inserts into the tuber calcanei.

The gracilis muscle is supplied by branches of the femoral artery, by the saphenous artery and by the obturator nerve.

Besides its main action of adduction of the thigh, it is also an extensor of the hip joint by means of its caudal tendinous insertion.

*M. semitendinosus* (3,5,15,18).

This lies in the caudal part of the thigh and provides its caudal contour (Figs. 1B,1C). It arises on the ischiatic tuberosity and has two distal insertions: cranially by means of a strong, flat tendon which passes under the aponeurosis of the *m. gracilis*, and caudally (Fig. 5) becomes part of the chorda magna (6).

The semitendinosus muscle is supplied by the proximal caudal femoral artery and the ischiatic and tibial nerves.

It is an extensor of the hip and stifle joint.

Several dissections which were performed on German Shepherd dog cadavers revealed that the two muscles insert distally at three different points: cranially on the tibial plate, distally on the medial tibia and caudally on the tuber calcanei as part of the chorda magna.

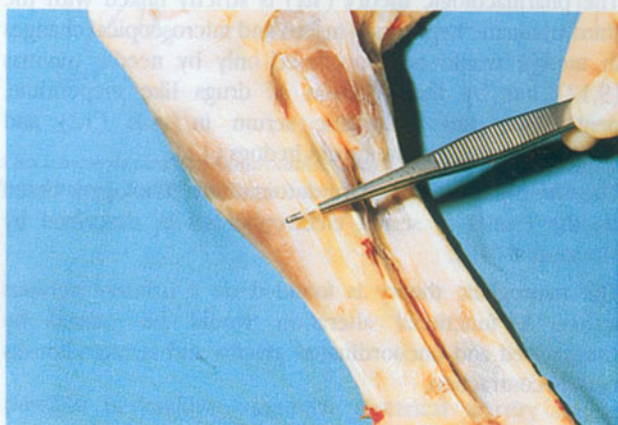


Fig. 3: The cranial insertions of *m. gracilis* and *m. semitendinosus* in the crural fascia, which inserts in the medial surface of the tibia.

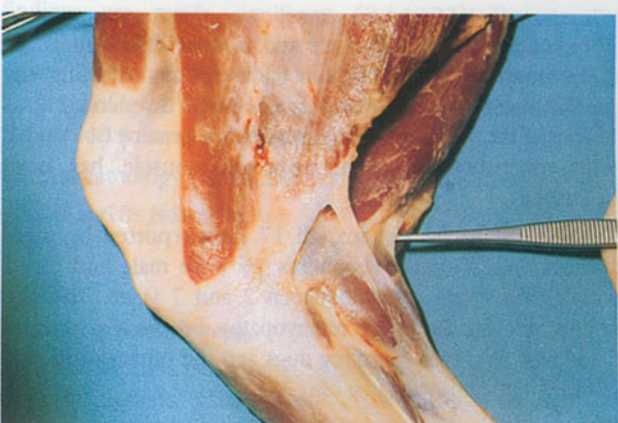


Fig. 5: The caudal insertion of *m. semitendinosus* is shown.



# AETIOPATHOGENESIS

The aetiology and pathogenesis of this muscle lesion have not been completely explained and it is necessary to consider each possible pathogenetic hypothesis. Traumatic, pharmacologic and inflammatory theories may account for the pathogenesis of myopathies.

The traumatic theory has three differing possibilities. The first, argued from the similar aetiology of contracture of infraspinatus muscle (2,19,23) suggests that the cause is an anatomical change following a single acute trauma, either exogenous or endogenous. If blood vessels or nerves are severed, this trauma would determine primary or secondary necrosis of muscle fibres. Necrosis would be followed by fibrosclerotic repair, resulting in a contracture.

Another theory supposes the effect of microtraumas in causing the myopathy. According to Vaughan (22) microtraumas would be caused in dogs with an extremely active life. Extreme mechanical effort would tear muscle fibres, followed by repair and then contracting phenomenon. The sequelae are widespread muscular microhaemorrhages due to fibre ruptures.

The third hypothesis concerns microtraumas due to repeated intramuscular injections, similar to that described in human medicine (9,12).

The pharmacologic theory (12) is strictly linked with the third traumatic hypothesis: macro and microscopical changes in muscle would not be caused only by needle traumas (9,12) but by the injection of drugs like meperidine, pentazocine and antitetanic serum in man (12) and lincomycin, and spectinomycin in dogs (12).

The chronic inflammatory or autoimmune aetiology is based on the results of serum electrophoresis as described by Moore et al (16).

The neurogenic theory is founded on a primary nervous lesion. A functional alteration would be caused by exaggerated and uncoordinated gracilis and semitendinosus muscle contraction.

# INCIDENCE

This condition has been reported in the United Kingdom (22) Sweden (21) and Italy (6) as lesions affecting the gracilis muscle and in the U.S. (16) and Sweden (21) as lesions of the semitendinosus muscle. In Germany (11) a dog with myopathy of semimembranosus muscle has been described.

Referring to breed and sex, all 17 dogs reported by these authors were German Shepherds 16 were male and one a female. Their age varied between 2 and 7 years. This age relates not to the onset of myopathy but to the time of diagnosis. Two were working dogs and the others were pets but they lived a very active life (22).

Out of the 17 reported cases, 14 had a unilateral lesion and 3 bilateral. In none of the dogs were both muscles affected.

# CLINICAL FEATURES

"Gracilis — semitendinosus muscle complex" myopathy is characterized by a pathognomonic lameness of the hindlimb.

The clinical signs may arise in dogs 2-3 years old as well as in those 7-9 years old. As far as their history is concerned, previous muscular or skeletal trauma to the hindlimb was not mentioned by the owner.

The typical movement, or "goose stepping" (16), occurs when the hindlimb is lifted and taken forward in a very peculiar way (Figs. 6, 7) and is observed by watching the dog from the side. This pathognomonic gait, however, is better recognized from behind. When the dog moves its limb forwards it performs a quick movement of hock hyperflexion and outward rotation, while the foot is rotated inwards (16,22). This typical movement has been called a "jerky gait" (22) (Figs. 8, 9) and is emphasised when the dog walks fast or if both hind legs are affected. The "jerky gait" is presumed to be due to abnormal contracture of one or both of the muscles drawing on the hock by means of their tendinous insertions causing hock hyperflexion and outward rotation, which is balanced by inward rotation provided by the tendinous insertions on the medial side of the hindleg.

On the medial and rear aspects of the thigh, it is possible to feel the swelling of the muscle belly which is increased in size, taut, hard, cold and not painful (22). Passive movements of limb abduction are sometimes reduced.

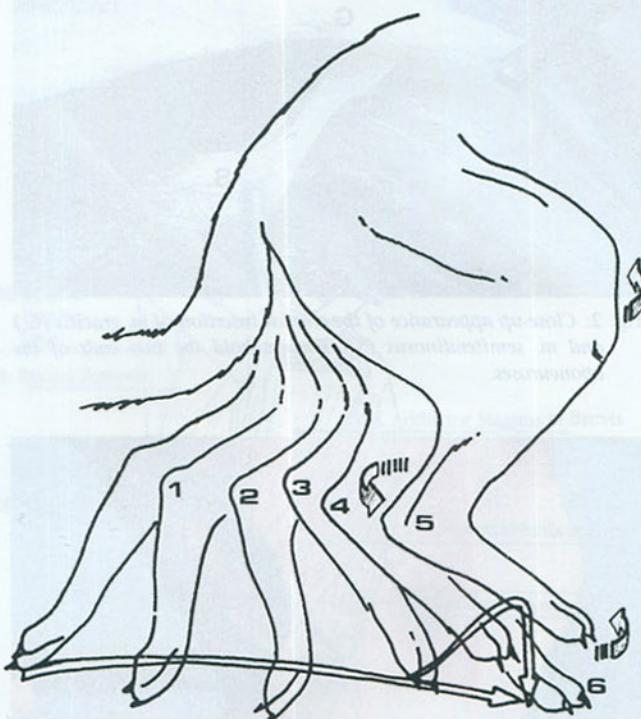


Fig. 6: "Goose stepping" (from Moore et al. (16) modified). The typical "goose stepping" is characterized by a change in hindlimb forward movement; caudal phase (1,2) is prolonged with the cranial phase being completed by a faster movement (3,4,5). The "jerky gait" is represented in 4 and 5 (see Figures 8 and 9), thereafter weight bearing is normal.





Fig. 7: "Goose stepping". Kinetic instant refers to n. 5 of Fig. 6.



Fig. 9: "Jerky gait". Kinetic instant refers to n. 5 of Fig. 8.

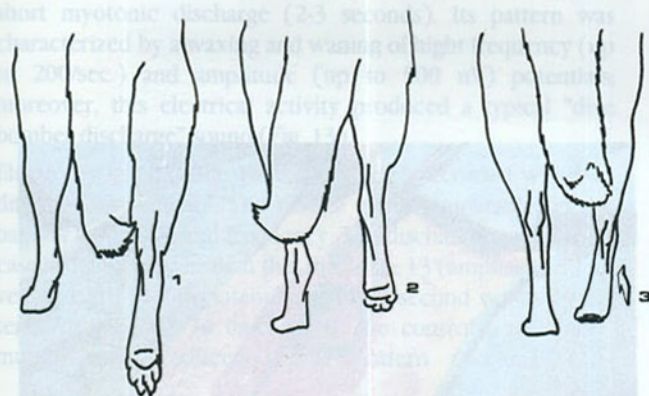
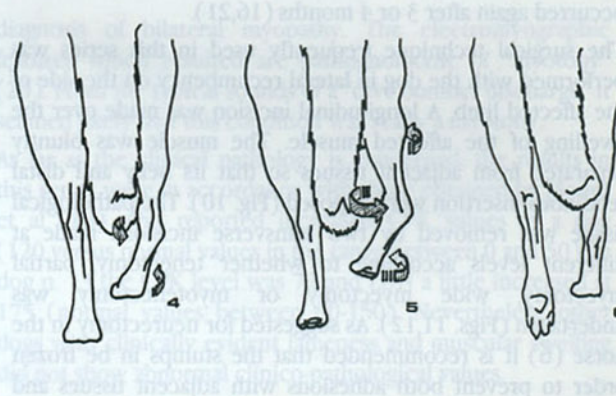


Fig. 8: "Jerky gait" (from Moore (15) modified). The typical "jerky gait" is due to hock hyperflexion and extrarotation, with the stifle and foot inwardly rotated (4,5). The dog performs this jerky movement when it takes the hindlimb forward (see Fig. 6). This is balanced by an opposite movement, so that weight bearing is normal (6).



## DIAGNOSIS

In severe cases, gracilis and/or semitendinosus muscle myopathy is readily diagnosed.

In order to obtain more information and to localise the affected muscle, it is useful to perform other examinations such as electromyography, radiography and clinical-pathology.

Electromyography enables the electrical activity of the gracilis and semitendinosus muscle to be evaluated.

In this study electromyography was carried out on two dogs. The electrodes were applied to the muscle affected with the unpremedicated patient standing on a table. Superficial needle electrodes were used which caused little discomfort and as the standing position is natural for the dog, it is particularly indicated. Another useful position is lateral recumbency because it allows the dog to contract the limb spontaneously by stimulating the foot. In this way it is

possible to evaluate muscular electrical activity during voluntary contraction. It is important to perform electromyography also on normal muscle, with a preference for the contralateral gracilis or semitendinosus.

Radiology is seldom useful for the diagnosis, because the findings are rarely significant (2,4,19,23). Occasionally radiodensity of the muscular belly and/or the distal tendon which inserts on the tibial medial plate may be detected. Such radiological features may however be absent even when the clinical features are very marked.

Clinical pathology (16) can help with the diagnosis especially:

- LDH and its isoenzymes;
- SGOT;
- CPK;
- serum electrophoresis.

If increased, CPK is the most important in its specificity for muscular lesions. LDH is less important because of its presence everywhere in the animal, unless a study referred exclusively to its isoenzymes is performed.



## THERAPY

Treatment of the lesion may be either conservative or surgical.

Distal tendon incision, tenectomy, partial or wide myectomy, and finally myotenyectomy are the surgical techniques which have been described.

In 6 cases of gracilis myopathy, Vaughan (22) performed simple myotomy. The contracture disappeared immediately but in every case lameness recurred from 3 to 5 months after surgery, because of healing of the severed tendon. A second surgical procedure was followed by further relapse.

Later, Moore et al. (16) and Thoren (21) proposed other surgical techniques which either removed part of the muscular belly or excised the entire muscle and its distal tendon. Although a period with complete remission of clinical signs followed surgery, a lameness of lesser or equal degree occurred again after 3 or 4 months (16,21).

The surgical technique frequently used in this series was performed with the dog in lateral recumbency on the side of the affected limb. A longitudinal incision was made over the swelling of the affected muscle. The muscle was bluntly separated from adjacent tissues so that its belly and distal tendinous insertion were exposed (Fig. 10). The pathological tissue was removed by two transverse incisions made at different levels according to whether tenectomy, partial myectomy, wide myectomy or myotenyectomy was undertaken (Figs. 11,12). As suggested for neurectomy in the horse (6) it is recommended that the stumps in be frozen order to prevent both adhesions with adjacent tissues and stump reunion.

Conservative treatment has been advised after unsuccessful results obtained with different surgical techniques. It consists of rest only, without drug administration or use of external appliances. For working dogs, reduction of activity is recommended. Good results of normal activity for 2-3 years are reported.

## PROGNOSIS

Poor functional recovery after surgery or conservative treatment is the most frequent result. Postoperatively there are two different sequelae: in some there is a nearly complete functional recovery soon after surgery, and in others there is early relapse. Most dogs again show the typical lameness from two weeks to six months later. This recurrence is due to rejoining of the muscle stump, and also the extension of the myopathy to adjacent muscles. This reunion is surprising, considering the amount of pathologic muscular tissue which is excised.

The overall prognosis is good because this mild lameness does not compromise the quality of life of the patient. Nevertheless, guard and hunting dogs and those which perform intensive activity have a compromised lifestyle; in these cases, partial or total reduction of their activity is mandatory.

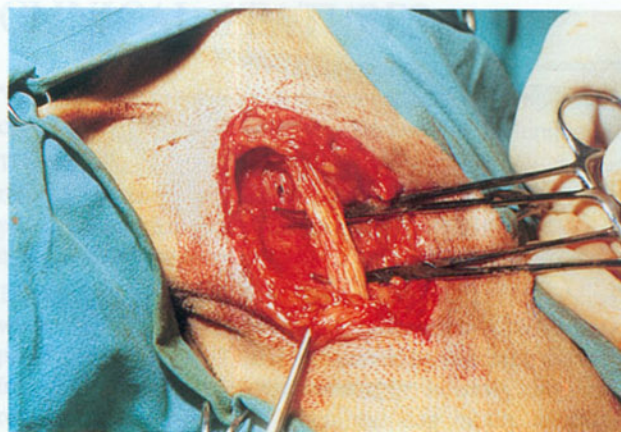


Fig. 10: Case n. 13. Gross appearance of marked fibrotic changes of distal m. semitendinosus.

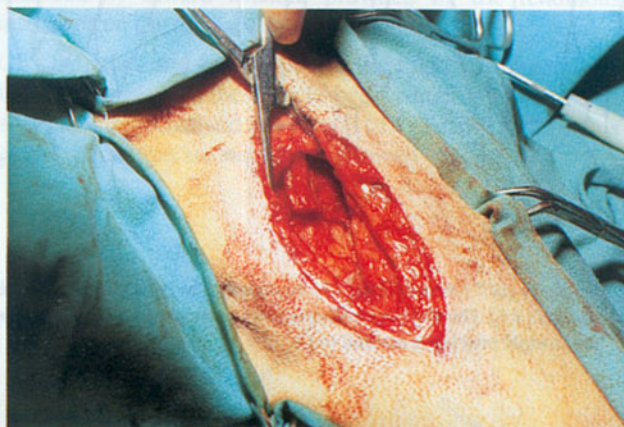


Fig. 11: Case n. 13. The m. semitendinosus proximal stump shown after resection.

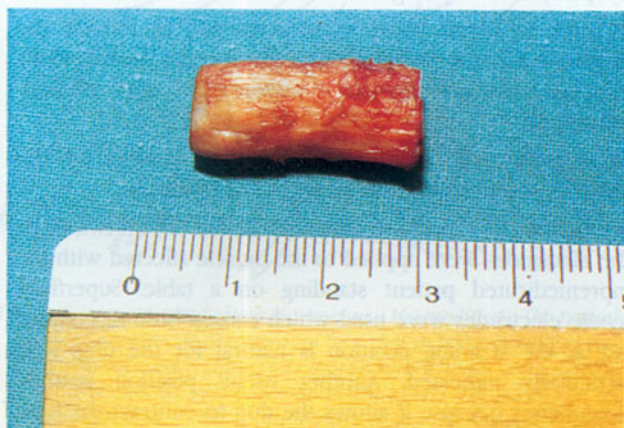


Fig. 12: Case n. 13. The excised pathologic tissue.



## CASE REPORTS

The dogs examined in our study are listed in Table 2. Out of 13 dogs examined during the last 9 years (5 of which were observed between May '88 — April '89) 10 were German Shepherds. Other breeds, standard or large size, were recorded (n. 1,2,3). Age did not seem to be important, male dogs were more affected than females. None of these had a particularly active life. Myopathy was unilateral in 9 (7 right, 2 left) and bilateral in 4 dogs. In one dog (n. 11) both muscles of the same limb were affected; while in another bilateral case (n. 12), the right m. semitendinosus and left m. gracilis were affected.

The dogs which underwent electromyography were shown to be affected on the gracilis muscle bilaterally (n. 10) and right semitendinosus (n. 13). After nervous stimulation, both muscles showed abnormal electrical activity, consisting of a short myotonic discharge (2-3 seconds). Its pattern was characterized by a waxing and waning of high frequency (up to 200/sec.) and amplitude (up to 500  $\mu$ V) potentials; moreover, this electrical activity produced a typical "dive bomber discharge" sound (Fig. 13).

Electromyograms (Fig. 14 and 15) were recorded with two different instruments. The results were comparable on the basis of amplitude and frequency. The discharge registered in case n. 10 was lower than that in case n. 13 (amplitude 50  $\mu$ V versus 120-150; 5-6 potentials/tenth of second versus 10-12/tenth of second). In case n. 10, the controlateral gracilis muscle also produced similar pattern confirming the

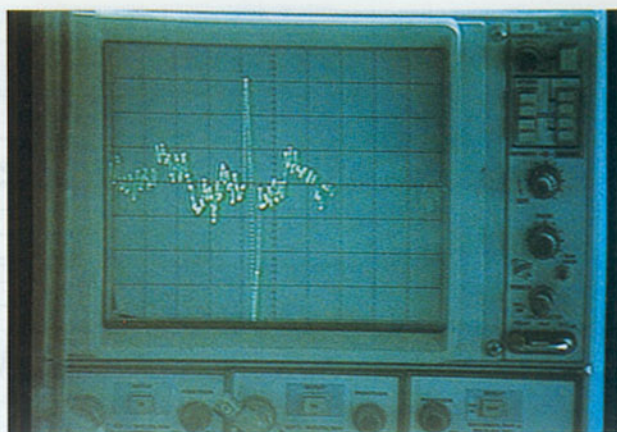


Fig. 13: Case n. 10. Electromyographic examination. Myotonic potential.

diagnosis of bilateral myopathy. The electromyographic features which resulted are pathognomonic of "myotony" (20). From the typical sounds of a "dive bomber discharge" it seemed likely that this condition was really a myotony.

As far as the clinical pathology is concerned, the results in this series were in accordance with those obtained by Moore et al (16) who reported increased CPK values in a dog (120 versus normal values in the range between 0 and 50). In dog n. 13 the CPK level was 72 and LDH a little increased at 175 (normal values between 60-150). Nevertheless, other dogs with clinically evident lameness and muscular swelling did not show abnormal clinico-pathological values.

10 out of 13 dogs underwent surgery. As 3 dogs underwent surgery twice and one dog presented bilateral myopathy, 14 surgical procedures were performed. Two dogs (n. 9 and 10) were treated conservatively and another (n. 12) was lost to follow-up.

Regarding surgical techniques, at first tenotomy alone (case 1) was carried out; then tenectomy (case 2) was chosen. Recurrences suggested a change to myectomy and finally the excision of the entire muscle belly (case 8). This dog exhibited complete remission for a period of six months, then the typical lameness recurred. Surgical techniques performed on the last two dogs produced a positive improvement. Dog n. 12 firstly underwent a total excision of the gracilis muscle together with proximal stump freezing and then, because of extension of the myopathy to the semitendinosus muscle, a second surgical excision. After two months it showed a slight degree of "jerky gait" but less than on initial presentation. Dog n. 13 underwent myotectomy and the leg was bandaged to the thorax in order to separate the two stumps (Fig. 16). After two days, because of haematoma formation at the surgical site and leg oedema, it was necessary to remove the bandaging. The follow up after two weeks showed a complete remission. Two months later, the dog was affected by a coagulopathy, which probably caused post-surgery complications and it was destroyed. At necropsy both the semitendinosus muscle, clinically diagnosed as affected by myopathy and the gracilis muscle were intact. Between the semitendinosus and semimembranosus muscles was found a white and yellow

Table 2: Clinical data for 13 dogs (1982-1989)

| N. | BREED            | SEX | Age (Years) | Date                     | Side           |
|----|------------------|-----|-------------|--------------------------|----------------|
| 1  | Bobtail          | F   | 5           | 27.01.1982               | Right          |
| 2  | Belgian Shepherd | M   | 3           | 27.01.1982<br>04.06.1982 | Left<br>Left   |
| 3  | German Shepherd  | M   | 4           | 08.09.1982               | Right          |
| 4  | German Shepherd  | M   | 9           | 06.06.1983<br>02.08.1983 | Right<br>Right |
| 5  | Boxer            | F   | 3           | 20.02.1984               | Right          |
| 6  | German Shepherd  | F   | 2           | 13.06.1984               | Left           |
| 7  | German Shepherd  | M   | 4           | 15.06.1984               | Right          |
| 8  | German Shepherd  | M   | 7           | 04.07.1986               | Right          |
| 9  | German Shepherd  | M   | 3           | 05.09.1988               | Right          |
| 10 | German Shepherd  | M   | 5           | 13.09.1988               | Right<br>Left  |
| 11 | German Shepherd  | M   | 3           | 08.02.1989<br>11.04.1989 | Right<br>Right |
| 12 | German Shepherd  | F   | 2           | 12.04.1989               | Right<br>Left  |
| 13 | German Shepherd  | F   | 2           | 04.05.1989               | Right          |



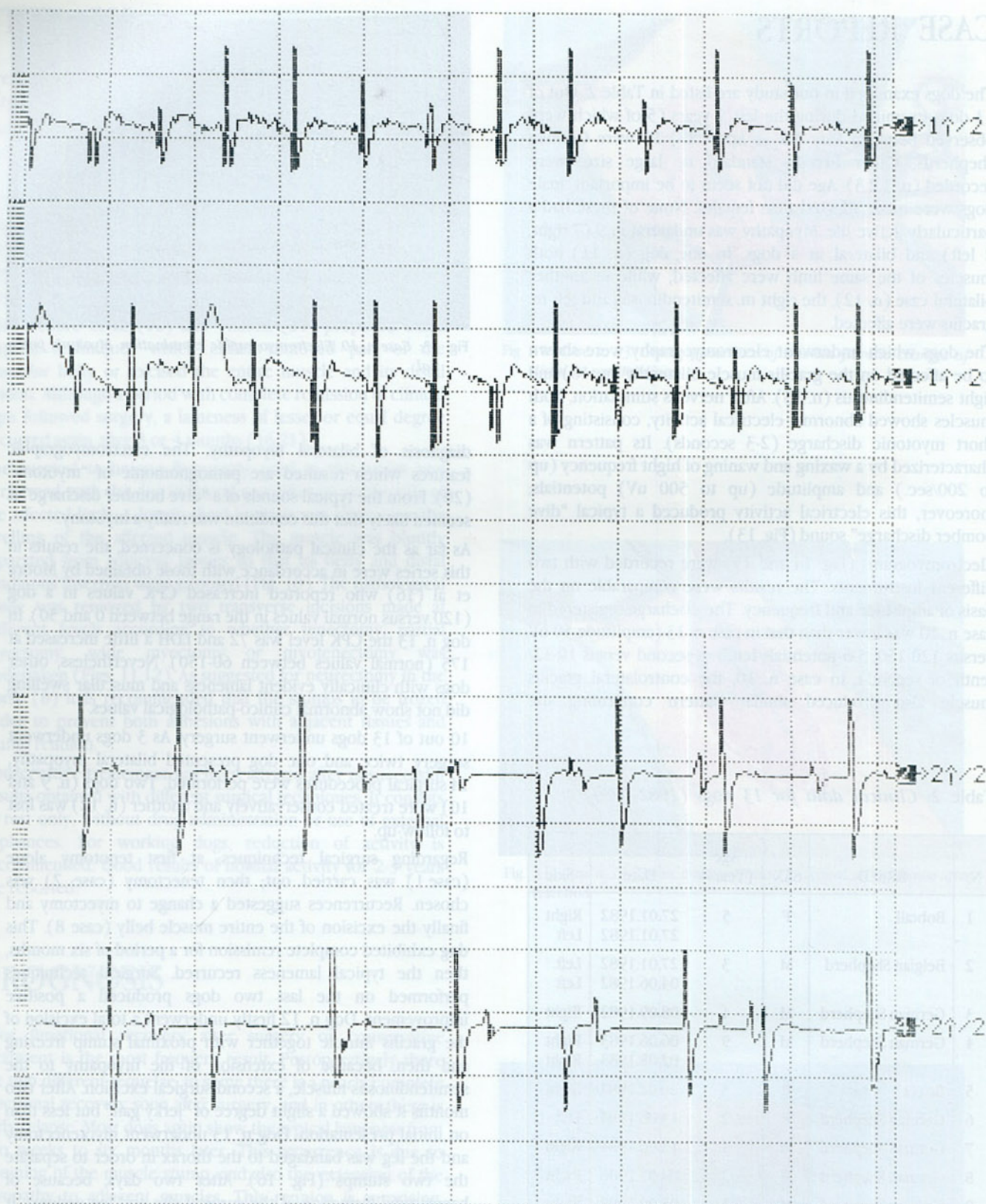


Fig. 14: Case n. 10. Electromyographic patterns. Above: normal motor unit potentials. Below: myotonic discharge (amplitude 50  $\mu$ V, frequency 50-60/sec.).



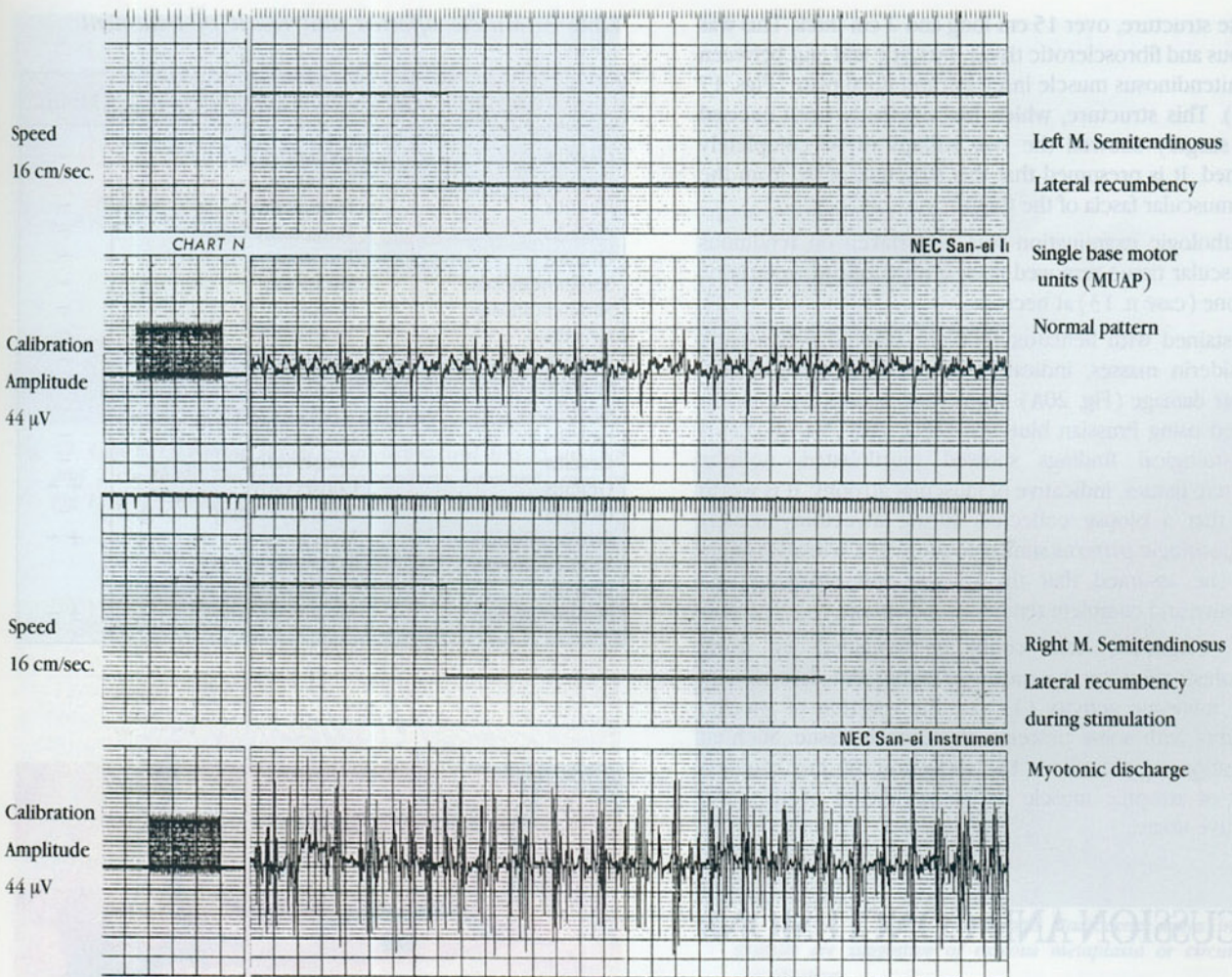


Fig. 15: Case n. 13. Electromyographic patterns. Above: normal motor unit potentials. Below: myotonic discharge (amplitude 120-150  $\mu$ V, frequency 100-120/sec.).



Fig. 16: Case n. 13. Stiff bandaging with the right hindlimb fixed to the thorax.



cord-like structure, over 15 cm long and 2 cm thick. This was tendinous and fibrosclerotic tissue, inelastic and taut between the semitendinosus muscle insertion and tibial plate (Figs. 17 and 18). This structure, which had evidently been excised during surgery showed the two stumps to be completely reattached. It is presumed that this structure arose from the medial muscular fascia of the thigh.

Histopathologic examination was undertaken on tendinous and muscular tissue removed from 9 dogs following surgery, and in one (case n. 13) at necropsy.

Tissue stained with hematoxylin-eosin showed perivascular haemosiderin masses, indicative of previous bleeding and muscular damage (Fig. 20A). Pigmented masses were better evaluated using Prussian blue stain (Fig. 20B). In two cases the histological findings showed plurilobated, nodular connective tissues, indicative of muscular atrophy. It is worth noting that a biopsy collected during myectomy usually shows histologic patterns similar to those of a normal tendon. It may be assumed that the muscle was changing into progressive and complete tendinous metaplasia.

Further diagnostic data could be obtained by using immunohistochemistry, by means of either the detection of ATP-ase myosinic activity (14) or the reaction of specific antisera with some proteins of muscular tissue. Such an investigation would allow the detection of the slightest amount of atrophic muscle in the middle of proliferating connective tissue.

## DISCUSSION AND CONCLUSIONS

In spite of only eight sporadic cases of myopathy being referred to the Department from 1982 to 1987, a further 5 cases were diagnosed within eleven months (May 88-April 89). This apparently increased prevalence is probably due to improved skill in detecting its characteristic features and to better knowledge of the lesion itself. Moreover, it is likely that in the past such lameness, whose aetiology and pathogenesis still remains unknown, has probably been identified as some other pathology.

The aetiopathogenesis of this lesion is open to question. The numerous hypotheses were not proven and the possibility of it being myogenic or neurogenic in origin was not solved. It is important, therefore, to further investigate its electromyographic features, by which it might be possible to explain the aetiology and devise a better therapy.

The clinical diagnosis is obtained by palpation of the affected muscles and by watching the animal from the rear. Electromyography helps to confirm the diagnosis.

Radiographic and clinico-pathologic findings are seldom useful, because negative results are found in dogs with clinically evident myopathy. Radiology can however help in unusual cases (Fig. 21) where myopathy of the gracilis muscle is associated with the presence of radiodense areas, suggestive of osseous metaplasia or circumscribed calcification of muscular tissue.

Xeroradiography could provide more detailed muscular studies.

Table 3: Muscle affected and result of treatment

| MUSCLE         | TREATMENT      | RESULT |
|----------------|----------------|--------|
| Gracilis       | Tenotomy       | -      |
| Gracilis       | Tenotomy       | -      |
| Gracilis       | Tenectomy      | -      |
| Gracilis       | Tenectomy      | -      |
| Gracilis       | Tenotomy       | -      |
| Semitendinosus | Myectomy       | -      |
| Semitendinosus | Myectomy       | -      |
| Gracilis       | Tenectomy      | -      |
| Gracilis       | Tenectomy      | -      |
| Gracilis       | Tenectomy      | -      |
| Gracilis       | Total myectomy | -      |
| Gracilis       | Conservative   | -      |
| Gracilis       | Conservative   | -      |
| Gracilis       | Conservative   | + -    |
| Gracilis       | Total myectomy | -      |
| Semitendinosus | Myectomy       | + -    |
| Semitendinosus |                |        |
| Gracilis       |                |        |
| Semitendinosus | Myotectomy     | + -    |

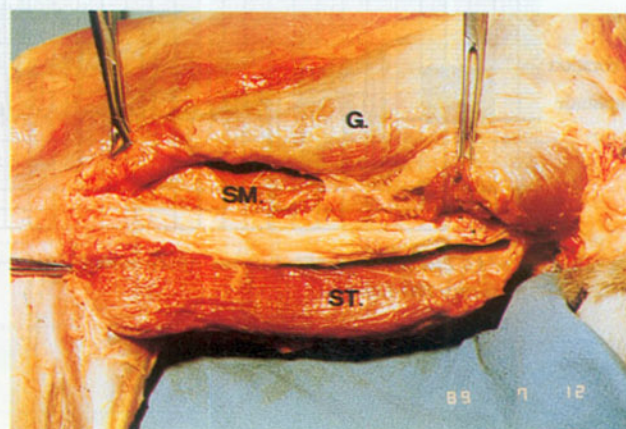


Fig. 17: Case n. 13. Fibrous strand and its connections with m. gracilis (G.), m. semimembranosus (SM.) and m. semitendinosus (ST.) found at necropsy.

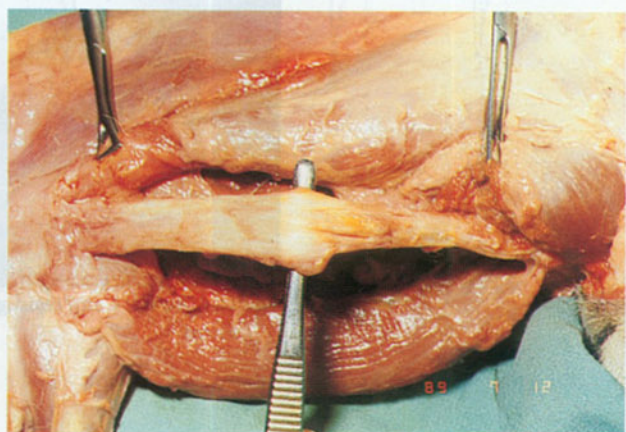


Fig. 18: Case n. 13. The tension of the fibrous strand is evident.



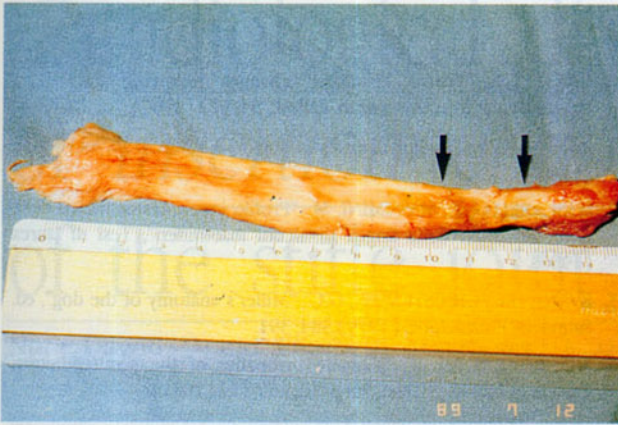
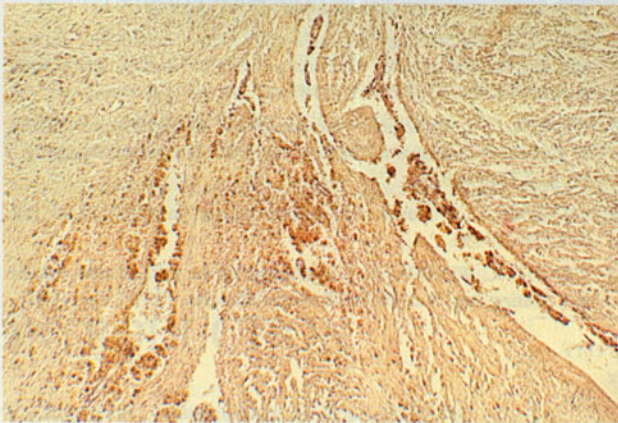
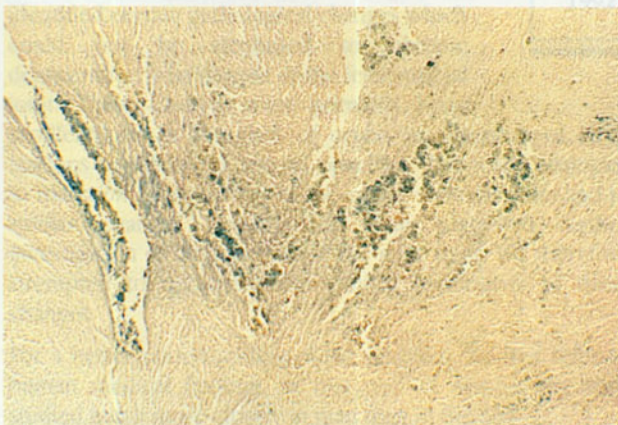


Fig. 19: Case n. 13. Fibrous strand completely excised. It is 15 cm long and 2 cm thick. The tract of myotectomy (arrowed) seen in Fig. 12 shows the two stumps to be completely united.



20a



20b

Fig. 20: Case n. 13. Perivascular hemosiderin pigment indicative of previous haemorrhages. A. Hematoxylin and eosin. B. Prussian blue.

Even if significantly increased, CPK values only indicate a general muscular lesion and are not pathognomonic of this localized myopathy.

Finally histopathology can only confirm clinical diagnosis, during or before surgery. Immunohistochemistry would add further useful information.



Fig. 21: Case n. 1. Lateral radiograph. Radiodense areas in the m. gracilis are suggestive of osseous metaplasia or circumscribed calcification.

The last consideration concerns the failure of both surgical and conservative management. Although even partial reduction of clinical signs could be considered successful, it is conceivable that good long term results could be obtained by performing a selective neurotomy, as used for similar neuromuscular lesions in other species, such as spastic paresis in the calf (6).

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