Acquired muscle contractures in the dog and cat.
A review of the literature and case report

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Summary
Canine and feline muscle contracture is reported to affect several different muscles, is associated with a number of predisposing factors, and a varying prognosis depending upon which muscle is affected. Most patients suffer some form of trauma weeks to months before the contracture is present. The clinical signs include: lameness, pain, weakness, decreased range of motion, a firmness noted throughout the entire muscle, and usually a characteristic gait. Pre-disposing factors for muscle contracture include: compartment syndrome, infection, trauma, repetitive strains, fractures, infectious diseases, immune-mediated diseases, neoplasia, and ischaemia. There does appear to be some breed and age predilection, however, the sex of the animal does not have an appreciable influence. In general, muscle contractures of the forelimb respond better to treatment and carry a better prognosis than muscle contractures of the hindlimb.

Keywords
Muscle contracture, brachialis, myopathy

Introduction
The purpose of this article is to review the literature concerning muscle contractures in the dog and cat, and then to present a case of brachialis muscle contracture in a cat. Most contractures have a history of acute injury or lameness weeks to months before the onset of contracture. The lameness and initial swelling usually subsides with supportive treatment, however, clinical signs of the contracture usually start to present a few weeks later. Clinical signs for most contractures include: lameness, pain, weakness, a firmness noted throughout the affected muscle and a characteristic gait. Reported muscle contractures in the dog and cat include, but are not limited to, most commonly the infraspinatus and quadriceps, and less commonly the supraspinatus, teres minor, sartorius, gracilis, and semitendinosus. Any muscle, however, can theoretically be affected. Compartment syndrome, infection, trauma, repetitive strains, fractures, infectious diseases (20, 22), immune-mediated diseases, ischaemia (22), eosinophilic myositis (20), and neoplasia may all lead to irreversible muscular contraction and fibrosis. Another disease, myositis ossificans, may cause contracture as well. Other reasons for temporary or permanent muscle contracture include: eclampsia, strychnine toxicity, tetanus, spastic paresis and Schiff-Sherrington syndrome (6).

Muscle contracture is defined as, 'The abnormal shortening of muscle tissue, rendering the muscle highly resistant to stretching' (19). A contracture can lead to permanent disability (19). There is a significant difference between muscle contraction and muscle contracture. Contraction refers to the normal physiological process of muscle shortening, resulting in work. Contracture refers to the abnormal pathologic process resulting in fibrosis and permanent damage to a muscle. It is important to remember that a muscle can have a significant amount of scar tissue from prior injury and still not be considered a contracture based on its ability to function. The pathological process of muscle contracture is characterized by replacement of most or all of the entire muscle and/or associated tendon with fibrous connective tissue. This process can take weeks to months and leads to shortening of the affected muscle or muscle group. A muscle contracture can also severely affect the associated joints by fixing them in flexion or extension.

Skeletal muscle tissue cells have a very limited ability for regeneration, and differ somewhat from cardiac muscle cells which are classified as nondividing cells that cannot undergo further mitotic division in the postnatal life, and differing greatly from smooth muscle cells which are classified as quiescent cells that have a moderate capacity for regeneration and can be driven into the G1 mitotic phase of regeneration after damage (18). Repair to damaged muscle tissue begins early in the inflammatory cascade and involves two processes: regeneration of injured tissue by parenchymal cells of the same type, and replacement by connective tissue (fibroplasia) (18). The balance of the two dictates how well new skeletal muscle tissue is produced and how much scar tissue is formed. It appears to be this limitation for skeletal muscle cell regeneration that drives the formation of scar tissue and inevitable fibrosis after skeletal muscle trauma instead of regenerating new cells and replacing the damaged.

Most muscle contractures can be accurately diagnosed by a thorough history and physical examination. A complete chemistry panel and CBC are usually within normal limits, however, creatinine phosphokinase (CK) may be elevated. Radiographs of the affected area and associated joints...
should be obtained. Most radiographs taken are often unrewarding yet they may show a decrease in joint space or increased soft tissue density in the area of the contracture. Ultrasonography has proven to be a useful tool in the diagnosis of muscle contracture and in determining the specific muscle(s) affected (17).

Compartment syndrome is a disease process most commonly seen in humans, however it does occur in dogs and horses (8). The pathology to muscle tissue is caused by an increase in pressure in a confined, inelastic space that leads to muscle inflammation with no room to expand (8). Muscle necrosis will occur when the pressure rises to >30 mm Hg (normal ~2 to 8 mm Hg) in a single compartment with no relief (8). A compartmental pressure >120 mm Hg may lead to complete and irreversible nerve block (2). Causes for an increased pressure within a compartment include: haemorrhage, post-ischaeamic tissue swelling, external pressure (bandage), or intramuscular injection (7). In elastic barriers that pre-dispose to compartment syndrome there are structures such as fascia and bone (8). Four different compartments have been described in the dog: femoral, cranialateral crus, caudal crus and caudal antebrachial (2,8).

It appears that compartment syndrome may play a significant role in the contracture of the muscles of the shoulder joint. If an injury is sustained to the infraspinatus muscle which leads to moderate cellular inflammation, the muscle begins to swell and has no room to expand. Inflammation within the boundaries of the deltoids, scapula and surrounding fascia will increase the compartmental pressure and inhibit venous blood flow initially causing congestion, then arterial blood flow, nervous innervation as well as diminishing the cellular response to damaged tissue. It is the non-forgiving pressure that leads to further cellular damage. The intolerance of cellular swelling causing pressures to rise >30 mm Hg is what seems to lead to necrosis and/or fibrosis of the affected muscles. Early treatment is focused at surgical decompression by fasciotomy, however, if contraction has already occurred, tenectomy or myectomy may be necessary (3).

Muscles and tendons are visco-elastic tissues, meaning that the colder the temperature, the more inelastic and prone to injury they become. A warm-up period before vigorous exercise, in order to increase the temperature of the muscle, will have a protective effect over muscle injury and help to prevent trauma and contracture, especially in cold conditions (7,12).

**Muscle contractures of the forelimb**

**Infraspinatus contracture**

Infraspinatus muscle contracture was first reported in the Dutch literature in 1970 (15) and is one of the two most frequently reported contractures in the dog, along with quadriceps contracture (13,29). Unilateral contraction of the infraspinatus muscle is most common, however, bilateral contractures have also been reported (15,16,22).

Most cases are working or hunting dogs that present with an acute onset of shoulder lameness with or exercise (11,13,15–17,22,26). Acutely, the shoulder is swollen and very painful. The initial trauma usually resolves with rest and/or supportive treatment, however, the affected limb begins to show signs of contracture several days to weeks after the initial trauma (26). Physical examination in the chronic case usually shows a bright, alert dog, without signs of any systemic illness. There is usually a weight bearing lameness of the affected forelimb and a characteristic gait. Palpation and manipulation of the contracted limb does not usually elicit a pain response (11,26). The remaining shoulder muscles, mainly the supraspinatus (26) and deltoid muscles, may show signs of disuse atrophy or be contracted as well (13,15,17,24). A substantial decrease in range of motion of the shoulder joint is appreciated (24). The affected forelimb is usually held with the shoulder in abduction and the elbow in adduction against the thoracic wall (11). The distal forelimb and carpus is externally rotated and held in abduction (16,26). Gait abnormalities usually consist of a weight bearing lameness and circumduction (11,15,17,24,26) of the affected forelimb with a characteristic flip-like action of the paw when placing the foot (16,24,26). When the patient is placed in lateral recumbency with the affected limb up, a true infraspinatus contracture will cause the distal forelimb to remain in an abducted and externally rotated position off the floor and away from the body (9).

Siems et al. reported a series of ultrasonographic findings of two traumatized infraspinatus muscles in the same dog at the same time, one of which resolved without any complications, and the other which resulted in contracture (17). The main ultrasonographic description of the contracted infraspinatus muscle is increased echogenicity when compared to a normal supraspinatus muscle (16,17).

Infraspinatus muscle contracture carries an excellent prognosis with partial tenectomy of the infraspinatus tendon (5,24,26). Lateral or cranialateral approaches to the shoulder both provide good exposure of the infraspinatus tendon (14,15,26). Complete transection of the infraspinatus tendon with partial tenectomy is required to restore motion in the affected limb and most often proves curative with an immediate release of contracture and return to function of the shoulder joint (16,21,24). It is common to find adhesions to the infraspinatus tendon and joint capsule during surgery, however, once dissected away they seldom cause further problems (14,24). Post-operative bandages need not be applied and physiotherapy should start when sutures are removed to prevent the formation of further adhesions (24,26). In one case, simultaneous tenectomy was performed on both the supraspinatus and infraspinatus muscles in the same shoulder of a dog (24). The dog had immediate return of function of the shoulder joint without joint instability or any other problems post-operatively (24).

The exact cause of infraspinatus muscle contracture is not known, however, trauma appears to be the most likely cause. Steiss postulated that an acute compartment syndrome, preventing the infraspinatus muscle from being able to handle an increased compartmental pressure and cellular swelling during heavy exercise, may be the reason why the infraspinatus muscle is most commonly affected in the shoulder (12).
**Supraspinatus contracture**

Contracture of the supraspinatus muscle occurs less frequently than infraspinatus contracture, however, the two share similar clinical signs and have occurred simultaneously. In 1979, Vaughan reviewed 15 cases of shoulder muscle contracture, only one of which turned out to be due to the supraspinatus muscle (24). Mineralization within the supraspinatus tendon is usually an incidental finding near the greater tubercle of the humerus, however, it can cause mechanical irritation of the biceps tendon sheath (3). Overuse and repetitive trauma are the most likely causes of mineralization (8).

Supraspinatus muscle contracture usually has the same history and physical findings as infraspinatus contracture, however, tangential radiographic views taken of the intertubercular groove of the shoulder joint should help to identify the affected tendon if mineralization is present.

Surgical treatment is recommended and consists of complete transaction of the supraspinatus tendon with partial tenectomy and release of all adhesions via a craniolateral or lateral approach. There is usually an immediate return to function. Physiotherapy should be initiated one to two weeks after surgery. Supraspinatus muscle contractures usually carry an excellent prognosis with the proper surgical treatment (24).

**Teres minor myopathy**

In 1997, Bruce et al. reported teres minor myopathy as a cause of lameness in a dog (28). A 5-year-old Labrador Retriever presented with a chronic, non-progressive lameness of eight months duration. The lameness persisted despite medical treatment with antibiotics, and non-steroidal and steroidal therapy. Physical examination showed a 6/10 lameness on the affected forelimb with suprascapular muscle atrophy. The teres minor muscle was painful and band-like on palpation. The shoulder was painful and decreased flexion in a sagittal plane was noted, however full flexion was possible when the distal humerus was supinated. Ultrasonography confirmed an increased echogenicity within the painful band palpated. A craniolateral approach to the shoulder was performed and the teres minor was found to be abnormal in appearance. Complete myectomy of the teres minor muscle and separation of adhesions resulted in immediate release of contracture and normal range of motion in the shoulder joint. Histopathology did not show any evidence of fibrosis, however, focal areas of inflammation were found with mononuclear cell infiltrate. The dog underwent passive physiotherapy starting two weeks after surgery and was found to be clinically sound six months later (28).

**Muscle contractures of the hindlimb**

**Quadriceps contracture**

Quadriceps muscle contracture occurs less commonly today due to better surgical technique and a wider knowledge of preventative measures (2). The normal range of motion in the stifle joint is from 180° at full extension to 30° at full flexion. During the contracted state in chronic cases the stifle may be fixed at full extension with no ability to flex. The condition primarily affects young dogs with distal femoral fractures, however, it has affected cats suffering from mid diaphyseal fractures of the femur (2, 27). Quadriceps contracture can also occur with severe quadriiceps trauma, as a congenital condition (2, 22, 24, 26, 27), and has been reported as a complication from toxoplasmosis (2, 22). There does not appear to be a breed or sex predilection. Most cases are due to prolonged immobilization of the affected limb in extension after a distal femoral fracture, inadequate fracture stabilization, excessive tissue trauma during surgery, or any combination thereof (20, 22, 29). Stifle joint extension, fibroplasia of the quadriceps muscles, and degenerative changes within the stifle joint develop as a result of fibrous adhesions between the quadriceps muscle and the fracture site (21, 29). Severe chronic cases have led to genu recurvatum (2, 22, 24) or ankylosis of the stifle joint (2, 8).

Clinical signs include a stiff stilted gait and carrying the limb in full extension (2, 22). The hip may be somewhat flexed, the stifle fixed in extension, and the hock extended with a limited range of motion (2). The quadriceps muscles in the chronic case are atrophied, non-painful and palpate as thick cords (2, 22, 29). The stifle joint can be fully extended, however, it may only be able to flex 30° initially. Flexion may gradually decrease until the stifle is rigidly fixed in extension, and may appear to be hyper-extended (genu recurvatum) (22, 29). Gait abnormalities include: an inability to completely extend the hip, inability to flex the stifle and hock, and a stiff stilted gait with circumduction (2). Chronic cases will form adhesions between the distal femur and the stifle joint capsule, further limiting flexion ability (29).

The diagnosis of quadriceps muscle contracture can be made by a thorough history and physical examination alone. The blood chemistry and CBC are usually within normal limits, however, an elevated CK may be present. Radiographs of the hind limb may show a stifle in extension with an old or healing femoral fracture. Early osteophyte formation and decreased joint space in the stifle joint may also be seen (2). The long bones of the affected limb may show signs of disuse osteoporosis depending on the length of time the limb has been in a contracted state (2). The cranial thigh muscles appear more radiopaque than the surrounding musculature and smaller than normal. Ultrasonography of the affected muscles will show an increased echogenicity throughout the entire quadriceps group.

This condition is better prevented because treatment often leads to poor results, particularly once the condition is well established. Rigid fracture stabilization and early return to function are the most important key elements in preventing the condition. Medical management is only effective in early cases where a 90°-90° flexion splint may be applicable (7, 29). Advanced cases of contracture carry a poor prognosis and most commonly result in a non-functional leg. Treatment regimes for chronic cases include: resection or dissection of fibrotic tissues, restabilization of femoral fractures, quadriceps myoplasty and physical therapy (7, 22).

Surgical treatment for quadriceps contracture focuses on restoring limb function by releasing all of the fibrous adhesions be-
between the distal femur and joint capsule, as well as between the quadriceps muscles and femur (22). Autogenous fat grafts, ophthalmic Gelfilm and silastic sheeting have all been used to prevent adhesions between the femur and quadriceps muscles (20, 21, 27). In some cases, the femur of the affected limb is shorter in length when compared to that of the other limb (2). If stifle range of motion is not satisfactory after adhesions are released (cannot be flexed greater than 40°), lengthening of the quadriceps will be required (29). Z-plasty of the quadriceps muscles is the preferred method for lengthening, however, release of the muscular origins can also be performed (22). In order to prevent post-operative recurrence of contracture, preventative measures must be taken. Transarticular external fixators that maintain the stifle in a passive state of flexion, a 90°-90° flexion splint, or intense physical therapy are all adequate options (29). More recently, successful management of quadriceps contracture has been reported in a cat after release of adhesions, placement of an autogenous fat graft between the quadriceps and femur, and the use of a dynamic flexion apparatus in combination with intense physiotherapy (27).

The prognosis for quadriceps contracture depends on the severity of disease before surgery. If an adequate range of motion was achieved and good post-operative care is taken, the prognosis is fair given that contracture recurrence is still a possibility. It is important to remember that surgical treatment is aimed at a favorable return to function for the limb and that a normal range of motion may never be achieved (21). One source quoted surgical success achieved if 50 to 75 percent use of the affected limb is returned (22). If surgical intervention is not satisfactory, arthrodesis or amputation may be necessary (21).

**Semitendinosus contracture**

Semitendinosus muscle contracture most commonly affects highly active young male German Shepherd Dogs (8). It can occur by itself, or with gracilis muscle contracture (24). The exact cause is unknown, however, repetitive trauma, immune mediated disease, fibrotic myopathy or secondary to neuropathy and gracilis muscle contracture have all been postulated as causes (2, 8).

Usually there is a history of lameness in the affected limb prior to contracture. Physical examination findings are generally unre- markable except for a thickened fibrous band in the area of the semitendinosus muscle (8). Gait and posture abnormalities include: a shortened stride, a rapid medial rotation of the paw, external rotation of the hock and internal rotation of the stifle during mid stride (2, 29). The blood chemistry and CBC are generally unremarkable, however, an elevation in CK may be seen. Radiographs of the affected hind limb are generally unrewarding but will help to rule out other causes of lameness.

Treatment of semitendinosus muscle contracture consists of supportive therapy during the initial phase of injury, however, surgical resection of the contracted muscle is not recommended because the condition usually returns in two to four months (2, 8, 24, 29). The animals are not in pain and can function, somewhat normally, with an abnormal gait.

**Gracilis contracture**

Gracilis muscle contracture generally affects highly active German Shepherd Dogs between the ages of three and seven years (8, 12, 26). Gracilis muscle rupture, also known as ‘dropped thigh’ (20, 21), is the most commonly diagnosed muscle injury of racing greyhounds and is more common with the right hindlimb (22, 23, 26).

Gracilis muscle contracture can be effectively diagnosed with a thorough history and physical examination. Physical exam may be normal, except for a firm mass in the area of the gracilis muscle. History consists of a single or repeated strain injury to the gracilis muscle (8) with a gait abnormality developing afterwards. Gait abnormalities usually start abruptly and progress over a period of six weeks to months (8, 26), at which time it becomes static. During locomotion, the affected leg is raised in a jerky fashion with the hock hyperflexed and rotated laterally, the metatarsus is rotated medially, and the overall stride is shortened (8, 26).

Current treatment is not recommended. Montgomery and Fitch reported a series of 19 cases treated by medical means only, as well as medical and surgical means, with every case recurring within a few months (8). Treatments attempted included prednisilone, azathioprine, surgery, and drug-surgery combinations (8). Affected dogs do remain active despite their lameness and appear not to be in pain. Myectomy of the entire gracilis muscle still has a poor prognosis because the gait abnormality will still return within three to five months due to semitendinosus involvement (26). Fibrotic myopathy may play a role in gracilis muscle contracture (12).

**Sartorius contracture**

Sartorius muscle contracture in the dog was reported in an adult male German Shepherd Dog in 1994 by Lobetti and Hill (10). The dog presented with a chronic, non-painful, non-weight bearing lameness of the right hind limb. The dog had shown signs of acute lameness in the right hind limb four weeks prior to the onset of contracture, however, the lameness did not respond to non-steroidal therapy or corticosteroids. Physical examination was within normal limits except for the right hind limb abnormality where a hard band-like structure was palpated in the cranio-lateral aspect of the thigh. The posture at rest consisted of the limb in slight adduction, with flexion of the hip and stifle and a slight inward rotation of the paw. The stifle could not be fully extended and pain was elicited when it was attempted. Radiographs showed moderate laxity of the hip joint without periosteal reactions. Mild periosteal new bone formation was seen along the shaft of the ilium. A band of soft tissue with increased density was noted in the area of the sartorius muscle.

Treatment consisted of a medial approach to the thigh directly over the thickened band. The sartorius was severed and all adhesions broken down. Immediate relief of the contracture was felt. Pathology reported the muscle as showing extensive fibroblastic hypertrophy with an increased amount of collagen. Replacement fibrosis and muscle contracture was diagnosed. The dog showed immediate recovery post-operatively. Two-month follow-up showed a clinically sound dog (Table 1).
**Muscle contracture**

Other diseases affecting muscle function

Myositis ossificans

Myositis ossificans is a generalized or localized progressive muscle disease of large breed, middle aged active dogs, characterized by non-neoplastic heterotopic bone formation of fibrous connective tissue and skeletal muscle mainly near the hip joints that forms most likely secondary to trauma and/or haemorrhage (3, 7, 20, 22). It has been known to occur in the cervical region, the shoulder and the quadriceps (7). Patients with von Willebrand’s disease or other coagulopathies may be pre-disposed (3, 8). The animals may or may not exhibit pain, however, if present, pain is usually exhibited after exercise. The clinical signs include: chronic lameness, muscle atrophy, and a swollen muscle mass. The lesions may be inflammatory in origin, or may be the result of trauma that forms a haematoma and later undergoes calcification (20, 22). Calcification occurs two to four weeks after the initial trauma and may appear radiographically as a well-defined, calcified mass with a radiolucent centre (20, 22).

Progressive ossifying fibrodysplasia is a similar disease seen in young to middle aged cats that affects the musculature predominantly on the neck and back. Those clinical signs consist of: mild gait stiffness, enlargement of the proximal limb muscles, pain and a decreased range of motion. Progression may take two weeks to several months for disability (3).

An accurate diagnosis can usually be made with a thorough history, physical examination, radiographs and histopathology. The patient should be screened for thyroid disease, von Willebrand’s disease, as well as other coagulopathies since haemorrhage appears to be a pre-disposing factor. General chemistry and CBC are generally within normal limits, however, an elevated CK may be found.

Medical management may be beneficial in mild cases, however, local injections of steroids are usually unrewarding (7). Care should be taken when giving anti-inflammatories for pain and inflammation because it may exacerbate bleeding tendencies (3). Treatment of more severe lesions is aimed at complete excision of the affected musculature (2, 8). Incomplete excision may provide palliative relief and correct any lameness, however, recurrence is a problem (2, 8). Amputation may be necessary in some cases (2). An effective treatment in cats for progressive ossifying fibrodysplasia (4) currently does not exist.

Ischaemia

Muscle contractures related to ischaemia occur in humans as well as in animals. Volkman’s Ischaemic Contracture (22, 25) describes a condition in humans suffering from flexion deformities of the wrist and fingers. The underlying cause is due to a decrease in blood flow via the brachial artery, resulting in enough blood flow to prevent necrosis, however, insufficient to support function (25). The same clinical signs can be seen in animals. Compartment Syndrome can also elevate ischaemia by increasing the pressure within a muscle to the point where it causes vascular occlusion. Immediate treatment is focused on re-establishing circulation in either case (25).

**Case report**

A 13-year-old, indoor/outdoor, neutered Domestic Shorthair cat was referred for a chronic, non-painful weight bearing lameness of the right forelimb. The owner reported that there was neither a history of injury nor of trauma. Physical examination was within normal limits, except for an inability to extend the right forelimb past a 90º angle. The cat stood with the right elbow in

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**Table 1  Overview of muscle contractures in the dog and cat.**

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Species</th>
<th>Breed</th>
<th>Predisposing factors, Pathogenesis</th>
<th>Treatment</th>
<th>Outcome</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infraspinatus</td>
<td>Canine</td>
<td>Working</td>
<td>Compartment syndrome, Trauma</td>
<td>Partial tenectomy, Adhesion release</td>
<td>Immediate return to function</td>
<td>Excellent</td>
</tr>
<tr>
<td>Supraspinatus</td>
<td>Canine</td>
<td>Working</td>
<td>Compartment syndrome, Trauma</td>
<td>Partial tenectomy, Adhesion release</td>
<td>Immediate return to function</td>
<td>Excellent</td>
</tr>
<tr>
<td>Teres minor</td>
<td>Canine</td>
<td>Labrador Retriever</td>
<td>Idiopathic, Trauma</td>
<td>Complete myectomy, Adhesion release</td>
<td>Immediate return to function</td>
<td>Good</td>
</tr>
<tr>
<td>Brochialis</td>
<td>Feline</td>
<td>Domestic shorthair</td>
<td>Idiopathic, Fibrotic myopathy</td>
<td>Partial myectomy, Adhesion release</td>
<td>Immediate return to function</td>
<td>Fair</td>
</tr>
<tr>
<td>Quadriceps</td>
<td>Canine</td>
<td>Any</td>
<td>Young animals with distal femoral fractures</td>
<td>Preventative, Surgery as needed</td>
<td>Partial return to function</td>
<td>Guarded</td>
</tr>
<tr>
<td>Semitendinosus</td>
<td>Canine</td>
<td>German Shepherd</td>
<td>Highly active young males, Repetitive trauma, Fibrotic myopathy</td>
<td>Supportive, Surgery not recommended</td>
<td>Use of limb with long term gait abnormalities</td>
<td>Poor</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>Canine</td>
<td>German Shepherd, Greyhound</td>
<td>Highly active, Trauma, Fibrotic myopathy</td>
<td>No treatment effective</td>
<td>Use of limb with long term gait abnormalities</td>
<td>Poor</td>
</tr>
<tr>
<td>Sartorius</td>
<td>Canine</td>
<td>German Shepherd</td>
<td>Idiopathic</td>
<td>Partial myectomy, Adhesion release</td>
<td>Immediate return to function</td>
<td>Fair</td>
</tr>
</tbody>
</table>

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flexion, supporting some weight. A shortened stride was the only gait abnormality present. The right shoulder palpated normally and had a normal range of motion. A taut, non-painful muscle band was palpated over the cranio-lateral aspect of the brachium. The range of motion in the elbow was limited; full flexion to 90° at full extension. General chemistry and CBC were within normal limits. FeLV and FIV tests were both negative. Radiographs of both forelimbs were normal except for a very mild increase in soft tissue density in the cranio-lateral aspect of the right brachium. Ultrasonography was not used. A tentative diagnosis of brachialis muscle contracture was diagnosed and the cat was taken to surgery for treatment. A cranio-lateral approach to the brachium over the brachialis muscle was used and provided good exposure. The brachialis muscle was normal in colour, yet smaller than normal and very firm. The muscle and inserting tendon were sectioned and immediately provided relief of the contracture with a normal range of motion. Routine closure was performed and recovery was uneventful. After recovery, a normal gait and range of motion was present. The cat was discharged with orders to begin physiotherapy 10 days after surgery and was reported to be doing well a few weeks later.

Histopathology revealed replacement of skeletal muscle with dense fibrous connective tissue. Mild, diffuse edema was present and the normal architecture of the tendon was obliterated by bundles of fibroblasts and collagen arranged perpendicularly to angioblasts. There was also a mild infiltrate of lymphocytes, plasma cells and neutrophils. A histopathological diagnosis of muscle degeneration fibrosis and granulation tissue was made. Indications of neoplasia, infectious organisms, autoimmune or immune mediated disease were not seen.

An examination eight months later showed that the contracture had once again returned to the same degree at which it was first seen. A similar fibrotic band involving the brachialis muscle was detected on palpation. A second surgery was performed and release of the brachialis muscle and tendon once again provided immediate postoperative relief of the contracture. Histopathology of the sectioned muscle and tendon showed a normal architecture obliterated by bundles of fibroblasts and collagen arranged perpendicularly to angioblasts. There was a mild to moderate infiltrate of lymphocytes, plasma cells and neutrophils. A histopathological diagnosis of granulation tissue was made but neither neoplasia nor evidence of infectious organisms were seen. The cat was once again discharged and ordered to begin physiotherapy 10 days post-operatively. A single injection of triamcinolone (Vetalog, Fort Dodge, IO, USA) was given intramuscularly at 2.2 mg/kg to help prevent further adhesions. The owner was contacted five months later and reported the cat to be doing well with normal range of motion and was not lame.

This case most likely represents a muscle contracture secondary to trauma, chronic irritation, or fibrotic myopathy. At the time of the last follow-up, the cat was reported to be doing well, was not lame, and possessed normal range of motion of the limb. This case is interesting in that it shows a muscle contracture not yet reported in the cat.

**Discussion**

Acquired muscle contractures in the dog and cat are diseases that share similar history and clinical signs, yet differ in treatment and prognosis with different muscles affected. There is usually a history of trauma causing an acute lameness and painful swelling that subsides with supportive care, however, specific gait abnormalities associated with each contracture appear days to weeks later. Most animals do not show any signs of systemic illness either on physical examination or serum chemistry and CBC. Radiographs of the affected limb are often un rewarding, however, they may show a decrease in joint space that the muscle spans, as well as an increased soft tissue density in the area of the affected muscle. The use of ultrasonography in diagnosing muscle contractures does seem to produce consistent results in finding an increase in echogenicity of the affected muscle.

Compartment syndrome may play a critical role in the development of muscle contractures affecting the shoulder joint.

Treatment of muscle contractures depends on which muscle is affected. Initial medical management for lameness upon first occurrence of the trauma consists of rest and supportive treatment with non-steroidal or steroid therapy. Currently, surgical treatment is only recommended for the treatment of teres minor myopathy, as well as contractures of the: supraspinatus, infraspinatus, brachialis and quadriceps. Surgical release of the supraspinatus, infraspinatus teres minor and brachialis muscles carry much better prognoses than surgical correction of the quadriceps, gracilis and semitendinosus.

Also, current treatment recommendations for contractures affecting the gracilis and semitendinosus muscles remain strictly medical. Supportive care and rest with initial injury are the best treatments early in the condition. Most animals can live with the chronic gait abnormalities, however, if it becomes a chronic problem, amputation of the affected limb is the recommended salvage procedure.

**References**


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