Suprascapular nerve decompression for treatment of neuropathy in a bucking bull

Courtney Griffin and Cathleen A Mochal-King DVM, MS, DACVS

From the Department of Clinical Sciences (Mochal) and the Department of Pathobiology and Population Medicine, College of Veterinary Medicine, Mississippi State University, P.O. Box 6100, Mississippi State, MS 39762

Supported by the Department of Clinical Sciences and Department of Pathobiology and Population Medicine, Mississippi State University, College of Veterinary Medicine

Correspondence: Cathleen Mochal-King, Department of Clinical Sciences, Mississippi State University, Mississippi State, MS 39762

Tel: (662) 541-1979

Fax: (662) 662-0243

Email: mochal@cvm.msstate.edu

Case Description: A 3-year old, 639-kg American bucking bull presented for a 4-day history of right forelimb lameness after sustaining an injury to the right shoulder exiting the cattle chute.

Clinical Findings: Upon evaluation, a 10 x 10 cm soft-tissue swelling was present over the right shoulder. Ultrasound findings indicated contour of the spine of the scapula, bicipital bursa, bicipital tendon, and greater tubercle of the humerus were within normal limits. The swelling revealed a hematoma overlying the distal scapula. No external wounds, palpable joint effusion or swellings were noted on examination of the distal limb. The bull developed atrophy of the supraspinatus and infraspinatus muscles with a gait change to the characteristic “sweeny appearance” with his scapulohumeral joint abducting laterally. Electromyography demonstrated decreased innervation to the supraspinatus and infraspinatus muscles consistent with a diagnosis of suprascapular nerve paralysis.

Treatment and Outcome: The suprascapular nerve was surgically decompressed with corresponding resection of the scapula and regional administration of 40 mg dexamethasone prior to closing. The bull was discharged 5 days following surgery. No lameness was present at discharge. He was restricted to a stall or small pen for 6 weeks. At 14-day recheck there was moderate incisional swelling, but no residual lameness. Four months following surgery the bull returned to bucking with improving musculature. At 12 month follow-up the bull is still in performance.

Clinical Relevance: Suprascapular nerve decompression can improve suprascapular nerve function, muscle atrophy, and gait.

Keywords: Suprascapular neuropathy, Electromyogram, Sweeny, Lameness, Muscle atrophy

Case Description:

A 3-year old, 639-kg American bucking bull presented for a 4-day history of right forelimb lameness. The owner of the bull reported that he was initially non-weight bearing lame after sustaining an injury to the right shoulder exiting the cattle chute during a rodeo. The lameness improved to weight bearing 24 hours after the injury, but was consistently present in the right forelimb. Upon presentation the bull exhibited a grade III/V lameness in the right forelimb that progressively worsened to a grade IV/V lameness during evaluation. A large 10 x 10 cm focal area of soft-tissue swelling was apparent over the right shoulder, caudal and proximal to the greater tubercle of the humerus.

Clinical Findings:

The bull was not amenable to physical examination and was restrained with a hydraulic squeeze and tilt table and sedated with 20 mg xylazine[a] (0.05 mg/kg IV) and 10 mg acepromazine[b] (0.08 mg/kg IV) administered intravenously via the tail vein. An ultrasound examination of the right scapulohumeral joint, scapula, and bicipital bursa was performed with a 7.5 MHz linear probe. Ultrasound findings indicated contour of the spine of the scapula, bicipital bursa, bicipital tendon, and greater tubercle of the humerus were within normal limits. The large soft-tissue swelling in the shoulder region revealed a hypoechoic and edematous triceps with a hematoma present overlying the distal scapula. The bull was tilted into lateral recumbency. His claws and distal limbs were thoroughly evaluated and ruled out as the source of his lameness. No external wounds, palpable joint effusion or swellings were noted on the examination of the distal limb. It was determined that general anesthesia would be necessary to facilitate radiographs of the right shoulder and the bull was hospitalized.

The bull exhibited very agitated and aggressive behavior, he was moved to a stall and placed on acepromazine[b] (0.08 mg/kg PO q12h). Anti-inflammatory and cold therapy treatments were initiated and the bull was allowed to acclimate to the hospital. The bull was treated with meloxicam[c] (0.5 mg/kg PO q24h for 3 days, then continued at 0.5 mg/kg PO q48h). The bull was restrained in a squeeze stall twice daily for 20 minutes for hydrotherapy treatments of the right shoulder region. He responded positively to anti-inflammatories and hydrotherapy over the next 48 hours; swelling decreased significantly and the right forelimb lameness became intermittent. Based on this initial improvement, radiographic evaluation of the right shoulder was postponed.

Four days after initiating medical management (8 days post-injury), the bull was no longer improving and he began to exhibit noticeable atrophy of the right supraspinatus and infraspinatus muscles. His right forelimb lameness was still intermittent, however, his gait changed dramatically to the characteristic “sweeny appearance” with his scapulohumeral joint abducting laterally with each step. Radiographic evaluation and electromyography (EMG) were elected at this time. The bull was withheld from food for 24 hours prior to anesthesia.

The bull was anesthetized using premedication with xylazine[a] (0.05 mg/kg IV) and butorphanol[d] (0.025 mg/kg IV), induction with ketamine hydrochloride[e] (2.2 mg/kg IV) and maintenance with isoflurane in oxygen.

Due to the bull’s size and body mass complete radiographic evaluation of the scapula could not be obtained. Radiographic imaging of the scapulohumeral joint, greater tubercle of the humerus and proximal humeral diaphysis demonstrated no evidence of fractures.

Electromyography was performed on the supraspinatus, infraspinatus, biceps, lateral and middle triceps, extensor carpi radialis and flexor carpi ulnaris. Ground, reference, and recording electrodes were placed and each muscle group was sampled multiple times. Both the

supraspinatus and infraspinatus muscles had obvious signs of muscle atrophy as evidenced by varying degrees of coarse fibrillation potentials with occasional positive sharp waves (Figure 1). These findings were considered specific for pathologic spontaneous activity due to the patient being anesthetized. The recording electrodes placed in the biceps, lateral and middle triceps, extensor carpi radialis, and flexor carpi ulnaris demonstrated short insertion potentials followed by electrical silence, characteristic of a normal muscle response.

A stimulation electrode placed near the suprascapular nerve delivered pulses, resulting in some contraction of the supraspinatus and infraspinatus muscles. Recordings were consistent with polyphasic motor unit action potential (MUAP), indicating some attempts at reinnervation. EMG findings were consistent with a diagnosis of suprascapular nerve paralysis. The bull recovered uneventfully. Following diagnostics, surgical intervention was recommended and the bull underwent suprascapular nerve decompression.

Treatment and Outcome:

Feed was withheld for 48 hours and water for 12 hours before surgery. Flunixin meglumine[f] (1.1 mg/kg IV) was administered for its analgesic and anti-inflammatory effects prior to surgery. Florfenicol[g] (20 mg/kg IM) was administered as the pre-operative antibiotic. The bull was anesthetized and placed in left lateral recumbency. The region was clipped free of hair and sterilely prepped. A 30-cm curvilinear incision was initiated over the spine of the scapula and directed craniodistally towards the greater tubercle of the humerus. The incision was deepened to transect the cutaneous trunci muscle. There was a large blackcurrant like hematoma overlying the spine of the scapula. The hematoma was debrided until the fascia covering the spine of the scapula could be incised. The brachiocephalicus muscle was elevated from the spine of the scapula with a 12mm wide flat periosteal elevator and retracted cranially with hand-held

retractors. The suprascapular neurovascular bundle was identified and carefully dissected free from the scapula and surrounding fascia with a small periosteal elevator. Once the neurovascular bundle was elevated from the scapula, the cranial margin of the scapula was rasped smooth. The neurovascular bundle and brachiocephalicus muscle were replaced. A 3.5 inch 18-gauge spinal needle was placed through the brachiocephalicus muscle overlying the nerve and its location was confirmed with palpation. The fascia was closed with 1-polyglactin 910. Prior to closing the superficial layers the region was medicated with 40 mg of dexamethasone SP[h] via the pre-placed spinal needle. The remaining surgical incision was closed routinely. While recovering from general anesthesia, the bull experienced a large volume of regurgitation.

Four days post-operation he became febrile (104.0°F) and exhibited bilateral muco-purulent nasal discharge. He was treated with florfenicol[g] (40 mg/kg SC) for suspected aspiration pneumonia.

The muscle atrophy was very apparent when standing and combined with the hair removal at surgery (Figure 2). The degree of muscle loss did not change for the remainder of the bull's hospitalization period. Moderate swelling of the incision was present 48 hours following surgery.

The bull recovered from the aspiration pneumonia. He was discharged 5 days after surgery with instructions to return in 14 days for suture removal. No lameness was present at discharge. It was advised that the bull be restricted to a stall or small pen for the next 6 weeks. He was continued on meloxicam every other day for 14 days. The owners were informed of a recommended slaughter withdrawal of 21 days from the last dose of Meloxicam administered. He returned in 14 days for suture removal and although there was still swelling present at the distal aspect of the incision, he exhibited no residual lameness. Four months following surgery

the bull returned to bucking with the owner reporting that the muscle atrophy had improved significantly. At 12-month follow-up the bull travels well and is still able to perform competitively, but does exhibit a minor decline in performance if asked to compete consecutive weekends.

Discussion:

Injury to the suprascapular nerve occurs commonly in the equine patient, but is not well documented in the bovine species.[5] Only 2 cases of bovine suprascapular neuropathy have been reported in the literature. The first occurred secondary to Streptococcal meningoradiculitis in a cow[10]; treatment was not attempted. The second case occurred in a 1.5-year old Angus bull treated with counter irritant with no reported outcome.[3] Damage to the suprascapular nerve most commonly occurs as a result of direct trauma and may result in paralysis and subsequently atrophy of the supraspinatus and infraspinatus muscles. Lateral stability of the shoulder joint is compromised, resulting in a characteristic gait deficit, commonly referred to as “Sweeny” in horses.[1,9] Medical and surgical management have both provided successful case outcomes, however surgical decompression resulted in faster return to athletic function and improved cosmetic outcomes.[8]

The anatomical features of the suprascapular nerve, particularly its reflection around the cranial border of the scapula, make it predisposed to chronic inflammation and acute inflammation secondary to traumatic injury.[7] Arising from the 6^{th} and 7^{th} cervical spinal cord segments, the suprascapular nerve innervates the supraspinatus and infraspinatus muscles via the brachial plexus. Accompanying the suprascapular artery, it passes between the subscapularis and supraspinatus muscles before its reflection around the cranial border of the scapula. At this site of reflection, a small tendinous band extends over the nerve.[6] Damage to the suprascapular nerve

occurs less commonly in ruminants because anatomically the nerve lies deeper within the musculature.[3] Although not demonstrated in the bovine species, histopathologic evaluation of the suprascapular nerve in 14 horses revealed evidence of a chronic neuropathy at the site of reflection in animals that were not clinical for suprascapular nerve damage at the time of death.[7]

A systematic approach to lameness evaluation is important in working up these patients, especially in those cases that do not display significant atrophy of the supraspinatus and infraspinatus muscles at the time of presentation. Typically, atrophy of these muscle bodies and subsequent prominence of the scapular spine is not documented until 7-10 days after injury.[6] History of direct trauma in the shoulder region is helpful, although damage to C6-C7 spinal cord segments and injury to the brachial plexus should also be considered in light of clinical signs.[5] As 90% of bovine lameness can be localized to the foot, it is important to rule out sole abscesses, which may result in significant lameness.[4] Other important rule outs include bicipital bursitis, fracture, scapulohumeral joint luxation, and septic arthritis.[5,6]

Careful evaluation of the distal limb and claw is necessary to rule out foot conditions. Radiographic evaluation of the limb will facilitate fracture diagnosis and evaluation of the joint spaces. Spontaneous activity of the suprascapular nerve on electromyographic evaluation of the supraspinatus and infraspinatus muscles 5-7 days after injury is confirmatory for a diagnosis of suprascapular nerve paralysis. Careful evaluation of additional muscles of the limb should be considered to distinguish suprascapular nerve injury from injury to the brachial plexus.[6]

Medical management recommended for horses consists of strict stall confinement until resolution of lateral instability of the shoulder joint-the characteristic, circumferential gait deficit. This is typically followed with 2-4 months of pasture confinement before returning to work.

Resolution of the lameness is achieved in most cases, however mean time to resolution ranged from 2-12 months.[8,9]

Surgical decompression of the suprascapular nerve has been well described in the equine patient. A scapular notch resection, or suprascapular nerve release, involves a subtotal ostectomy at the cranial border of the scapula at the level of the scapular notch. The nerve is freed from the overlying tendinous band and gently retracted before an osteotome or wire saw (both methods have been described) facilitates removal of a small section of bone. The suprascapular nerve is then released and allowed to traverse the newly notched out section of bone, minimizing entrapment and subsequent compression.[1,2] Post-operative therapy routinely included non-steroidal anti-inflammatories and stall rest and results are good with 80% of patients in one study[2] and 90% of patients in a second study attaining normal muscle function post operatively.

Clinical Relevance:

The bull represented in this case report demonstrated the classic appearance of suprascapular neuropathy with subsequent muscle atrophy and lameness associated with the injury. The lameness improved with medical management but did not resolve completely until surgical treatment. The bull returned to his intended use 4 months following surgery. In conclusion, suprascapular nerve release was successful in a 3-year-old American bucking bull.

Footnotes

a. Xylazine, Akorn, Inc., Decatur, Illinois, USA
b. Acepromazine maleate, Henry Schein Animal Health, Dublin, Ohio, USA
c. Meloxicam, Unichem Laboratories. LTD. Pilerne Ind. Estate. Pilerne, Bardez, Goa, India
d. Butorphanol, Fort Dodge Animal Health, a division of Wyeth, Pfizer Inc, New York, New York, USA
e. Ketamine, Boehringer Ingelheim Vetmedica, Inc., St. Joseph, Missouri, USA

f. Flunixin meglumine, Intervet Inc., Merck & Co. Inc. Whitehouse Station, New Jersey, USA
g. Flurofenicol, Intervet Inc., Merck & Co. Inc. Summit, New Jersey, USA
h. Dexamethosone-SP, Bimeda-MTC Animal Health Inc. Cambridge, Ontario, Canada

Figure Legend

Figure 1: Figure 1 depicts varying degrees of coarse fibrillation potentials (FP) and positive sharp waves (PSW) consistent with spontaneous pathologic activity of the supraspinatus and infraspinatus muscles obtained via electromyography. These findings indicate damage to the suprascapular nerve and would be consistent with a diagnosis of “Sweeny.”

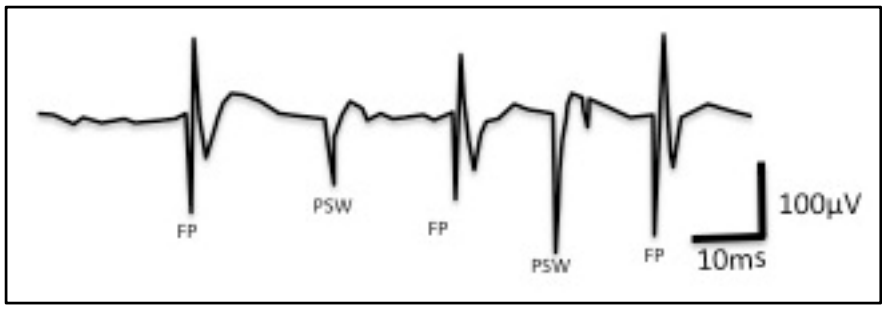

Figure 2: Image A, demonstrates the lower, slightly rolled outward appearance of the right shoulder. Image B, depicts the loss of muscle present over the scapula as apparent from the dipped appearance and lack of rounding on the abaxial surface of the scapula. Image C, further demonstrates the loss of muscling, as the raised incision at the greater tubercle of the humerus is significantly elevated when compared to the more proximal scapular region.

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