Gait abnormalities caused by selective anesthesia of the suprascapular nerve in horses

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Objective—To assess gait abnormalities associated with selective anesthesia of the suprascapular nerve (SSN) achieved by use of perineural catheterization and thereby determine the function of that nerve as it relates to gait in horses.

Animals—3 adult horses with no preexisting clinically apparent lameness at a walk.

Procedure—Each horse was anesthetized; the right SSN was exposed surgically for placement of a perineural catheter to permit delivery of 1 mL of 2% mepivacaine hydrochloride. Six hours after recovery from anesthesia, each horse was videotaped while walking (50-step data acquisition period) before and after administration of mepivacaine. Videotapes were reviewed and the proportion of abnormal steps before and after selective SSN anesthesia was assessed. A step was considered abnormal if a marked amount of scapulohumeral joint instability (ie, lateral luxation of the proximal portion of the humerus) was observed during the weight-bearing phase of the stride.

Results—Clinically apparent gait dysfunction was detected in all 3 horses following perineural administration of the local anesthetic agent. Anesthesia of the SSN resulted in scapulohumeral joint instability as evidenced by consistent lateral excursion of the shoulder region during the weight-bearing phase of gait at a walk. The proportion of abnormal steps before and after SSN anesthesia was significantly different in all 3 horses.

Conclusions and Clinical Relevance—These data support the role of the SSN in shoulder joint stability in horses and define SSN dysfunction as 1 mechanism by which the syndrome and gait dysfunction clinically referred to as sweeny may develop. (*Am J Vet Res* 2006;67:834–836)

The clinical syndrome known as sweeny is characterized by instability of the shoulder joint and atrophy of the supraspinatus and infraspinatus muscles. The condition is reported to be the result of trauma-induced SSN dysfunction.[1] The development of atrophy of the supraspinatus and infraspinatus muscles as a consequence of denervation supports this concept. It has been reported[2] that experimental transection of the SSN in an adult horse caused atrophy of the supraspinatus musculature but no gait abnormality. This implicates brachial plexus dysfunction as a cause of gait abnormality.[2] Surgical management of sweeny involves decompression of the SSN by scapular notch resection with or without suprascapular ligament transection and neurolysis.[3,4] Results of previous studies[3,4] indicate favorable responses to both conservative[5] and surgical treatments.

To our knowledge, no scientific study has been performed to define the clinical effects of transitory SSN anesthesia in horses. The purpose of the study reported here was to assess gait abnormalities associated with selective anesthesia of the SSN achieved via perineural catheterization and thereby determine the function of that nerve as it relates to gait in horses. We hypothesized that selective anesthesia of the SSN would result in clinically apparent shoulder joint instability.

Abbreviations

SSN	Suprascapular nerve

Materials and Methods

Animals—Three healthy adult horses (1 mare and 2 stallions), acquired from a non-University independent provider, were selected for use in the study. The horses weighed 395 to 467 kg (mean weight, 426 kg). All horses were evaluated for existing gait abnormalities, and no preexisting clinical lameness at a walk was detected. The experimental protocol for this project was approved by the Institutional Animal Care and Use Committee at Oklahoma State University.

Surgery—The day before surgery, each horse was physically examined; a venous blood sample was collected for assessment of PCV and total plasma protein and BUN concentrations. Eight hours prior to surgery, food was withheld from the horses but free access to water was permitted. Immediately before surgery, the right shoulder region of each horse was clipped and penicillin G potassium (22,000 U/kg, IV, [repeated after 6 hours]) and phenylbutazone (4.4 mg/kg, IV, once) were administered. Anesthetic premedication consisting of xylazine hydrochloride (0.44 mg/kg, IV) and butorphanol tartrate (0.02 mg/kg, IV) was administered. Anesthesia was induced by use of diazepam (0.1 mg/kg, IV) and ketamine hydrochloride (2.2 mg/kg, IV). After orotracheal intubation, anesthesia was maintained by use of sevoflurane vapor delivered via positive-pressure ventilation. Each horse was positioned in left lateral recumbency, and the right shoulder region was aseptically prepared. A 14-cm skin incision was made 1 cm cranial and parallel to the scapular spine. The incision was centered about the distal extent of the scapular spine. Surgical dissection advanced to the supraspinatus muscle, which was then divided to permit

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exposure of the SSN along the dorsal margin of the scapular neck. A 16-gauge (1.7-mm-diameter), 20.3-cm-long, flexible polyurethane catheter[a] was positioned with the tip of the catheter between the nerve and the scapula. The catheter was anchored and secured by use of absorbable sutures into the deep fascia of the supraspinatus muscle in the surgical wound. Catheter security and patency were confirmed by gentle traction and flush of the catheter with sterile saline (0.9% NaCl) solution, respectively. Closure was performed in multiple layers by use of synthetic absorbable material. The exposed injection extension portion of the catheter exited from the most dorsal aspect of the wound and was secured to the skin adjacent to the surgical wound. The surgical wound and catheter were protected during anesthetic recovery by use of stent bandage and an adhesive incisional drape.

Data collection—Data acquisition consisted of video documentation initiated 6 hours following recovery from anesthesia. Baseline control data were collected as the horses were walked by hand in a straight line at a controlled rate of 1.40 to 1.45 m/s. Videotape footage of each horse was collected before chemical denervation. Anesthesia of the SSN was achieved by use of 1 mL (20 mg) of 2% mepivacaine hydrochloride delivered via perineural catheterization. Ten minutes after the injection, experimental data were obtained and videotape footage recorded. The video data collected were compiled and randomized to avoid bias and were reviewed by 2 investigators (DVD and HWJ) who were unaware of the treatment condition for each horse. Each step was characterized and recorded as normal or abnormal. A step was considered abnormal if a marked amount of shoulder instability, as evidenced by lateral luxation of the proximal portion of the humerus, was observed during the weight-bearing phase of the stride. The number of abnormal steps in a 50-step data acquisition period was determined.

Statistical analysis—The proportion of abnormal steps before and after the chemical denervation was compared for each horse with a Fisher exact test[6] (performed by use of computer software[b]). Statistical significance was determined at a value of $P < 0.05$.

Results

Before the horses were anesthetized for purposes of this study, a physical examination was performed and PCV and total plasma protein and BUN concentrations were evaluated; all findings were within reference limits for all 3 horses. No baseline lameness or gait abnormalities attributable to surgery or catheter placement were detected in any of the horses prior to data collection.

For all 3 horses, 50 consecutive abnormal steps (as indicated by marked clinically apparent shoulder instability) were observed during the data acquisition sessions following initiation of the anesthetic blockade. The proportion of abnormal steps before and after the chemical denervation was significantly ($P < 0.001$) different for each of the 3 horses; similarly, analysis of the combined data for all 3 horses revealed a significant ($P < 0.001$) difference between the proportion of abnormal steps before and after chemical denervation. Physical examination of the horses at 24 and 48 hours and 2 weeks after the experimental procedure revealed little observable discomfort and no permanent neurologic dysfunction.

Discussion

These data support the role of the SSN in provision of shoulder joint stability and define the role of the SSN as 1 mechanism involved in the etiopathogenesis of the clinical syndrome referred to as sweeny. However, these data are not consistent with findings of a previous study[c] in equids, which indicated that no shoulder instability resulted from transection of the SSN. The authors would like to emphasize that the previous study[c] obtained inconsistent results after performing SSN neurectomy on 1 horse and 2 ponies. Two of the 3 animals (the horse and first pony) had slight lameness postoperatively. Muscle atrophy was inconsistent; the horse and the second pony had marked atrophy by 10 days and the first pony had slight atrophy at 14 days after surgery. Atrophy was detected in the supraspinatus muscle only in the horse and both the supraspinatus and infraspinatus muscles in first pony, and the affected musculature was unspecified in the second pony. This inconsistent pattern of muscle atrophy suggests accessory innervation of these muscle groups as a result of biological variation or incomplete transection. Slight shoulder instability developed in the second pony that was not reported to be lame postoperatively. Therefore, results of SSN neurectomy were highly variable among those 3 experimental animals.

Differences between our data and findings of the previous study[c] could be explained by variability in anatomic sites of surgical transection and local anesthetic blockade. Also, biological or anatomic variation in the innervation of the muscles involved and the degree of surgery-induced pain following neurectomy could have resulted in lameness and affected results. Lameness in the forelimb that underwent surgery could have potentially masked shoulder joint instability if the horse was incompletely loading or guarding the limb.

The data obtained in the present study were consistent and reproducible. Experimental limitations included diffusion of the local anesthetic agent, catheter migration, and the small number of experimental animals. Anesthetic diffusion was minimized by use of a small volume of solution (1 mL) and a short interval (10 minutes) between administration of the agent and posttreatment evaluation. Also, the anatomic arrangement of the nerve and position of catheter would require that the agent diffuse around the cranial aspect of the scapula bone to affect centrally located neurons. A period of 6 hours elapsed before initiation of the experiment to ensure that no residual gait abnormalities attributable to surgery or anesthesia were clinically detectable. This time frame also decreased the likelihood of catheter-related problems including neural damage, excessive inflammation, and loss of patency, migration, or removal of the device by the animal. Theoretically, catheter migration was a potential complication. However, it is the authors' opinion that catheter migration did not occur. We believe this because of the suture technique used to secure the catheter. Catheter security was assessed intraoperatively by placing the catheter under traction and was deemed appropriate at that time in all horses. The number of horses used in the present study was small (n = 3); however, the absolute consistency of the results obviated further investigation and the use of additional experimental animals. The results substanti-

ate the role of the SSN in development of the abnormal gait observed in horses with sweeny.

a. Central venous catheterization set, Arrow International Inc, Reading, Pa.
b. PROC FREQ in SAS, version 8.2, SAS Institute Inc, Cary, NC.
c. Dyson S. *The differential diagnosis of shoulder lameness in the horse*. Diploma of Fellowship of the Royal College of Veterinary Surgeons thesis, Royal College of Veterinary Surgeons, Newmarket, UK, 1986.

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