GAIT ABNORMALITIES CAUSED BY SELECTIVE ANESTHESIA OF THE SUPRASCAPULAR NERVE IN HORSES

By

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Submitted to the Faculty of the
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in partial fulfillment of
the requirements for
the Degree of
MASTER OF SCIENCE
May, 2006

GAIT ABNORMALITIES CAUSED BY SELECTIVE ANESTHESIA OF THE SUPRASCAPULAR NERVE IN HORSES

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ACKNOWLEDGEMENTS

I would like to extend my sincere appreciation to Oklahoma State University for the financial tuition assistance which allowed me the opportunity to complete this degree. I would also like to thank the Research Advisory Committee for funding of this research.

I would like to acknowledge my graduate advisor and surgical residency program director, Dr. Hank Jann not only for his intellectual and physical commitments to this project but also for his undaunted encouragement and tutelage throughout my residency.

I would like to thank the other members of my graduate committee, Drs. Jerry Ritchey and Todd Holbrook. They have both been of great assistance on many different levels during my studies. I am proud to consider them both personal friends.

I would like to acknowledge Ms. Diana Moffeit for all that she has done to "keep me on track" with adherence to university paperwork, deadlines and requirements. Without her assistance the dual task of surgical resident / graduate student would be infeasible.

Lastly, I would like to thank all of the OSU staff and final year veterinary students who assisted with the surgical and data acquisition phases of this work for their efforts and cooperation.

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CHAPTER I

INTRODUCTION

Suprascapular nerve injury or Sweeny is a condition which affects horses of all ages, sizes and breeds. The hallmark indicator of this condition is atrophy of the spinatus muscles of the scapula. Most often this develops with an attendant gait deficit which is characterized by a lateral excursion of the shoulder joint during the load-bearing phase of ambulation. This deficit is commonly described as “shoulder slip”. In most instances it is the result of blunt trauma or supposed stretching of the nerve fibers as they course over the cranial aspect of the scapular neck. The nerves intimate association with the scapular bone and relatively superficial location anatomically render it vulnerable to injury.

CHAPTER II

REVIEW OF LITERATURE

Description

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 traumatically induced suprascapular nerve (SSN) dysfunction. This can result from blunt trauma, sharp transection or stretching of the SSN inducing neuronal damage. The condition can affect any age, breed or sex of equid.[1,2] The development of atrophy of the supraspinatus and infraspinatus muscles as a consequence of denervation supports this concept. Denervation atrophy generally becomes clinically apparent 10 to 14 days following injury.

Anatomy

The anatomical arrangement of the equine shoulder is similar to other quadrupeds. The shoulder, or scapulohumeral, joint is comprised of the distal articulation of the glenoid of the scapula bone with the proximal humeral head. The overlying musculature is responsible for the stabilization of the spheroidal joint. Although rotational movement is theoretically possible, the movements of the joint are normally restricted to forward hinge motion during ambulation. The tendons of the muscles about the shoulder are responsible for stabilization of the joint and in turn prevent excessive transverse movement of the shoulder (*Figure 1*).[3]

The neural origins of the SSN are cervical segments C6 and C7 which then converge at the cranial aspect of the brachial plexus. [1,3] After coursing through the plexus, the nerve (now the SSN) courses lateral and contours itself to the cranial waist of the scapular neck prior to proceeding caudad and a providing its motor function to its dependent muscles (*Figures 2 & 3*). The muscles innervated by the SSN include the supraspinatus and infraspinatus, which anatomically originate from the lateral paraspinous fossae of the scapula bone. The arrangement of these two muscles clearly demonstrates their biomechanical functions as lateral stabilizers of the scapulohumeral joint which is devoid of collateral ligaments.

Treatment

Both surgical and medical therapies have been reported for management of sweeny. Surgical management involves decompression of the SSN by scapular notch resection with or without suprascapular ligament transection and neurolysis.[4,5] Medical therapies involve anti-inflammatory agent administration and strict confinement. [1,4,6] Results of previous studies[4,5] indicate favorable responses to both conservative[6] and surgical treatments.

Purpose of Study

It has been reported[7,c] that experimental transection of the SSN in an adult horse caused atrophy of the supraspinatus musculature but no gait abnormality. This implicated brachial plexus dysfunction as a cause of gait abnormality.[7]

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.

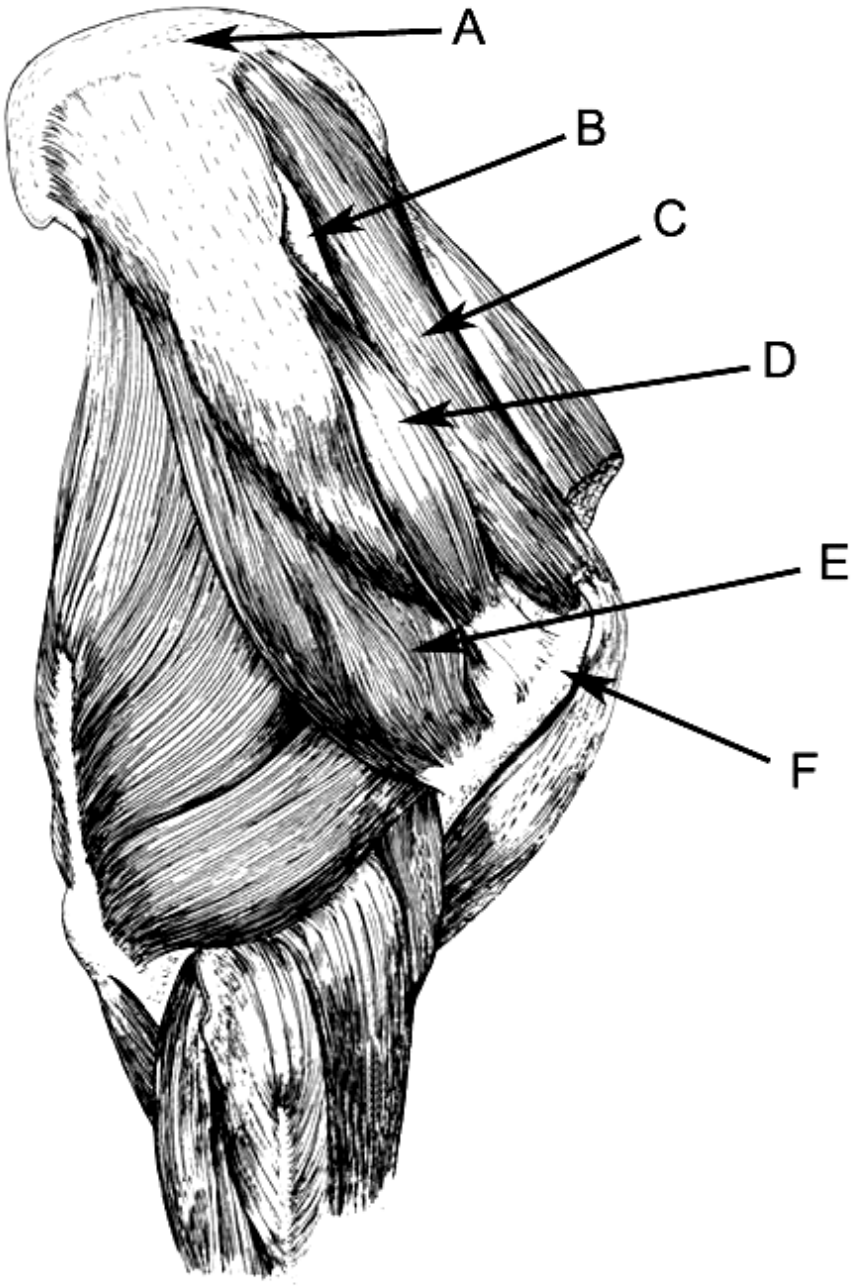

Figure 1. Superficial anatomy of the right shoulder (lateral view).

A. dorsal boarder of scapula; B. spine of scapula; C. supraspinatus muscle;

D. infraspinatus muscle; E. deltoideus muscle; F. greater tubercle of humerus.

Redrawn from Dyce, Sack Wensing[3]

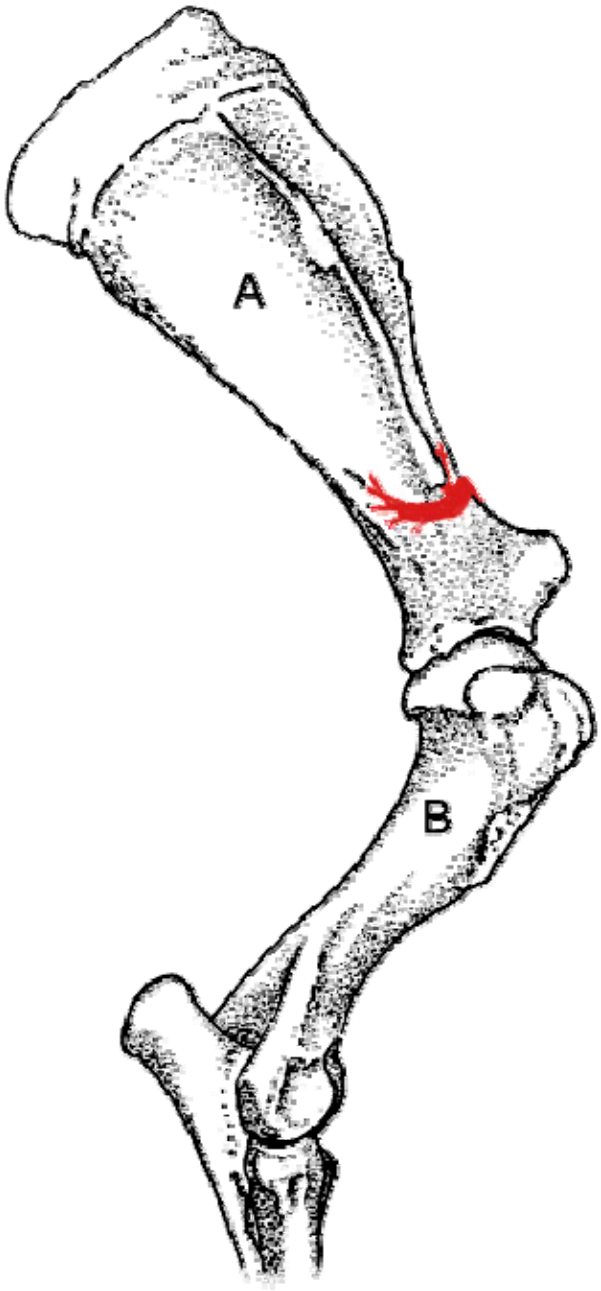

Figure 2. Skeletal anatomy of the right shoulder (lateral view).

A. Scapula; B. Humerus; The suprascapular nerve is depicted in red.

Redrawn from Dyce, Sack Wensing[3]

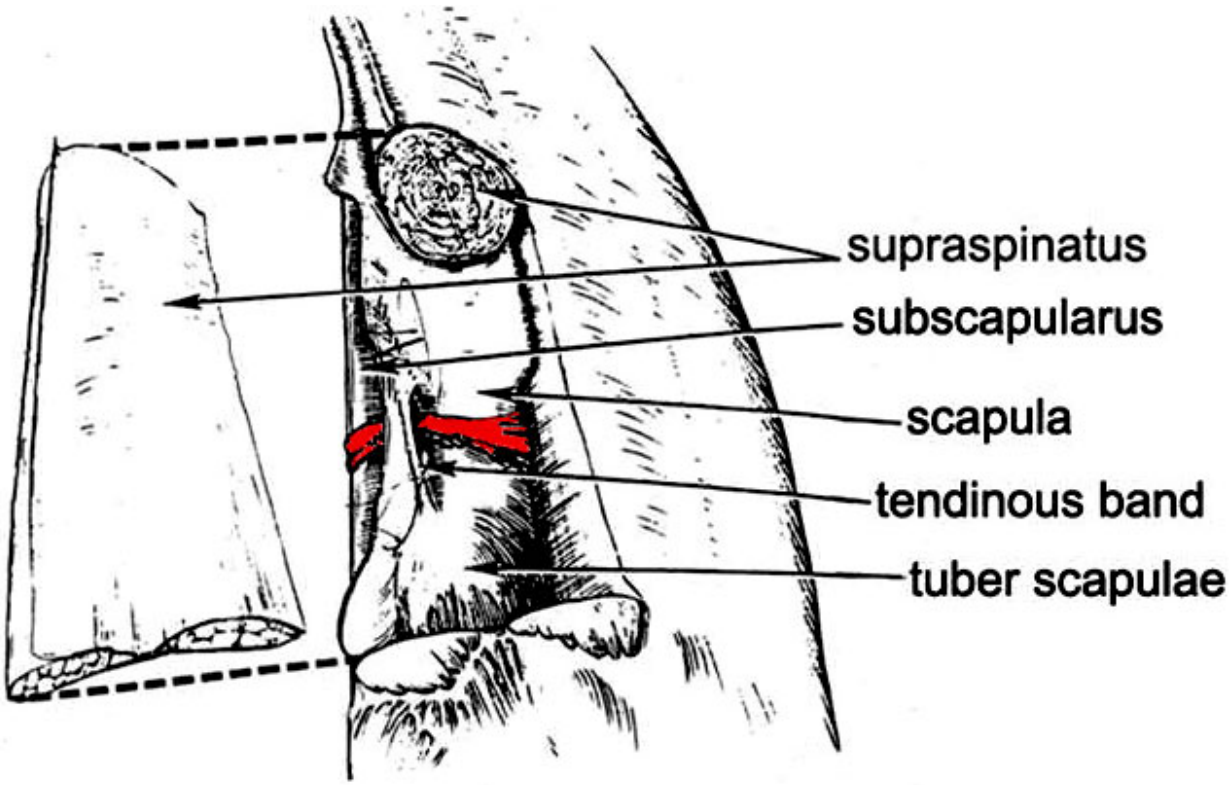

Figure 3. Drawing demonstrating anatomical relationship of structures of the cranial scapula (cranial view).

Redrawn from Schnider,Bramlage[4]

CHAPTER III

METHODOLOGY

Animals

Three healthy adult horses, acquired from an outside independent provider, (1 mare and 2 stallions) 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 pre-existing 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 prior to 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 (*Figure 4*). 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 (*Figure 5*). Surgical dissection advanced to the supraspinatus muscle, which was then divided to permit exposure of the SSN along the dorsal margin of the scapular neck (*Figure 6*). 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 (*Figure 7*). The catheter was anchored and secured by use of absorbable sutures into the deep fascia of the supraspinatus muscle in the surgical wound (*Figure 8*). Catheter security and patency was 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 suture material (*Figures 9 & 10*). 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 (*Figure 11*). The surgical wound and catheter were protected during anesthetic recovery by use of stent bandage and an adhesive incisional drape (*Figure 12*)

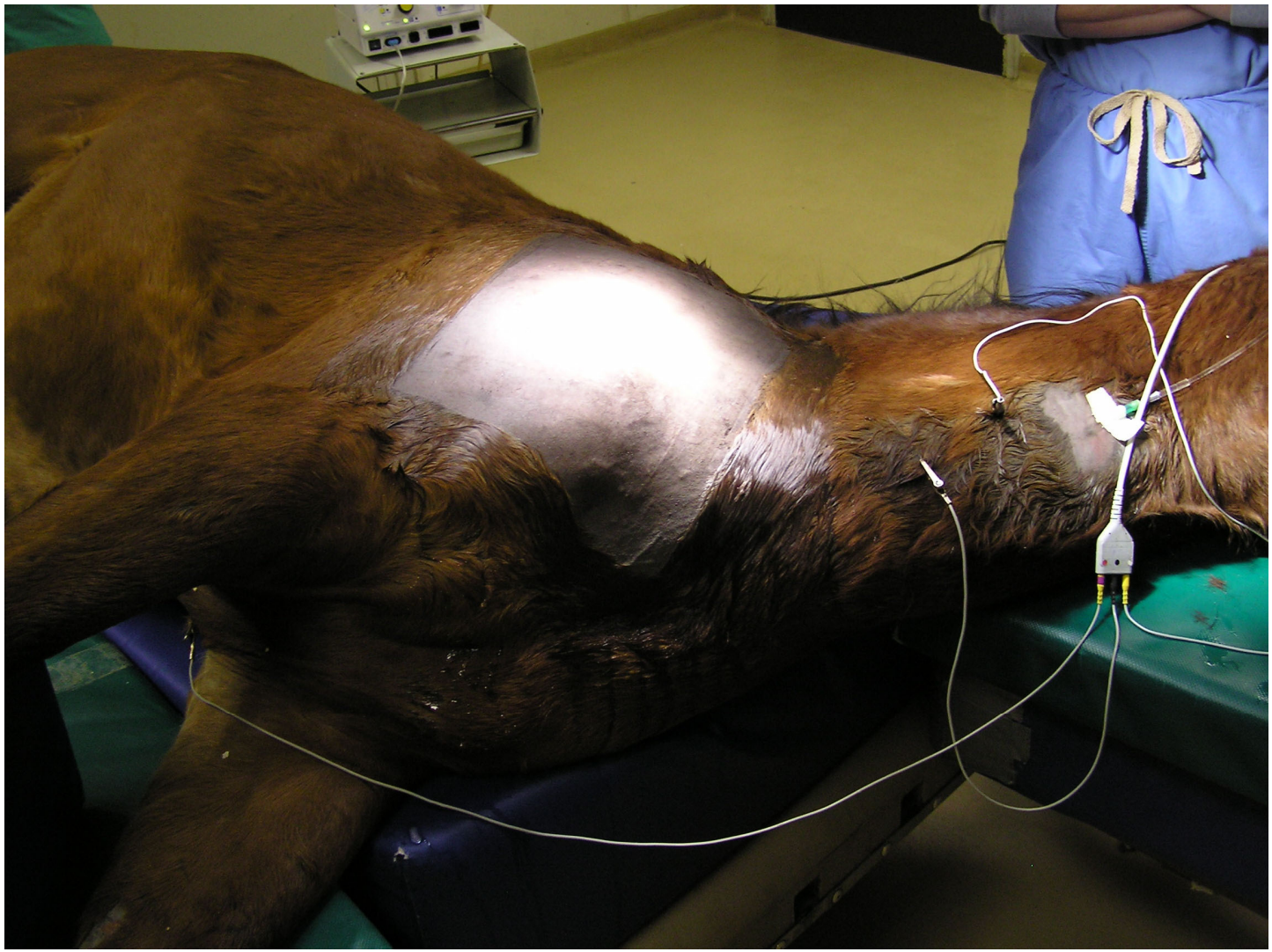

Figure 4. Surgical positioning and aseptic preparation of surgical field.

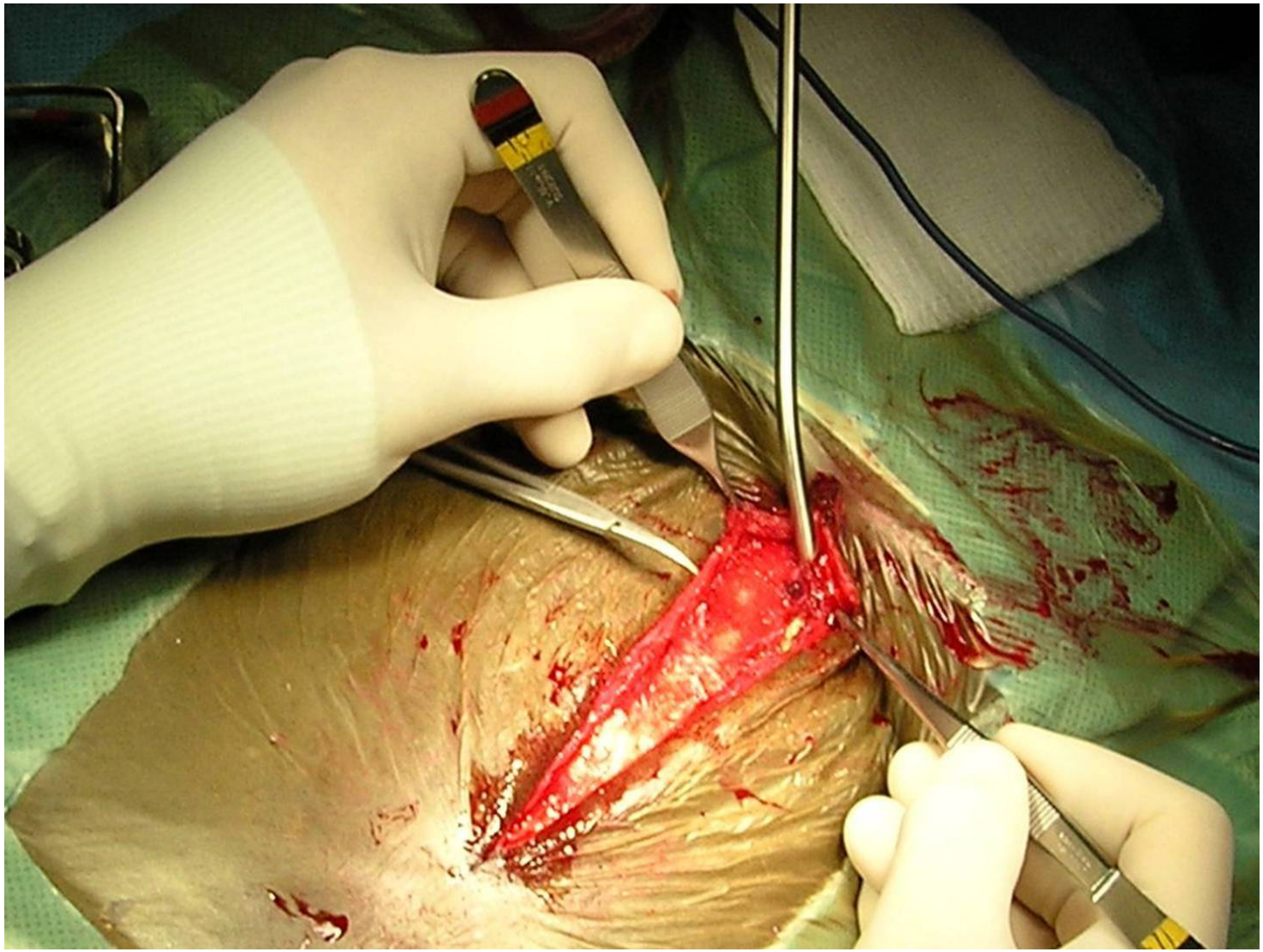

Figure 5. Surgeons view of approach to cranial scapula

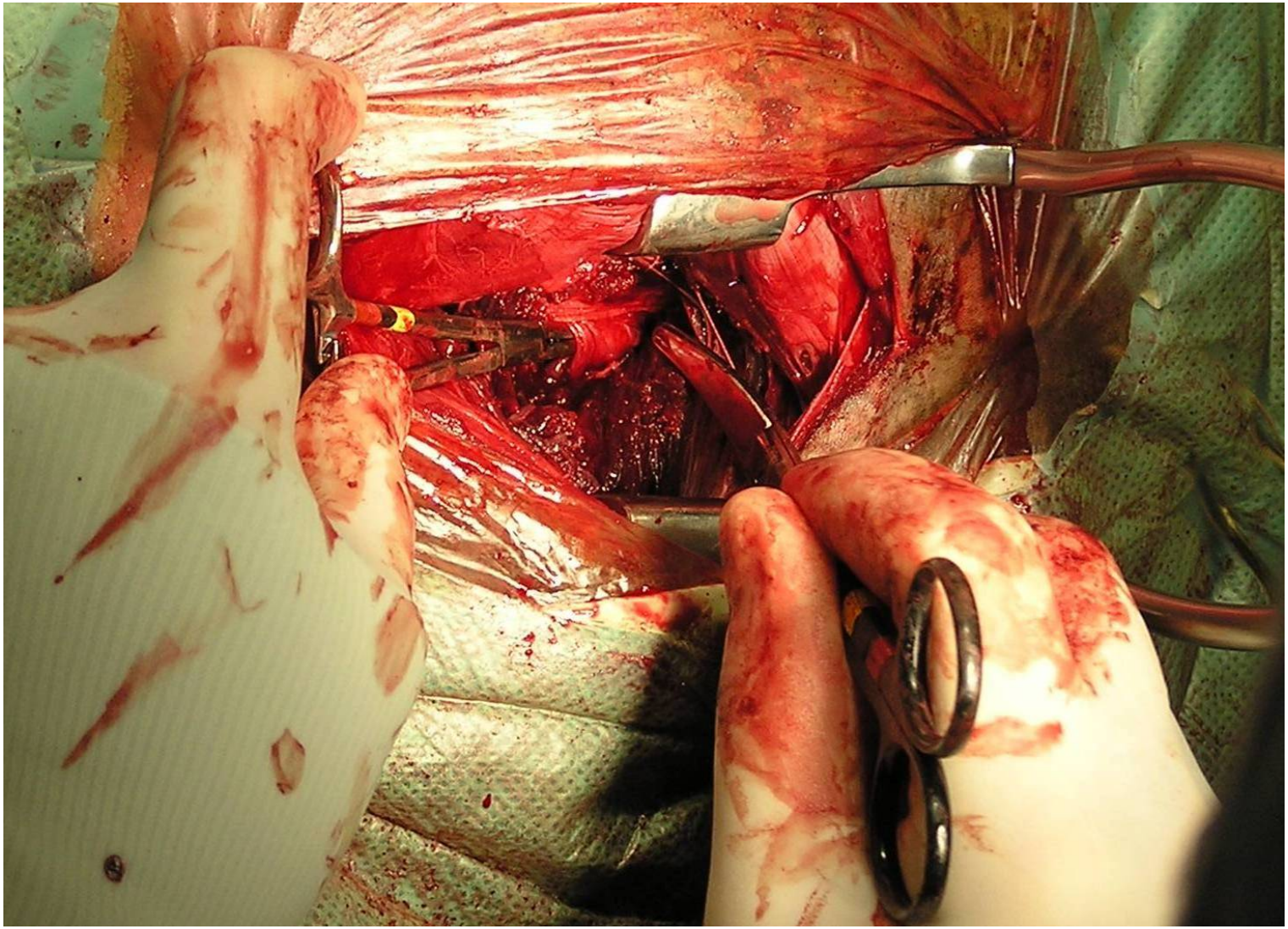

Figure 6. Surgeons view demonstrating isolation of the suprascapular nerve at cranial aspect of right scapula.

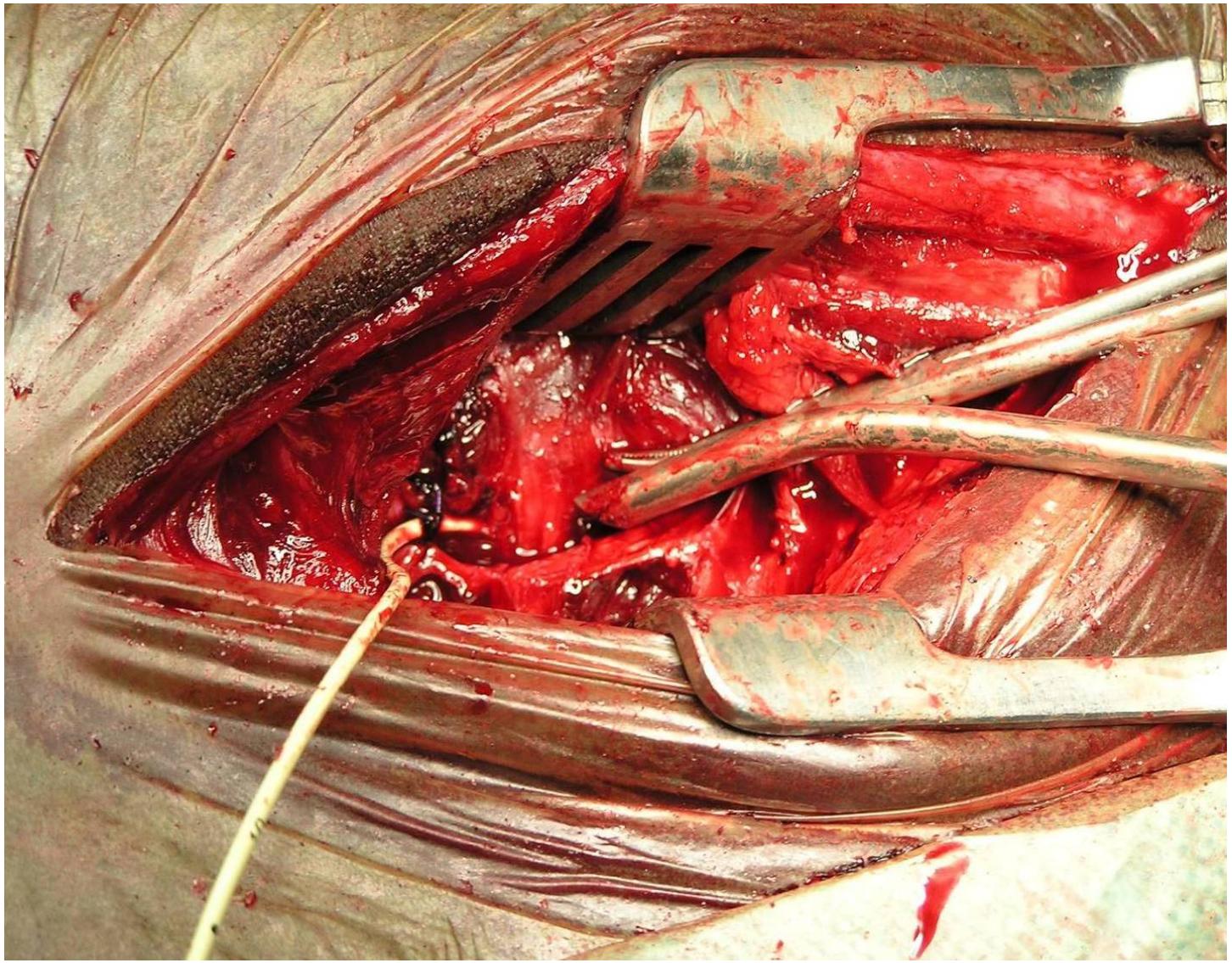

Figure 7. Surgeons view demonstrating placement and anchorage of catheter tip just beneath suprascapular nerve.

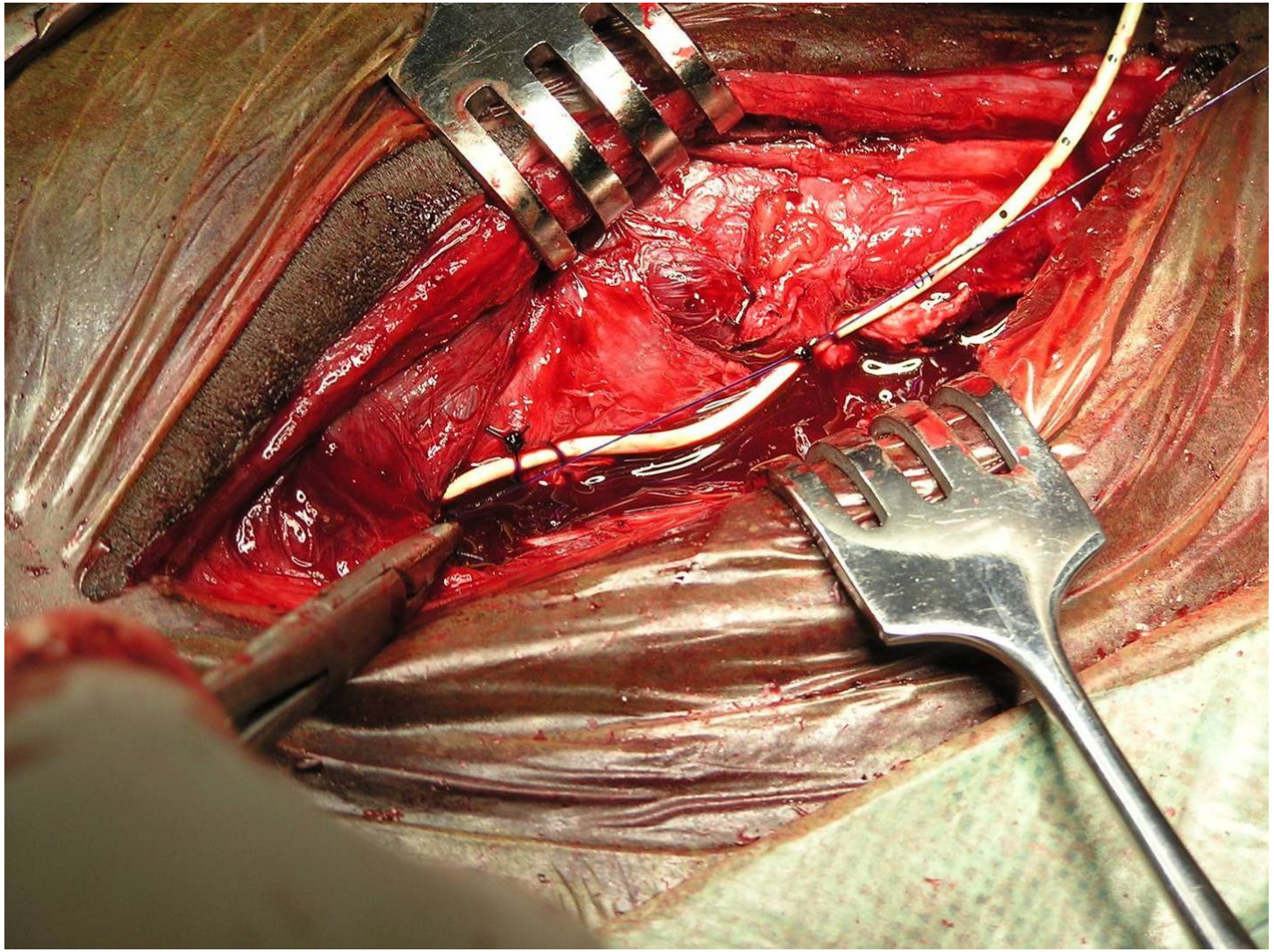

Figure 8. Surgeons view showing technique of catheter fixation to the superficial fascia of the supraspinatus muscle using absorbable suture material.

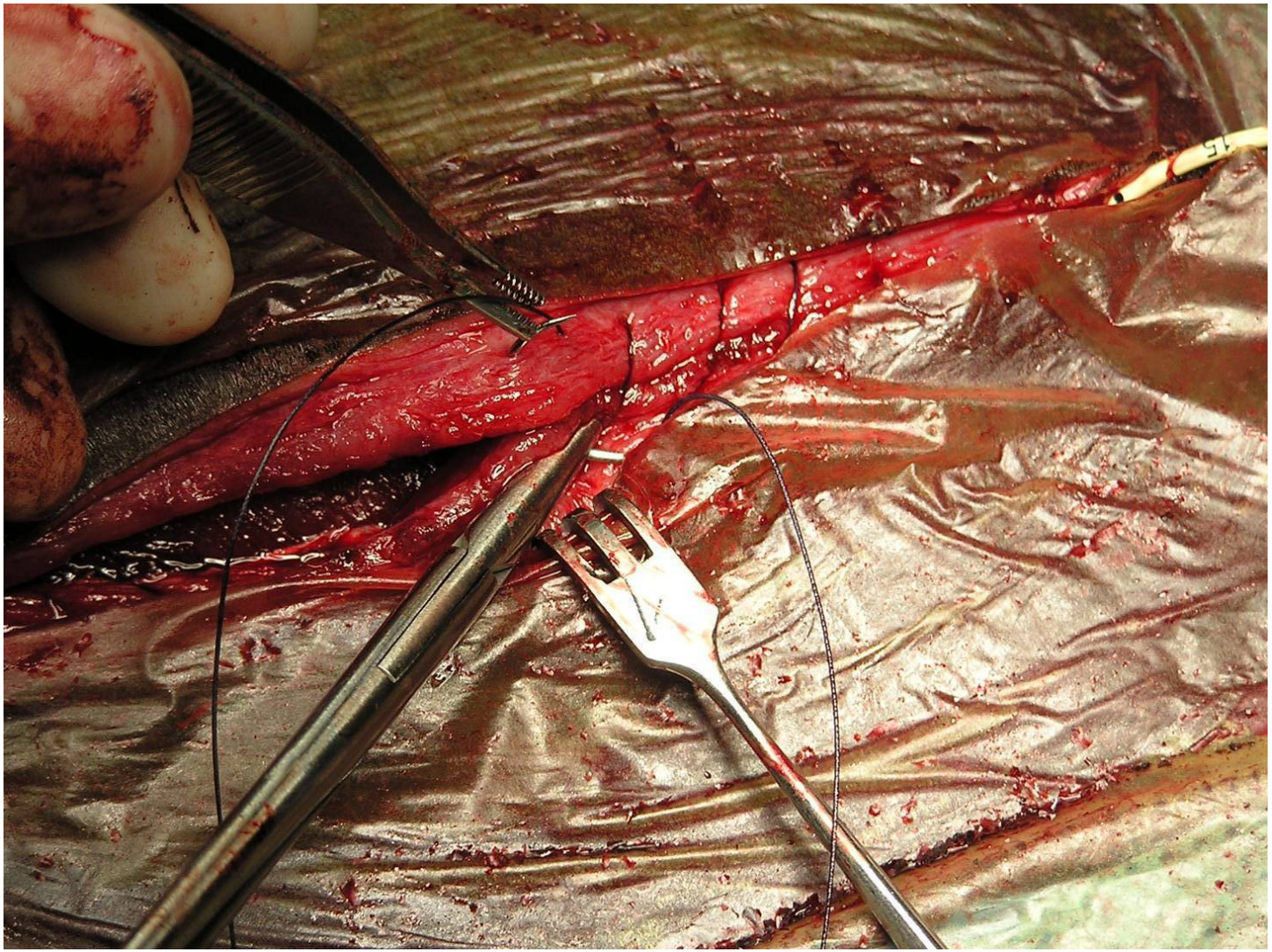

Figure 9. Closure of the superficial muscle layers using absorbable suture.

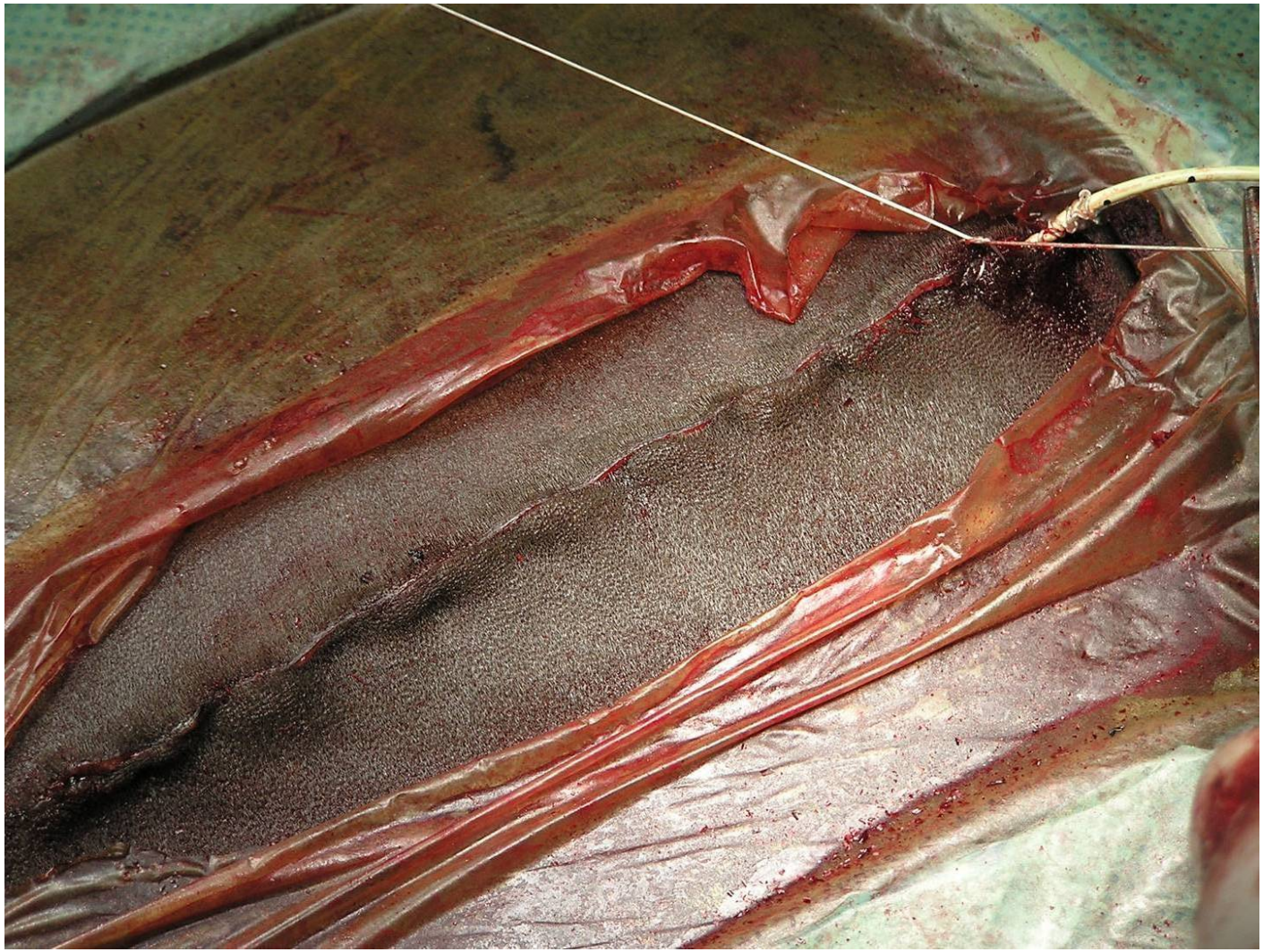

Figure 10. Closure of the subcuticular layer of the surgical wound demonstrating dorsal exit point and final fastening of catheter in wound.

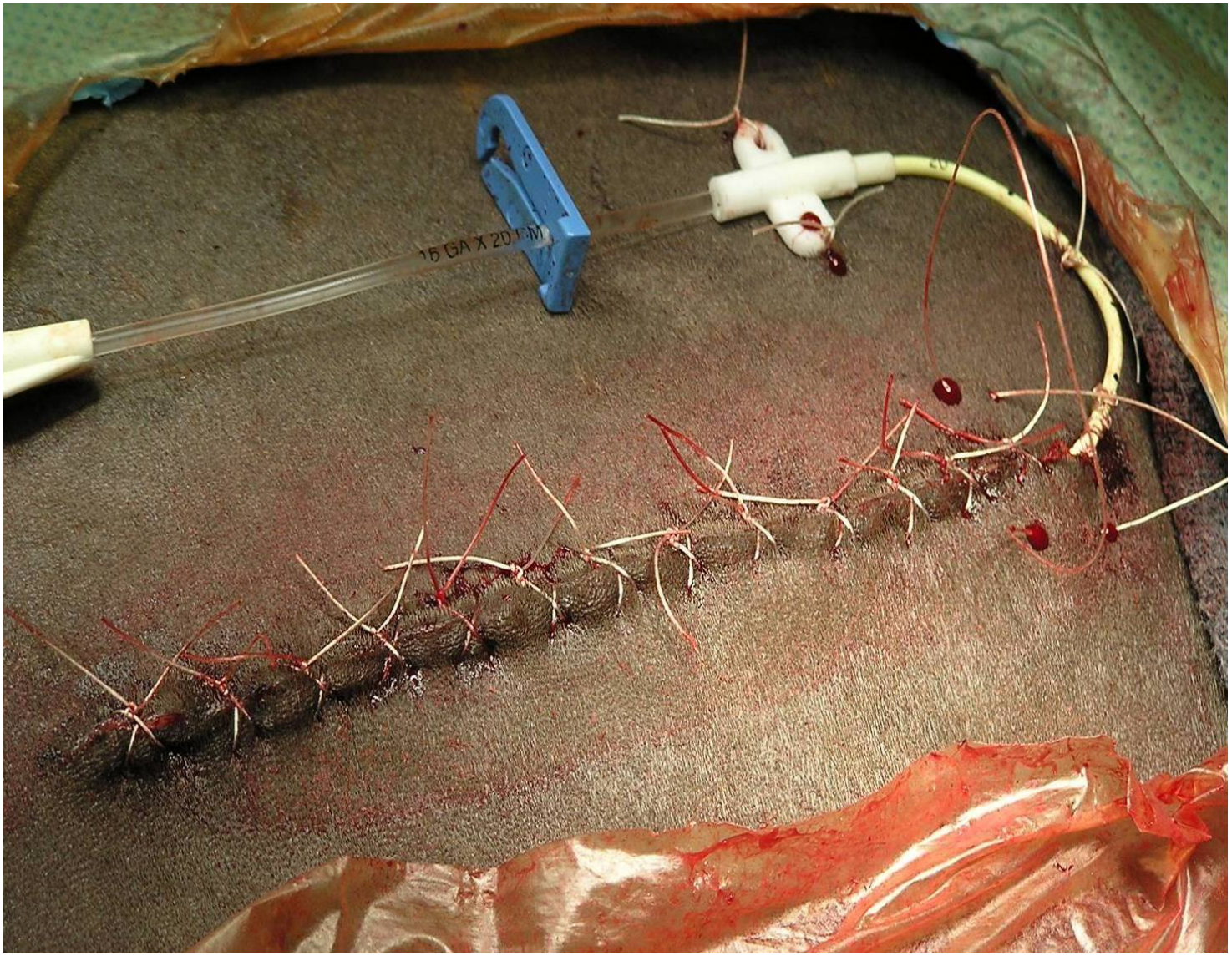

Figure 11. Final closure of surgical wound demonstrating skin closure, superficial catheter fixation and accessibility.

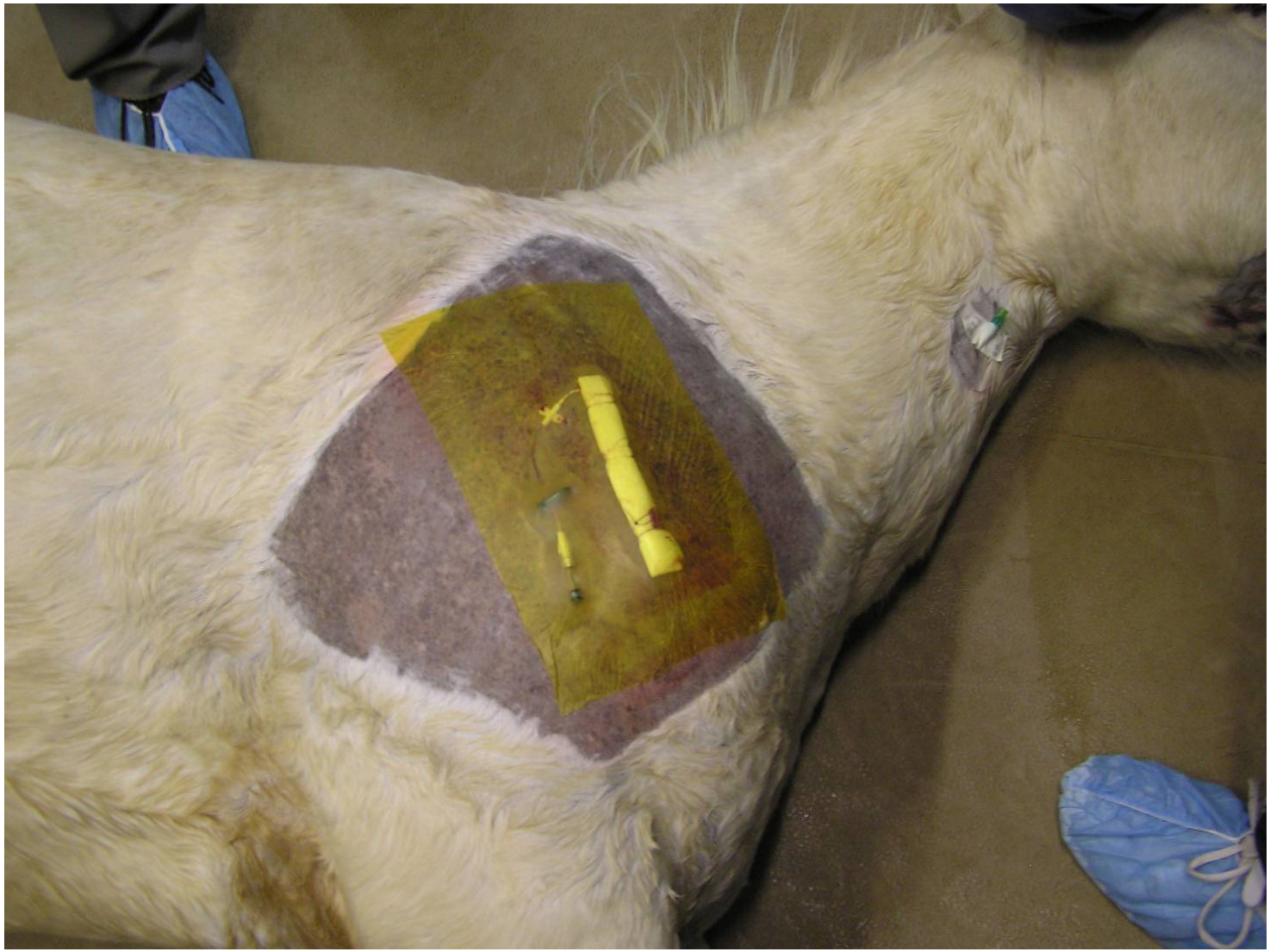

Figure 12. View of shoulder demonstrating protective oversewn stent and draping for recovery from anesthesia.

CHAPTER IV

FINDINGS

Data collection

Data acquisition consisted of video documentation initiated 6 hours following recovery from anesthesia. Baseline control data was collected as the horses were walked by hand in a straight line at a controlled rate of 1.40 to 1.45 m/sec. 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 was compiled and randomized to avoid bias and was blindly reviewed by the authors (DVD, HWJ) without knowledge of the treatment condition. 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[8] (performed by use of computer software[b]). Statistical significance was determined at a value of $P < 0.05$.

Results

Pre-anesthetic screening consisting of physical exam, PCV, total plasma protein and BUN concentrations were within reference limits for all 3 horses. No baseline lameness or gait abnormalities attributable to surgery or catheter placement were observed 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 three horses revealed a significant ($P < 0.001$) difference between the proportion of abnormal steps before compared to after chemical denervation. Physical examination of the horses 24, 48 hours and 2 weeks after the experimental procedure revealed very little observable discomfort and no permanent neurologic dysfunction.

CHAPTER V

CONCLUSION

These data support the role of the SSN in provision of shoulder joint stability and define the role of the SSN as one 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 in equids,[c] 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 biologic 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, biologic 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 post treatment 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, 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 substantiate the role of the SSN in development of the abnormal gait observed in horses with sweeny.

FOOTNOTES

a. Central venous catheterization set, Arrow International Inc, Reading, Pa.

b. PROC FREQ in SAS, version 8.2, SAS Institute, 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. Newmarket, UK, Royal College of Veterinary Surgeons, 1986.

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6. Dutton DM, Honnas CM, Watkins JP. Nonsurgical treatment of suprascapular nerve injury in horses: 8 cases (1988-1998). *J Am Vet Med Assoc* 1999;214:1657-1659.

7. Dyson SD. The elbow, brachium, and shoulder. In: Ross MW, Dyson SD, eds. *Diagnosis and management of lameness in the horse*. St. Louis: Saunders, 2003;414.

8. Agresti A. Inference for Two-Way Contingency Tables. In: Agresti A. *Categorical data analysis*. New York: John Wiley & Sons, 1990;59-62.

APPENDIX

Example video collected from third subject.

VITA

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Major Field: Veterinary Biomedical Sciences

Scope and Method of Study:

It is generally accepted that sweeny, (atrophy of the supraspinatus and infraspinatus muscles with an attendant gait abnormality), is due to traumatically induced dysfunction of the suprascapular nerve (SSN). The development of atrophy of the supraspinatus and infraspinatus muscles as a consequence of denervation supports this concept. On this basis, current surgical management of sweeny involves decompression of the SSN. This is achieved by scapular notch resection and/or dorsal suprascapular ligament transection and neurolysis. Previous studies report favorable response to both conservative and surgical therapies. Previous work describing experimental transection of the SSN resulted in atrophy of the supraspinatus and infraspinatus muscles without producing a gait abnormality yet, to this date; no scientific study has been performed to define the clinical effects of transitory SSN anesthesia. This thesis describes the methodology and clinical effects of specific SSN anesthesia by selective perineural catheterization (SPC) in three adult horses. Chemical denervation of the SSN was achieved using 1 ml of 2% mepivacaine hydrochloride delivered via SPC.

Findings and Conclusions:

Statistically significant scapulohumeral instability as evidenced by consistent lateral excursion at the walk during the weight-bearing stride phase occurred in all three subjects. These data support the role of the SSN in provision of shoulder stability and define the role of the SSN in the etiopathogenesis of the clinical syndrome referred to as sweeny.

ADVISER'S APPROVAL: Henry W. Jann