
Anne M. Walker, DVM; Debra C. Sellon, DVM, PhD, Diplomate ACVIM; Melissa T. Hines, DVM, PhD, Diplomate ACVIM; Claude A. Ragle, DVM, MS, Diplomate ACVS; and Noah Cohen, VMD, PhD, Diplomate ACVIM

Temporohyoid osteoarthropathy is a poorly described neurologic disorder of horses characterized by acute onset of cranial nerve dysfunction with bony proliferation of the temporohyoid joint and proximal stylohyoid bone. A retrospective study of 21 cases revealed that affected horses have a fair to good prognosis for return to athletic function, although most horses have mild residual cranial nerve dysfunction. Authors’ addresses: Department of Veterinary Clinical Sciences, College of Veterinary Medicine, Washington State University, Pullman, WA 99164 (all); and Department of Large Animal Medicine and Surgery, College of Veterinary Medicine, Texas A&M University, College Station, TX 77843 (Cohen). © 2001 AAEP.

1. Introduction
Temporohyoid osteoarthropathy is a neurologic disorder of horses characterized by acute onset of cranial nerve dysfunction with bony proliferation of the temporohyoid joint and proximal stylohyoid bone, ultimately leading to fusion of the temporohyoid joint.1–7 Fusion of the joint predisposes horses to fracture along the base of the skull or the shaft of the stylohyoid bone. These fractures may occur secondary to normal tongue and laryngeal movement. There is little information in the current veterinary literature concerning long-term follow-up and prognosis for affected horses. A retrospective study with client follow-up was conducted to determine prognosis for life and return to athletic function, presence of residual deficits, and likelihood of recurrence in affected horses.

2. Materials and Methods
The case records of horses admitted to Washington State University and Texas A&M University between 1993 and 2000 were reviewed. Diagnostic results of skull radiographs, guttural pouch endoscopy, magnetic resonance imaging, and computed tomography were examined. Horses with asymmetry, inflammation, or osseous proliferation in the region of the temporohyoid joint or stylohyoid bone were included in the study. Follow-up information was obtained by telephone conversation with the owner, veterinarian, or trainer.

3. Results
Twenty-one horses were identified with clinical evidence of temporohyoid osteoarthropathy. The mean age of affected horses was 10.5 years. There was no obvious breed or sex predilection. Horses
presented for a variety of complaints including head shaking, acute neurologic dysfunction, ear infections, and corneal ulceration. At initial presentation, 10 horses had signs consistent with a right-sided lesion and 11 horses had signs consistent with a left-sided lesion. Diagnostic evaluation ultimately revealed that 2 horses with unilateral complaints actually had bilateral disease.

Twenty horses had some degree of facial nerve (cranial nerve VII) deficits at the time of presentation; 17 horses had signs of vestibulocochlear nerve (cranial nerve VIII) involvement. Of 8 horses that had Schirmer tear tests performed, 7 had decreased tear production. Nine horses had corneal ulceration. Of 19 horses with skull radiographs, 16 had evidence of osseous proliferation of the temporohyoid joint or stylohyoid bone. Of 20 horses that underwent endoscopy of the guttural pouches, all showed abnormalities of the stylohyoid bone (osseous proliferation or asymmetry), or inflammation in the associated soft tissue structures on the affected side. Three horses had asymmetry or inflammation in the area of the temporohyoid joint without obvious osseous enlargement of the proximal stylohyoid bone. These horses also had no radiographic evidence of osseous changes in the temporohyoid region. Abnormalities were observed in all horses that underwent magnetic resonance imaging (n = 4) or computed tomographic evaluation (n = 2).

Treatment of these horses was aimed primarily at eliminating infection and relieving inflammation. All horses were treated with systemic anti-inflammatory therapy. Trimethoprim sulfa was used in 19 of the 21 horses. Eighteen horses were also treated with anti-inflammatory medications, most commonly phenylbutazone. On average, the horses in this study were treated for 30 days. Nine horses were treated for corneal ulceration with topical antibiotics. Five of the 9 horses eventually required tarsorrhaphy for the affected eye. Three horses underwent surgery for partial stylohyoidostectomy.1

Follow-up information was available for 20 horses. Mean follow-up time was 3.6 years. One horse died acutely secondary to a skull fracture. Two horses were euthanized within 6 months of initial diagnosis for lack of improvement in clinical signs. On necropsy, these 3 horses had evidence of fractures along the base of the skull. One horse was euthanized for laminitis, unrelated to the temporohyoid problem, but was observed to have improvement of cranial nerve dysfunction prior to euthanasia. Fifteen of the 20 horses (75%) were considered to have greatly improved clinical signs. These horses were considered suitable for athletic use and the majority had returned to their intended use (11 of the 15). Athletic pursuits included dressage competition, barrel racing, competitive roping, showmanship, and reining. Of the 16 surviving horses, 15 had some degree of residual cranial nerve deficit. Twelve horses had facial nerve deficits and 11 had vestibulocochlear nerve deficits. Three horses had one episode of corneal ulceration after initial discharge from the hospital. One of the 16 surviving horses was considered unimproved and not safe to ride due to vestibular deficits and risk of falling.

4. Discussion

In this study, horses with temporohyoid osteoarthropathy had a fair to good prognosis for return to some form of athletic function, with 75% of the horses in this study athletically usable within a mean follow-up time of 3.6 years. It may take months to years for maximal improvement in clinical signs. The horses in this study had a range of 6 months to 2 years before they returned to use. In a previous report of this condition, total resolution of clinical signs was observed in 5 horses within 30–60 days.2 In a separate report, 22 of 26 horses recovered with no clinically apparent neurologic dysfunction unless blindfolded.3 All but one horse in the study reported here had some residual cranial nerve deficits reported by the owner, trainer, or veterinarian. These deficits did not seem to limit the athletic function of the horses in most cases. Recurrence or acute worsening of clinical signs was not reported for any horse. Consistent with a previous report, endoscopy was more sensitive than radiography for detection of lesions.4

Horses with temporohyoid osteoarthropathy have a fair to good prognosis for return to some type of athletic function. However, most cases will have some residual cranial nerve dysfunction and it may take a year or longer for maximal improvement to occur. Although the potential for recurrence of acute clinical signs exists, this does not appear to be common in affected horses.

References