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Neurologic Deficits Including Auditory Loss and Recovery of Function in Horses with Temporohyoid Osteoarthropathy

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Background: Auditory loss is a common deficit in horses with temporohyoid osteoarthropathy (THO), however, recovery of function is unknown.

Hypothesis/Objectives: To investigate neurologic function with emphasis in audition in horses with THO after treatment. To describe anatomical alterations of the petrous temporal bone that might result in auditory loss.

Animals: Twenty-four horses with a clinical diagnosis of THO.

Methods: Prospective study. A brainstem auditory evoked response (BAER) study was done as part of the criteria for inclusion in horses with a clinical diagnosis of THO from the years of 2005 to 2014. Physical and neurologic status and BAER findings were recorded. Brainstem auditory evoked response variables were compared by using Wilcoxon sign test. Fisher's exact test was also used. Significance was set at $P < 0.05$.

Results: The most common signs included auditory loss (100% of horses), vestibular and facial nerve dysfunction (83%), and exposure ulcerative keratitis (71%). Concurrent left laryngeal hemiparesis was observed in 61% of horses through endoscopy. Auditory dysfunction was bilateral in 50% of the cases (complete and partial), and unilateral affecting more commonly the right ear (R = 8, L = 4). Short- and long-term follow-up revealed persistent auditory loss in all horses based on abnormal response to sound, and further confirmed through a BAER in 8 horses.

Conclusions and Clinical Importance: Auditory dysfunction appears to be a permanent neurologic deficit in horses diagnosed with THO despite overall neurologic improvement.

Key words: Brainstem auditory evoked response; Deafness; Equine; Hearing.

Temporohyoid osteoarthropathy is a disorder of the temporohyoid joint formed by the stylohyoid and petrous temporal bones.[1] The disorder is characterized by bony proliferation, fusion of the joint, and potential fracture of the involved or adjacent bones.[1–5] Proposed etiologies include inflammation, infection of the middle/inner ear secondary to a hematogenous or ascending infection from the upper respiratory tract, extension of external ear infection, and primary degenerative process.[6,7] Clinical signs vary from head shaking, apparent resentment of manipulation of the head or ears, resistance to the bit, difficulty eating, and neurologic deficits mainly consisting of facial and vestibulocochlear nerve dysfunction.[1,8,9] Auditory loss appears to be a common neurologic abnormality in these horses.[8–11] Evaluation of auditory function solely based on physical examination could be difficult to interpret in horses with partial hearing loss or complete hearing loss of one ear with hearing in the contralateral ear. As long as one ear has some function, the horse might not appear deaf. However, localization of sound requires that both ears remain functional.[9] In addition, horse's attention, cooperation, or behavior might interfere with accurate interpretation of hearing by the examiner.

Abbreviations:

BAER	brainstem auditory evoked response
CHO	ceratohyoid ostectomy
SHO	stylohyoid ostectomy
THO	temporohyoid osteoarthropathy

Vestibular and facial nerve dysfunction is commonly observed in horses with THO;[1] however, both are not always identified.[1,5,7,8,12] Most studies have reported deficits of the vestibulocochlear nerve;[1,13–15] however, there are only descriptions of the vestibular dysfunction with no reference to auditory. Therefore, complete evaluation of the vestibulocochlear nerve was not done. Further, studies referring to neurologic recovery after medical or surgical management have been reported with no mention of auditory function.[1,12–16] One study reported up to 70% (n = 14/20) return to previous physical activity.[1] However, the authors did not specify which nerve function recovered and how many of these horses had medical or surgical intervention.[1] An earlier study of surgical management in 24 horses with THO found 87 and 89% clinical improvement 1 year after partial stylohyoid ostectomy (SHO) and ceratohyoid ostectomy (CHO), respectively; with most improvement occurring within the first 6 months after surgery.[15] Studies with complete evaluation of the neurologic status (including hearing) with long-term follow-up to determine recovery of function (including hearing) in

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horses with THO are not available. Therefore, the purpose of this study was to investigate the neurologic status with emphasis in auditory function after therapeutic intervention in horses diagnosed with THO. Further, anatomical alterations of the petrous temporal bone that might result in auditory loss will be described.

Materials and Methods

Animals

This prospective study included horses with a clinical diagnosis of THO through a neurologic and imaging examination from January 2005 until December 2014. Physical and neurological parameters were recorded, and basic blood work (CBC, chemistry panel) was performed. Auditory function was investigated in these horses through a brainstem auditory evoked response examination on admission and was mandatory to be included in this study. Horses underwent medical or surgical therapy to alleviate clinical signs. Short- and long-term follow-up included physical, neurologic, and BAER examinations. The study was approved by an animal care and used protocol from our institution, and owner consent was obtained.

Brainstem auditory evoked response

Brainstem auditory evoked response testing was done using two evoked potentials systems,[ab] upon availability as described elsewhere.[8] Horses were placed in examination stocks and sedated with xylazine hydrochloride at a dosage of 0.3 to 0.4 mg/kg IV. Insert earphones (add 0.9 milliseconds to latencies) were placed deep into the external ear canal.[8] Subcutaneous needle electrodes were placed at the vertex (V), left mastoid (LM), right mastoid (RM), and on the dorsal midline at the level of C2 vertebra (C2) for recording the BAER.[8] For one of the evoked systems[a], a ground electrode (Z) was placed between the occipital protuberance and C2 vertebra.[8] For the newer system[b], the contralateral mastoid served this purpose.[9] Specifications for BAER recording and labeling of peaks were the same as those described elsewhere.[8] Each BAER recording was the average of a minimum of 400 responses over a 10 msec epoch. An alternating (rarefaction plus condensation) broadband click stimulus at 90 dB normal hearing level (nHL) was applied individually to each ear. A simultaneous masking sound was used on the contralateral side with an offset of −30 dB nHL.[8] All BAER studies were done in duplicate and 2 derivations were recorded simultaneously: 1) vertex to ipsilateral mastoid (V-M) and 2) vertex to C2 (V-C2).[8] The 2 derivations were recorded simultaneously and facilitated the identification of the peaks, particularly in cases of low amplitude responses.[8]

Determination of normal versus abnormal BAER was based on latency and amplitude for peaks I, III, and V measured in milliseconds and microvolts (μV), respectively; interpeak intervals (IPI) for latencies between peaks I–III, III–V, and I–V; and, amplitude ratio by dividing peak V by peak I on the vertex to C2 derivation.[8] The study was determined as complete (absence of identifiable BAER peaks) or partial auditory loss (increased peak latency beyond 2 standard deviations [SD] from previously published reference values[8], or difficulty identifying peaks).[8,9,17,18]

Diagnostic imaging

Imaging studies were reviewed by a board-certified radiologist independently and blinded to the neurologic and BAER examination. A radiographic diagnosis of THO was made if the proximal aspect of one or both stylohyoid bone was thickened, periarticular remodeling of the temporohyoid joint, or both could be identified.

Computed tomographic (CT) studies consisted of a minimum of one transverse series of images through the caudal skull obtained in a bone algorithm with slice thickness ranging from 0.625 to 2 mm. Osteoproliferation at the periphery of the temporohyoid joint was graded from 0 to 3 as follows: grade 0 represents absence of proliferation, grade 1 = proliferation not bridging the joint, grade 2 = proliferation bridging the joint, but extending over less than 1 cm, and grade 3 = proliferation over more than 1 cm. Grade 1 and higher were considered positive for a diagnosis of THO. The ear canal, tympanic membrane, tympanic cavity, cochlea, ossicles, and facial canal were carefully evaluated for the presence of osseous alterations or the presence of abnormal soft tissue.

Statistical analysis

Latency for peaks I, III, and V, interpeak intervals (I–III, III–V, I–V), and amplitude ratio (V/I) were compared from initial and follow up BAER. The BAER variables were checked for normality by using the Shapiro–Wilk test. Variables were compared between variables from the BAER examinations by the paired sample Wilcoxon Signed Rank Test, using commercial software.[c] Fisher's exact test was used to evaluate the association between the loss of auditory function and the CT grades. Significance was set at $P < 0.05$.

Results

Animals

Sixty horses were diagnosed with THO during the study period. Of these, BAER testing was performed at first visit in 24 horses. These horses were of Quarter horse (n = 16), Thoroughbred (n = 5), Warmblood (n = 2), and Morgan (n = 1) breeds. There were 12 females, 10 geldings, and 2 stallions. Their ages ranged from 7 to 21 years (mean 12.3, median 11.5 years). The presenting complaint included acute unbalanced gait (n = 15), droopy face (n = 9), head tilt and leaning (n = 7), nonhealing corneal ulcer (weeks to 2 months, n = 6), acute corneal ulcer (n = 3), head tossing (n = 2), seizure-like episode (n = 1), recumbency (n = 1), suspected deafness (n = 1), and draining tract from parotid gland (n = 1). Seven horses were treated with oral trimethoprim-sulfas and phenylbutazone or flunixin meglumine for 7 to 15 days before referral with no resolution of signs. All horses with corneal ulceration were treated with ophthalmic antimicrobials and atropine, but no resolution was observed.

Neurologic examination

Physical examination was within normal limits except for the neurologic status. Neurologic examination was abnormal in all 24 horses (Table 1). There were no alterations in mentation and behavior (horses were bright, alert, and responsive to the environment). Menace response and palpebral reflex were reduced (n = 13) or absent (n = 5) because of facial nerve dysfunction (neurologic examination ruled out other locations). Dazzle reflex was absent in 5 horses because of facial nerve dysfunction. Corneal reflex was not performed in horses with exposure keratitis and ulceration (n = 17) because of risk of further injury (eg, perfora-

Table 1. Neurologic deficits. R = right side, L = left side, complete = complete auditory loss, partial = partial auditory loss, C/P = complete/partial auditory loss on each ear, respectively. Grades of ataxia (1–5, 5 = recumbent). Endoscopy: A: Horses identified with THO based on endoscopy, B: Horses identified with left laryngeal hemiparesis based on endoscopy. Grades of laryngeal dysfunction (1–4) noted on endoscopy.

		Unilateral			
Horses (N = 24)	Bilateral	R	L	Abnormal	Normal
Facial nerve dysfunction	5	10	5	20	4
Neurogenic exposure keratitis/Sicca	1	11	5	17	7
Vestibular disease	2	12	6	20	4

	Complete	Partial	C/P	R	L	Abnormal	Normal
Auditory loss (BAER) (N = 24)	0	0	12	8 (C: 7, P:1)	4 (C: 3, P: 1)	24	0

	1	2	3	4	5	Abnormal	Normal
Ataxia (Grade)	0	4	6	8	1	19	5

Endoscopy (N = 18)	Bilateral	Unilateral		Abnormal	Normal
A. THO	9	R: 5	L: 4	18	0

	Grade					
	1	2	3	4	Abnormal	Normal
B. Left laryngeal hemiparesis	7	6	5	0	11	7

tion). One of these horses had had an enucleation caused by corneal perforation at presentation. Corneal reflex was present (retraction of eye globe present but with reduced blink) in the remaining 8 horses. Horses with chronic nonhealing ulceration had an exaggerated retraction of the eye globe upon light stimulation. Anisocoria was observed in 15 horses and thought to be secondary to exposure keratitis with ulceration with the affected eye (miotic because of apparent pain, or midriatic pupil because of dilation with atropine) being ipsilateral to the facial, vestibular deficits, or both. A Schrimer's tear test was performed in 15 horses and showed a reduced tear production in the affected eye ranging from 5 to 19 mm/min versus 20 to 35 mm/min in the contralateral eye. All horses appeared to be visual and had normal facial nociception. Facial paresis or paralysis was observed in 20 horses. Vestibular dysfunction characterized by nonpositional conjugate horizontal (fast phase away from the head tilt) or rotatory nystagmus, head tilt and leaning (16 horses), and ataxia (15 horses) were observed. Blindfold was performed in 10 horses, and vestibular dysfunction became apparent in 5 horses with mild ptosis and corneal ulceration, and exacerbated in 6 horses. Horses with moderate to severe ataxia (n = 15 horses, grades 3 to 5) according to a published grading system[19] were not blindfold because of safety concerns. Bilateral vestibular disease characterized by lack of physiologic and pathologic nystagmus, wide-based stance, and ataxia was observed in 2 horses. A sound test consisting of clapping or ringing keys from one side with the examiner hiding from the horse revealed suspected deficits such as horses not turning the head toward or moving away from sound or acting startled. This was repeated for consistency. A feed trial (cookies, hay, pellets, grain) and water were offered as part of the neurologic examination. Difficulty or reduced prehension with packing of feed in the affected side was observed in 7 horses. Mastication and swallowing were apparently normal. Tongue movement and tone were normal.

Brainstem auditory evoked response

Auditory loss was bilateral in 12 horses and unilateral in 12 horses (Table 1). Horses with bilateral disease had complete (right ear n = 6/12, left n = 6/12) and partial auditory loss on each ear. Complete auditory loss was ipsilateral to the side with the most profound signs of either facial or vestibular signs. Unilateral auditory loss involved the right ear in 8 and the left ear in 4 horses, respectively.

Clinical laboratory

A CBC did not reveal abnormalities except in 2 horses on which the PCV was 25 and 27% (reference range 30 to 46%), respectively. Abnormal findings included elevated total bilirubin (n = 7, range 2.4 to 3.8 mg/dL; reference range 0.5 to 2.3 mg/dL), and elevated creatine kinase (n = 6, range 380 to 2,940 U/L; reference range 119–287 U/L). Serum immunofluorescent antibody test for *S. neurona* and *N. hughesi*, serum IgM-capture ELISA for West Nile virus, and PCR on a nasal swab for equine herpes virus 1 were performed in 10 horses and were negative. Cerebrospinal fluid analysis was performed in 4 horses and was normal in 3 horses. Marked histiocytic pleocytosis and hemorrhage was found in a horse with a seizure-like episode.

Endoscopy

Bilateral guttural pouch endoscopy was done in 18 horses, 9 of which had bilateral, and 9 unilateral abnormalities consisting of thickening of the stylohyoid bone and suspected fracture. Left laryngeal hemiparesis was observed in 11 horses (grade 2, n = 6; grade 3, n = 5; according to published grading scale[20], Table 1). Three of the 11 horses with laryngeal dysfunction had a history of poor performance. These 11 horses were of Quarter Horse (n = 7) and Thoroughbred (n = 4) breeds.

Diagnostic imaging

Skull radiographs were performed in 21 horses and revealed bilateral THO in 12 horses, right sided disease in 6 horses, left sided in 1 horse, and no radiographic abnormality in 2 cases. The abnormalities consisted of thickening of the stylohyoid bone, and osseous proliferation of the temporohyoid articulation. Of the 12 horses with bilateral radiographic disease, 8 had bilateral, and 4 unilateral auditory loss. Of the 3 other cases with bilateral auditory loss, 2 had no radiographic abnormalities, and 1 had right-sided radiographic abnormalities. Five cases had matching unilateral radiographic disease and unilateral auditory loss, but one case had left-sided radiographic abnormalities and right-sided auditory loss.

Computed tomography was performed in 11 horses (22 sites: 2 sites per horse [right and left sides]) and showed bilateral and unilateral THO in 9 and 2 horses, respectively. Most bilateral cases were asymmetric with one side more severely affected than the other side. Two horses diagnosed with unilateral disease based on radiography and endoscopy were found to have bilateral disease when evaluated through a CT scan and 1 horse with normal radiographic findings was found to have left-sided disease on CT. Four cases were diagnosed with fractures of the temporal bone. Two of these 4 cases had severe bilateral abnormalities and the other 2 were asymmetric with the fracture ipsilateral to the most severely affected side. All fractures were not displaced and had an oblique rostrodorsal to caudoventral orientation. In 3 of the cases, the fractures extended through the facial canal and in 1 the cochlea was concurrently involved. An additional horse had extension of the fracture to the cochlea, but not the facial canal. Seven cases had increased hypoattenuating tissue surrounding the auditory ossicles. This finding was unilateral in all 7 cases and associated with increased soft tissue at the tympanic membrane (n = 6/7), fluid in the external ear canal (n = 5/7), and fluid in the tympanic cavity (n = 4/7).

Of the 9 cases with bilateral abnormalities on CT, auditory loss was bilateral in 5 horses, and unilateral in 4 horses (2 on each side). Regarding the 2 cases with unilateral lesion on CT, one had ipsilateral auditory loss, but the other one had bilateral auditory loss. All cases with temporal bone fracture or increased soft tissue surrounding the auditory ossicles had ipsilateral auditory loss. However, 9 of 17 sites (out of 22 sites [11 horses: 11 left and 11 right sites]) on which auditory loss was identified; had no evidence of CT abnormalities in the external ear canal, tympanic membrane, tympanic bulla, facial canal, or cochlea.

Therapeutic management

Three horses were euthanized as per owner election because of severity of signs and finances. One additional horse was euthanized because of other concurrent problems (multiple melanomas, anaerobic parotid abscess). Treatment consisted of medical and surgical treatment in 7 and 13 horses, respectively. Medical treatment consisted of trimethoprim sulfas, flunixin meglumine or phenylbutazone, intravenous fluids, and monitoring. Twelve horses had a ceratohyoid ostectomy (bilateral in 4 horses), and 1 horse had ipsilateral stylohyoid ostectomy. Postoperative complications included laryngeal swelling and paralysis (observed on endoscopy at a later time) in 2 horses with resulting dyspnea that required an emergency tracheotomy. These 2 horses developed dysphagia that resolved within 5 days. These horses had left sided laryngeal hemiparesis (grade 2 and 3, each) observed at endoscopy on admission. Transient exacerbation of vestibular disease (marked ipsilateral leaning, head tilt, and bilateral horizontal nystagmus with fast phase contralateral to the head tilt) was observed in 9 of 13 horses.

Follow-up

Short-term follow-up examinations (1–3 months after discharge) were done in 20 horses at our institution or at the owners' property. Clinical evaluation of auditory function (clap, ringing keys or bell) showed variable results ranging from no head turn or ear prick to inconsistent response to failure to localize sound in all 20 horses. This altered behavioral response to sound was supportive of auditory loss. Neurologic improvement (vestibular, facial nerves deficits) was noted after 30–32 days in 2 of 7 horses treated medically, but they later had recurrent exacerbation of clinical signs. One of these horses was euthanized 3 months later because of the lack of resolution of signs. The other 5 horses did not show major improvement and had recurrent exacerbation of signs resulting in election of euthanasia of 3 horses. Vestibular and facial nerve deficits were still present, but substantially improved (from marked head tilt and facial droop to almost straight head posture and mild facial droop) in horses treated surgically (n = 13/13) versus being mildly improved (minimal change in observed signs) in horses treated medically (n = 3/7, the remaining 4 were euthanized).

Of the 16 surviving horses, long term follow-up (>6 months to 7 years) was possible in 15 horses (medical, n = 3/3; surgical, n = 12/13). One horse treated surgically (CHO) was euthanized because of exacerbation of clinical signs on the side contralateral to the surgery. One horse treated medically was euthanized a year after discharge caused by the lack of resolution with periods

of exacerbation of signs. The remaining 14 horses improved substantially, but had residual vestibular (n = 7) and/or facial (n = 10: mild = 6, marked = 4) nerve deficits. One horse with a 5-year follow-up developed deviation of the muzzle to the ipsilateral side which was presumed to be because of fibrosis in the ipsilateral side; facial expression and brainstem reflexes involving the facial nerve of the contralateral side were all normal. Two horses had ongoing chronic exposure keratitis which resulted in enucleation in one horse. Vestibular disease observed in 7 horses was considered mild (subtle head tilt, no apparent pathologic nystagmus, no ataxia) and did not interfere with daily activity or locomotion. Six horses that had CHO (5 with no apparent vestibular disease) were ridden again. The remaining horse that had CHO was being ridden at owner's election despite having subtle head tilt and facial droop.

Behaviorally, all 14 of the remaining horses had persistent auditory loss. Recheck BAER examinations were performed within 1–4 years after discharge in 8 horses and showed no improvement of auditory function on the affected side (bilateral [n = 5], unilateral [n = 3]). Further, auditory loss progressed (increased peak latencies and decreased amplitudes) in the remaining hearing ear in 3 horses with bilateral disease. One horse had medical treatment and 2 had CHO (unilateral CHO = 1, bilateral CHO = 1 horse). Ears with complete auditory loss remained unchanged in all 8 horses. Three horses were treated medically, and 5 surgically (unilateral stylohyoid ostectomy = 1 horse, and 4 had ceratohyoid ostectomy [unilateral = 1, bilateral = 3 horses]). Rechecking of endoscopic evaluation of the upper airway was declined by the owners because of financial constraints.

Discussion

Auditory loss is a common neurologic alteration in horses with temporohyoid osteoarthropathy.[8,9] Based on this study, auditory loss was the most common neurologic deficit (100%) identified followed by vestibular and facial nerve dysfunction (83% each). Neurogenic exposure keratitis was observed in 71% of the cases that resulted in corneal perforation and enucleation in 2 horses. Auditory loss was bilateral or unilateral in 50% of the horses with THO. In horses with bilateral BAER deficits, auditory loss was complete in one ear and partial in the contralateral ear. In horses with unilateral BAER deficits, auditory loss was complete except for one horse with partial loss. Despite observed overall neurologic improvement (facial and vestibular function) in diseased horses, auditory dysfunction persisted (clinical evaluation: short-term n = 19/19, long-term n = 16/16; BAER evaluation: long-term n = 8/8). Further, auditory loss progressed in 3 of 5 horses with bilateral disease. However, this deficit did not appear to interfere with the horses' daily activities (other than getting startled when surprised). This deficit appeared to be permanent based on a subjective interpretation of abnormal response to sound (lack of response or failure to localize sound) with subsequent confirmation with a BAER in a limited number of horses (n = 8) on a long-term follow-up. Despite the low numbers of horses and limited follow-up in this study, it appears that surgical management provided the best results in terms of neurologic function, avoided recurrence or exacerbation of signs, and in some cases resumption of previous athletic activity despite mild neurologic dysfunction, but did not improve outcome for auditory function. Further, survival was higher for horses undergoing surgical (n = 12/13) than medical (n = 2/7) intervention.

The severity of the imaging findings assessed in this study were not good indicators of presence or absence of auditory function. Two cases with bilateral auditory loss had no radiographic changes and 1 case had auditory loss contralateral to the radiographic abnormalities. This further supports the lack of sensitivity of radiographs for the diagnosis of THO.[1,2,8] Although CT has been shown to be more sensitive for the diagnosis of THO than radiography or endoscopy,[2,8] its potential use to predict auditory loss is poor as demonstrated in this study. Further, there was no association between severity of CT abnormalities and auditory loss, although all horses with CT alterations of the auditory structures had auditory loss.

Results from this study support the importance of performing a full neurologic examination which should include a behavioral hearing testing followed by BAER testing in horses with suspected compromised vestibulocochlear nerve function such as that observed in horses with THO. Normal response to sound includes turning the head or ears toward the source of sound, or moving away from the sound.[9] Subjective evaluation of hearing might detect if auditory function is present or absent, but subtle or unilateral dysfunction will likely go undetected. Failure to localize sound should raise a suspicion of auditory dysfunction as was the case in these horses. Reports of neurologic recovery in horses with THO are limited and lack assessment of auditory function; therefore full evaluation of neurologic recovery was not determined.[1,14]

The cause of THO remains unknown and various etiologies have been postulated.[1,6,7] Regardless of cause, in order to understand the association between THO and auditory deficits; the auditory pathway in relation to the temporohyoid joint must be reviewed. The auditory pathway consists of the conductive and neural pathways.[21] The conductive pathway comprises the external and middle ear.[22,23] The neural pathway consists of peripheral and central components.[23] The peripheral part includes receptors of hearing in the hair cells of the spiral organ within the cochlea, spiral ganglion, and cochlear nerve all within the petrosal portion of the temporal bone.[22] The central part comprises the cochlear nuclei (dorsal and ventral nuclei on each side in the caudal medulla oblongata), olivary nuclei (medulla), trapezoid body (pons), lateral lemniscus (pons), caudal colliculus (midbrain), medial geniculate nuclei (ventral nuclei in thalamus), and auditory cerebral cortex (temporal cortex).[24] Failure of conduction of sound waves from the external ear canal to the inner

ear is termed conductive deafness; whereas alterations of the neural structures of the auditory pathway is known as sensorineural deafness.[25] The altered anatomy of the petrous temporal bone in horses with THO results in sensorineural deafness because of the involvement of the structures of the inner ear. However, extensive anatomical alterations could also disrupt the bony ossicles.[9]

The petrous temporal bone also houses structures that regulate equilibrium (semicircular canals, utricle, and saccule), and part of the facial nerve.[3,22,26] Therefore, in addition to auditory loss, alteration of the anatomy of the petrous temporal bone can also result in vestibular and facial nerve dysfunction as observed in this study. Further, it is usually the presence of vestibular, facial, or both nerve deficits (including resulting exposure keratitis) that alerts the clinician of possibly dealing with THO. A possible explanation as to why auditory loss was found to be the most common deficit might relate to the more ventral location of the auditory structures compared to the more dorsal location of the vestibular part and tract of the facial nerve.[26] Therefore, it could become more readily obliterated by pathological changes in the ventral part of the petrous temporal bone associated to the affected temporohyoid joint. Abnormal findings of the petrous temporal bone include proliferative changes, thickening, sclerosis, remodeling, and fracture.[1,2] This permanent altered anatomy would explain why auditory loss remains despite overall neurologic improvement. Based on limited follow-up, the neurologic status of affected horses in this study improved substantially with some horses having mild deficits not noted by the owner, but noticed by the examiner (MA). Despite these deficits, horses were functional and some resumed athletic activity.

The hyoid apparatus consists of various bones (stylohyoid [2], ceratohyoid [2], basihyoid [1], lingual process [1], thyrohyoid [2]) and is an essential anatomical and functional mobile structure that supports the pharynx, larynx, and tongue.[13] Fusion of the temporohyoid joint alters biomechanical forces in horses with THO resulting in remodeling of adjacent bony structures and secondary alteration of contained soft tissue structures. Therefore, releasing this abnormal mechanical force through surgical intervention could prevent further alteration of bony and soft tissue structures.[13,15] This might explain the observed better outcome in horses undergoing surgery versus medical treatment. In addition, vestibular system adaptation could also have contributed to the observed improvement. Comparisons of outcome based on the types of surgery (SHO versus CHO) was not possible because only 1 horse had SHO versus 12 horses that had CHO.

Of interest in this study was the finding of 61% of horses (n = 11/18) with some degree of left laryngeal dysfunction as evidenced by upper airway endoscopy. Left laryngeal hemiplegia is commonly caused by left recurrent laryngeal neuropathy in horses of various breeds.[20] In this study only Quarter Horses and Thoroughbreds had this abnormal finding. This finding was contralateral to the more affected side of THO (unilateral = 2, bilateral = 7 horses), and likely coincidental. However, an association with THO cannot be entirely ruled out and might merit further investigation. Two of the horses that required emergency tracheotomy during recovery from anesthesia had pre-existing laryngeal dysfunction grades 2 and 3, respectively. Independent of pre-existing laryngeal dysfunction, trauma from surgical manipulation might result in local inflammation and dysfunction of local nerves.[27] Clinicians and surgeons must be aware of this pre-existing concurrent finding if surgical management is considered in horses with THO. Ceratohyoid ostectomy has been reported to be technically easier than stylohyoid ostectomy.[13] Further, CHO presents a low risk of bony regrowth of the ostectomy site and minimizes potential complications such as damaging the glossopharyngeal and other nerves.[13]

In summary, auditory loss is a common neurologic deficit in horses with THO that appears to be permanent regardless of medical or surgical intervention. A complete neurologic examination including a behavioral hearing test in horses with THO is essential. However, unilateral, partial, or mild auditory impairment could go unnoticed unless a BAER is performed. Further, imaging alterations are not reliably associated with auditory function. A BAER could be used as a screening test to evaluate for possible bilateral disease and determine the indication for bilateral surgical intervention. A better outcome for other neurologic deficits such as vestibular and facial nerve dysfunction was noted in horses undergoing surgical intervention than in horses treated medically. Further, faster improvement of neurologic function was noted in horses treated surgically. Based on the limited number of cases presented here, the prognosis for life in horses treated surgically appeared to be good and favorable for function that in some cases return to previous athletic activity (n = 6/13) was possible. Recurrence or exacerbation of signs was the most common reason for euthanasia in horses treated medically (n = 5/7). Prompt recognition and therapeutic intervention of disease might improve long-term outcome for life and function. However, owners must be advised that auditory function might not recover despite overall neurologic improvement and therapeutic modality. However, a larger number of horses are needed to investigate this assumption.

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Footnotes

[a] Viking IVD, Nicolet Biomedical Inc., Madison, WI
[b] VikingQuest, Nicolet Biomedical Inc., Madison, WI
[c] SPSS 10.0, SPSS Inc., Chicago, IL

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Off-label Antimicrobial Declaration: Authors declare no off-label use of antimicrobials.

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