Differential diagnosis of Facial Nerve Paralysis in horses

Harry Carslake

Philip Leverhulme Equine Hospital, University of Liverpool, Neston, Cheshire UK.

[hbc@liv.ac.uk](mailto:hbc@liv.ac.uk)

Introduction

Facial nerve paralysis is one of the more commonly encountered cranial nerve abnormalities in the horse. Unilateral paralysis has a characteristic clinical presentation and is usually relatively easy to recognise. Locating the lesion, identifying any concurrent neurological deficits and providing a prognosis are often challenging however.

This article presents an overview of the anatomy of the facial nerve, followed by a discussion of both focal and generalised causes of its dysfunction.

**Neuroanatomy**

The facial nerve (cranial nerve VII) arises from nuclei located in the ventrolateral brainstem. The proximal axons, in close proximity to those of the vestibulocochlear nerve (CN VIII), enter the medial aspect of petrous temporal bone via the internal auditory meatus. Within the petrous temporal bone the facial nerve enters the facial canal and then divides. The main somatic motor nerves continue in the facial canal exiting the skull through the stylomastoid foramen, located just caudoventral to the external acoustic meatus. Smaller visceral branches exit the temporal bone through different routes, and supply parasympathetic innervation to the lacrimal and mandibular and sublingual salivary glands, and sensory innervation to a small area of taste receptors.

After exiting the skull, the first branches of the free portion of the facial nerve are the caudal and internal auricular nerves, which innervate the muscles of the external ear. The digastric branch has minor branches to the hyoid apparatus, middle ear and caudal belly of the digastricus muscle (Fig 1).

The auriculopalpebral branch leaves from the main trunk of the facial nerve before it emerges superficially. It innervates the orbicularis oculi muscle (which closes the eye), levator anguli oculi medialis muscle (partially responsible for raising the upper lid) and the rostral muscles of the external ear. The palpebral branch crosses the zygomatic arch where it can be palpated and blocked to facilitate examination of the orbit (the palpebral nerve block).

The main trunk of the facial nerve emerges from between the parotid salivary gland and caudal masseter muscle, after which it divides into the dorsal and ventral buccal branches. In fine-haired horses these can often be seen subcutaneously traversing the masseter muscle (Fig 2), and the nerve is particularly susceptible to external trauma at this point. The dorsal and ventral buccal branches divide further to innervate the muscles of the cheeks, lips, muzzle and nostrils. Many of these branches are joined by the sensory branches of the trigeminal nerve.

**Clinical signs**

Complete loss of function of the entire facial nerve, or the buccal branches is usually easily recognised (Fig 3) but partial loss of function or paresis can produce more subtle clinical signs (Fig 4), especially if the horse is excited and has generalised increased muscle tone. Observing the horse over the stable door or administering mild sedation will sometimes help to overcome this problem.

Lesions of the buccal branches:

Horses have a very mobile muzzle in comparison to other species and marked deviation of the muzzle away from the affected side is one of the most recognisable features of unilateral paralysis of the dorsal buccal branches. The ventral buccal branch causes the lower lip to droop and together these may cause drooling, protrusion of the tongue and difficulty with feed prehension (Fig 5). Dysphagia and feed packing in the cheek can also result through an inability of the buccal musculature to move food from the cheeks to the central mouth. Loss of function of the muscles in the nostrils will cause the alar fold to droop and may cause inspiratory stridor, especially during exercise.

Noxious stimulation of the skin on the face of a normal horse should cause a local muscle twitch as well as movement of the whole head. A lack of facial twitching in response to stimulation indicates either trigeminal nerve (sensory) dysfunction, facial nerve (motor) dysfunction or both. If the sensory pathway is intact but not the facial innervation, the horse will normally move its head but not twitch its facial muscles.

Lesions of the auriculopalpebral branch

Paralysis of the orbicularis oculi muscle causes an inability to close the eye actively. Paradoxically, the normal clinical presentation of palpebral branch dysfunction in horses is mild ptosis (Fig 6), as the auriculopalpebral branch also innervates the levator anguli oculi medialis muscle, which is partially responsible for raising the upper eyelid. Dysfunction of this branch may be demonstrated by lack of resistance to raising the upper eyelid, even when gently touching the eyelid margins or cornea. In response to corneal stimulation the horse will usually quickly learn to blink with the 3rd eyelid, by retracting the globe. This helps compensate for the loss of eyelid function and impaired distribution of tears (see below). Lesions of the proximal auriculopalpebral branch will also cause reduced ear movement.

Lesions of the internal and caudal auricular branches

On the affected side the normally erect pinna will droop and there will be a reduced Pryor response (movement and rotation in response to auditory stimuli).

Lesions proximal to the stylomastoid foramen

Because of the proximity of CN VIII, lesions at this level are often accompanied by vestibulocochlear dysfunction, usually presenting as head tilt, circling, nystagmus and ventral strabismus. In addition to the loss of innervation of all the muscles of facial expression there will be sometimes be loss of parasympathetic innervation of the ipsilateral lacrimal gland and mandibular and sublingual salivary glands. Decreased tear production, accompanied by impaired tear distribution caused by paralysis of orbicularis oculi muscle can cause keratitis sicca and secondary corneal ulceration (Fig 7). Often the corneal lesion is not immediately apparent, as blepharospasm and epiphora are reduced or absent.

Paralysis of the caudal belly of digastricus muscle and the 2 small muscles within the hyoid apparatus and middle ear are unlikely to be of any clinical significance.

Central lesions

While New Zealand remains free of equine protozoal myeloencephalitis (EPM) and West Nile virus (WNV), lesions in the brainstem are very unlikely to be sufficiently focal to affect only the facial nuclei. Clinical signs of facial nerve dysfunction at the level of the facial nucleus will be always be accompanied by other central nervous system abnormalities.

Loss of the upper motor pathways in the rostral brainstem that control the motor nucleus of the facial nerve will also be accompanied other abnormalities of the central nervous system, but can result in a bland, grimacing expression (Fig 8). Hypertonia and hyperreflexia of the facial muscles may also be detectable with upper motor neurone lesions.

An intermittent, unilateral and usually mild spasm of the facial muscles (often termed ‘facial tic’) has been recognised in horses. It is usually most apparent over the muzzle or eyelids. In people it can be caused by compression of the peripheral nerve, brainstem lesions, or vascular lesions, but in horses (and people) it is usually idiopathic.

In addition to clinical signs specific to the facial nerve, other neuropathies might be evident, especially if the lesion is central or generalised. A full neurological examination should be performed whenever facial nerve dysfunction is detected.

**Differential diagnoses of facial nerve paralysis in horses**

1. Peripheral nerve trauma

The superficial course of the buccal and auriculopalpebral branches of the facial nerve renders them highly susceptible to external trauma. Most commonly this will be caused by a horse pulling back on its head collar, by blunt trauma to the head or by prolonged lateral recumbency during general anaesthesia. Trauma resulting in fracture of the petrous temporal bone is also likely to cause facial nerve dysfunction, normally accompanied by peripheral vestibular signs. Paralysis caused by trauma is most often unilateral and its duration depends on the degree of peripheral nerve damage (see box 1). A brief period of twitching and hyperreflexia may occasionally be seen as a precursor to paralysis.

Box:

**Peripheral nerve trauma**

Any peripheral nerve trauma can be classified into 3 syndromes, based on the degree of injury.

1. **Neurapraxia** is a transient interruption of nerve function in the absence of structural change, usually caused by concussion or interrupted blood supply. Function usually returns spontaneously after a few days, although it can take up to 3-4 weeks. There is no permanent loss of function.
2. **Axonotmesis** is when the axon is damaged but the endoneurium and myelin sheath (Schwann cells) remain intact. The distal axon degenerates (Wallerian degeneration) and is followed by regeneration from the site of the lesion at a rate of approx. 2 cm per month. Neurogenic atrophy of the paralysed muscle will result, which can lead to fibrosis, contracture and long term dysfunction. Adequate function normally returns with facial nerve lesions, however for a lesion at the proximal buccal branch this could take several months.

3. **Neurotmesis** refers to complete severance of the axon and its nerve sheath. It is rare unless there is penetration of the skin or fracture of an adjacent bone. Regeneration of some branches may occur, at a rate of approx. 2cm per month. This is unpredictable however and at best only partial function of the nerve returns.

1. Compressive or inflammatory lesions adjacent to the nerve

In addition to the facial canal, the petrous temporal bone also contains the middle and inner ear. Otitis media +/- interna can result in damage to adjacent structures, including the facial nerve (Fig 9). Space-occupying or infiltrative neoplasms or other lesions can also result in facial nerve paralysis. (Fig 10). The proximity of the facial canal to the guttural pouch means that lesions (such as mycotic plaques) affecting the dorsal pouch can also cause facial nerve paralysis.

1. Congenital

There is one report of congenital unilateral facial nerve paralysis in a Warmblood filly (Schon et al 2017).

4. Temporohyoid osteoarthropathy (THO) (Fig 11)

This condition is most commonly seen in middle-aged horses, and its aetiology is not fully understood. A proliferative osteoarthropathy of the temporohyoid joint progresses to ankylosis and subsequent periarticular stress fractures of the petrous temporal bone. Otitis interna/media has been strongly suspected as the primary cause in some cases. In others a non-septic degenerative arthropathy, proposed to be caused by excessive movement of the joint and/or head trauma, leads to proliferative ankylosis and fracture of the petrous temporal bone.

Prior to the onset of neurological signs, clinical signs of THO may include pain on palpation of the base of the ear, difficulty masticating, headshaking or other behavioural abnormalities. Subsequent fracture of the petrous temporal bone is usually accompanied by acute onset facial nerve dysfunction affecting all branches, and often concurrent peripheral vestibular dysfunction. Haemorrhage into the inner and middle ear is common and suppurative meningitis may rarely occur via extension of septic otitis interna.

Diagnosis of THO is based on observation of the clinical signs described above and use of diagnostic imaging. Endoscopy of the guttural pouches will frequently reveal enlargement of the proximal end of the stylohyoid bone. Dorsoventral radiographs of the head are also useful for detecting bony changes in the temporohyoid region and the tympanic bulla. If available computed tomography (CT) provides superior imaging of this region. If fever and depression accompany signs of THO, cerebrospinal fluid analysis is advisable to exclude secondary septic meningitis.

Medical treatment of THO consists of anti-inflammatory drugs and antimicrobial drugs in cases of otitis interna/media or septic meningitis. Ipsilateral partial stylohyoidectomy, or more recently ceratohyoidectomy have been described as surgical treatments also for THO.

4. Polyneuritis equi (PNE)

PNE (previously referred to as neuritis of the cauda equine) is a progressive granulomatous inflammatory disorder affecting the cauda equina, cranial nerves and occasionally other peripheral nerves. An immune mediated aetiology secondary to an infective agent has been proposed but not validated as the initiating cause.

PNE usually affects the cauda equina first, starting with hyperaesthesia and tail rubbing which is frequently be missed by the owner. This normally progresses to paralysis and anaesthesia of the tail, anus, rectum, perineum and bladder. Poor tail tone, faecal and urinary incontinence, and prolapse of the penis are common presenting complaints.

Cranial nerve dysfunction normally occurs after cauda equina deficits, however they can sometimes be the presenting sign. The most commonly affected cranial nerves are V, VII and VIII, producing atrophy and weakness of the muscles of mastication, anaesthesia of the face and vestibular signs; as well as the clinical signs of facial nerve dysfunction described above. Diagnosis of PNE is by exclusion and treatment is palliative.

5. Generalised peripheral neuropathies

**Equine Motor Neurone disease (EMND)**

First described in 1990, EMND is a degenerative disorder affecting the somatic lower motor neurones. Muscle wasting and generalised weakness are the most common presentations, and facial nerve paralysis, if present, is likely to be of secondary importance. Diagnosis antemortem is challenging and is usually based on clinical presentation, histopathology of the tail head muscle or the spinal accessory nerve and sometimes deposition of brown lipofuscin in the tapetal fundus.

**Lead poisoning**

Horses are selective eaters and so acute lead poisoning is unusual. Chronic, low dose exposure is more common and usually occurs via pasture contamination. Pharnygeal and laryngeal paralysis are common early presenting signs, causing dysphagia and roaring. Generalised motor weakness (including the facial nerve), weight loss, and depression can also occur.

**Botulism**

This polyneuropathy is usually associated with ingestion of *Clostridium botulinum* toxin-contaminated feed. The toxin blocks the release of acetylcholine, resulting in motor and parasympathetic nerve dysfunction. Weakness of the muscles supplied by the facial nerve can be seen, but usually as part of widespread lower motor neurone weakness. Dysphagia is a common presenting sign. Muscle trembling often occurs, and in foals the condition has been referred to as ‘Shaker foal syndrome’.

5. Central Lesions

Central nervous system (CNS) lesions are rarely focal enough to cause dysfunction of the facial nerve only; the exceptions being equine protozoal myeloencephalitis (EPM) and West Nile virus (WNV) which are both exotic to New Zealand. Central lesions are likely to be associated with other signs such as other cranial nerve deficits, altered mentation and generalised sensory, motor or autonomic deficits.

Head trauma may cause a range of neurological signs attributable to the central nervous system. Basioccipital, basisphenoid and petrous temporal bone fractures, normally occurring when the horse flips over backwards and hits its poll, commonly cause facial paralysis at the level of the brainstem or proximal peripheral nerve. Blood and/or cerebrospinal fluid from the ears or nose is highly suggestive of a fracture in this region, which can be difficult to detect radiographically.

Brain stem neoplasia, abscessation, and verminous meningoencephalitis are rare causes of facial nerve paralysis, and are very likely to be accompanied other signs of CNS dysfunction. If EPM or WNV are suspected cerebrospinal fluid CSF) cytological analysis and serum and CSF immunological testing can be indicated to help reach a diagnosis.

Box 2

**Horner’s Syndrome (Fig 12)**

Horner’s syndrome doesn’t actually involve any of the cranial nerves; however it is commonly confused with facial nerve paralysis. It describes a collection of clinical signs associated with loss of sympathetic innervation to the head. The lesion may occur in the brainstem, the cervicothoracic spinal cord, the cervical sympathetic trunk or the postganglionic sympathetic fibres of the head. A common aetiology is perivascular injections affecting the cervical sympathetic trunk. One of the most consistent clinical signs is ipsilateral ptosis and lowering of the upper eyelash angle, caused by loss of tone in the sympathetically innervated smooth muscle of the upper eyelid and which can be confused with loss of motor innervation of orbicularis oculi muscle. Ipsilateral miosis and localised sweating are the other clinical signs of Horner’s syndrome in horses, which are not normally seen with facial nerve dysfunction.

# Summary

Paralysis of the facial nerve normally has a characteristic clinical presentation. It is frequently part of a wider neuropathy, so in each case a full neurological examination is recommended to provide an accurate diagnosis and prognosis.

**Acknowledgement**

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**Figure Legends**

Fig 1. Branches of the free portion of the left facial nerve after it emerges from the stylomastoid foramen

Fig 2. Buccal branches of the facial nerve visible subcutaneously over the masseter.

Fig 3. Paralysis of the left facial nerve caused by fracture of the petrous temporal bone secondary to temporohyoid osteoarthropathy. There is ventral deviation of the left ear, ptosis of the left eye and deviation of the muzzle to the right.

Fig 4. Right-sided facial nerve paresis, with subtle muzzle deviation and asymmetric ear and eyelid positions.

Fig 5 Paralysis of the right buccal branches, caused by trauma to the face more than 2 years previously. Drooping of the lower lip and muzzle deviation result in the inability to maintain the tongue in the mouth.

Fig 6. Ptosis of the left eye and muzzle deviation after temporary paralysis of the facial nerve at the caudal ramus of the mandible for ophthalmic examination. Note the markedly steeper eye lash angle on the left side.

Fig 7. Corneal ulceration and secondary anterior uveitis after right-sided temporohyoid osteoarthropathy. Paralysis of the eyelid reduces distribution of the tear film.

Fig 8. A horse with a lesion in the diencephalon causing dysfunction of the upper motor neurones of the facial nerve. The horse has a bland facial expression, but normal reflexes and tone. Photograph: Joe Mayhew

Fig 9. A transverse CT image at the level of the petrous temporal bones showing increased attenuation in the right ear canal and tympanic cavity, consistent with right-sided otitis media. The arrow marks the right external acoustic meatus. Soft tissue swelling and erythema were visible around the dorsal stylohyoid bone on endoscopy of the guttural pouch. The horse presented with right-sided facial nerve paralysis, and secondary corneal ulceration.

Fig 10a Horse presenting with acute onset right-sided facial nerve paralysis and dysphagia of 2 weeks’ duration.

Fig 10b Endoscopic image of the left guttural pouch showing a normal stylohyoid bone.

Fig 10c Endoscopic image of the right guttural pouch showing swelling around the proximal stylohyoid bone and its articulation to the petrous temporal bone.

Fig 10d Transverse CT image at the level of the petrous temporal bones, demonstrating soft tissue swelling in the temporohyoid region and extensive lysis of the temporal bone. On post-mortem examination the soft tissue mass was consistent with a squamous cell carcinoma.

Fig 11a. Horse presenting with left-sided peripheral facial nerve paralysis affecting all the peripheral branches.

Fig 11b. Endoscopic image of the right guttural pouch, showing a normal temporohyoid region.

Fig 11c Endoscopic image of the left guttural pouch showing marked, diffuse thickening of the stylohyoid bone.

Fig 11d Cropped 3D reconstruction of a CT of the caudal skull and hyoid apparatus showing bony thickening of the left stylohyoid bone and ankylosis of the temporohyoid joint.

Fig 12a Unilateral sweating and ptosis consistent with left sided Horner’s syndrome.

Fig 12b: Mild miosis of the left pupil compared to the right (Fig 12c)

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