Trigeminal-mediated headshaking: A diagnostic challenge

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The term ‘equine trigeminal-mediated headshaking’ (TMHS) has replaced the previously used diagnosis of ‘idiopathic headshaking’ to reflect the clinical signs, which are considered to characterise trigeminal neuropathic pain (Pickles et al., 2014). This diagnosis is reserved for horses with a presumed functional, rather than structural, disorder of the trigeminal nerve, as distinct from horses such as those described in the case series by Ogden et al. (2022) where gross pathology, in this case trauma of trigeminal nerve branches, induces clinical signs of headshaking.

Other pathological or behavioural causes of headshaking behaviour, as listed by Ogden et al. (2022), have also been described. It is only by exclusion of these other causes, or via invasive neurophysiological studies which are outwith the realm of clinical practice, that a diagnosis of TMHS can be confidently assigned. A gross pathological cause of headshaking behaviour is only rarely diagnosed (Pickles et al., 2014), and therefore, it can be tempting to try and take shortcuts in the diagnostic procedure; however, if we do not look for these other causes, we will surely not find them. Indeed, Ogden et al. (2022) report that the traumatic skin lesion in one of their cases was disregarded as self-inflicted from headshaking-induced muzzle rubbing by the initial treating veterinary surgeon. Careful anamnesis, physical examination and often diagnostic imaging are therefore required for correct diagnosis (Pickles et al., 2014).

Previously, ‘idiopathic headshaking’ was diagnosed in 97% of headshakers presenting to equine hospitals; however, with the advent of advanced imaging modalities such as computed tomography (CT), causative pathology is identified in up to 10% (Fairburn et al., 2022). This emphasises the merits of these ancillary diagnostic techniques and suggests that, perhaps, we may even still be overdiagnosing true, functional, trigeminal-mediated headshaking.

Conclusive involvement of the trigeminal nerve in headshaking has only been confirmed within the last 10 years (Aleman et al., 2013; Newton, 2001; Roberts et al., 2017) and suggests that, perhaps, we may even still be overdiagnosing true, functional, trigeminal-mediated headshaking.

The aberrant trigeminal nerve function documented in TMHS, the seasonality of clinical signs in many headshaking horses and the absence of gross or histopathological lesions in the trigeminal nerves and ganglia from headshaking horses (Aleman et al., 2013) allows accurate mapping of the areas of altered sensory perception (Maier et al., 2010). A standardised test is performed to determine thresholds for thermal and mechanical stimuli, pain thresholds for several stimulus modalities, suprathreshold pinprick tests and wind-up, plus a specific assessment for dynamic mechanical allodynia and paradoxical heat sensation. Calibrated stimuli are applied to capture perception and pain thresholds providing information on patterns of sensory loss (for the functioning of the thick and thin nerve fibres), as well as a gain of function (hyperalgesia, allodynia and hyperpathia) with simultaneous detection of cutaneous and
deep tissue sensibility, which can be compared against a reference population (Mücke et al., 2021). Such testing would be ideal to aid the diagnosis of TMHS; however, as it entails the cooperation of the subject being examined to report findings, it is challenging in the horse. The author has had difficulty achieving consistent results (unpublished data); however, Veres-Nyéki et al. (2021) report reliable measurements for QST of the equine face of healthy horses, which was well-tolerated. The face did not require shaving or clipping, but age had a significant effect on tactile sensory and mechanical and thermal nociceptive threshold (p = 0.001) with threshold values increasing with age. The most consistent values were reported over the nostril (tactile sensory threshold), temporomandibular joint (mechanical nociceptive threshold) and supraorbital foramen (thermal nociceptive threshold). Such testing of horses requires a subjective assessment of the horse’s response to stimulation by an observer, rather than reporting by the individual themselves, and thus has limitations compared with humans. However, based on the results reported by Veres-Nyéki et al. (2021), the assessment of trigeminal sensory function, by von Frey filament stimulation of the nostril area, in control horses and horses with TMHS is warranted.

The hunt is now on for the cause of the aberrant trigeminal nerve activity in equine TMHS, which, thus far, remains frustratingly elusive. The role of gonadotrophin fluctuations (Sheldon et al., 2019a), dietary cation-anion balance (Sheldon et al., 2018, 2019b, 2019c) and caecal microbiota (Aleman et al., 2022) has recently been investigated for involvement in the aetiology of TMHS. Finally, after decades of inactivity, there has been a resurgence of interest in demystifying this syndrome.

CONFLICTS OF INTEREST
No conflicts of interest have been declared.

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REFERENCES


