London College of Animal Osteopathy

Thesis

Title:

Osteopathic manipulative therapy in the management of Trigeminal mediated headshaking in horses.

A narrative literature review undertaken via a systemic process.

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Abstract

Background

Trigeminal Mediated Headshaking (TMHS) in horses is thought to be a severe neuropathic facial pain with an unknown aetiology. It is identified by clinical signs such as a vertical, involuntary head ‘flicking’, nasal irritation and striking out with the forelimb. Most are aggravated by exercise, although some are affected at rest. TMHS horses largely have a poor prognosis. Therapeutic treatment currently includes supplementation, EquiPENS, pharmacological and surgical intervention, however if no significant therapeutic benefit is found, euthanasia is indicated and advised.

Osteopathy is a manual therapy modality used to treat dysfunction of the neuromusculoskeletal system. It seeks to identify somatic dysfunction in the body that may contribute to a change in physiological function, consequently causing clinical signs and symptoms of disease.

The aim of this review is to understand the current pathophysiology of tissues associated with THMS and understand how Osteopathic treatment (OMT) may aid the return to healthy physiology.

Methods

Search terms used:

("trigeminal mediated head shaking" OR "idiopathic head shaking" OR "TMHS") AND ("horses" OR "equine")

Databases searched include PubMed, Science Direct, Google Scholar. Anatomy and Neurology books were sought to understand the anatomical relevance in TMHS.

Results

The appraisal of associated tissues and apparent dysfunction identified in THMS combined with the pathophysiology of somatic dysfunction helps to understand a reasonable hypothesis for the aetiology of disease.

Conclusion

Information gathered forms a hypothesis on the aetiology of TMHS and the pathophysiological changes apparent in somatic dysfunction suggesting a connection between the two subjects, potentiating a secondary hypothesis that OMT could be considered in restoring pathological physiology to health.

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Methods

Search terms used for the subject of the review were as follows:

("trigeminal mediated head shaking" OR "idiopathic head shaking" OR "TMHS") AND ("horses" OR "equine")

Databases such as PubMed, Scopus, Google Scholar and Science Direct were reviewed, within 2014-2025. References associated with search findings were also reviewed.

List of Abbreviations

TMHS	Trigeminal Mediated Headshaking
SD	Somatic dysfunction
OMT	Osteopathic Manipulative Treatment
CNV	Trigeminal Nerve
V1	Ophthalmic branch
V2	Axillary branch
V3	Mandibular branch
HRE-S	History, Rest, Exercise - Score
Mg	Magnesium
Ca	Calcium
OAJ	Occipitoatlantal Joint
C1-4	Cervical Vertebrae 1-4
PN	Peripheral Nerve
CNS	Central Nervous System
SCG	Superior Cervical Ganglia
CNX	Vagus Nerve
GOT	General Osteopathic Treatment
ECOP	Educational Council of Osteopathic Principles
TART	Texture changes, Asymmetry, Restriction, Tenderness
GIT	Gastrointestinal Tract

1.0 Introduction

1.1 Purpose

The purpose of this academic thesis is to review existing literature on the potential for osteopathic medicine to be considered in the management of TMHS in horses.

This review will cover pathophysiology along with current diagnostic and treatment methods available for TMHS with consideration to the apparent physiological changes. Consequently, leading the discussion into the relevant physiological effect of osteopathic somatic dysfunction (SD), treatment methods including osteopathic manipulative treatment (OMT) and how they can be applied to affected neuromusculoskeletal regions.

These topics combined suggest the hypothesis that OMT on the relevant anatomical connections associated with TMHS, could aid pathological tissue to return to its' normal healthy physiology, thus reduction if not resolution of disease.

1.2 Overview

TMHS is thought to be a severe neuropathic facial pain of the horse of idiopathic origin, closely compared to Trigeminal Neuralgia in humans.

It is characterised by clinical symptoms such as mild-violent, frequent, involuntary, vertical flicking or jerking motions of the head. There can be hyperalgesia, localised or globalised allodynia, with irritability of the sinuses indicated by rubbing, snorting/ sneezing, inflamed mucosa, sensitivity to light, wind and in severe cases, striking with the forelimb. Some show signs of elevated stress, anxious expression with a change in head carriage, in some cases becoming unrideable and difficult to handle (Roberts, 2019).

2.0 Literature review

2.1 *Prevalence*

There is currently very little understanding of TMHS, affecting approximately 1-1.5% (Roberts, 2019) of the equine population in the UK, the aetiology and pathophysiology are currently unknown.

Research has collated triggers of TMHS symptoms including bright sunlight, wind, pollen and seasonal changes which were reported in exacerbation of symptoms. Most horses are

precipitated by exercise, only a small portion are affected at rest. The population shows a mean age of onset as 9.6 years, geldings more affected than mares, while all breeds can be affected (Bell et al., 2024).

2.2 Existing Research

Current research is indicative of functional abnormality within the neurological communication and function of the Trigeminal Nerve (CNV). Therefore, suggesting atypical physiology present at its origin, causing downstream irritation of associated structures (Aleman et al., 2013; Pickles et al., 2011).

Objective research has shown the maxillary branch (V2) of the CNV can be affected with abnormal electrophysiology present in TMHS horses. Studies have shown anaesthesia via the V2 posterior ethmoidal nerve, gave 90-100% improvement of symptoms. The threshold for infraorbital branch firing was significantly reduced, confirming hyperexcitability of the nerve is involved in the aetiology of disease (Nessler et al., 2024).

There is evidence showing lack of structural change, such as demyelination of the nerve root, which has been shown in cases of human Trigeminal Neuralgia. Although, the sample size for this study was small, and results were found via postmortem examination (Roberts et al., 2017).

Incidental findings of dietary factors, seasonal changes and photophobic components also exist, suggesting a functional alteration of the sensory pathway (Aleman et al., 2013; Nessler et al., 2024). Given the varying symptoms recorded by cases of THMS, we can establish an understanding that any segment of CNV may be affected due individual presentation of clinical signs.

Considering TMHS horses can be affect by seasonal change, research has been explored examining Luteinizing hormone concentrations in TMHS horses. However, there were no difference in levels to the control group (Sheldon et al., 2019). Therefore, hormones may not have a direct affect to the nerve hyperexcitability.

Studies have demonstrated dorsal root ganglion neurons have properties that can fire abnormally. An increase in subthreshold oscillations in resting membrane potential of A-type neurons can increase activity, subsequently activating adjacent hyperexcitable C fibres. If enough neurons fire together, a pain signal can be generated. Repeated firing can cause progressive damage to nerve endings, known as central sensitisation (Aleman et al., 2015).

2.3 Diagnosis

Primary TMHS otherwise known as ‘idiopathic headshaking’ is diagnosed by eliminating other pre-existing differential diagnoses that could contribute to head shaking. Differentials include oral, ophthalmologic, orthopaedic, nuchal bursitis, neurological pathology, infectious, parasitic, viral manifestation, poor training/riding/tack fit, which then treated, TMHS symptoms were eradicated. Suggesting symptoms were secondary to underlying pathology or management (Perrier et al., 2023).

Kloock et al., 2022 analysed the impact of diagnostic procedures for primary TMHS, where clinically relevant findings were efficiently identified via computed tomography (CT) and causality found via local anaesthesia/ targeted therapy to identify potential affected regions.

Kloock et al., 2024 researched the use of History, Rest and Exercise Score (HRE-S) to evaluate disease severity. HRE-S is shown to be a valid tool in evaluating severity of TMHS disease, irrespective of the observer’s experience.

Similarly, an accelerometer, analysing gait has been used successfully to objectively measure TMHS and can help to differentiate between TMHS and non-TMHS horses (Pickles et al., 2024). Although could be considered an ethical debate due to exacerbation of symptoms.

2.4 Current Treatment Methods

Neuromodulation via percutaneous electrical nerve stimulation (EquiPENS) uses a probe inserted in the infraorbital foramen. This is the accessible, sensory part of the maxillary branch of the CNV and is currently first-line treatment. EquiPENS is thought to work via gate control theory but requires greater understanding of aberrant neurophysiology (Pickles, 2019).

Limited by the unknown aetiopathogenesis and current level of understanding of neuromodulation, there are no predictors of outcome. However, 50-53% of participants went into remission for 2 days-156 weeks (Roberts et al., 2016, 2020), which reinforces that symptoms originate from a neuropathological state of hyperexcitability (Pickles et al., 2011).

Newton et al., 2000 reviewed varying approaches to treatment. Infraorbital anaesthesia and rhizotomy had no effect in 6/7 cases, tinted lenses showed no long-term benefit, suggesting photic aetiology unlikely to be present. Cyproheptadine has proven ineffective, though with addition of or use of carbamazepine proved effective in 88% of cases. Dosage, duration and

outcome cannot yet be predicted due to a lack of pharmacokinetic research for horses (Roberts et al., 2009). Limitations could be as per the cost of this line of treatment.

Studies have shown intravenous magnesium sulphate (Mg) or oral magnesium with or without boron supplementation has shown great effect, increasing plasma total and ionised Mg concentrations thus decreasing headshaking symptoms (Sheldon et al., 2019). Advocating using elements to support systemic nerve function can have a beneficial therapeutic effect.

DeClue Equine 2019 reviewed chronological existing research on history and treatment methods, giving front-line experience and their opinion on aetiology of disease. Treatment involves a (non-specified) injection of the occipitoatlantal joint (OAJ) with the addition of shockwave therapy, stating they are treating horses with a 100% success rate by addressing the origin of disease.

3.0 Physiological and anatomical considerations

3.1 *Anatomical relevance - TMHS*

Eliminating pre-existing pathology measurable by clinical diagnostics with knowledge on the anatomical regions, function, and visceral connections affected by primary TMHS gives rise to investigation of structural and functional pathways of the nerve origin to discover not only symptomatic relief, but to treat the origin of which it may arise.

3.2 Anatomy

All anatomical components must be considered to fully begin to understand pathology including bone, myofascial, neurological, cutaneous, ligamentous and all other structures. The anatomy associated with TMHS is extensive, only key parts are discussed as per limitations of this review. Further investigation is necessary to obtain a comprehensive understanding of anatomy and physiology.

3.2.1 The cranium

The poll, part of the cranium, consists of the occipital bone articulating with the first cervical vertebrae (C1) or atlas. The External occipital protuberance lies between the nuchal crest and foramen magnum at the top of the poll. The nuchal ligament attaches here and consists of two parts. Dorsally the funicular part, ventrally the laminar part extending from the external occipital protuberance to Thoracic 3-5 vertebrae. Dorsal to the atlas/axis lies the nuchal

bursae. Ventral to the occiput, the foramen magnum, exit for the spinal cord, basilar artery, spinal accessory nerves, vertebral and spinal venous system through the occipital cavity, and jugular foramen where cranial nerves IX, X and XI pass through, in close anatomical relationship of the occiput (Budras et al., n.d.).

3.2.2 Myofascial physiological characteristics

Recent study of myofascial kinetic lines by Schultz DVM et al., 2021, shows seven kinetic chains that attach to the occiput. Fascia can contract as a protection mechanism from acute stretch, compression or overuse in attempt to stabilise the structure. Dysfunction by trauma within the fascia can cause fibroblast differentiation into myofibroblasts, long-duration contractility without nervous stimulation, initially known from wound healing. Differentiation of myofibroblasts can cause stiffness within fascia thus mechanical tension. Production of pharmacological components such as histamine, oxytocin and nitric oxide, together with cytokines and growth factors exists. A low pH of the tissue causes contraction, so diet may be indicated here. Sympathetic stimulus can also induce contractility, so a stressful horse may present with fascial stiffness.

3.2.3 The nervous system

The nervous system is made up of the central and peripheral nervous systems. Peripheral nerves (PN) are extensions of the central nervous system (CNS), the brain and spinal cord. PN's have many sensory receptors which detect internal and external stimuli, transmitting ascending impulses to the CNS for interpretation. Ascending information sends feedback from nociception, temperature, light touch, proprioception and autonomic impulses such as baroreceptors and chemoreceptors. All signals for the head travel through the CNV, which is then processed through the trigeminal sensory nucleus of the brainstem (Aleman et al., 2015).

3.2.4 Trigeminal Nerve

The Trigeminal Nerve is a peripheral nerve. The largest of the cranial nerves, with sensory and motor function. It has three major divisions, Ophthalmic (V1), Maxillary (V2) and Mandibular (V3), each differentiating into subdivisions. V3 innervates cutaneous and oral mucus membrane, carrying motor fibres to muscles of mastication including pterygoid muscles, tensor tympani and tensor veli palatini of the ear. Extending branches supply the mylohyoideus, digastric muscles, lower teeth and skin of the lip. Further sensory branches supply the buccal, sublingual and mandibular glands and the auriculotemporal branch

supplies the cutaneous temporal region and parotid gland. V2 is sensory via the infraorbital nerve situated in the infraorbital foramen, a common site for anaesthesia, midway along the maxilla. V2 supplies the upper teeth, lips, nostrils and nasal vestibule. The masseteric nerve, a branch of V3, enters the masseter immediately rostral to the temporomandibular joint (Budras et al., n.d.).

3.2.5 The Superior Cervical Ganglia

The superior cervical ganglia (SCG) are a bundle of nerve fibres stemming from the nerve roots from C0-4. SCG traverses in the neck in close association with the vagosympathetic trunk, located in the retro styloid space near the base of the skull, in the bifurcation of the common carotid artery, extending from cranial base to fourth cervical vertebrae (C4) (Aleman et al., 2015).

SCG is bordered by various structures. Laterally digastricus and sternocleidomastoid muscles, lying against transverse processes C2-3 and prevertebral muscles. Anteriorly styloid muscles and the jugular carotid neurovisceral bundle, medially the pharynx (Barral & Croibier, 2009; Gardner & Bunge, 2005).

It provides sympathetic innervation to lacrimal, salivary, pineal and thyroid glands and the carotid plexus which controls blood vessels of the head regulating vasoconstriction, including eyes for pupillary dilation. Lesions in this area can affect the Vagus nerve (CNX), causing hyperaemia of conjunctiva, retina, nasopharyngeal membranes, inner and middle ear, meninges, cerebral cortex and pituitary gland. Motor disturbances to the heart, diaphragm, and stomach. Dilated pupils, increased acidity of gastric secretions due to increase in pyloric sphincter tone (Burns & McConnell, 2022).

Research has shown trauma, in this case a fracture of the occipital condyle produced a rotation of the atlas and a bone fragment dorsal to the guttural pouches on radiological examination. Due to the anatomical situation, the trauma caused delayed onset paralysis of the cranial nerves affected, distinguished by clinical presentation (Martin-Giménez et al., 2019).

This is indicative that trauma (i.e. spinal lesions) can directly affect pathophysiology. Together with physiological adaptations from spinal lesions discussed below (4.0), this review will focus on the potential for trauma inhibiting CNV function.

3.3 Neurology

Anatomy and function of the nervous system is key in understanding functional vs dysfunctional to recognise clinical signs of disorder and identify their location. The nervous system is composed of neurons.

3.3.1 Neurons

Neurons communicate via chemical and electrical signalling. There are three parts to a neuron that convey an electrical impulse: dendrites, cell body and the axon. Conduction from one neuron to another is initiated by charged particles across the synaptic cleft. A presynaptic neuron releases a neurotransmitter which is received by the appropriate neurotransmitter receptor. Chemicals critical in maintaining homeostasis for the nervous system and resting membrane potential include Calcium (Ca) and Magnesium (Mg), which preserve stability and excitability of the nerve cell. (Barral & Croibier, 2009; Gardner & Bunge, 2005).

3.3.2 Magnesium

Magnesium is essential for glutamate regulation, an excitatory neurotransmitter. Low levels of Mg may potentiate glutaminergic neurotransmission, leading to neuronal excitotoxicity and excitation of nerves. As previously discussed, a dysfunction identified in TMHS with CNV hyperexcitability, a decreased threshold to which it fires (Kloock et al., 2022). This correlates to the supplementation of Mg to increase plasma levels (Sheldon et al., 2019).

3.3.3 Calcium

Calcium is essential in nerve conduction. Voltage gated Ca channels open on the neuron membrane causing an influx into the cell. Increase in intracellular Ca triggers neurotransmitters to fuse with the membrane at the synapse, which travel across the synaptic cleft and bind to specific receptors which can either excite or inhibit the postsynaptic neuron. Ca mediated processes ensure rapid and efficient neuronal transmission (Südhof, 2012; Vizi, 1979).

Low Ca levels, hypocalcaemia can cause peripheral nerve excitability, thus spontaneous firing. Further pathogenesis can lead to Tetany due to electrolyte imbalance, peripheral nerve degeneration or neuropathy (Wu et al., 2023).

3.4 The Gut

Facilitation of the SCG can affect CNX, increasing acidity of gastric secretion in the gut. Increasing research on the gut-brain-axis reveals the connection between stasis of gut microbiota and its relationship to immune, nervous and metabolic systems, including production or consumption of neurotransmitters and their effect on host physiology. In humans, studies have shown without required microbiota, an increased response to stress. The gut bacteria can influence the hypothalamic-pituitary-adrenal axis, CNX stimulation, effect permeability of the blood-brain-barrier, secretion of short-chain fatty acids and finally modulation of neurotransmitters (Strandwitz, 2018).

3.4.1 Gut Bacteria

Specific gut bacteria produce enzymes that help synthesise or are a precursor to excitatory neurotransmitters such as Glutamate. Some of which include, *lactobacillus plantarum, Bacteroides vulgatus, Escherichia coli and staphylococcus*. Most of which, can survive within an acidic gut (Chen et al., 2021; Smith, 2003).

Research has shown caecal microbiota in TMHS horses was the same to that of the control group. However, they were significantly different with an abundance of Methanocorpusculum spp. (Aleman et al., 2022).

Gut microorganisms produce metabolites which are vital for host metabolic processes, including function of the gastrointestinal tract (GIT). Methanocorpusculum spp produces methane by converting carbon dioxide and hydrogen generated by fermentation of carbohydrates into methane and promotes ATP synthesis in anaerobic bacteria in the gut microbiota (Guindo et al., 2020; Krishnamurthy et al., 2023).

These findings could suggest that TMHS horses have an acidic GIT, which could have a reciprocal relationship to spinal lesioning at the OAJ and SCG. With the interrelationship of excitatory neurotransmitter production this could potentiate hyperexcitability of the nervous system.

3.4.2 Diet

Carbohydrates are mostly broken down and absorbed in the small intestine. Excessive carbohydrates in the diet pass to the hindgut causing accumulation and production of lactic acid, creating a significant reduction in pH. Accumulation of lactic acid can irritate and

damage gut mucosa and alter the permeability to toxins. A decrease in pH can lead to hindgut acidosis, if the pH remains below 5.8 over an extended period, the epithelial lining may be damaged, thus nutrients are not fully absorbed, with significant fermentation of the caecum (Dicks et al., 2014).

This proposes future research of the horses GIT stasis and pH balance due to its potential effect on the nervous system.

4.0 The Osteopathic approach

4.1 Background

In 1874, Dr Andrew Taylor Still, founder of Osteopathy suggested the hypothesis that "rational therapy is based upon an understanding of body unity, self-regulatory mechanisms and interrelationship of structure and function" (Paulus, 2013).

4.2 Principles

The principles and philosophy of OMT formed a basis for General Osteopathic Treatment (GOT), which can be used to diagnose and directly treat somatic dysfunction (SD), also known as an Osteopathic lesion. The Educational Council on Osteopathic Principles (ECOP) defines SD as "impairment or altered function of the somatic system, including the skeletal, arthrodial, myofascial and related vascular, lymphatic and neural components". SD can be identified via using the principal 'TART', texture changes, asymmetry, restriction of motion and tenderness. The altered structure of SD is reciprocally related to the fluidity and efficiency of function (Grolaux et al., 2021).

4.3 Spinal lesion - pathophysiology

Allan, n.d. conducted a study by artificially inducing a spinal lesion to examine the physiological changes associated with SD.

4.3.1 Nerve pathology

Research demonstrated that nerve roots, branches, and sympathetic supplies, mostly vasomotor, at the affected spinal level experienced pathological changes. Various cell groups within the grey matter were disrupted, some showing atrophy or inflammatory changes. Axon degeneration was observed extending above and below the lesion site, traced back to nerve

centres of the spinal cord, posterior root ganglion, and sympathetic nerves, affecting up to two-thirds of the axons (Allan, n.d.).

4.3.2 Vascular pathology

Pathological changes were noted in blood vessels, specifically the outer adventitia of arterioles, capillaries, veins, and arteries. Blood vessels were damaged throughout their layers, from endothelial cells to muscle fibres and surrounding tissues. Blood corpuscles, including plasma and leukocytes were found to have escaped due to diapedesis and haemorrhage, indicating adaptive or pathological local changes. Hyperaemia, primarily affecting the grey matter of the cord, was especially pronounced in the posterior and medial anterior horns leading to disturbance in both sensory and motor nuclei. Hyperaemic arteritis was noted in smaller arteries due to local vasomotor changes, while vascular disturbances were apparent in sympathetic blood vessels. Circulatory changes affected organs innervated by these vasomotor nerves, circulatory disturbances varied among different cell groups, with ischemia being most pronounced at the lesion site and gradually diminishing above and below it. Congestion, along with diapedesis was evident in the anterior and posterior spinal cord, disrupting afferent sensory impulses and causing reflexive segmental disturbances in efferent vasomotor fibres (Allan, n.d.; Burns & McConnell, 2022).

4.3.3 Muscular pathology

Acute pathological disruptions were observed without any pre-mortem changes. Muscle contractures, caused by interstitial myositis caused increased connective tissue and muscle fibre atrophy. Associated nerves and their arterioles and veins were also degenerated (Allan, n.d.; Burns & McConnell, 2022).

4.3.3 Combined pathological findings

Congestion and inflammation, leading to vascular disturbances, were consistently found. The initial pathological changes in muscular, fascial, ligamentous, and osseous tissues interfered with normal afferent signalling to the spinal cord. This interference, caused by the lack of joint freedom of movement, initiating changes within the spinal cord segment, disrupting the afferent feedback system and impairing vasomotor control. Vessel dilation and congestion were prominent. Arterioles, capillaries, and veins suffered disturbed innervation, leading to compromised blood flow, endothelial tissue, plasma exudation and subsequent diapedesis, with small areas of haemorrhage, particularly in nerve centres of the cord and ganglia. Local

nutrition was compromised, resulting in parenchymal degeneration. During the first few months following a lesion, hyperaemia was followed by chronic vascular congestion with blood being cleared by the lymphatic system or undergoing coagulation and scar tissue formation. The second year and thereafter, progressive fibrosis occluded capillaries, venules, and arterioles leading to varying degrees of ischemia. Nerve centres, especially those connecting the ganglia to the cord and sympathetic system, were highly sensitive to circulatory changes, causing functional impairments. Lesions negatively impacted the health of tissues and organs innervated by affected nerves (Allan, n.d.; Burns & McConnell, 2022).

4.4 Summary

Louisa Burns D.O. summarises pathological changes following osteopathic lesions with initial hyperaemia leading to oedema. Congestion of tissues with subsequent haemorrhages, fibrosis, thus ischemia and hypertension (Burns 1986).

Her hypothesis suggests bony lesioning causes disease in distant parts. Correction of the lesion could lead to partial recovery at least, with recovery dependant on the extent of pathological changes present.

This may potentially explain the aetiology of the pathological disturbances and clinical symptoms associated with TMHS.

5.0 Neurophysiological basis of Osteopathic manipulative treatment

5.1 Clinical Neurophysiological Response to Trauma

As described, (4.3) a SD develops inflammation, producing an influx of pro-inflammatory cytokines with localised tenderness, heat and swelling. These neurochemicals act on the receptor membrane, lowering the threshold which it will fire, thus causing hypersensitivity and pain.

Trauma stimulates nociceptors, sending information of threat to the CNS to be interpreted. The ventral horn of the spinal cord is also affected, causing efferent motor stimuli thus muscle spasm, identified by palpation. Further ischemia and hypertension can be felt by palpation from alteration in skin texture due to sympathetic stimulus, causing cutaneous vasoconstriction, relative to the ‘TART’ acronym (4.2) (Chila D.O. et al., 2011; Paulus, 2013).

5.2 OMT assessment

Osteopaths observe dynamic and static assessments looking for fibrotic changes, hypertension and joint restriction along with pain, sensitivity, swelling, muscles spasm and tissue texture changes as indicators of SD. Decreased muscle bulk can be a sign of neurological deficit, or poor joint or spinal mobility (Pusey et al., 2010; Burns & McConnell, 2022).

5.3 OMT Techniques

OMT techniques aim to address SD by improving joint mobility and neuromusculoskeletal function.

5.3.1 Soft Tissue, Myofascial Release and Positional Release

Soft tissue techniques address abnormal tone resulting from SD. Cross-fibre at 90-degree orientation or longitudinal muscle stretch is applied to improve tone, nutrition to tissue, pliability and decompress associated joints. Pressure and amplitude applied may be altered to suit patient's tolerance and physiological state (Pusey et al., 2010).

Direct (restrictive barrier engaged) and indirect (position of ease) Positional release and myofascial release techniques induce relaxation of the relative tissue to find homeostasis. Fascia responds to biomechanical stress through collagen fibres producing microelectrical potential changes which occur in cellular, neural, vascular and lymphatic components (O'Connell 2011).

5.3.2 Muscle Energy Technique (MET)

Reciprocal inhibition and post-isometric relaxation are methods of MET. Controlled positioning of joints, muscle and related fascia are applied to their restricted barrier, then a counterforce produced by the patient is applied. Range of motion can be increased due to the refractory state of the myotatic reflex, or via reflex relaxation of antagonist muscle groups (Chila et al., 2011).

5.3.3 Articulation, Mobilisation

Gentle repetitive movements through a restrictive barrier, improving proprioception, increasing large fibre afferent input to the CNS which can inhibit incoming pain signalling (Pain Gate Theory) (Mendell, 2014).

Mobilisation of joints and surrounding tissue helps stimulate local release of hormones associated to insulin related growth factor, increasing the length of sarcomeres which form muscle fibres. Restoring mobility can restore proprioceptive receptibility from Golgi Tendon Organs and muscle spindles subsequently improving balance and co-ordination (Anthony Pusey et al., 2010)

5.3.4 High Velocity Low Amplitude Thrust

Restoration of range of motion to a restricted arthrodial joint. Applicable where there is segmental irritation with local oedema, tightening of myofascial/capsular components and muscular hypertonicity. Engaging a restrictive barrier in one or more planes of motion, the practitioner applies a brief, rapid force within the anatomical range of motion within the joint. There may or may not be a an audible 'pop' sound, the mechanism for which is still up for debate (Hohner; Cymet, 2011).

5.3.5 Cranial Osteopathy

A complex system of treatment of the inherent motion of the CNS, fluctuation of cerebral spinal fluid, mobility of associated membranes, cranial bones and involuntary motion between cranium and sacroiliac complex (Chila *et al*., 2011; Pusey *et al*., 2010; Dowling, 2000).

6.0 Summary

6.1 Summary

TMHS exhibits abnormal function of the neuromusculoskeletal system associated with relative anatomical structures. Symptoms of disease indicate exploration to various other contributing lifestyle factors, although none of which have yet been shown to have significant therapeutic longevity.

The use of OMT has not currently been explored as a treatment method of TMHS in horses.

6.2 Suggestions

Dysfunctional physiological changes apparent with SD could correlate to those that could exist with TMHS horses. Research of SD of the OAJ, the origin of CNV may be implied to understand and be able to treat the source of TMHS in horses.

Consideration of TMHS affected horses' diet and systemic pH balance could be examined to obtain maximum therapeutic benefit.

TMHS horses that are affected at exercise but not at rest indicates research into the biomechanical demands/ changes on the neuromusculoskeletal system in dynamic movement as opposed to static positioning.

Currently diagnosis involves a CT scan of the head, however the anatomy and physiology of CNV, originating from C0-C3, could justify a CT of the upper cervical to assess the full anatomical pathway of the trigeminal nerve, with a measure of vascular health by Doppler Ultrasound, for example.

6.3 Conclusion

Information gathered forms a hypothesis on the aetiology of TMHS and the pathophysiological changes apparent in somatic dysfunction suggesting a connection between the two subjects. A secondary hypothesis implies OMT could be considered in restoring pathological physiology to health.

Limitations

- Population – UK based statistics based on only those who are diagnosed by a vet. No differentiation between cases has been made i.e. ridden/ non-ridden horses. Aetiology could be indicated with significance to those that are ridden or used for human recreation/ sport – Variability between multiple systemic and physiological factors are unable to be measured on a case-by-case basis.
- Aleman et al., 2013 study was based on a small population of 9 TMHS horses.
- EquiPENS study had no control group, and results were obtained by owners, leaving them open to subjectivity and variability.
- Diagnosis – CT scans are currently taken of the head. Anatomy and physiology of CNV originates from C0-C3.
- All studies included are of the English language.
- Restricted access to some research databases was a limiting factor for this review.
- There are no pharmacokinetic studies for the use of pharmaceutical intervention of the horse, similarly microbiota and the effect on the peripheral nervous system are largely on humans thus far. Further research needs to be undertaken for equids.

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