Is a Definitive Diagnosis of Cervical Vertigo Possible in 2020? A Narrative Review and a Proposed Diagnostic Algorithm

Jianli Jan Wu.

SAERA. School of Advanced Education Research and Accreditation

ABSTRACT

Vestibular symptoms are a non-specific, world wide phenomenon with potential for lasting health and economic impact. Vestibulopathy can vary in etiology from peripheral to central and can be influenced by vascular, neurologic and musculoskeletal comorbidities. The cervical spine has defined neuroanatomical links to the vestibular system. However, vestibular symptoms attributed to cervical spine sources has been a contentious diagnostic theory. Cervical vertigo and cervicogenic dizziness are some of the proposed diagnoses. This paper reviews the history of the diagnoses and the established anatomical interconnections between the cervical spine and vestibular system. The current literature is also reviewed for evidence regarding: etiology; patient characteristics, and; for the existence of sufficiently powerful tests to diagnose cervicogenic vestibular symptoms. Based on the recent literature, a new diagnostic algorithm is proposed and an attempt is made to assess the ability to definitively diagnose Cervical Vertigo.

INTRODUCTION

Basic science, animal and human clinical research has demonstrated evidence for the labyrinth and cervical afferent influence over somatic motor control in human movement. The multi modal sensory integration required in this system is complex. Multiple afference inputs allow for redundancy and adaptation. However, failure to compensate can result in clinically relevant and functional disabilities that have social and economic impact. Dizziness and vertigo are some of the most common complaints among patients presenting to primary care physicians, neurologists, and otolaryngologists. These symptoms are nonspecific vestibular symptoms (Bisdorff et al., 2009) and include a wide range of differential diagnosis. The following paper serves to capture the current state of the literature with regard to diagnostic considerations and presents an evidence informed clinical algorithm of what has been historically and clinically known as cervical vertigo or cervicogenic dizziness.

Epidemiology

The incidence of dizziness with or without neck pain has been well documented in the scientific and medical literature (Ferrer-Peña et al., 2019), with a worldwide incidence rate in adults of 15–20% (Neuhauser, 2016) and prevalence rate of 17-30% (Murdin & Schilder, 2015). Common attributed causes were cardiovascular and otological/peripheral with up to 80% of cases classified as “no specific diagnosis possible” (Bösner et al., 2018). Vestibular related vertigo accounts for 1/4 of dizziness complaints, this increases with age and with a 3:1 female gender prevalence (Neuhauser, 2016).

Vestibular related reductions in quality of life outcomes are common in addition to substantial economic burdens (Agrawal et al., 2018; Bronstein et al., 2010; Ferrer-Peña et al., 2019; Murdin & Schilder, 2015). Direct (healthcare related) and indirect (sick leave and disability) costs have recently been estimated at 64,929 USD per patient lifetime or a lifetime societal burden of 227 billion USD for the US population over 60 years of age (Kovacs et al., 2019). This represents a significant functional and economic burden on the affected patient, their families and on society as a whole.

In a study of 288 chiropractic practices in Australia, dizziness was present in 28% of all neck pain patients with 24.1% being diagnosed with cervicogenic vertigo (Vindigni et al., 2019). However, there exists a paucity of quality epidemiological studies on specific vestibular disorders (Murdin & Schilder, 2015). Dizziness can be general manifestations of neurologic, cardiovascular, psychiatric, or vestibular disorders. However, dizziness and neck pain can also be symptoms of other more life-threatening diseases. It can be part of the prodrome of arterial dissections (Ohshima et al., 2019), tumours or masses (Darouassi et al., 2017), or rotational vertebral artery occlusion (Bow Hunters Syndrome) (Zaidi et al., 2014).

These global statistics highlight the need and benefits of prompt diagnosis of vestibular symptoms. Grill et al. (2018) commented on inappropriate and insufficient diagnostic accuracy and treatment timeliness in Europe. He attributes this to a lack of interdisciplinary collaboration, insufficient knowledge of standards in diagnosis and therapy and a lack of prospective multicentric studies.

Early cases and a historical view of Cervical Vertigo

Cervical Vertigo as a clinical entity was first described in a series of four cases by Ryan and Cope (1955). In their seminal case study, they had isolated instances of Cervical Vertigo from patients that had 1) cervical spondylosis 2) patients treated with neck traction and 3) patients that had certain types of neck injury. Their clinical characteristics observed included posterior neck pain or stiffness, suboccipital pain and postural vertigo. Symptom reproduction and nystagmus were also observed with neck bending.

From the outset, there existed multiple competing aetiologies of cervical vertigo: Ryan and Cope as described above with their 3 distinct mechanisms; Maran (1963) discussed aetiologies of arthritis, vertebral artery occlusion, stenosis of the basilar artery and proprioceptive connections, and; Weeks and Travell (1955) and Gray (1956), who reported on myofascial pain syndrome and extra-labyrinthine causes of vertigo and its symptomatic relief with intramuscular injections of anaesthetic.

Nomenclature

Currently naming conventions of cervical vertigo have been debated; proposed clinical aetiologies have expanded beyond spondylosis, and; a paucity of high-powered diagnostic tests have hampered the acceptance of this clinical entity. Cervical vertigo is considered a diagnosis of exclusion due to a lack of reliable and valid diagnostic tests and its incidence in a tertiary audiology clinic was reported at 17% (Somefun et al., 2010). For the purpose of this essay, cervicogenic; cervical; and neck are all deemed to be equivalent in the symptomatic description of vestibular symptoms attributed to the cervical region. There exists a paucity of: direct causal evidence for, and; diagnostic specificity for this diagnosis. Until such time that one is formalized, the next appropriate naming convention should rely on specific symptomatic descriptors. Previously used definitions of dizziness included: vertigo, disequilibrium, presyncope, or light-headedness.

This nomenclature is an often semantically driven source of much divisiveness in the medical community (Stanton et al., 2007; Lystad et al., 2011).

Cervicogenic Vertigo, in its evolution of nomenclature from cervical vertigo or cervical proprioceptive vertigo has slowly given way to cervical/cervicogenic dizziness. Recently a new non-hierarchical consensus classification has been presented by the Bárány Society towards an international classification of vestibular disorders (ICVD). This classification includes: symptom definitions that are purely phenomenological without reference to a theory on pathophysiology for a particular disease; definitions that are non-overlapping and non-hierarchical but allow one or more symptoms to coexist in a particular patient; and consideration was given in the choice of terminology to ease of translation to languages beyond English, given current word usage patterns (Bisdorff et al., 2009). This new classification prioritizes the future evidence informed clinical and research usage of symptom timing, duration, triggers vs inconsistent subjective descriptors (Stanton et al., 2007; Caplan, 2007). Accordingly, vertigo and dizziness, indeed all vestibular symptoms, are non-hierarchical and can both be used

individually or concurrently as valid vestibular symptom descriptors (Bisdorff et al., 2009).

Bisdorff et al. (2009) defined the following: (internal) vertigo is the sensation of self-motion when no self-motion is occurring or the sensation of distorted self-motion during an otherwise normal head movement; (non-vertiginous) dizziness is defined as the sensation of disturbed or impaired spatial orientation without a false or distorted sense of motion, and; postural symptoms are balance symptoms related to maintenance of postural stability, occurring only while upright (seated, standing, or walking) contrary to symptoms linked to changing body posture with respect to gravity (e.g., standing up). These latter symptoms are referred to as "orthostatic".

On the necessity of the monumental undertaking of an ICVD, the authors note: Having structured criteria for diagnosis is obviously mandatory for disciplines which rely heavily on symptom-driven syndromic diagnosis, such as psychiatry and headache, where often there is no histopathologic, radiographic, physiologic, or other independent diagnostic standard available. However, diagnostic standards and classification are also crucial in areas of medicine such as epilepsy and rheumatology, where, although confirmatory tests do exist, there is substantial overlap in clinical features or biomarkers across syndromes (Bisdorff et al., 2009, p1).

This classification scheme is still in its infancy and with a diagnostic conundrum that exists in vestibular symptoms (vertigo or dizziness) attributed to the cervical region, these steps cannot come fast enough. A valid and reliable operational definition is vital to further research, clinical knowledge synthesis and knowledge translation. The term Cervicogenic Vestibular Symptoms is utilised here as an evidenced informed general categorisation.

Neuroanatomical and physiological basis of cervicogenic vestibular symptoms

"The brain integrates information from multiple sensory modalities to generate appropriate motor output and create perceptual experiences of the environment." (Yakusheva, 2019, p21).

Spatial orientation to self and the environment is necessary for survival. The phylogenetic response in vertebrates is a complex sensory neuro integration mechanism involving varied interconnections of afferent and efferent signals to/from the vestibular nuclei complex. Vision, proprioception and vestibular sensory systems provide information about self and the environment. The integration of this information via central vestibular neurons encodes for accurate effective head, eye, neck and trunk control through vestibulo-mediated reflexes for spatial awareness, perception and coordinated movements.

In addition to the primary vestibular receptors of the labyrinth, the vestibular nuclei receive and combine input from: cortical; cerebellar; oculomotor and; spinal sources, including cervical proprioception. (Figure 1) The central lateral, medial and descending (spinal) vestibular nuclei play a prominent roll integrating cervical spine afferents and coordinating efferents between

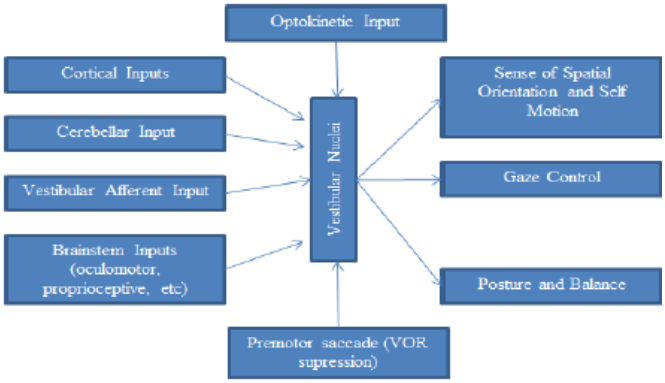
various ipsi and contralateral vestibular, oculomotor, cerebellar and cervical spine nuclei implicated in control of posture via vestibulospinal tracts, the stabilization of

gaze via the medial longitudinal fasciculus/ventral tegmental tract) and higher order self-motion and spatial orientation perception via ascending thalamocortical tracts.

It is important to note that faster moving stimuli is ideally reliant on vestibular pathways as visual and proprioceptive mechanism are too slow acting (Vidal, 2015; Highstein & Holstein, 2006; Hamid & Sismanis, 2006; Albernaz et al., 2019). Saglam & Lehnen (2014) also highlighted the importance of feedforward in addition to feedback mechanisms for stabilising active and passive head movements.

Figure 1.

Multimodal integration within the vestibular nuclei. Adapted from Vidal, 2015.



Cervical spine contributions to gaze stability: The vestibulo-ocular reflex

Gaze stability is the process of ensuring a still image on the retina. Improved information processing and visual perceptions require the minimization of image movement across the retina. The vestibulo-ocular reflex and the cervico-ocular reflex directly involve the cervical spine to act in concert to maintain images on the retina during active and passive head movements. Another visually mediated eye movement is the optokinetic reflex that help to fix images on the retina and; saccades, smooth pursuit, vergence and fixation that fix the image on the fovea, the area of highest visual acuity (Straka et al., 2005).

The vestibulo-ocular reflex (encompassing rotational vestibulo-ocular reflex, translational vestibulo-ocular reflex and the ocular counter roll reflex) is triggered by high frequency head on neck movements that stimulate the peripheral vestibular afferents to produce equal and opposite conjugate eye movements to maintain image stability on the retina (Hamid & Sismanis, 2006; Vidal, 2015). The peripheral vestibular afferents are 2 sets of 3 orthogonally placed semi-circular canals that are sensitive to angular acceleration and 1 pair of otolith organs sensitive to linear accelerations. This reflex relies on the vestibular afferents which has a latency of 7-15ms vs the visual afferents of 100ms (Albernaz et al., 2019), but because the labyrinth is only tuned to acceleration in head movement the visually mediated reflex is integral to slow and constant velocity head motion (visual-vestibulo-ocular reflex).

Additional neuronal inputs (Figure 1) are also involved in the vestibulo-ocular pathways including; complementary inputs from the optokinetic and proprioceptive systems; a behaviourally gated inhibitory input from the premotor saccadic pathway during gaze shifts (vestibulo-ocular reflex cancellation), and; efference copies of the motor command (Vidal, 2015; Cullen, 2012).

Recent findings indicate increased vestibulo-ocular reflex gain in patients with reported vestibular symptoms (Zamyslowska-Szmytke et al., 2019). Nevertheless, Grande-Alonso et al. (2018) to the contrary, reported

that clinical testing of the vestibulo-ocular reflex via video head impulse testing was found to be normal in patients with reported vestibular symptoms.

Cervical spine contributions to gaze stability: The Cervico-ocular Reflex

The cervico-ocular reflex is triggered by lower velocity neck on trunk movements that stimulate the cervical spine proprioceptive afferents to produce equal and opposite conjugate eye movements to maintain image stability on the retina. Animal studies by Neuhuber et al. (1989) and Matsushita et al. (1995) demonstrated confirmed evidence of upper cervical primary proprioceptive inputs to the vestibular nuclear complex and secondary inputs form the cervical nuclei. They hypothesised that these afferents enabled them to contact a variety of precerebellar, vestibulospinal, and pre-oculomotor neurons.

It is hard to make a case for the impact of cervico-ocular responses in typical real-life head-on neck/truck movements as it is difficult to ascertain the functional relevance of isolated laboratory generated cervico-ocular responses. In human studies, Dorn et al. (2017) investigated if cervical spine afferents would induce cervico-ocular reactions. They cited from Holzl et al. (2009) that in healthy individuals, cervical spine stimulation via a 3d-trunk-excursion chair would produce a diagnostically relevant upbeat nystagmus. Cervical transcutaneous electrical nerve stimulation was additionally used to disrupt sensory afferents produced confirmatory modulation of cervico-ocular nystagmus reactions.

Recent studies have reported on increases in cervico-ocular reflex gain in whiplash trauma to the cervical spine and with age with an associated negative correlation with vestibulo-ocular responses (Kelders, 2003; Kelders, 2005). Cervico-ocular reflexes are also increased in subjects with nonspecific and traumatic neck pain with no relative differences in vestibulo-ocular reflexes (Ischebeck et al., 2016).

Additionally, in labyrinthine deficient subjects, cervico-ocular gains: are increased (Norre, 1987; Huygen et al., 1991); replace the vestibulo-ocular reflex (Bronstein & Hood, 1986), and; are reversible after vestibular function improvement (Bronstein et al., 1995). The adaptability of the cervico-ocular reflex has been further documented by Rijkaart et al. (2004) and Kasai and Zee (1978). However, though adaptable, the presence of a cervico-ocular reflex has proven to be highly variable in normal, vestibular deficient, presbyvertigo and traumatic subjects (Norre, 1987; Kelders et al. 2003; Schubert et al., 2004; Ischebeck et al., 2016).

Cervical spine contributions to head in space control: The vestibulo-colic reflex

The vestibulo-colic reflex refers to the “activation of neck muscles induced by labyrinthine (vestibular) stimulation”. It generally functions to stabilise the position of the head in space (Keshner, 2009). Neurophysiological studies by Forbes et al. (2014) indicated that these reflexive responses are evoked regardless of the requirement of the vestibulum to maintain control of head on trunk during postural tasks. They concluded that, “vestibulo-colic reflex coupling... suggest functions through its closed-loop influence on head posture to ensure cervical spine stabilization.”

In fact, this short latency reflexive connection is the basis for the now over 2

decades use of the cervical vestibular-evoked myogenic potential neurophysiological test for saccule otolith function. And the effects reported, "...are likely to represent otolith-dependent vestibulo-colic and vestibulo-ocular reflexes, both linear and torsional." (Rosengren & Colebatch, 2018).

Like other reflexes centred around the vestibular nuclei complex, most neurons involved in the vestibulo-colic reflex are mono, di and trisynaptic between: the vestibular labyrinth (otoliths and semi-circular canals); neck muscles (via vestibulospinal pathways); the cerebellum (adjusts the gain of the vestibulo-colic reflex), and; somatosensory neck afferents. Responsiveness to velocity of head movements and polymyogenic connections enables multiple degrees of freedom to reflexively compensate for passive and active head perturbations (Keshner, 2009). Compensatory reticulospinal pathways also exist to produce short latency vestibulo-colic behaviours in the event of vestibulospinal compromise (Wilson & Schor, 1999).

Cervical spine contributions to head on trunk postural control: The cervico-colic reflex

The cervico-colic reflex is a compensatory response of the neck muscles that is driven by neck proprioceptive inputs during motion of the body to stabilize the head on the body. The upper cervical versus lower cervical spine segments have shown to be particularly associated with a higher level of afferent input to the vestibular nuclei. This had been demonstrated clinically and functionally by Bare in 1926 (Hamid & Sismanis, 2006) and de Jong et al. in 1977.

The cervico-colic or neck stretch reflex produces patterns of neck muscle response similar to vestibulo-colic reflex, however at 10x less strength and both the vestibulo-colic and cervico-colic reflexes "tune the head response" and diminish the duration and amplitude of head oscillations (Peng et al., 1999). The vestibulo and cervico-colic (muscular stretch) reflexes are additive in nature and functionally sensitive to muscle (stretch) length (Dutia, 1988).

Happee et al. (2017), demonstrated cervico-colic involvement in head on trunk stabilization with reduction in head rotation and translation at a lower frequency. They concluded that the cervico-colic reflex was responsible for intersegmental stability and to prevent spinal buckling. Keshner (2009) also documented on an increased contribution from the cervico-colic reflex as a compensatory mechanism to maintain head in space stability. Of note is Keshner's (2009) conclusion:

"Thus, the vestibular system does not appear to be directly implicated in generating the initial head stabilizing response during functional motion. The role of the vestibular system may actually be one of damping the response to the mechanics of the system and of monitoring the position of the head and trunk in space, secondary to feedback from segmental proprioceptors, in order to minimize the sustained effects of destabilization and maintain orientation in space." (p. 4223-4224)

Cervico-vestibulo-sympathetic connections

The past 25 years have seen an emergence of data relating to the existence vestibulo sympathetic connections in the so called vestibulo sympathetic reflex of the cardiovascular system (Shortt and Ray, 1997; Kawanokuchi et al., 2001; Carter and Ray, 2008; Yates et al., 2014) and direct

connections between cervical spinal and sympathetic ganglia (Zuo et al., 2014).

It was reported that the vestibular systems are connected to the sympathetic system via muscle sympathetic activity (muscle pump) to the exclusion of skin sympathetic activity (skin sweat rate, galvanic response or blood flow). Changes were noted in calf blood flow and calf vascular resistance with no change to mean arterial pressure. The authors suggest that the vestibular system may assist the baroreflex against orthostatic challenges and blood flow regulation in dynamic exercise and that its responses are age dependent (Ray & Monahan, 2002).

Kawanokuchi et al. (2001) demonstrated an account of muscle sympathetic activity being suppressed by the vestibulo-sympathetic reflex and more recently, studies on otolith stimulation in an upright posture have shown a greater sensitivity of the vestibulo-sympathetic reflex (Sauder et al., 2008). There have been observed and demonstrated direct and multi synaptic connections from the vestibular nuclear complex to the medulla that stimulates the preganglionic sympathetic neurons in animals (Holstein et al., 2011, Yates et al., 2014). These connections have shorter latencies and act in a complementary function to the carotid baroreceptors that are sensitive to reductions in blood pressure.

Bolton et al. (1998) investigated the role and necessity of the vestibular nuclei in the neural pathway that mediate the cervical-sympathetic response. Their results indicated cervical afferents (including muscle spindle and golgi tendon) influences sympathetic preganglionic neuronal activity. This activity persisted in animals with transected brain stems suggesting an alternative non vestibular pathway for sympathetic outflow. Human clinical investigations have also proposed extra vestibular, gravi and proprioceptive control mechanisms of the vestibulo-sympathetic reflex (Normand et al., 1997) and in patients with bilateral vestibular loss (Kuldavletova et al., 2019).

A hypothesized theory of developmental tuning of posturo-cardiovascular responses involving neck afferent inputs in the first few months of life has been proposed. This is in consideration of the presence of the co-occurring neck somato-motor reflex (tonic neck righting reflex) in human neonates (Yates et al., 1999).

The vertebrobasilar arterial system provides for both the peripheral and central auditory vestibular system. The labyrinth is supplied by the labyrinthine artery, a branch of the anterior inferior cerebellar artery (AICA). There is no redundancy to the labyrinthine artery and its supplied structures are sensitive to minimal disruptions (Hamid & Sismanis, 2006). Occlusion of the anterior inferior cerebellar artery can cause total deafness and loss of vestibular function however one of its branches, posterior vestibular artery has many collaterals and disruptions to its supply may not lead to total loss of saccular and posterior and lateral canal function.

The posterior inferior cerebellar arteries branch off the vertebral arteries to supply the inferior cerebellar hemispheres, the dorsolateral medulla and the inferior aspects of the vestibular nuclear complex. The basilar artery is the primary artery of the pons (Wilkinson, 1992).

Stenosis, dissection and occlusion of any arterial vessels can lead to loss of function, infarct and ultimately cell death in the supplied structures.

Efference copy/corollary discharge as a functional pathophysiological framework

In a more global systems view, altered proprioceptive input has been regarded as the foremost mechanism for cervicogenic vestibular symptoms that is grounded in anatomy and physiology (Karlberg et al., 1996; Magnusson & Malmstrom, 2016; Dieterich & Brandt, 2015).

Efference copy/corollary discharge has been presented as a direct or indirect functional neuro-physiological framework for self-motion perception (Goldberg & Cullen 2011; Cullen 2012). Efference copy, as defined by von Holst and Mittelstaedt in 1950 for the opto-motor response in flies, is a negative copy of a motor command. It is identical to the principle of corollary discharge defined by Roger Sperry in 1950 while studying the opto-motor response in fish (Binder et al., 2019). Efference copy and corollary discharge are part of the Reafference Principle that describes how a sensory system can cope with self-induced (i.e., "re-afferent") sensory input. If the efference copy and re-afferent signal are of equal magnitude, the subtraction of the efference copy from the reafference will cancel the sensory input signal (Bridgeman & Stark, 1991). This removes conflict between self-movement perception and external sensory input.

In her paper, Cullen (2012), attempts to answer the question of, "How is the processing of self- motion information adjusted to meet the needs of specific tasks?" She presents a scheme (Figure 2) to demonstrate the interaction of reafferent information, other extra vestibular proprioceptive information and direct vestibular afferents concluding that this sensory integration takes place early in processing by comparator mechanisms in the cerebellum to allow for accurate motor output and upstream spatial perception. Vestibular reafference has also been utilized as a pathophysiological framework for symptoms associated with vestibular incongruence (Brandt & Huppert, 2016).

In light of the profound interconnected elements between cervico-vestibular connections and potential pathophysiological mechanisms, this narrative review attempts to identify key diagnostic tests that rule-in Cervicogenic Vestibular Symptoms.

(See Figure 2.)

HYPOTHESIS AND OBJECTIVES

As evidenced in the literature to date, there is substantial functional patient, and economic societal impact to vestibular symptoms. In 1950, the scientific community was introduced to an alternative source of vestibular symptoms that arose from the cervical spine. Since then, anatomical, neurobiological, neurophysiological and neurophysiological studies have theorised, investigated and confirmed the coordinated interplay between the cervical spine and the vestibular system. As of the date of the initial search, it has not been clinically possible to definitively rule-in the diagnosis of cervical vertigo and its confirmation has only been possible after excluding all other potential differential diagnoses. This research paper seeks to answer the clinical question, "Is a definitive diagnosis of cervical vertigo possible in 2020?". The objectives are as follows: i. To review the current literature for diagnosis of Cervicogenic Vestibular Symptoms; ii. To investigate updates in aetiology, patient characteristics, and

diagnostic criteria of Cervicogenic Vestibular Symptoms; iii. To identify sufficiently powerful diagnostic tests to rule-in Cervicogenic Vestibular Symptoms, iii. In the absence of adequately powered tests, to propose a new evidenced informed clinical algorithm, and; iv. To establish considerations for future research investigations.

METHODOLOGY

This is a narrative review of the current literature. The primary aim is to review the diagnosis of Cervicogenic Vertigo: its historical concepts; basic science foundations, and; current theories. This will serve as the foundation for our investigation of the most promising diagnostic tests, patient characteristics and diagnostic criteria for the creation of a clinical decision-making algorithm.

Search strategy

The primary question of this research was "Do sufficiently powerful diagnostic tests to rule-in Cervicogenic Vestibular Symptoms exist?" Secondarily, this research sought to identify the current state of the science on Cervicogenic Vestibular Symptoms. An online Medline (Ovid) database (ALL 1946 to January 29, 2020) search was conducted on October 12, 2019 and updated on January 30, 2020. The keywords utilised were "cervic*" and including "dizz*" or "vertig". Wildcards were used to capture all iterations of the key words. Proximity and Boolean operators were also utilised. All searches were limited to the English language and peer reviewed publications. Concussion and whiplash associated disorders were excluded from the search to achieve a great degree of specificity. Hand searching for treatment and other non-diagnosis related studies were found and excluded.

The results were exported to Mendeley reference manager for further review and assessment.

Table 1.

Inclusion and Exclusion Criteria for Article Selection.

Inclusion	Exclusion
English Language	Concussion
Peer-reviewed publications	Whiplash
	Treatment

Primary search history

1. ((cervic* adj3 (dizz* or vertig*)) not (whiplash or concussion)).mp. [mp=title, abstract, original title, name of substance word, subject heading word, floating sub-heading word, keyword heading word, organism supplementary concept word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier, synonyms], Results: 250

2. Limit 1 to English language, Results: 149

3. Hand Screen, Results: 125

4. Hand Screen to exclude treatment, Results: 63

Data extraction and analysis

The data was synthesised and aggregated into general themes of: clinical aetiologies; patient characteristics; diagnostic criteria; and diagnostic tests. No further exclusions were made based on publication types to ensure a wide breadth of available data. Additional reference and citation searches were conducted for completeness and confirmation. Background themes include: epidemiology; historical cases; vestibular nomenclature; and, neuroanatomical and pathophysiological basis for cervicogenic vestibular symptoms.

RESULTS

Clinical aetiology

Cervical Spondylosis is a common and pervasive finding in the reporting of clinical aetiologies for cervical vertigo (Takahashi, 2018; Zheng et al., 2019; Li et al., 2014; Wang et al., 2011; Yang et al., 2018; Yang et al., 2017).

In a retrospective study, Takahashi (2018) reviewed over 1000 patient visits in a 15-month period that had a chief complaint of dizziness. In these patients, over 90% were found to have cervicogenic general dizziness and their mean age was 62. In addition, 91% of those that underwent cervical spine MRI had comorbid cervical spinal canal stenosis; 80% was able to recall an abnormal cervical spine load; and, occupational and prolonged cervical spine postures were frequent triggers.

Zheng et al. (2019) investigated imaging differences of patients with cervical spondylosis with cervical vertigo. This was a retrospective study of a group of 31 patients with concomitant cervical vertigo and spondylosis (myelopathy, radiculopathy and mixed) that underwent total disc replacement surgery. The surgery resulted in the successful reduction of vertigo with the most reduction seen in cases where a herniated disc was the source of compression. This was followed by a more modest reduction in cases where an osteophyte was the source of compression. It was hypothesized that the results were due to the stimulation of sympathetic fibres located within the posterior longitudinal ligament and a network of sensory fibres around the degenerated cervical disc.

Yang et al. (2017) recently suggested that Ruffini corpuscles (a type is implicated in the pathogenesis of cervicogenic vertigo. In their study, degenerated cervical discs were noted to contain an increase in number and ingrowth of Ruffini corpuscles. Ruffini corpuscles are mechanoreceptors that are thought to be sensitised in the presence of inflammatory mediators to contribute to aberrant proprioceptive signals. These findings were associated with the incidence of vertigo in the patients with cervical spondylosis along with the comorbid presence of free nerve endings (pain). In a follow-up clinical study, Yang et al. (2018), confirmed their histopathological findings in a group of patients that showed marked dizziness and pain relief with surgery (anterior cervical discectomy and fusion) in comparison to conservative care controls.

The human posterior longitudinal ligament has been found to contain sympathetic post ganglionic nerve fibres (Li et al., 2014). Recent studies provide support to the hypothesis that irritation to sympathetic nerves contribute to symptoms of vertigo in

patients with cervical degeneration and pain (Wang et al., 2011; Li et al., 2019).

In an observational study of cervical vertigo subjects with and without pain, Morinka (2009) attributed the significant findings of increased psychiatric dysfunction, orthostatic hypotension and brainstem dysfunction to recurrent and chronic pain influence on the limbic system. Musculoskeletal disease was found as a common comorbidity in cervical vertigo with: arthritic deformity of the knee joints; head and neck; and, cervical spondylosis being the most commonly observed.

Similarly, experimentally acutely induced cervical pain led to impaired sensorimotor control during and after the pain had waned. This was also accompanied by dizziness (Malmstrom et al., 2013).

Specific to the anatomy of the cervical spine: vascular insufficiency related to the patency through the paired vertebral and carotid arteries, and; sympathetic deregulation of cardiovascular control involving the cervico-vestibular-sympathetic reflexes are additional potentially contributing mechanisms to vestibular symptoms.

Since 1950, there now exists a proposed organisational framework that provides an explanation of how extra vestibular sensory (muscle spindle afferents from the eye or cervical spine) information and direct vestibular afferents can interact to allow for adaptation in the event of an efference copy and internal model mismatch (no cancellation signal). This theory has subsequently been implicated as a basis for the proprioceptive theory of cervical vertigo (Brandt & Huppert, 2016) in the generation of cervicogenic vestibular symptoms. Cullen (2012) has also implicated this model as an explanation of motor learning and plasticity.

Symptoms and patient characteristics

A systematic review conducted by Knapstad et al., (2019) consolidates the clinical characteristics of patients with cervicogenic dizziness despite methodological variability of the included studies. They reported an expectation of a gradual onset of vestibular symptoms aggravated by neck pain and movements rather than head movements.

Wrisley et al. (2000) also reported that the symptoms of cervicogenic dizziness were associated with injury or other cervical spine disease but their onset may be immediate or gradual from days to years following injury. These symptoms are typically episodic lasting from minutes to hours and are increased with neck movements or neck pain and decreased with treatments that relieve neck pain. Table 2 lists other clinical characteristics that have identified.

(See Table 2.)

Thompson-Harvey and Hain, (2019) created a 41-question survey based on questions from the Dizziness Handicap Inventory and the Neck disability Index. They found that these questions failed to show differences between patients with cervical vertigo from vestibular or migraine patients. They concluded that, there is substantial overlap in the symptoms reported by subjects with cervical vertigo and those with other vestibular disease or migraine.

Diagnostic screening

The Dizziness Handicap Inventory (DHI) is a 25-question self-reported questionnaire utilised to quantify the impact of dizziness on physical, emotional and functional aspects of

daily life. Reid et al. (2017) conducted a case-controlled study of cervicogenic dizziness and general dizziness patients. Using the DHI, they attempted to distinguish the 2 dizziness populations and report on the 3 questions that had the most discriminative ability. A score of 9 or more on these 3 questions accurately classified the cervicogenic dizziness group. These questions are: does looking up increase your problem; do quick movements of your head increase your problem, and; because of your problem, are you afraid to leave your home without having someone with you. The authors do note some overlap of 2 questions with a previous study on the DHI to identify patients with Benign Paroxysmal Positional Vertigo.

Diagnostic tests

Clinical physical examinations and para-clinical diagnostic tests are performed to improve diagnostic uncertainty regarding reported symptomology captured during the Clinical History and Interview and to rule in or out various differential diagnoses.

L'Heureux-Lebeau et al. (2014) investigated the diagnostic value of: Videonystagmography in various cervical movements (Smooth Pursuit Neck Torsion Test and Cervical Torsion Test); the Cervical Relocation Test; and, a dynamic balance evaluation (timed 10 metre walk with head turns). They compared the performance of these tests between a Cervicogenic Dizziness group (clinical diagnosed via exclusion of differential diagnosis, associated dizziness with neck pain and cervical spine pain/trauma/disease) and Benign Paroxysmal Positional Vertigo group. They arrived at these conclusions: The Cervical Relocation Test was more specific and sensitive when a 4.5-degree error was made in any 1 position; the dynamic balance evaluation revealed no differences between groups; the sensitivity and specificity for the Cervical Torsion Test was higher than the Smooth Pursuit Neck Torsion Test protocol. In terms of test discrimination between the Cervicogenic Dizziness and Benign Paroxysmal Positional Vertigo groups, higher discriminating ability was found with a combination of the Cervical Relocation Test and the Cervical Torsion Test. L'Heureux-Lebeau et al's protocol regarding these two significant tests are as follows:

1. The Cervical Relocation Test: The subject is seated in a darkened room with a laser pointer attached to the top of their head. With their eyes closed the subject is asked to bring their head to and from any cervical spine position back to centre. The variation (joint position error) in degrees to the centre point is recorded. L'Heureux-Lebeau et al. (2014) report a joint position error of 4.5 degrees as diagnostic.

2. The Cervical Torsion Test: The subject is seated with ocular fixation removed and eye movements measured. The head is fixed by the examiner and the body is turned to various degrees of rotation: 90 degrees (L'Heureux-Lebeau et al., 2014); 45-90 degrees (Treleaven et al., 2019). The positions are held for a time as eye movements (nystagmus) are recorded. L'Heureux-Lebeau

et al. (2014) report a more than 2 degrees per second nystagmus as diagnostic.

Other tests being investigated for the diagnosis of Cervicogenic Vestibular Symptoms are posturography and its variants (Karlberg et al., 1996; Wrisley et al., 2000;

Micarelli et al., 2019) and the Head-Neck Differentiation Test (Treleaven et al., 2019).

1. Static and dynamic posturography studies typically involve a force platform where sway and centre of pressure is recorded in eyes open or closed conditions. The more comprehensive sensory organization test involves static posturography in a virtual field with the addition of 4 more dynamic conditions: eyes opened on sway referenced visual surround; eyes open and closed on a sway referenced support surface and eyes open on a sway referenced support surface and surround. Scoring and analysis can identify ankle or hip contributions to balance and the relative impairments of the vestibular, proprioceptive or visual systems in balance.

2. The Head-Neck Differentiation Test is a clinical test that attempts to separate the vestibular and cervical spine contributions to the patient's subjective symptoms. The patient is asked to rotate just their head and their symptoms are recorded. If symptoms are provoked by this manoeuvre, it is thought to come from vestibular sources. Second, with the patient's head stabilised, they are asked to

rotate their body on a swivel chair. Symptoms provocation in this manner is thought to be attributed to the cervical spine. A rotation en bloc manoeuvre that is thought to stimulate the vestibular receptors in isolation has recently been reported (Reiley et al., 2017).

Normative data for clinical tests are essential to establish quantitative criteria to ensure accurate diagnosis. Based on the emergence of evidence for the utility of para-clinical tests especially in combination (L'Heureux-Lebeau et al., 2014) and the lack of resource availability for advanced testing in a clinical environment, Treleaven et al., (2019) attempted to identify normative qualitative responses of the Cervical Torsion Test and the Head-Neck Differentiation Test.

Treleaven et al., (2019), reports 35(23%) participants experienced symptoms in response to 1 or more of the 6 test conditions. Most of these tested experienced their symptoms during the Head-Neck Differentiation Test. The specificity for torsion component in the Cervical Torsion Test was 98.64% and a combined specificity for both tests was 100%. This represented the ability of the tests to successfully rule out Cervicogenic Dizziness because those who experienced symptoms identified qualitative descriptors unlike those of Cervicogenic

Dizziness patients. They conclude that further studies are warranted to investigate subjective symptoms in the target (Cervicogenic Dizziness) population (Treleaven et al., 2019).

Diagnostic criteria

In their 2019 systematic review, Knapstad et al. concluded that the diagnostic criterion of Cervicogenic Dizziness was, "based on the patient simultaneously reporting neck pain and dizziness as well as the exclusion of other neurological or neuro-otological disorders. The distinction between vertigo and dizziness was not considered essential for the diagnosis…" (p10).

As the current clinical consensus is to treat Cervicogenic Vestibular Symptoms as a diagnosis of exclusion, the diagnostic inclusion criteria reported in the available studies are highly variable and dependant on the study authors. Table 3 outlines some

utilised diagnostic criteria and their study authors.

(See Table 3.)

DISCUSSION

Diagnosing a non-specific entity

The process of arriving at a defined medical diagnosis is an iterative one. The process is ideally subject to multiple rounds of refinement towards minimizing diagnostic uncertainty and maximizing certainty. Multiple clinical diagnoses (Differential Diagnoses) are not unheard of and even the less precise 'Clinical Impression' is commonly used in the diagnostic process.

Towards improving diagnosis in health care, the Committee on Diagnostic Error in Health Care (n.d.) concluded that "the diagnostic process is a complex, patient-centred, collaborative activity that involves information gathering and clinical reasoning with the goal of determining a patient's health problem. This process occurs over time, within the context of a larger health care work system that influences the diagnostic process. (para. 3)"

Ultimately the accuracy of this clinical diagnostic process improves patient expectation, therapeutic intervention and overall outcome. This is arguably the most important clinical process involved in the management of health conditions, and is the weakest aspect of validating Cervicogenic Vestibular Symptoms. Essential elements that factor into the diagnostic process include a valid aetiology that has evidential support of basic and clinical sciences, identifiable symptoms and patient characteristics, adequately powered diagnostic tests and quantifiable criteria that must be met for inclusion.

Aetiology

There is convincing anatomical, physiological and clinical support for labyrinth and cervical afferent contribution to somato-motor control, human spatial perception, movement and locomotion. Of late, there has been a relative abundance of scientific investigations in the subset of spondylotic patients that present with vestibular symptoms: a proprioceptive network of: mechanoreceptors in degenerated discs (Yang et al, 2017; Yang et al, 2018);

sympathetic post ganglionic fibres in the posterior longitudinal ligaments (Li et al, 2014); and, muscle spindles in deep cervical muscles (Cullen, 2012) link the clinical symptoms of pain and cervicogenic vestibular symptoms in the cervical spondylosis population. This potentially establishes another line of evidence that the Cervicogenic Vestibular patient may just be a subset of those with degenerative changes that affect the biomechanics and morphology of the cervical spine structures.

Additionally, there exists a theoretical basis for the functional role of several cervical structures in the generation of vestibular symptoms in the global population. In general: disruptions, injury and other pathology of the associated blood supply, neurological pathways, receptors or processing centres can cause vertigo, dizziness, unsteadiness and other vestibular symptoms (de Jong et al., 1977; Albernaz et al., 2019) Proprioceptive, pain, vascular, sympathetically mediated, central vestibular, carotid body dysfunction, spondylosis and arthropathies have previously been

suggested as potential explanatory mechanisms of cervicogenic vestibular symptoms. However, fracture, ligamentous instability, vestibular, visually mediated and other differential aetiologies must also be considered as competing diagnosis that also result in vestibular symptoms.

The APA Dictionary of Psychology (Retrieved January 21, 2020) defines preafference as: a central brain process in which the somatosensory area is primed to expect the particular sensory inputs that are predicted as the consequence of an intended motor action. The concept of preafference applied to cervicogenic vestibular symptoms is a novel explanation of how inappropriate sensorimotor learning may occur in the presence of dysfunctional and inappropriately learned patterns of movement. It can be suggested that previously learned movements based on sensory afferents serve as a foundation for improved speed of movement generation, timing, sequencing and output. This process of motor learning has a role in acquiring new movement skills, refining already acquired ones and more importantly may be essential in the generation of the internal model.

As somatosensation fluctuates as a result of continued cervical spine degeneration, trauma or pain, pre-efference will always result in an ever-changing internal model. It can be supposed that the somatosensory area will never be able to predict relatively quick changes in the expected sensory inputs. This presents until now, an unmentioned concept of how sensory mismatches between the predicted consequence of dysfunctional neck movements in the somatosensory areas for self-motion and spatial orientation perception can lead to a self-sustaining pattern of chronic vestibular symptomatology. These concepts of pre-efference, efference copy and internal model helps to unify the multifactorial aetiology associated with the complex clinical diagnosis of cervicogenic vestibular symptoms.

Patient symptoms and characteristics

Efforts to quantify key patient characteristics in higher quality studies have been hampered by variability in diagnostic criteria and poor methodology (Knapstad et al, 2019; Treleaven et al, 2019). However there have been recent advancements in the ability to screen potential cases (Thomson-Harvey & Hain, 2019; Reid et al, 2017). In the absence of viable clinical diagnostic tests, the identification of key clinical characteristics via subjective interviews or questionnaires are necessary for screening patients for further paraclinical diagnostic testing.

Diagnostic tests

In consideration that is a near impossibility to isolate neck and head movements in real world conditions, Ryan and Cope (1959), makes historical mention of: De Kleijn's plaster cast; Tait and McNally's tilt table and; Nylen's posture table as experimental attempts to isolate cervical spine motion. Ryan and Cope (1959) themselves utilized a "collar test" involving a plaster collar and a modified version of the protocols established by Dix and Hallpike in 1952, Cawthorne in 1954 and Gordon in 1954 to experimentally isolate the components of postural induced cervical vertigo. In contrast, Brandt and Bronstein (2001) eloquently reasoned that "The perception of head rotation is mediated by vestibular, proprioceptive, or visual receptors. Vertigo should therefore be induced by stimulation of any of these

systems." And that it is "virtually impossible" to have a selective postural test for neck function.

A recent case-controlled study (L'Heureux-Lebeau et al, 2014) was identified that attempted to quantify para-clinical tests in the diagnosis of cervicogenic dizziness. Para-clinical tests are categorised so because they require specialised equipment that may not be feasible in a strict primary care or rehabilitation clinical setting due to technical or logistical considerations. No other higher quality studies of clinical or paraclinical diagnostic tests were identified in this research review.

There is currently no established reliable clinical test for the entity of cervicogenic vestibular symptoms and in addition there are often better-established clinical diagnoses to explain these symptoms. The diagnostic process attempts to identify key patient characteristics of a condition in order to rule a particular diagnosis in or out. At present, the entity of cervicogenic vestibular symptoms cannot be diagnostically ruled in (Treleaven et al, 2019; Yacovino & Hain, 2013). This inability to rule cervicogenic vestibular symptoms in as a diagnosis subsequently requires one to rule out all competing diagnosis and thus making cervicogenic vestibular symptoms a diagnosis of exclusion.

A diagnosis of exclusion demands a higher level of clinical diagnostic fortitude to rule out more serious differential diagnosis and places more importance on the diagnostic process as a whole. A non-exhaustive list of differential diagnoses to rule out before ruling in Cervicogenic Vestibular Symptoms appears in Table 4.

(See Table 4.)

While advances have been made in the field by combining existing diagnostic tests to

improve overall test power, the apparent lack of consistency in laboratory and clinical cervico-vestibular-ocular data and patient presentations reviewed in this research report implies an inherent adaptability in the cervico-vestibular system. This may serve to explain the unreliability of subjective symptom reporting, patient characteristics and the resulting variability in diagnostic test results.

Diagnostic criterio

Higher quality sensitive and specific diagnostic tests that allow for the diagnosis of cervicogenic vestibular symptoms have been "elusive" and increase the challenge of diagnosis. In order to guide clinical diagnosis and research, the diagnosis of cervicogenic dizziness is suggested by the utilising criteria. Wrisley (2000) proposed the following criteria: (1) a close temporal relationship between neck pain and symptoms of dizziness, including time of onset and occurrence of episodes; (2) a previous neck injury or pathology, and; (3) the elimination of other causes of dizziness.

As noted previously, there exists in studies a great variety with respect to their diagnostic criteria. This methodological variability will only improve with common definition, nomenclature and a method of specific diagnosis. In the absence of any sufficiently powerful diagnostic tests, we must be reliant on diagnosis ex juvantibus (Magnusson & Malmstrom, 2016). This is considered a top-down approach where temporal association of dizziness and neck pain and the amelioration of dizziness or other vestibular symptoms is observed with treatment of the associated cervical spine dysfunction.

Reliable and valid diagnostic tests are vital in the establishment of diagnostic criteria. Paradoxically, in order to adequately validate diagnostic tests and further scientific inquiry, reliable and valid diagnostic criteria is also vital to methodologically sound studies.

Proposing a new diagnostic algorithm

Due to the lack of evidence for sufficiently powerful diagnostic tests (Knapstad et al, 2019); no systematic review has been successfully attempted regarding the diagnosis of this clinical entity. In its place several authors have suggested clinical decision-making algorithms to best guide the clinical diagnosis of this condition (Wrisley et al, 2000; Reiley et al 2017).

I propose the following algorithm based on the most recent evidence. This new clinical algorithm allows for a logical progression through the 3 most common patient clinical characteristics and the upholding of the medical principle of "primum non nocere" by ruling out immediate life-threatening diagnosis.

(See Figure 3.)

In addition: the inclusion of newer screening tools that have shown potential to discriminate between competing diagnosis (BPPV or Cervicogenic Vestibular Symptoms); combining Cervical Relocation and Torsion Tests to improve diagnostic power, and; identifying specific patient symptom descriptors and comorbidities differentiates this from previously published algorithms. It will undoubtedly require further verification and validation.

To date, this is the only clinical algorithm to take into account the most recent scientific discoveries. I expect that this will improve the discrimination and identification of patients that present with Cervicogenic Vestibular Symptoms. Ultimately, the overall utility of the presented algorithm will be subject to validity and reliability of the new inclusions. Retrospective analysis of this algorithm on a large enough cohort of "confirmed" cases of Cervicogenic Vestibular Symptoms would serve to add further validation.

Future research considerations

Future studies must prioritize the following considerations. First, inconsistencies should be reduced with regard to patient characteristics for improved internal validity and the ability to pool subjects in systematic reviews. Second, a common diagnostic criterion is considered high priority for future research to improve methodological consistency and add statistical power. Third, research in promising clinical and paraclinical tests (Cervical Neck Torsion, Head-Neck Differential Test, Cervical Repositioning and posturography) also needs consistency in its methodologies to establish clinical validity. This may also serve as a method to establish and measure the adaptive capacity of the cervical spine in relation to the onset of vestibular symptoms. Fourth, new evidence suggests that it is reasonable to attribute cervical spondylosis as a primary factor or patient characteristic in the incidence of cervicogenic vestibular symptoms. The necessary level of degenerative change to evoke vestibular symptoms and a way to objectively measure these levels in the general cervicogenic vestibular population is yet to be established.

CONCLUSION

A newly proposed etiological framework based on pre-afference and efference copy should help to organize and drive future basic science and top-down clinical research, although questions still remain regarding the potentially etiologic but often coincidental findings of vestibular symptoms and neck dysfunction or headache. Indeed, a bottom-up approach highlights fallacy in building an etiologic mechanism on the supposed cervico-ocular responses. The balance of evidence suggests that diagnosis of vestibular symptoms attributed to the cervical spine cannot be definitively made. As such, the covenant between patient and physician dictates implicitly, that priority is placed on the health and safety of the patient. In the case of a patient presenting with vestibular symptoms and distinct or associated neck dysfunction, any course of care should rule out the causes with life threatening and severe consequences. A course of care for the relatively minor cervical spine dysfunction and associated vestibular symptoms can then be implemented. Ultimately, while the etiological basis of this diagnosis is important, the reality of its clinical diagnostic relevance is likely inconsequential as the management of neck dysfunction (pain, proprioceptive or otherwise) with or without associated vestibular symptoms is equivalent in either case. Cervicogenic vestibular symptoms remain a diagnosis of exclusion as we await scientific and clinical studies performed with sufficiently higher levels of rigor.

REFERENCES

1. APA Dictionary of Psychology. (n.d.). Retrieved January 21, 2020, from https://dictionary.apa.org/

2. Agrawal, Y., Pineault, K. G., & Semenov, Y. R. (2018). Health-related quality of life and economic burden of vestibular loss in older adults. *Laryngoscope Investigative Otolaryngology*, *3*(1), 8–15. https://doi.org/10.1002/lio2.129

3. Albernaz, P. L. M., Zuma e Maia, F., Carmona, S., Cal, R. V. R., & Zalazar, G. (2019). *The New Neurotology*. Springer International Publishing. https://doi.org/10.1007/978-3-030-11283-7

4. Bisdorff, A., Von Brevern, M., Lempert, T., Newman-Toker, D. E., Bertholon, P., Bronstein, A., Kingma, H., Antonio Lopez Escamez, J., Magnusson, M., Minor, L. B., As Pérez, N., Perrin, P., Suzuki, M., Waterston, J., & Yagi, T. (2009). Classification of

5. vestibular symptoms: Towards an international classification of vestibular disorders. *Journal of Vestibular Research*, *19*, 1–13. https://doi.org/10.3233/VES-2009-0343

6. Bolton, P. S., Kerman, I. A., Woodring, S. F., & Yates, B. J. (1998). Influences of neck afferents on sympathetic and respiratory nerve activity. *Brain Research Bulletin*, *47*(5), 413–419. https://doi.org/10.1016/S0361-9230(98)00094-X

7. Bösner, S., Schwarm, S., Grevenrath, P., Schmidt, L., Hörner, K., Beidatsch, D., Bergmann, M., Viniol, A., Becker, A., & Haasenritter, J. (2018). Prevalence, aetiologies and prognosis of the symptom dizziness in primary care – a systematic review. *BMC Family Practice*, *19*(1), 33. https://doi.org/10.1186/s12875-017-0695-0

8. Brandt, T. (1996). Cervical vertigo--reality or fiction? *Audiology & Neuro-Otology*, *1*(4), 187–196.

9. Brandt, T., & Bronstein, A. M. (2001). Cervical vertigo. *Journal of Neurology Neurosurgery and Psychiatry*, *71*, 8-12. https://doi.org/10.1136/jnnp.71.1.8

10. Brandt, T., & Huppert, D. (2016) A new type of cervical vertigo: Head motion-induced spells in acute neck pain. *Neurology*, *86*(10), 974–975. https://doi.org/10.1212/WNL.0000000000002451

11. Bridgeman, B., & Stark, L. (1991). Ocular proprioception and efference copy in registering visual direction. *Vision Research, 31(11), 1903–1913.* https://doi.org/10.1016/0042-6989(91)90185-8

12. Bronstein, A. M., Morland, A. B., Ruddock, K. H., & Gresty, M. A. (1995). Recovery from bilateral vestibular failure: Implications for visual and cervico-ocular function. *Acta Oto-Laryngologica, 115*(S520), 405–407. https://doi.org/10.3109/00016489509125283

13. Bronstein, A. M., & Derrick Hood, J. (1986). The cervico-ocular reflex in normal subjects and patients with absent vestibular function. *Brain Research*, *373*(1–2), 399–408. https://doi.org/10.1016/0006-8993(86)90355-0

14. Bronstein, A. M., Golding, J. F., Gresty, M. A., Mandalà, M., Nuti, D., Shetye, A., & Silove, Y. (2010). The social impact of dizziness in London and Siena. *Journal of Neurology*, *257*(2), 183–190. https://doi.org/10.1007/s00415-009-5287-z

15. Caplan, L. R. (2007). Dizziness: How Do Patients Describe Dizziness and How Do Emergency Physicians Use These Descriptions for Diagnosis? *Mayo Clinic Proceedings*, *82*(11), 1313–1315. https://doi.org/10.4065/82.11.1313

16. Carter, J. R., & Ray, C. A. (2008). Sympathetic responses to vestibular activation in humans. *American Journal of Physiology - Regulatory Integrative and Comparative Physiology*, *294*(3), R681-R688. https://doi.org/10.1152/ajpregu.00896.2007

17. Chan, Y. (2009). Differential diagnosis of dizziness. *Current Opinion in Otolaryngology and Head and Neck Surgery*, *17*(3), 200-203. https://doi.org/10.1097/MOO.0b013e32832b2594

18. Committee on Diagnostic Error in Health Care. (2015). The Diagnostic Process. In E. P. Balogh, B. T. Miller, & J. R. Ball (Eds.), *Improving Diagnosis in Health Care.* National

Academies Press (US). Retrieved from https://www.ncbi.nlm.nih.gov/books/NBK338593/

19. Cullen, K. E. (2012). The vestibular system: multimodal integration and encoding of self-motion for motor control. *Trends in Neurosciences*, *35*(3), 185–196. https://doi.org/10.1016/j.tins.2011.1

20. de Jong, P. T. V. M., de Jong, J. M. B. V., Cohen, B., & Jongkees, L. B. W. (1977). Ataxia and nystagmus induced by injection of local anesthetics in the neck. *Annals of Neurology*, *1*(3), 240–246. https://doi.org/10.1002/ana.410010307

21. Dieterich, M., & Brandt, T. (2015). The bilateral central vestibular system: Its pathways, functions, and disorders. *Annals of the New York Academy of Sciences*, *1343*(1), 10–26. https://doi.org/10.1111/nyas.12585

22. Dorn, L. J., Lappat, A., Neuhuber, W., Scherer, H., Olze, H., & Hölzl, M. (2017). Tonic Investigation Concept of Cervico-vestibular Muscle Afferents. *International Archives of Otorhinolaryngology*, *21*(1), 46–57. https://doi.org/10.1055/s-0036-1583759

23. Dutia, M. B. (1988). Interaction between vestibulocollic and cervicocollic reflexes: Automatic compensation of reflex gain by muscle afferents. In O. Pompeiano & J. Allum (Eds.), *Progress in Brain Research* (Vol. 76, pp. 173–180). Elsevier Science Publishers. https://doi.org/10.1016/S0079-6123(08)64503-3

24. Eva-Maj, M., Hans, W., Per-Anders, F., Mikael, K., & Måns, M. (2013). Experimentally induced deep cervical muscle pain distorts head on trunk orientation. *European Journal of Applied Physiology*, *113*(10), 2487–2499. https://doi.org/10.1007/s00421-013-2683-y

25. Ferrer-Peña, R., Vicente-De-Frutos, G., Flandez-Santos, D., Martín-Gómez, C., Roncero-Jorge, C., & Calvo-Lobo, C. (2019). Patient-reported outcomes measured with and without dizziness associated with non-specific chronic neck pain: Implications for primary care. *PeerJ*, *2019*(8). https://doi.org/10.7717/peerj.7449

26. Forbes, P. A., Siegmund, G. P., Happee, R., Schouten, A. C., & Blouin, J. S. (2014). Vestibulocollic reflexes in the absence of head postural control. *Journal of Neurophysiology*, *112*(7), 1692–1702. https://doi.org/10.1152/jn.00343.2014

27. Girasoli, L., Cazzador, D., Padoan, R., Nardello, E., Felicetti, M., Zanoletti, E., Schiavon, F., Bovo, R. (2018). Update on Vertigo in Autoimmune Disorders, from Diagnosis to Treatment. *Journal of Immunology Research*, *2018*, 5072582. https://doi.org/10.1155/2018/5072582

28. Goldberg, J. M., & Cullen, K. E. (2011). Vestibular control of the head: Possible functions of the

vestibulocollic reflex. *Experimental Brain Research, 210,* 331–345. https://doi.org/10.1007/s00221-011-2611-5

29. Grande-Alonso, M., Moral Saiz, B., Mínguez Zuazo, A., Lerma Lara, S., & La Touche, R. (2018). Biobehavioural analysis of the vestibular system and posture control in patients with cervicogenic dizziness. A cross-sectional study. *Neurologia*, *33*(2), 121–128.

30. Gray, L. P. (1956). Extra Labyrinthine Vertigo Due to Cervical Muscle Lesions. The *Journal of Laryngology & Otology*, *70*(6), 352–361. https://doi.org/10.1017/S0022215100053032

31. Grill, E., Akdal, G., Becker-Bense, S., Hübinger, S., Huppert, D., Kentala, E., Strobl, R., Zwergal, A., Celebisoy, N. (2018). Multicenter data banking in management of dizzy patients: first results from the DizzyNet registry project. *Journal of Neurology*, *265*, 3–8. https://doi.org/10.1007/s00415-018-8864-1

32. Hamid, M., & Sismanis, A. (2006). *Medical otology and neurotology: a clinical guide to auditory and vestibular disorders*. New York: Thieme.

33. Happee, R., de Bruijn, E., Forbes, P. A., & van der Helm, F. C. T. (2017). Dynamic head-neck stabilization and modulation with perturbation bandwidth investigated using a multisegment neuromuscular model. *Journal of Biomechanics*, *58*, 203–211. https://doi.org/10.1016/j.jbiomech.2017.05.005

34. Highstein, S. M., & Holstein, G. R. (2006). The Anatomy of the vestibular nuclei. In *Progress in Brain Research* (Vol. 151, pp. 157–203). Elsevier. https://doi.org/10.1016/S0079-6123(05)51006-9

35. Holstein, G. R., Friedrich, V. L., Kang, T., Kukielka, E., & Martinelli, G. P. (2011). Direct projections from the caudal vestibular nuclei to the ventrolateral medulla in the rat. *Neuroscience*, *175*, 104–117. https://doi.org/10.1016/j.neuroscience.2010.12.011

36. Huygen, P. L. M., Verhagen, W. I. M., & Nicolasen, M. G. M. (1991). Cervico-ocular reflex enhancement in labyrinthine-defective and normal subjects. *Experimental Brain Research*, *87*(2), 457–464. https://doi.org/10.1007/BF00231863

37. Ischebeck, B. K., De Vries, J., Van Der Geest, J. N., Janssen, M., Van Wingerden, J. P., Kleinrensink, G. J., & Frens, M. A. (2016). Eye movements in patients with Whiplash Associated Disorders: A systematic review. *BMC Musculoskeletal Disorders*, *17*(1). https://doi.org/10.1186/s12891-016-1284-4

38. Karlberg, M., Johansson, R., Magnusson, M., & Fransson, P. A. (1996). Dizziness of suspected cervical origin distinguished by posturographic assessment of human postural dynamics. *Journal of Vestibular Research: Equilibrium*

and Orientation, *6*(1), 37–47. https://doi.org/10.3233/VES-1996-6105

39. Kasai, T., & Zee, D. S. (1978). Eye-head coordination in labyrinthine-defective human beings. *Brain Research*, *144*(1), 123–141. https://doi.org/10.1016/0006-8993(78)90439-0

40. Kawanokuchi, J., Fu, Q., Cui, J., Niimi, Y., Kamiya, A., Michikami, D., Iwase, S., Mano, T., & Suzumura, A. (2001). Influence of vestibulo-sympathetic reflex on muscle sympathetic outflow during head-down tilt. *Environmental medicine: annual report of the Research Institute of Environmental Medicine, Nagoya University*, *45*(2), 66–68.

41. Kelders, W. P. A., Kleinrensink, G. J., van der Geest, J. N., Feenstra, L., de Zeeuw, C. I., & Frens, M. A. (2003). Compensatory increase of the cervico-ocular reflex with age in healthy humans. *Journal of Physiology*, *553*(1), 311–317. https://doi.org/10.1113/jphysiol.2003.049338

42. Kelders, W. P. A., Kleinrensink, G. J., Van Der Geest, J. N., Schipper, I. B., Feenstra, L., De Zeeuw, C. I., & Frens, M. A. (2005). The cervico-ocular reflex is increased in whiplash injury patients. *Journal of Neurotrauma*, *22*(1), 133–137. https://doi.org/10.1089/neu.2005.22.133

43. Keshner, E. A. (2009). Vestibulocollic and Cervicocollic Control. In *Encyclopaedia of Neuroscience.* (pp. 4220–4224). Springer Berlin Heidelberg. https://doi.org/10.1007/978-3-540-29678-2_6308

44. Knapstad, M. K., Nordahl, S. H. G., & Goplen, F. K. (2019). Clinical characteristics in patients with cervicogenic dizziness: A systematic review. *Health Science Reports*, *2*(9), e134.

45. Kovacs, E., Wang, X., & Grill, E. (2019). Economic burden of vertigo: A systematic review. *Health Economics Review*, *9*(37). https://doi.org/10.1186/s13561-019-0258-2

46. Kuldavletova, O., Denise, P., Quarck, G., Toupet, M., & Normand, H. (2019). Vestibulo-sympathetic reflex in patients with bilateral vestibular loss. *Journal of Applied Physiology*, *127*(5), 1238–1244. https://doi.org/10.1152/japplphysiol.00466.2019

47. L'Heureux-Lebeau, B., Godbout, A., Berbiche, D., & Saliba, I. (2014). Evaluation of paraclinical tests in the diagnosis of cervicogenic dizziness. *Otology & neurotology: official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology*, *35*(10), 1858–1865. doi:10.1097/MAO.0000000000000506

48. Li, H., Ma, X., Wu, X., Liu, F., Yu, T., Yue, B., Xiang, H., & Chen, B. (2014). Morphological observation of sympathetic nerve fibers in the human posterior longitudinal ligament. *Spine*, *39*(26), 2119–2126.

49. Lystad, R. P., Bell, G., Bonnevie-Svendsen, M., & Carter, C. V. (2011). Manual therapy with and without vestibular rehabilitation for cervicogenic dizziness: A systematic review. *Chiropractic and Manual Therapies*, *19*(21). https://doi.org/10.1186/2045-709X-19-21

50. Magnusson, M., & Malmström, E. M. (2016). The conundrum of cervicogenic dizziness. In *Handbook of Clinical Neurology* (Vol. 137, pp. 365–369). Elsevier B.V. https://doi.org/10.1016/B978-0-444-63437-5.00026-1

51. Malmström E, Karlberg M, Melander A, Magnusson M, & Moritz U. (2007). Cervicogenic dizziness - musculoskeletal findings before and after treatment and long-term outcome. *Disability & Rehabilitation*, *29*(15), 1193–1205.

52. Maran, A. G. (1963). Cervical vertigo-report of a case. *The Journal of Laryngology and Otology*, *77*, 1032–1037. Retrieved from http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=medc3&NEWS=N&AN=14100982

53. Matsushita, M., Gao, X., & Yaginuma, H. (1995). Spinovestibular projections in the rat, with particular reference to projections from the central cervical nucleus to the lateral vestibular nucleus. *Journal of Comparative Neurology*, *361*(2), 334–344. https://doi.org/10.1002/cne.903610210

54. Micarelli, A., Viziano, A., Augimeri, I., Micarelli, B., Capoccia, D., & Alessandrini, M. (2019). Diagnostic route of cervicogenic dizziness: usefullness of posturography, objective and subjective testing implementation and their correlation. *Disability and Rehabilitation*. https://doi.org/10.1080/09638288.2019.1680747

55. Morinaka, S. (2009). Musculoskeletal diseases as a causal factor of cervical vertigo. *Auris Nasus Larynx, 36(6), 649–654. https://doi.org/10.1016/j.anl.2009.04.009*

56. Murdin, L., & Schilder, A. G. M. (2015, March 26). *Epidemiology of balance symptoms and disorders in the community: A systematic review. Otology and Neurotology. Lippincott Williams and Wilkins.* https://doi.org/10.1097/MAO.0000000000000691

57. Neuhuber, W. L., & Zenker, W. (1989). *Central distribution of cervical primary afferents in the rat, with emphasis on proprioceptive projections to vestibular, perihypoglossal, and upper thoracic spinal nuclei. The Journal of Comparative Neurology, 280(2), 231–253.* https://doi.org/10.1002/cne.902800206

58. Normand, H., Etard, O., & Denise, P. (1997). *Otolithic and tonic neck receptors control of limb blood flow in humans. Journal of Applied Physiology, 82(6), 1734–1738.*

https://doi.org/10.1152/jappl.1997.82.6.1734

59. Norre, M. E. (1987). Cervical vertigo. *Diagnostic and semiological problem with special emphasis upon "cervical nystagmus." Acta Oto-Rhino-Laryngologica Belgica, 41(3), 436–452.* Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/3673567

60. Ohshima, T., Miyachi, S., Isaji, T., Matsuo, N., Kawaguchi, R., & Takayasu, M. (2019). *Bilateral Vertebral Artery Dissection and Unilateral Carotid Artery Dissection in Case of Ehlers-Danlos Syndrome Type IV. World Neurosurgery, 121, 83–87.* https://doi.org/10.1016/j.wneu.2018.09.236

61. Peng, G. C. Y., Hain, T. C., & Peterson, B. W. (1999). *Predicting vestibular proprioceptive and biomechanical control strategies in normal and pathological head movements. IEEE Transactions on Biomedical Engineering, 46(11), 1269–1280.* https://doi.org/10.1109/10.797986

62. Ray, C. A., & Monahan, K. D. (2002). *The vestibulosympathetic reflex in humans: Neural interactions between cardiovascular reflexes. In Clinical and Experimental Pharmacology and Physiology (Vol. 29, pp. 98–102).* https://doi.org/10.1046/j.1440-1681.2002.03614.x

63. Reid, S. A., Callister, R., Katekar, M. G., & Treleaven, J. M. (2017). *Utility of a brief assessment tool developed from the Dizziness Handicap Inventory to screen for Cervicogenic dizziness: A case control study. Musculoskeletal Science and Practice, 30, 42–48.* https://doi.org/10.1016/j.msksp.2017.03.008

64. Reiley, A. S., Vickory, F. M., Funderburg, S. E., Cesario, R. A., & Clendaniel, R. A. (2017). *How to diagnose cervicogenic dizziness. Archives of Physiotherapy, 7(1).* https://doi.org/10.1186/s40945-017-0040-x

65. Rijkaart, D. C., Van Der Geest, J. N., Kelders, W. P., De Zeeuw, C. I., & Frens, M. A. (2004). *Short-term adaptation of the cervico-ocular reflex. Experimental Brain Research, 156(1), 124–128.* https://doi.org/10.1007/s00221-004-1878-1

66. Rosengren, S. M., & Colebatch, J. G. (2018). *The Contributions of Vestibular Evoked Myogenic Potentials and Acoustic Vestibular Stimulation to Our Understanding of the Vestibular System. Frontiers in Neurology, 9.* https://doi.org/10.3389/fneur.2018.00481

67. Ryan, G. M., & Cope, S. (1955). *Cervical vertigo. Lancet, 269(6905), 1355–1358.* Retrieved from http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=medc3&NEWS=N&AN=13279136

68. Sağlam, M., & Lehnen, N. (2014). *Gaze stabilization in chronic vestibular-loss and in cerebellar ataxia: Interactions of feedforward and sensory feedback mechanisms.*

Journal of Vestibular Research, 24(5,6), 425–431. https://doi.org/10.3233/VES-140538

69. *Sauder, C. L., Leonard, T. O., & Ray, C. A. (2008). Greater sensitivity of the vestibulosympathetic reflex in the upright posture in humans. Journal of Applied Physiology, 105(1), 65–69.* https://doi.org/10.1152/japplphysiol.90347.2008

70. Schubert, M. C., Das, V., Tusa, R. J., & Herdman, S. J. (2004). *Cervico-ocular reflex in normal subjects and patients with unilateral vestibular hypofunction. Otology and Neurotology, 25(1), 65–71.* https://doi.org/10.1097/00129492-200401000-00013

71. Shortt, T. L., & Ray, C. A. (1997). *Sympathetic and vascular responses to head-down neck flexion in humans. American Journal of Physiology, 272(4 PART 2).* https://doi.org/10.1152/ajpheart.1997.272.4.h1780

72. Stanton, V. A., Hsieh, Y. H., Camargo, C. A., Edlow, J. A., Lovett, P., Goldstein, J. N., Abbuhl, S., Lin, M., Chanmugam, A., Rothman, R.E., & Newman-Toker, D. E. (2007). *Overreliance on symptom quality in diagnosing dizziness: Results of a multicenter survey of emergency physicians. Mayo Clinic Proceedings, 82(11), 1319–1328.* https://doi.org/10.4065/82.11.1319

73. Straka, H., Vibert, N., Vidal, P. P., Moore, L. E., & Dutia, M. B. (2005). *Intrinsic membrane properties of vertebrate vestibular neurons: Function, development and plasticity. Progress in Neurobiology, 76(6), 349–392.* https://doi.org/10.1016/j.pneurobio.2005.10.002

74. Takahashi, S. (2018). *Importance of cervicogenic general dizziness. Journal of Rural Medicine, 13(1), 48–56.* https://doi.org/10.2185/jrm.2958

75. Thompson-Harvey, A., & Hain, T. C. (2019). *Symptoms in cervical vertigo. Laryngoscope Investigative Otolaryngology, 4(1), 109–115.* https://doi.org/10.1002/lio2.227

76. Treleaven, J., Joloud, V., Nevo, Y., Radcliffe, C., & Ryder, M. (2019). *Normative Responses to Clinical Tests for Cervicogenic Dizziness: Clinical Cervical Torsion Test and Head-Neck Differentiation Test. Physical Therapy, 100(1), 192–200.* https://doi.org/10.1093/ptj/pzz143

77. Vidal, P. P. (2015). *The Vestibular System. In G. Paxinos (Ed.), The Rat Nervous System (pp. 803–861).*

78. Vindigni, D., Zark, L., Sundberg, T., Leach, M., Adams, J., & Azari, M. F. (2019). *Chiropractic treatment of older adults with neck pain with or without headache or dizziness: analysis of 288 Australian chiropractors' self-reported views. Chiropractic & Manual Therapies, 27(1), 65.* https://doi.org/10.1186/s12998-019-0288-1

79. Wang, Z., Wang, X., Yuan, W., & Jiang, D. (2011). *Degenerative pathological irritations to cervical*

PLL may play a role in presenting sympathetic symptoms. Medical Hypotheses, 77(5), 921–92

80. Weeks, V. D., & Travell, J. (1955). *Postural vertigo due to trigger areas in the sternocleidomastoid muscle. The Journal of Pediatrics, 47(3), 315–327.* https://doi.org/10.1016/S0022-3476(55)80006-6

81. Wilkinson, J. L. (1992). *Neuroanatomy for medical students. Oxford: Butterworth-Heinemann.*

82. Wilson, V. J., & Schor, R. H. (1999). *The neural substrate of the vestibulocollic reflex. What needs to be learned. Experimental Brain Research, 129(4), 483–493.* https://doi.org/10.1007/s002210050918

83. Wrisley, D. M., Sparto, P. J., Whitney, S. L., & Furman, J. M. (2000). *Cervicogenic dizziness: A review of diagnosis and treament. Journal of Orthopaedic and Sports Physical Therapy. Movement Science Media.* https://doi.org/10.2519/jospt.2000.30.12.755

84. Yacovino, D. A., & Hain, T. C. (2013). *Clinical characteristics of cervicogenic-related dizziness and vertigo. Seminars in Neurology, 33(3), 244–255.* https://doi.org/10.1055/s-0033-1354592

85. Yahia, A., Ghroubi, S., Jribi, S., Mâlla, J., Baklouti, S., Ghorbel, A., & Elleuch, M. H. (2009). *Chronic neck pain and vertigo: Is a true balance disorder present? Annals of Physical and Rehabilitation Medicine, 52(7–8), 556–567.* https://doi.org/10.1016/j.rehab.2009.07.033

86. Yakusheva, T. A. (2019). *Multisensory Integration: Mathematical Solution of Inherent Sensory Ambiguities. In A. Shaikh & F. Ghasia (Eds.), Advances in Translational Neuroscience of Eye Movement Disorders (pp. 21–42). Cham: Springer International Publishing.* https://doi.org/10.1007/978-3-030-31407-1_2

87. Yang, L., Chen, J., Yang, C., Pang, X., Li, D., Wu, B., Wu, Y., Lu, X., Xu, J., Chen, X., & Peng, B. (2018). *Cervical Intervertebral Disc Degeneration Contributes to Dizziness: A Clinical and Immunohistochemical Study. World Neurosurgery, 119, e686–e693.* https://doi.org/10.1016/j.wneu.2018.07.243

88. Yang, L., Yang, C., Pang, X., Li, D., Yang, H., Zhang, X., Yang, Y., & Peng, B. (2017). *Mechanoreceptors in Diseased Cervical Intervertebral Disc and Vertigo. Spine, 42(8), 540–546.* https://doi.org/10.1097/BRS.0000000000001801

89. Yates, B. J., Aoki, M., Burchill, P., Bronstein, A. M., & Gresty, M. A. (1999). *Cardiovascular responses elicited by linear acceleration in humans. Experimental Brain Research, 125(4), 476–484.* https://doi.org/10.1007/s002210050705

90. Yates, B. J., Bolton, P. S., & Macefield, V. G. (2014). *Vestibulo-sympathetic responses. Comprehensive Physiology, 4(2), 851–887.* https://doi.org/10.1002/cphy.c130041

*91.*Zamyslowska-Szmytke, E., Adamczewski, T., Ziaber, J., Majak, J., Kujawa, J., & Sliwinska-Kowalska, M. (2019). *Cervico-ocular reflex upregulation in dizzy patients with asymmetric neck pathology. International Journal of Occupational Medicine and Environmental Health, 32(5), 723–733.* https://dx.doi.org/10.13075/ijomeh.1896.01428

92. Zheng, S., Muheremu, A., Sun, Y., Tian, W., & Wu, C. A. (2019). *Preoperative imaging differences of patients with cervical spondylosis with cervical vertigo indicate the prognosis after cervical total disc replacement. The Journal of international medical research, Advance online publication.* https://doi.org/10.1177/0300060519877033

93. Zuo, J., Han, J., Qiu, S., Luan, F., Zhu, X., Gao, H., & Chen, A. (2014). *Neural reflex pathway between cervical spinal and sympathetic ganglia in rabbits: Implication for pathogenesis of cervical vertigo. Spine Journal, 14(6), 1005–1009.*

APPENDIX

Figure 2. *Neural mechanism for the attenuation of vestibular reafference*

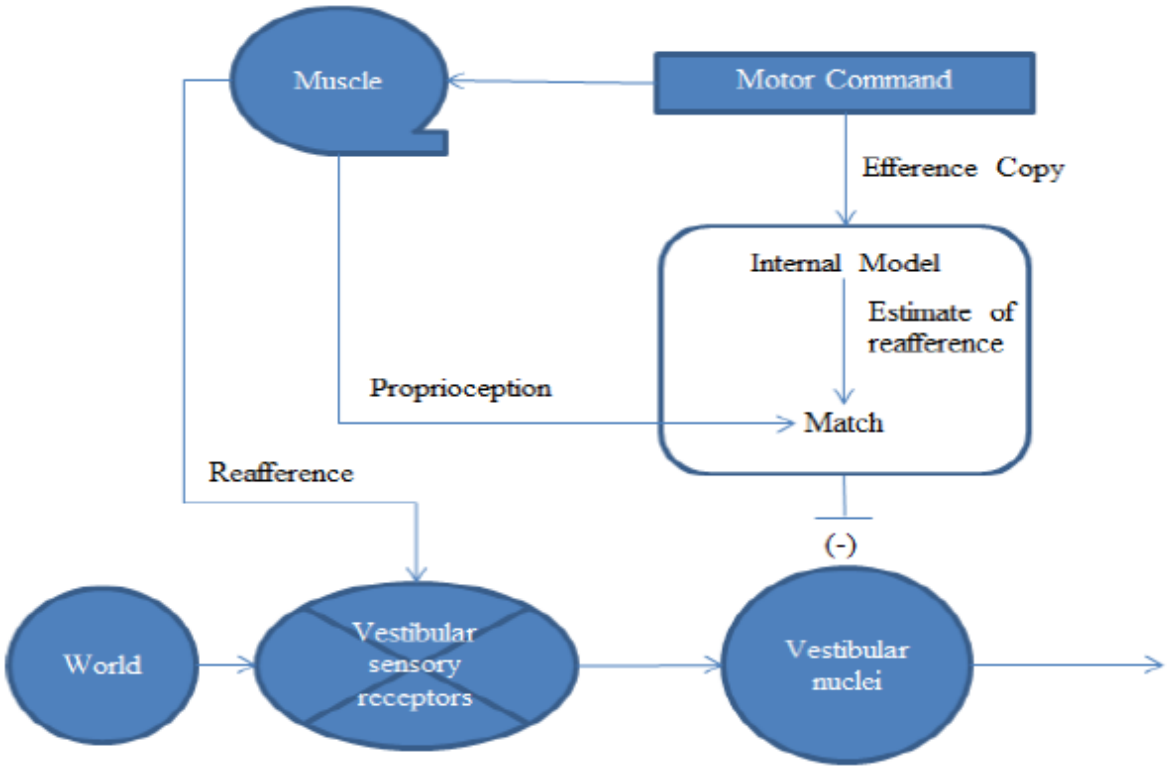


Table 2. *Studies that have identified clinical features of cervicogenic vestibular symptoms.*

Study Title	**Author**	**Clinical Features**
Cervical vertigo; Evaluation of paraclinical tests in the diagnosis of cervicogenic dizziness	Brandt & Bronstein, 2001; L'Heureux-Lebeau et al., 2014	Most common symptom descriptors of "drunkenness" or "light-headedness'"
Chronic neck pain and vertigo: Is a true balance disorder present? Manual therapy with and without vestibular rehabilitation for cervicogenic dizziness: A systematic review; Cervicogenic dizziness: A review of diagnosis and treatment	Yahia et al., 2009; Lytsad et al., 2011; Wrisley et al., 2000	Concurrent headache
Cervical vertigo; Evaluation of paraclinical tests in the diagnosis of cervicogenic dizziness Utility of a brief assessment tool developed from the Dizziness Handicap Inventory to screen for Cervicogenic dizziness: A case control study	Brandt & Bronstein, 2001; L'Heureux-Lebeau et al., 2014 Reid et al., 2017	Cervical trauma and spasm Cervical Stiffness
Cervicogenic dizziness - musculoskeletal findings before and after treatment and long-term outcome	Malmstrom et al., 2007	Cervical spine and muscle tenderness and tightness
Cervical vertigo; Chronic neck pain and vertigo: Is a true balance disorder present? Diagnostic route of cervicogenic dizziness: usefulness of posturography, objective and subjective testing implementation and their correlation	Brandt & Bronstein, 2001; Yahia et al., 2009; Micarelli et al., 2019	Limited cervical spine range of motion
Chronic neck pain and vertigo: Is a true balance disorder present?	Yahia et al., 2009	Symptoms of impaired balance
Cervical vertigo; Cervicogenic dizziness - musculoskeletal findings before and after treatment and long-term outcome; Evaluation of paraclinical tests in the diagnosis of cervicogenic dizziness	Brant & Bronstein, 1991; Malmstrom et al., 2007; L'Heureux-Lebeau et al., 2014	Pain on palpation to upper cervical spine

Table 3. *Commonly used diagnostic criteria in the study of cervicogenic vestibular symptoms*

Study Title	**Source**	**Criteria**
Clinical characteristics in patients with cervicogenic dizziness: A systematic review; Cervicogenic dizziness: A review of diagnosis and treatment	Knapstad et al., 2019; Wrisley et al., 2000	Vestibular symptoms correlated with neck pain
Evaluation of paraclinical tests in the diagnosis of cervicogenic dizziness	L'Heureux-Lebeau et al., 2014	Temporal association to trauma
Cervicogenic dizziness: A review of diagnosis and treatment; The conundrum of cervicogenic dizziness	Wrisley et al., 2000; Magnusson & Malmstrom 2016	An ex juvantibus confirmation of the diagnosis
Cervical vertigo--reality or fiction? The conundrum of cervicogenic dizziness; Cervicogenic dizziness: A review of diagnosis and treatment; Chronic neck pain and vertigo: Is a true balance disorder present? Evaluation of paraclinical tests in the diagnosis of cervicogenic dizziness; Utility of a brief assessment tool developed from the Dizziness Handicap Inventory to screen for Cervicogenic dizziness: A case control study; Biobehavioural analysis of the vestibular system and posture control in patients with cervicogenic dizziness. A cross-sectional study	Brandt, 1996; Magnusson & Malmstrom, 2016; Wrisley et al., 2000; Yahia et al., 2009; L'Heureux-Lebeau et al., 2014; Reid et al., 2017; Grande-Alonso et al., 2018	Exclusion of other vestibular and competing disorders

Table 4. *Differential diagnosis for Cervicogenic Vestibular Symptoms.*

Cervical spine fracture/ligamentous instability
Cerebrovascular disorder ● infarct ● haemorrhage ● dissection ● occlusion ● stroke/transient ischemic attack
Neurological ● cord compression syndromes ● cerebral herniation
Central vestibular disease ● tumour/mass effect ● multiple sclerosis ● hereditary ataxia ● vestibular migraine ● cerebellar atrophy ● axon damage (concussion)
Peripheral vestibular disease ● benign paroxysmal positional vertigo ● Meniere's disease ● labyrinthitis ● neuronitis ● infection ● tumour/mass effect ● persistent postural-perceptual dizziness ● otosclerosis ● bone dysplasia ● labyrinth concussion
Ototoxicity/alcohol/nutrient deficiency
Orthostatic hypotension
Autoimmune, metabolic, endocrine disease
Functional and psychiatric vestibulopathy

Adapted from Chan, 2009; Wrisley et al., 2000; Albernaz et al., 2019; Reiley 2017, and; Girasoli et al., 2018.

Figure 3. *Proposed clinical algorithm for cervicogenic vestibular symptoms.*