Cervicogenic and Vestibular Vertigo-Bridging the Gap

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ABSTRACT

The diagnosis and management of vertigo is still a challenge. Vertigo is managed by heterogenous group of specialists, including Otolaryngologists, Neurologists and Physiotherapists. The cause of vertigo could be vestibular, central or cervicogenic. The current review was undertaken as an attempt to highlight the various gaps in knowledge about vertigo of varied aetiologies. Cervicogenic vertigo is caused due to neck pathologies and is considered by some authors to be one of the most common vertigo syndromes. However, there is little mention of it in otolaryngology textbooks. Barriers across the specialities and lack of communication between the specialists are an impediment in vertigo management. This review has attempted to highlight the likely multifactorial aetiologies and fallacies in the pathophysiology of Meniere's disease. The clinical overlaps in cervicogenic and vestibular vertigo have been discussed, some of them being, the occurrence of aural symptoms and neck pain in both the entities. This review article brings out the need to revisit the pathophysiology of vestibular vertigo syndromes and include cervicogenic dizziness in the list of vertigo syndromes in Otolaryngology textbooks. The authors found a dire need of an interdisciplinary approach to elucidate the cause of vertigo of varied presentation with the goal of better patient management.

Keywords: Benign paroxysmal positional vertigo, Meniere's disease, Myofascial pain syndromes

INTRODUCTION

Dizziness or vertigo is one of the most common symptoms presented by the patients to a general practitioner or an otolaryngologist. Dizziness can be caused due to various vestibular or non vestibular aetiologies, which may include central, systemic and psychogenic or cervicogenic causes [1]. Vertigo syndromes are mainly treated by otolaryngologists and neurologists, considered as either peripheral vertigo by the former or central vertigo by the latter. Accordingly, dizziness has been categorised into four clinical syndromes namely vertigo, presyncope, disequilibrium and non specific dizziness [2]. However, in this classification there is no mention of cervicogenic dizziness as a clinical syndrome. This entity has not received much acceptance among the otolaryngologists and neurologists. Cervicogenic dizziness, according to physiotherapists, is dizziness which is associated with chronic neck pain and/or stiffness of neck with underlying pathology of the tissues of the cervical spine [3-6]. Cervicogenic dizziness is considered to be one of the most common vertigo syndromes by some studies [4,6,7]. However, whether it is always a separate clinical entity or can occur in association with other types of vertigo is controversial [6,7]. Jain S et al., noted overlaps in the vertigo syndromes of cervicogenic and vestibular dizziness [8]. This is supported by various studies, which have found significant association between vestibular vertigo and neck pain [8-10].

One of the previous study has observed that Meniere's disease is associated with neck pain and the symptoms of Meniere's disease can be precipitated with neck movement. [9]. There is an established relation between Benign Paroxysmal Positional Vertigo (BPPV) and neck pain with chronic fatigue, visual and cognitive dysfunctions and aural symptoms [10]. Classically, there are four vertigo syndromes which are most commonly accepted among otolaryngologists [Table/Fig-1] [11-19]. However, cervicogenic vertigo is missing from the list of these vertigo syndromes [8,11].

Pathophysiology of Vertigo and Balance Disorders

Balance system of our body is maintained by inputs from 3 systemsvestibular system, visual reflexes and proprioceptive system [12].

Type of dizziness	Presentation	Conditions associated with the type of dizziness
Vertigo	Sense of own self moving with respect to the surroundings or the surrounding moving with respect to the person [13]	Vestibulopathy (Meniere's disease, BPPV, Labyrinthitis) [12]
		Central Nervous system conditions like posterior circulation stroke and vertebrobasilar insufficiency [13,14]
		Cervicogenic dizziness (Barre Lieou Syndrome and Rotational Vertebral Artery Occlusion) [13-15]
Presyncope	Feeling of fainting and may be associated with excessive salivation, heat or diplopia lasting for seconds and minutes, precipitated on getting up from lying position.	Cardiovascular conditions [13,16,17].
Disequilibrium	Sensation of imbalance or falling down mostly while walking [18]	Neurological disorders like Parkinson's disease and peripheral neuropathy, cerebellar and spinal cord pathologies [11]
		Medication like benzodiazepines and Tri- cyclic antidepressant [13]
		Proprioceptive cervical vertigo [11,15]
Non specific dizziness	Vague complaints	Psychiatric disorders [13,17-19]

These inputs reach the vestibular nuclear complex located in the pontomedullary junction and cerebellum. These nuclei are connected to the cerebellum which helps in fine tuning and coordination of efferent motor impulses. These nuclei also relay sensory information to the cortical and subcortical structures which also control various components of balance system [12].

Inner ear serves as a major input in the maintenance of balance. Visual inputs and inputs from the muscle proprioceptors give additional information, however, there is also a significant input from the neck proprioceptors and are under-recognised [12].

The pathophysiology of vertigo and balance disorders can be broadly classified into [12]:

A. Disturbance in reflex function due to imbalance among various inputs.

There are different tract and reflexes which maintain the balance in the body. These tracts include the:

- 1. Vestibulo-ocular reflex which helps maintaining the image at the fovea.
- 2. Vestibulospinal reflexes which maintains the body position with respect to the head movements.
- 3. Reticulospinal tract which receives input from vestibular. Sensory and motor system and co-ordinates balance.
- 4. Vestibulocollic reflex which maintains the neck musculature with respect to head movements.
- 5. Cervical reflex is the one of the most important and is yet the most under recognised part in maintenance of balance system where the afferents originate from the neck musculature [20]. Abnormalities of neck tissue like degenerative diseases, neck tightening can also lead to vestibular dysfunction by failure of the following reflexes:
- Cervico-ocular reflex: It is a feedback mechanism which maintains eye movement which can supplement visual optokinetic reflex [21].
- Cervicospinal reflex: Maintains body position along with vestibulospinal and reticulospinal reflex. It causes extension ipsilaterally and flexion contralaterally of the limb [21].
- Cervico-colic reflex: This in an intrinsic reflex maintaining neck position. These reflexes are altered during inflammatory conditions or abnormalities of neck posture.

From above, it can be seen that inputs from both vestibular labyrinth and neck, are associated with various reflexes, responsible for maintaining body balance, and any alterations in these can lead to vertigo or imbalance [12].

Vertigo syndromes are caused due to abnormalities in these reflexes. Imbalance in the reflex input from any of the above mentioned sources leads to giddiness [8,12]. Increase or decrease in any of the three inputs (vestibular, visual or proprioceptive) leads to vertigo [8,12]. This is observed in physiologic conditions like sudden spinning of head, motion sickness, and hypofunction of vestibular labyrinth in conditions like suppurative labyrinthitis or irritative conditions of labyrinth like viral labyrinthitis. Vertigo which causes imbalance precipitated with movement, head position and body posture is also seen in proprioceptive cervical vertigo and neuropathies. This results from abnormal inputs from the body proprioceptors which may be excessive firing as seen in proprioceptive cervical vertigo secondary to neck pathologies or subnormal firing as seen in neuropathies. Neck has the highest density of proprioceptors in the body making cervical reflexes an important component of the balance system [12].

- Oculovestibular reflex: Visual reflexes are the second major afferents after vestibular afferents. They maintain the body position with respect the visual stimulus.
- Somatosensory reflexes which include the input from muscle tendon and sensory inputs from the feet which helps in maintaining the balance.
- 8. Higher integration, cortical and subcortical structures which integrate and coordinate the balance function [12].

B. Ischaemia of the end organs of balance

The end organs of balance comprise of the vestibular apparatus and the higher connections. Vascular ischaemia of the vestibular apparatus may lead to vertigo due to conditions of vestibular labyrinth like BPPV, Meniere's disease, and also certain cervicogenic vertigo syndromes like Barre Lieou syndrome and Rotational Vertebral Artery Vertigo (RVAO) described below [14].

Clinical Overlaps in Cervicogenic and Vestibular Dizziness and its Likely Pathophysiology

Cervicogenic dizziness: Cervicogenic dizziness is caused due to cervical spine pathologies and cervical muscle spasms and is closely associated with neck pain [22]. Cervicogenic vertigo is considered only when all the other causes of vertigo have been excluded, which includes vestibular, neurological and cardiovascular causes [23]. Cervicogenic dizziness is a chronic type of disease and is usually not caused by trauma to the cervical spine such as whiplash injuries. In conditions of cervical artery diseases which may include atherosclerosis or embolism, dizziness is usually continuous; however cervicogenic dizziness is episodic and may persist for a few minutes to a few hours [22]. Cervicogenic dizziness is associated with neck or postural abnormalities like forward neck posture, muscle spasms or neck stiffness, chronic degenerative conditions of the cervical spine like cervical spondylosis and is aggravated with neck movements and usually subsides with relief in neck pain [14,22].

Types of cervicogenic giddiness: The suggested types for cervicogenic giddiness include.

1. Proprioceptive Cervical Vertigo

Ryan and Cope gave the term "cervicogenic giddiness." Cervical joints harbour the highest density of proprioceptors among the spinal joints with the maximum density in the joint capsule of C1-C3. The gamma muscle spindle of the neck musculature also contains the highest density of mechanoreceptors. These receptors give afferent information to the CNS and are interconnected to the visual and vestibular system. Various factors like inflammatory conditions such as osteoarthritis, abnormal positioning of neck, spasms can alter the afferent impulses to the CNS causing vertigo. Manual therapy has been proven to be an effective measure for cervicogenic dizziness [24].

There are various studies which show that cervical spine sagittal configuration is altered in cases of neck spasms or pathologies which hastens the degenerative process of muscles, ligaments, nerves or bony processes [4,9]. Altered configuration of cervical spine causes abnormal vertebral kinematics or abnormal sensory inputs from the cervical proprioceptors [4]. Cervical spine curvatures can be measured by certain angles. In cervical Absolute Rotatory Angle (ARA), two tangent lines are drawn from the posterior border of the C2 and C7 vertebrae in a lateral X-ray of cervical spine and the angle between the two lines gives us information about the cervical spine curvature. Anterior Head Translocation (AHT) is measured by drawing a vertical line from the posterior inferior margin of the body of C7 vertebrae in a lateral cervical spine X-ray and measuring its distance from the posterior superior margin of the body of C2 vertebrae. Moustafa IM et al., measured the ARA and AHT of 252 asymptomatic individuals and found that 25 degree ARA was one standard deviation below the mean value and that 15 mm was the mean value for AHT [4].

The authors of this article have found that these angles are altered in cases of cervicogenic dizziness and also in some cases of vestibular dizziness like Meniere's Disease and BPPV from compilation of their unpublished data [8]. This fact again goes in favour of same origin for cases of cervicogenic dizziness and some cases of vestibular dizziness. Systematic review of literature shows that manual therapy is an effective measure for cervicogenic dizziness but it's benefit in the long term questionable [4]. However, in a study it is shown that long term improvement (more than 1 year) of cervicogenic dizziness was achieved by improving the altered cervical configuration which could be done using traction methods. This further proves the role of altered cervical configuration in causing cervicogenic dizziness [4].

2. Barre-Lieou Syndrome (Sympathetic Dysfunction)

Barre suggested that blood volume in the vertebral artery is sensitive to the sympathetic stimulus which is stimulated in response to the pathological changes in the cervical tissue and degenerative diseases causing reflex vasoconstriction of vestibulobasilar system. Barre Lieou syndrome comprises of dizziness, ringing sensation, headache, diplopia, nausea, vomiting, palpitation and Gl upset. It was hypothesised that compression or irritation of posterior longitudinal ligament and dura caused by pathological conditions of cervical tissue like cervical spondylosis stimulates the sympathetic system affecting the vertebral artery blood volume causing giddiness, thus resection of posterior longitudinal ligament may resolve the irritation or compression. Excellent results were obtained after anterior cervical dissectomy and fusion in a study [15].

3. Rotational Vertebral Artery Vertigo (Bow Hunter Syndrome)

Labyrinthine artery is an end artery thus making the labyrinth susceptible to Vertebrobasilar insufficiency. In response to ischaemia, depolarisation of the sensory cells leads to ringing sensation and dizziness. Atherosclerosis or thromboembolism may be the cause of vertebrobasilar insufficiency. Blood supply of the predominant vertebral artery could be affected by head rotation due to compression against atlantoaxial joint causing "Rotational Vertebral Artery Vertigo (RVAO)" [15].

4. Migraine-associated Cervicogenic Vertigo

Vertigo and migraine has been well documented and "Migraine associated Vertigo" has been widely identified. It is seen that vertigo is seen in 33% of cases of migraine and most of the cases was associated with neck and shoulder pain [15]. Migraine is seen associated with about 50% cases of Meniere's disease and the disease is more common in older age group [22].

Pathophysiology Underlying Aural Symptoms in Cervicogenic and Vestibular Vertigo

There is occurrence of aural symptoms like tinnitus and hearing loss in association with cervicogenic dizziness in syndromes like Barre Lieou and Rotational vertebral artery syndrome. Aural symptoms of cervicogenic giddiness associated with Barre Lieou Syndrome and RVAO, are considered to be attributable to vascular insufficiency related to sympathetic dysfunction, in the former and vertebral artery compression in the latter [1]. Again, certain idiopathic syndromes of vestibular vertigo like Meniere's Disease and idiopathic causes of sudden audio-vestibulopathy are said to be associated with ischaemia of labyrinth [25]. Extrapolating these observations, it can be considered that in some of the cases of idiopathic vertigo syndromes, cause of vestibular vertigo could originate from the neck, with effect on either the vertebral artery circulation, or sympathetic dysfunction [8]. Based on the above, it can be inferred that there is possibility of same underlying aetiology, likely in the neck, in certain cases, for both vestibular and cervicogenic vertigo.

The labyrinthine artery supplies the 8th nerve after which it divides into anterior vestibular and common cochlear artery. The labyrinthine artery is a branch of anterior inferior cerebellar artery which is a branch of Basilar artery formed by the fusion of both the vertebral arteries. The common cochlear artery divides into spiral modiolar artery (cochlear artery) and vestibulocochlear artery. Major supply to cochlea is by the cochlear artery (80 percent) and posterior vestibular artery (20 percent) which are end arteries supplying from basal to apical regions of the cochlea [26]. Thus, in vascular ischaemia the peripheral apical regions (low frequency regions) of the cochlea are affected, which is also seen in cases of Meniere's disease [25,27,28].

Pathophysiology of Meniere's disease: According to American Academy of Otolaryngology and Head and Neck Surgery (AAO-HNS) (1995), Barany society, European Academy of Otology and Neurotology, definite Meniere's disease is when there are two or more spontaneous episodes of vertigo each lasting 20 minutes to 12 hours, low to medium frequency sensorineural hearing loss in audiometry in the affected ear during or after the episode of vertigo, symptoms of fluctuating hearing loss, tinnitus and aural fullness which does not explain any other vestibular complaints [29]. In probable Meniere's disease in which the symptoms last for 12-24 hours with fluctuating aural symptoms not explaining any other condition [29,30]. Possible Meniere's disease is when there is episodic vertigo without documented hearing loss or sensorineural hearing loss which is fluctuating or fixed with disequilibrium but without definitive episodes and other causes have been excluded [29,30]. Lermoyez syndrome is characterised by reduction in tinnitus and hearing loss during or shortly after attacks of vertigo [31]. Some of the patients of Meniere's disease develop Tumarkin's otolithic crisis or drop attacks which occur without warning, loss of consciousness or associated neurological symptoms [32].

Meniere's disease is characterised by episodes of fluctuating hearing loss, tinnitus and vertigo along with gradually progressive sensorineural hearing loss [33]. According to AAO-HNS criteria the ubiquitous and highly specific finding in all cases of Meniere's disease is Endolymphatic hydrops or dilated endolymph [34]. In advanced cases of Meniere's disease, loss of hair cells, loss of dark cells of crista nerve fibres with neuroepithelial degeneration is seen with thickening of basement membrane along with atrophy of stria vascularis in ipsi- as well as contralateral ear [35-38].

Controversies associated with the pathophysiology of Meniere's disease: Initially vascular hypothesis of Meniere's disease was considered to be the underlying aetiology of the disease however autopsies did not show any signs of widespread ischaemia. Endolymphatic hydrops was considered to be the causative factor in Meniere's disease but not all temporal bones with endolymphatic hydrops had history of Meniere's disease [23,36,37]. Thus, Meniere's disease and endolymphatic hydrops could be caused due to another unknown factor and not all patients with endolymphatic hydrops suffer from Meniere's disease [37].

The pathophysiological features of Meniere's disease are not always consistent. Neck pain is found to be associated with Meniere's disease in various studies [8,9]. Thus, there might be a multifactorial aetiology for Meniere's disease. The cervical reflexes may contribute to vertigo and may also contribute to the ischaemia of the vestibular labyrinth and the cochlea [14].

Autoimmune, vascular, allergy, diet, viral infections, genetic variations and neck related factors have been suggested to cause endolymphatic hydrops [8,36,38,39]. Direct mechanical damage to inner ear cells due to hydrops has been seriously questioned [25,40,41]. Potassium intoxication theory and Ruptured membrane theory have been disapproved by previous studies [42-47]. Jain S et al., hypothesised that the CSF pressure may increase due to neck spasms with resultant increase in endolymphatic pressure [8]. It is seen that pressure of perilymph and endolymph increases with rise in intracranial pressure as there is no pressure difference between perilymph and endolymph. They suggested that apart from raised CSF pressure that is caused due to neck spasms which results in increased endolymphatic pressure, direct compression of vertebral artery resulting from cervical problems could also be the cause of ischaemic damage to the inner ear which gives rise to aural symptoms in Meniere's disease, a hypothesis which needs confirmation by further studies [8].

Inner ear damage has been reported in patients with Migraine [22,25]. There is a strong association between migraine and Meniere's disease. However, migraine alone is not sufficient to cause Meniere's disease, it is always associated with Meniere's disease. Neck spasm has been associated with both Meniere's disease and Migraine [9,15].

Vascular theory of Meniere's disease: Labyrinth is supplied by end artery making it vulnerable to ischaemia. It has been hypothesised that endolymphatic hydrops lowers the threshold of inner ear for ischaemia. In vascular conditions such as migraine, vascular malformations (in younger individuals), hypertension and hyperviscosity, vasculitis, traumatic raised intracranial tension, hydrocephalus, sleep apnea, anaemia, lung diseases may lower perfusion and cause chronic hypoxia. These factors decrease the perfusion of inner ear till just above the threshold and thus minor changes in inner ear pressure leads to ischaemia with the stria being most sensitive followed by distal process of sensory cells. This gives rise to symptoms of hearing loss, tinnitus and vertigo. The marginal zone for blood supply is the apex of the cochlea thus the low frequency will be affected first as seen in Meniere's disease [25]. Since an attack of Meniere's disease typically lasts for 5-60 minutes, there is no necrotic tissue and ischaemia reperfusion injury occurs only in severely affected hair cells which undergo apoptosis [22,25].

This hypothesis is supported by the fact that there is a significantly higher incidence of Meniere's disease in individuals with cardiovascular risk factors. The incidence of Meniere's disease and cardiovascular factors increases with age. Migraine is the most common risk factor for cerebrovascular ischaemia in younger individuals. There is a strong association between migraine and Meniere's disease [22,25].

Gaps in Meniere's disease pathophysiology: It has been seen that headache, pain or tightness of neck, abnormalities in neck position (forward posture) is common in patients of Meniere's disease and some cases of BPPV [8]. Considering the association of Meniere's patient with neck pain, ischaemia of the vestibular labyrinth in Meniere's disease could be an end organ pathology due to primary underlying pathology in the neck related to muscle spasms, vertebral artery compression or sympathetic dysfunction. Also, this could be the possible link between cervicogenic dizziness and other forms of vestibular dizziness (Meniere's disease and BPPV). It implies that the primary problem in both types of dizziness could underlie in the neck, in selected number of cases. Abnormal posture, certain activities or absence of activities, trauma to neck or other parts of the body may cause myofascial problems, which are represented as chronic stress in the neck and spasms of the neck which present as limited range of movements, with likely resultant change in the circulation of blood and cerebrospinal fluid, giving rise to symptoms of Meniere's disease and cervicogenic dizziness [8].

CONCLUSION(S)

This review has attempted to highlight the likely multifactorial aetiologies and fallacies in the pathophysiology of Meniere's disease. The clinical overlaps in cervicogenic and vestibular vertigo have been discussed, some of them being the occurrence of aural symptoms and neck pain in both the entities, with role of cervical reflexes in causation of vertigo. The present review article has attempted to highlight the gaps in the knowledge of pathophysiology of vertigo, and cervicogenic dizziness as an entity, not known to most otolaryngologists. The authors suggest the need to revisit the pathophysiology of vestibular vertigo syndromes and include cervicogenic dizziness in the list of vertigo syndrome in Otolaryngology textbooks. Interdisciplinary approach, including involvement of physiotherapy assessment, for elucidation of cause of vertigo of varied presentation with a goal of better understanding of pathophysiology and patient management.

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- iThenticate Software: Jul 30, 2021 (8%)

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