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"Cervical Vertigo-Pathophysiology and Management: An Update"

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Abstract: *There are many myths about cervical vertigo. Cervical vertigo is defined as vertigo from the cervical spine. In this review article, we outline the basic science and clinical evidence for cervical vertigo according to the current literature. So far, there are 4 different hypotheses explaining the vertigo of a cervical origin, including proprioceptive cervical vertigo, Barré-Lieou syndrome, rotational vertebral artery vertigo, and migraine-associated cervicogenic vertigo. Proprioceptive cervical vertigo and rotational vertebral artery vertigo have survived with time. Barré-Lieou syndrome once was discredited, but it has been resurrected recently by increased scientific evidence. Diagnosis depends mostly on patients' subjective feelings, lacking positive signs, specific laboratory examinations and clinical trials, and often relies on limited clinical experiences of clinicians. Neurological, vestibular, and psychosomatic disorders must first be excluded before the dizziness and unsteadiness in cervical pain syndromes can be attributed to a cervical origin. Treatment for cervical vertigo is challenging. Manual therapy is recommended for treatment of proprioceptive cervical vertigo. Anterior cervical surgery and percutaneous laser disc decompression are effective for the cervical spondylosis patients accompanied with Barré- Liéou syndrome. As to rotational vertebral artery vertigo, a rare entity, when the exact area of the arterial compression is identified through appropriate tests such as computed tomography angiography (CTA), magnetic resonance angiography (MRA), or digital subtraction angiography (DSA). Decompressive surgery should be the chosen treatment.*

Keywords: *Cervical vertigo, neck pain, whiplash injury, cervical spondylosis, manual therapy, vertebrobasilar insufficienc, vestibular rehabilitation.*

I. Introduction

The term vertigo or dizziness refers either to an unpleasant disturbance of spatial orientation or to the erroneous perception of movement. Vertigo involves a perceived movement either of one's own body, such as swaying or rotation, or of the environment, or both (1,2). In different studies have shown that dizziness (including vertigo and nonvestibular dizziness) ranks among the most common complaints in medicine, affecting approximately 20 – 30% of the general population (3,4). There are a various causes of vertigo including those arising from disturbances of the ear, nose, and throat (ENT); central nervous system (CNS); cardiovascular system; and benign positional paroxysmal vertigo (BPPV). Although diagnosis of the disorder can sometimes be difficult and require specialist facilities, these problems can often be successfully treated. However, a group of patients remains and it is suspected that the cause of their problem is a disorder of the cervical spine, known as cervical vertigo(5). In a study by Colledge et al (6) investigating the causes of dizziness in the elderly, the authors attributed dizziness to cervical spondylosis in 65% of cases. However, whether cervical vertigo is an independent entity remains controversial (7,8). Supporters of cervical vertigo usually believe it to be the most common vertigo syndrome; they confirm their diagnosis with a range of signs, symptoms, and tests, which are either inappropriate or irrelevant (7). At the same time, there is much evidence that cervicogenic dizziness is a distinct disorder. Several authors have demonstrated that anesthetic injections to the upper cervical dorsal nerve roots can produce dizziness and nystagmus (9-11). Electrical stimulation to cervical muscles has also been shown to induce a sensation of tilting or falling (12). Their opponents reject the diagnosis for 2 reasons.

In the first place, there is neither a reliable clinical test for the syndrome nor a typical time course for the condition. Secondly, reliable and well-established signs and tests can support a convincing alternative diagnosis in almost all patients presenting with vertigo (7). All clinical studies on cervical vertigo to date have 3 weak points: the inability to confirm the diagnosis, the lack of a specific laboratory test, and the unexplained discrepancy between patients with severe neck pain without vertigo and patients complaining of disabling vertigo with moderate neck pain (13). It was overemphasized in the past and is overlooked nowadays (8). There is a lack of consensus regarding its pathophysiology, diagnostic criteria, and optimal treatment (14).

Proprioceptive input from the neck helps in the coordination of eye, head, and body posture as well as spatial orientation. Based on this, it has been argued that a syndrome of cervical vertigo might exist (13,15). In studies conducted in humans using cervical affects such as endogenous (pain) (16), external chemical agents (local anesthesia) (9), and galvanic stimulation(12), it was possible to cause postural imbalance, nystagmus, and vertigo. In addition, manual therapy is effective for cervical vertigo, which provides indirect evidence supporting the existence of cervical vertigo (5,17-19). According to Wrisley et al (1), the diagnosis of cervical vertigo is dependent upon correlating symptoms of imbalance and vertigo with neck pain and excluding other vestibular disorders based on history, examination, and vestibular function tests. This concept is superior to the definition proposed by Yahia et al (20), which defined cervical vertigo as an association of thefollowing features: chronic cervical pain, vertigo after cervical rotation without nystagmus, cervical osteoar- thritis, and/or intervertebral disc degenerative changes, and acceptable by most clinicians. This paper reviews the pathophysiology and management of cervical vertigo according to the existing literature.

Pathophysiology

The pathophysiology of cervical vertigo is not clear. According to Heikkila (21) and Yacovino and Hain (22), there are 4 different hypotheses explaining vertigo of a cervical origin have been proposed .

Proprioceptive Cervical Vertigo

In 1955, Ryan and Cope (23) first introduced the term"cervical vertigo." They believed cervical vertigo was due to abnormal afferent input to the vestibular nucleus from damaged joint receptors in the upper cervical region. The proprioceptive system is extremely well-developed, because the cervical zygapophyseal joints are the most densely innervated of all the spinal joints (24). Hulse (25) found that 50% of all cervical proprioceptors were in the joint capsules of C1 to C3. In addition, there is an abundance of mechanoreceptors in the γ-muscle spindles of the deep segmental upper cervical muscles (26). The mechanoreceptor seems to be a critical component of the proprioceptive system. The dense network of mechanoreceptors in the soft tissues in the neck region not only controls multiple degrees of freedom of movements about each of its joints but, more importantly, gives the CNS information about the orientation of the head with respect to the rest of the body via direct neurophysiological connections to the vestibular and visual systems (5,27). It has been found that the afferent cervical activity is more likely controlled by neck mechanoreceptors in the upper cervical spine. These mechanoreceptors' function can be altered by direct trauma, muscular fatigue, degenerative changes, or direct effect of pain (28). Therefore, these anatomical bases may explain why upper cervical dysfunction can cause cervical vertigo.

Whiplash injuries often caused by cervical proprioception disorders, leading to cervical vertigo. Whiplash injuries are experienced by 0.1% of the population (29) and the incidence of symptoms of vertigo in whiplash sufferers has been variously reported as 20 – 58% (1), 25 – 50% (30), and as high as 80 – 90% (31). Rubin et al (32) compared posturography in 29 patients with whiplash injuries to 51 healthy subjects, and reported significantly greater abnormalities in the balance of the patients. Endo et al (33) found that patients with persistent vertigo or dizziness after whiplash injury ex- hibit specific body sway by posturography. There is clear evidence that patients with whiplash injuries always have postural control impairment (32-34), but the sole responsibility of cervical proprioception has never been demonstrated as abnormal vestibular function and asymmetric reduction of vertebral arterial flow can also occur after whiplash injury (13). In a study by Treleaven et al (34) in 2003, people with whiplash-associated dizzi- ness and/or unsteadiness (n = 102) were shown to have significantly greater joint position errors and a higher neck pain index than control subjects (n = 44), consis- tent with cervical mechanoreceptor dysfunction being a likely cause of the symptoms. Yacovino and Hain (22) considered that in whiplash-associated disorder, pain, limitation of movement, and strains of joint capsules, paravertebral ligaments, and cervical musculature could modify the proprioceptive cervical balance in a sustained way and produce mild but chronic vertigo.

In addition to whiplash injuries, cervical spondylosis (degenerative osteoarthritis) and cervical muscle spasms can also cause vertigo. Degenerative cervical spine diseases, depending on their nature, lead to different sensorial strategies in posture (35). Colledge et al (6) studied the causes of vertigo in the elderly, and found that 65% were caused by cervical spondylosis. And dizziness and pain were reduced with an injection of anesthetic into the posterior neck muscles (23). Therefore, cervical vertigo may be a result of whiplash injury, other forms of cervical spine dysfunction, or spasms in the cervical muscles.

Brown (15) demonstrated that there were close connections between the cervical dorsal roots and the vestibular nuclei with the neck receptors (such as pro- prioceptors and joint receptors), which played a role in eye-hand coordination, perception of balance, and postural adjustments via an experimental study in animals. With such close connections between the cervical receptors and balance function, it is understandable that traumatic, degenerative, inflammatory, or mechanical derangements of the cervical spine can affect the mechanoreceptor system and give rise to vertigo. Some experimental researches reported a "reversible" lesion in

the neck and observed deficits in balance function and vision, which provides further evidence to support the neck receptors playing a very important role in controlling the movement of the eyes and body posture. A study carried out by Biemond and de Jong (10) found that injecting anesthetic into the cervical region of rabbits could produce positional nystagmus.

Subsequently, Cohen (11) found that injecting anesthetic into the upper 3 cervical dorsal roots could cause dysfunctions in balance, orientation, and coordination in primates. In humans, injection of anesthetic into the cervical dorsal roots gave rise to disequilibrium (1,9). After sectioning C2 and C3 posterior nerve roots in patients suffering from torticollis, Biemond and de Jong (10) observed a strong postural nystagmus oriented to the side of the surgery. Later, Wapner et al (12) found that the electrical stimulation to the cervical muscle could cause equilibrium disturbance. Accordingly, the aberrant input from the cervical proprioception may be related to muscle spasms in the sternocleidomastoid and upper trapezius muscle (9,15). Hence, this evidence leads to the current theory that cervical vertigo results from abnormal input into the vestibular nuclei from the proprioceptors of the upper cervical region.

Neck pain may be related with cervical vertigo. Patients with neck pain, especially in the upper cervical spine, often have postural instability (36). A convincing mechanism of cervical vertigo is based on altered upper cervical somatosensory input associated with neck ten- derness and limitation of movement (7,13). Interstitial inflammatory mediators have been postulated to sensitize muscle spindles (37), and myofascial trigger points exhibit spontaneous electromyogram activity, which is compatible with hyperactive muscle spindles (38). Neck proprioception has been studied previously in patients with cervical vertigo as individuals with whiplash-associated unsteadiness and/or dizziness were shown to have significantly greater joint position errors and more severe neck pain than control subjects (27). If the firing characteristics (symmetric or asymmetric) of the cervical somatosensors change due to neck pain, a sensory mismatch between vestibular and cervical inputs would be expected to result in cervical vertigo (13,39,40).

Barré - Liéou Syndrome (Sympathetic Dysfunction)

As early as 1926, Barré (41) noted pathological changes of cervical vertebra might stimulate sympathetic nerve fibers and play a role in modifying the blood volume of the vertebral artery. Subsequently, he along with Liéou described a series of symptoms called the Barré-Liéou syndrome, including vertigo, tinnitus, headache, blurred vision, dilated pupils, nausea, vomiting, and so on (42). They suggested that the sympathetic plexus surrounding the vertebral arteries could be stimulated by cervical degenerative disease and this stimulation could contribute to reflexive vasoconstriction of vertebrobasilar system, thus accounting for the Barré - Liéou symptoms. Animal experiments showed that electrical stimulation of sympathetic path- ways appears to have little effect on cerebral blood flow (CBF) during normotension rats (43). A later study showed that acute or chronic sympathetic denervation does not alter distribution of CBF over a wide range of arterial pressure or during hypocapnia in anesthetized dogs (44). Baumbach and Heistad (45) found direct evidence that sympathetic stimulation increases resistance of large arteries, but resistance of distal vessels either tends to decrease or decreases significantly; thus, total cerebral vascular resistance remains unchanged dur- ing sympathetic stimulation. Therefore, sympathetic denervation has a minimal effect on CBF under normal conditions (46). Foster and Jabbour (47) thought that Barré-Liéou syndrome was discredited and not a useful eponym for 2 reasons. Firstly, its symptoms are not unique, being common to many vestibular disorders, all of which could be included in its spectrum. Cervical arthritis, tinnitus, and headache are extremely common and so a number of people will have this combination purely by chance. Vertigo, tinnitus, and headache associated in several different vestibular disorders, as noted by Meniere 60 years before Barré (46). Secondly, the cause of the syndrome has been discredited. Denervation has no apparent ill effects in normotensive individuals, and stimulation does not cause vasoconstriction. No sympathetic or vascular changes were subsequently identified that could account for these symptoms(44,46).

No scientific review of this syndrome has appeared in the English literature in the last 60 years, but it has been resurrected recently. Li et al (48) found a large number of sympathetic postganglionic fibers were distributed in the cervical posterior longitudinal ligament (PLL) of every segment in rabbits. The density of sympathetic fibers distributed in the intervertebral portion of PLL was more than that in the vertebral portion. Compared with the deep layer section, the nerve fibers in the superficial PLL layer section were thicker and more densely populated. Existence of sympathetic postganglionic fibers was also confirmed in human specimens. Those nerve fibers were mostly short and isolated in areatus form, with non-interwoven branches. It's also reported that the cervical uncovertebral joint capsule, PLL, rear of the annulus, and dural sac are distributed with sympathetic fibers in humans (49).

In a recent study conducted by Hong and Kawagu- chi (51), 39 patients who were diagnosed with cervical spondylosis with sympathetic symptoms and underwent anterior cervical discectomy and fusion (ACDF). After the operation, good to excellent clinical results were attained in 95% of these patients. They speculated that it may be the compression or stimulation of the sympathetic nervous system other than the

vertebral artery which induces symptoms such as vertigo, dizziness, headache, tinnitus, nausea and vomiting, heart throb, hypomnesia, and gastroenterologic discomfort. Compression of the dura mater and the PLL of the cervical spine may induce a sympathetic reflex. The activity may pass through the ganglia and the sympathetic trunk to the postganglia fibers arriving at the target organ, such as the vertebral artery, and subsequently inducing symptoms like vertigo.

After ACDF, the decompression of the dura and the PLL was clear and thus the sympathetic symptoms were relieved. Wang et al (52) hypothesize that: (1) there are sympathetic nerve postganglionic fibers distributed in the PLL or discs; (2) pathological changes secondary to degeneration of the intervertebral disc may cause irritation of sympathetic nerve fibers in the PLL or discs, leading to sympathetic symptoms via certain pathways; and (3) removal of the PLL or stabilization of the segment which decreases the irritation to the PLL will help to eliminate the sympathetic symptoms.

Rotational Vertebral Artery Vertigo (Bow Hunter Syndrome)

Generally, insufficient blood supply does not necessarily cause symptoms if there is sufficient collateral circulation, whereas a full range of symptoms commonly occur as a result of an insufficient terminal vessel. The vascular supply to the vestibulocochlear organ, being an end artery, makes this organ more susceptible to vertebrobasilar insufficiency (VBI). And neurons, axons, and hair cells in the vestibulocochlear system are known to respond to ischemia by depolarizing, causing transient hyperexcitability with ectopic discharges, manifesting as tinnitus, vertigo, and dizziness. The most common causes of VBI are atherosclerotic stenosis or thromboemboli, but extrinsic compression by bony structures and soft tissues has also been described (53). The vertigo caused by reversible stenosis or obstruction of the dominant vertebral artery at the atlantoaxial level upon head rotation is called "rotational vertebral artery vertigo." It was originally first considered physiologically by Toole and Tucker (54), who found com- promised blood flow in vertebral arteries due to head rotation in hemodynamic studies of cadavers. Noh et al (55) thought that isolated vertigo and nystagmus in patients with rotational vertebral artery vertigo (RVAO) may occur due to transient ischemia of the inferior cerebellum or lateral medulla via the cerebral angiography and transcranial doppler. However, some other studies found that in most patients with RVAO, the symptoms might be ascribed to asymmetrical excitation of the bilateral labyrinth induced by transient ischemia or by disinhibition from inferior cerebellar hypoperfusion (56,57). According to anatomic and histopathologic studies, the labyrinth is vulnerable to ischemia (58). On the one hand, the labyrinthine artery, an end artery with very little collateral, provides the arterial blood supply to the membranous labyrinth (59).

On the other hand, animal experiments suggested that the vestibular and cochlear nerve fibers turn into unexcitable within 20 seconds to several minutes after complete blood flow interruption (60). Therefore, Choi et al (61) and Brandt and Baloh (62) thought that vertigo, nystagmus, and tinnitus in patients with RVAO without any other brainstem signs and symptoms were due to labyrinthine rather than central brainstem ischemia. In many literature, RVAO is also called"bow-hunter syndrome"(22,61-64). This name was first used by Sorenson (63) in 1978 to describe a patient who had a brain stem stroke during archery practice and experienced this constellation of symptoms. Mechanical compression of bow-hunter syndrome can be induced by transverse foraminal stenosis, cervical osteophytes, fibrous bands, and other degenerative processes including cervical motion segment instability (64). Recently, Sarkar et al (53) reviewed the published literature and found that bow-hunter syndrome had often been reported at the C1-2 level, and the majority of cases occur in patients of older than 50 years because of degenerative osteophytes and contralateral atherosclerosis. Similar to bow-hunter syndrome, in 1992 Weintraub (65) reported 2 cases with cervical vertigo that occurred after shampoo treatment in a hairdressing salon and first named "beauty parlor stroke syndrome". The study found that the most likely pathophysiologic mechanism of the syndrome was stenosis of the vertebral artery caused by compression at the atlanto-occipital junction (66). Endo et al (66) also demonstrated that hyperextension of the neck with the head hanging during hair shampooing in a hairdressing salon is a major risk factor for VBI and found that patients with beauty parlor stroke syndrome are similar to those seen in patients that have been in traffic accidents and fall into the category of craniocervical injuries. Recently, research found that there were no significant changes in blood flow or velocity in the vertebral arteries of healthy young male adults after various head positions and cervical spine manipulations (67). However, many other researchers showed that the decrease of vertebral artery velocity was related to vertigo during head rotation. Machaly et al (68) found the patients with cervical spondylosis complaining of vertigo (71.4%) have significantly lower blood flow parameters than non-vertigo patients with cervical spondylosis (32.9%) during head rotation using color duplex sonography. Another study demonstrated a pathological decrease of vertebral artery flow velocity in patients with degenerative changes in the cervical spine (69). A meta-analysis of 9 studies concluded that vertebral artery blood flow velocity was compromised more in patients with VBI than in healthy individuals on contralateral rotation (70).

Migraine-Associated Cervicogenic Vertigo

In 2013, Yacovino and Hain (22) put forward the hypothesis of "migraine-associated cervicogenic vertigo." They postulated that this hypothesis could explain why some patients suffering from cervical pain have vertigo while others do not and thought migraine could be a link between cervical pain and cervicogenic vertigo (22). On the one hand, the association between migraine and vertigo has been well documented in the literature and the term "migraine-associated vertigo" has been widely recognized by the international com- munity (71-74). A study carried out by Selby and Lance (75) found one-third of people with migraine experience vertigo. On the other hand, cervical pain and stiffness is a typical finding of migraine. Blaschek et al (76) found that neck and shoulder pain was closely associated with migraine in adolescents and 63% of the group of migraine patients reported cervical and shoulder pain. However, Kelman (77) found that 39.7% of patients with migraine were reported to ex- perience neck pain. In a representative cross-section of migraineurs, neck pain was more commonly associated with migraine than nausea, a defining characteristic of the disorder (78). Subsequently, Yacovino and Hain (22) thought that there was a wide overlap between migraine-associated vertigo with cervical pain or stiffness related to migraine and the symptomatic defini- tion of cervical vertigo. They believed that a possible way to relate both entities in a bidirectional way would be the cervical trigeminal vestibular path. The reciprocal connections between the vestibular nuclei and the trigeminal nucleus caudalis can provide a mechanism in which the vestibular signals would influence the vascular trigeminal paths, widely related to the processing of the vestibular and trigeminal information during the migraine attacks (22). A similar activation of the cervical trigeminal path gave rise to migraine and then resulted in cervicogenic vertigo. But, this hypothesis of migraine-associated cervicogenic vertigo requires further research to confirm.

Diagnosis

Doctors knows that diagnosis of cervical vertigo can be challenging and controversial, and it is made only after other potential causes for dizziness or vertigo have been excluded (1,14). The symptom of neck pain for diagnosis of cervical vertigo is very important. If a patient has a chief complaint of vertigo, but it is not accompanied by neck pain, a diagnosis of cervical vertigo may first be excluded (13,15). Benign paroxysmal positional vertigo (BPPV) is often misdiagnosed as cervical vertigo. So, a Dix-Hallpike test for the patients with vertigo is necessary (13). The Dix-Hallpike test consists of a series of head movements conducted in order to stimulate the movement of the debris in the posterior semicircular canal. The patient starts in a sitting position and their head is turned 45º towards the side to be tested. The assessor then assists them to lie down quickly and extends their neck 20º over the end of the plinth, maintaining 45ºrotation. The assessor should be able to see the patient's eyes and should observe for nystagmus. A positive response is elicited if rotational nystagmus is noted (79,80). In addition, the vestibular functions must be tested for the patients with vertigo by the otolaryngologist. If the vestibular disorders are excluded, a diagnosis of cervical vertigo may be supported (1).

Patients with cervical vertigo usually have pain in the nape of the neck and occipital region, sometimes accompanied by stiffness of the neck. Symptoms of cervical vertigo may be reproduced, and nystagmus may be induced by bending the neck, but not by merely positioning the head in space (23). In addition, in 2 re- cent randomized controlled trials (RCTs), the symptom of neck pain has been included in criteria for cervical vertigo (17,81). Cervical vertigo typically occurs in episodic nature lasting minutes to hours (1,22). Symptoms resulting from cervical vertigo are often increased with neck movements or neck pain and decreased with interventions that relieve neck pain (1). Cervical vertigo may occur anywhere from days to months or longer after an injury of the head and neck (15). Neck pain often radiates to the temporal-parietal region in a banana- shaped distribution and may only be present during deep palpation of the neck. Thus, some patients may be unaware of neck pain until examined. Examination findings in cervical vertigo include reproducible vertigo with manipulation of the neck, pain with palpation of the suboccipital region, cervical transverse processes of C1 and C2, cervical spinous processes of C2 and C3, levator scapulae, upper trapezius muscle, splenius, rectus, and semi-spinalis muscles. These findings can often be missed during an otolaryngologist's routine examination (14).

To differentiate vertigo from rotational vertebral arterial syndrome, the use of MRA or computed tomography angiography (CTA) is particularly useful to identify a vertebral arterial compressive pathology (e.g., bow-hunter syndrome). The most reliable and essential test to identify the exact area of mechanical vertebral arterial compression and to make a surgical decision is digital subtraction angiography (DSA), especially on position with head rotation (rotation and extension). Nevertheless, since DSA is an invasive method requiring considerable iodine-containing contrast, it should be reserved for situations where suspicion is high, rather than as a screening test (22). Non-invasive techniques like cervical MRA avoid the risks of angiography and also the radiation risk of CTA. Dynamic MRA or CTA may be problematic because it requires patients holding the symptomatic posture for some minutes, which can be very difficult for these patients, and may even cause unconsciousness although both methods can show the stenotic area even without rotation (82,83).

If vestibular function is tested by vestibular stimuli and visual function by visual stimuli, then somatosensory cervical function should be tested with selective somatosensory stimulation. Complains of vertigo or unsteadiness on turning the head are much more likely to imply vestibular rather that cervical dysfunction(13). The neck torsion nystagmus test is considered by some researchers to identify cervical vertigo. This test requires the head of the patient to be stabilized while the body is rotated underneath. Theoretically, when neck proprioceptors are stimulated the inner ear structures must remain at their resting state (84). However, this test has not been demonstrated to be specific for cervical vertigo (1). It has been demonstrated that up to 50% of patients without cervical spine pathology have nystagmus with the neck torsion test (85). A positive response (nystagmus) may not indicate pathology, but may instead be a manifestation of the cervical ocular reflex (85). In addition, evidence of increased postural sway in patients with whiplash-associated vertigo or other cervical dysfunction has led some to consider using posturography as a diagnostic test. Endo et al (33) found patients with vertigo after whiplash injury had a special gait by posturography. Posturographic tests can be used to assess and confirm the body's imbalance in patients with whiplash injury (86). However, this test cannot be performed in the clinic without specialized equipment. The lack of a definitive diagnostic test increases the challenge of diagnosing cervical vertigo (1,84,87). Therefore, when diagnosis of cervical vertigo is suggested, the following relevant factors should be considered: a close temporal relationship between neck pain and symptoms of vertigo, previous neck injury or pathology, and elimination of other causes of vertigo(1).

Treatment

Management of cervical vertigo is challenging because the source of symptoms is difficult to identify(88).If vertigo originates from proprioceptive dysfunc- tion of the upper cervical spine, it was suggested that the management of this condition should be the same as for cervical pain (13). Humphreys and Peterson (89) compared adult neck pain patients with dizziness (n=177) to neck pain patients without dizziness (n =228) who presented for chiropractic treatment. After following up of 6 months, 80% of patients with dizziness and 78% of patients without dizziness reported clinically relevant improvement; in addition, there were no significant differences between patients with and without dizziness for any of the outcome measures.

Several authors have that proposed manual therapy is effective for vertigo of a cervical origin (5,17-19,88,90). We systematically reviewed the literature on manual therapy for the patients with cervicogenic dizziness, by evaluating RCTs published in English literature (see Table 2 for selected characteristics of these studies). In addition, in a systematic review of the literature, Reid and Rivett (5) concluded that there is limited evidence to support manual therapy for cervical vertigo. More- over, it was recommended that further research be conducted, especially RCTs, to provide more conclusive evidence of the role of manual therapy for cervicogenic dizziness. The systematic review published by Lystad et al (19) found that there is moderate (Level 2) evidence in a favorable direction to support the use of manual therapy for cervicogenic dizziness. The RCT carried out by Reid et al (17), which was deemed to be of good methodological quality, assessed the effectiveness of a specific type of spinal mobilization known as sustained natural apophyseal glides (SNAGs). They found signifi- cant improvement in dizziness severity and frequency, lower scores on the Dizziness Handicap Inventory (DHI), and decreased neck pain in the treatment group at both 6 and 12 weeks post-treatment. Thus, they concluded that SNAGs are a safe and effective manual therapy technique for the treatment of cervicogenic dizziness and pain. SNAGs were shown to have a clinically and statistically significant immediate and sustained effect in reducing dizziness, neck pain, and disability caused by cervical spine dysfunction (17). Recently, Reid et al (18) concluded that both SNAGs and Maitland mobilizations provide comparable immediate and sustained (12 weeks) reductions in intensity and frequency of chronic cervical vertigo. Manual therapy such as spinal manipulation may be effective in treating cervical vertigo by re- storing normal movement of the zygapophyseal joints, reducing pain and muscle hypertonicity, and thereby restoring normal proprioceptive and biomechanical functioning of the cervical spine (5,31).

Several authors encourage the implementation of vestibular rehabilitation in the treatment of dizziness of a cervical origin (1,91). Stability and posture of the cervical spine is achieved by a combination of reflexes mediated by vestibular, visual, and cervical sensory input, and the cerebellum plays an important role in integrating this sensory information (13,15,27). It can be hypothesized that a well-integrated vestibulo-cerebellar system would be more capable of compensating for the altered cervical sensory input in cases of cervicogenic dizziness. Thus, one can argue that when nor- mal cervical afferent input is compromised, vestibular rehabilitation may strengthen the vestibulo-cerebellar system to improve the ability to adapt to the situation(19). Published case studies have reported positive out- comes when combining manual therapy and vestibular rehabilitation (91). Lystad et al (19) thought that the evidence for combining manual therapy and vestibular rehabilitation in the management of cervical vertigo remains inconclusive. However, there is a reasonable rationale for utilizing manual therapy in conjunction with vestibular rehabilitation for cervical vertigo, and further research to elucidate the potential synergistic effects is strongly recommended. In addition, physical therapy is also an effective method for treatment of cervical vertigo (92).

Regarding the treatment of Barré-Liéou syn- drome, excising the lesions of the disc and PLL, and blocking abnormal sympathetic signal transduction may relieve symptoms of vertigo. Recently, Ren et al (93) reported 35 patients with cervical vertigo underwent percutaneous laser disc decompression (PLDD). After follow-up of 24 – 66 months, vertigo was improved; good to excellent clinical results were attained in 71.4% of these patients. They thought PLDD has many advantages, such as minimal trauma, high safety, and satisfactory mid-term efficacy with no significant difference in clinical efficacy between different age and gender groups. Moreover, PLDD technology can gasify the intervertebral disc using the heat energy of laser and form a hole, and then, with the intradiscal pressure decreased, extrusive inter-disc tissue can be partially retracted, thus releasing or re- ducing the compression or irritation on nerves. Mean- while, the heat treatment affection of the laser on the disc and its surrounding tissue can dilate blood vessels, reduce algogenic substance, and normalize the func- tion of the autonomic nervous system, all of which can release the inflammation in the disc-surrounding nerve tissue or increase the irritability threshold of the sympathetic nervous system thus improving the vertebralbasilar arterial blood supply (92). In a recent study by Li et al (94), 31 patients who were diagnosed as cervical spondylosis with sympathetic symptoms underwent anterior cervical fusion with PLL resection and were followed up for ≥ 5 years. At the final follow-up, good to excellent results were attained in 80.6% of these patients and the mean 20-point score decreased significantly from 7.3 ± 3.5 before surgery to 2.2 ± 2.7 ($P < 0.001$). They assumed that when these sympathetic nerves were excited, a sympathetic reflex was induced, which passed through the sympathetic reflex center located in the thoracic spinal cord and passing out into postganglionic sympathetic fibers innervated around the target organs including the vertebral artery, heart, and eyes etc., leading to sym- pathetic symptoms including vertigo, headache, blurry vision, and palpitation (94). Sympathetic nerve fibers distributed around the vertebral artery have been implicated in the autoregulation of vertebrobasilar artery (VBA) blood flow and CBF. It seems like sympa- thetic excitation roused by stimulation of periarterial neural structures of the vertebral artery can produce a decrease of blood flow of VBA and CBF. Stimulation of sympathetic nerves in the PLL may also produce sympathetic excitation, causing vertebrobasilar insuf- ficiency symptoms ie., vertigo (52). Hong and Kawagu- chi (51) also owe the improvement of cervical spondylosis patients with vertigo to resecting the cervical disc and PLL, and reducing the stimulation of sympathetic postganglionic fibers. Theoretically, the pathological factors induced by cervical spondylosis may affect the sympathetic nerves present in the PLL in the follow- ing 4 mechanisms. The first one is the direct irritation caused by mechanical compression resulting from the prolapsed degenerated intervertebral discs. The second one is that the IL-6, NO, PLA, and other inflammatory factors and chemical mediators induced by the prolapsed degenerated disc may irritate sympathetic nerves present in the PLL. The third is that cervical ver- tebral instability can irritate the PLL and stimulate the sympathetic nerves it contains. The fourth is that osteophytes on the posterior edge of the vertebral body may stimulate the PLL during neck movement (47,94).

As to RVAO, treatment options, introduced by acase report, range from lifestyle modification (avoid- ance of head turning), anticoagulation, endovascular, and stenting to surgical treatments (95). On the basis of their safety, effectiveness, and good long-term out- come, surgical treatments, including cervical decompression and/or cervical spine fusion, have been recommended as the first line treatment option of RVAO. However, it cannot be performed in patients with occlusion or hypoplasia of the unaffected vertebral artery(56). Furthermore, several authors have reported successful remission of symptoms with conservative treatment (96-98). Recently, a study carried out by Choi and colleagues (56) showed a favorable long-term outcome of conservative treatments in RVAO. Therefore, they thought that conservative treatments were safe and might be considered as a first-line treatment in RVAO.

II. Conclusion

Cervical vertigo is characterized by vertigo from the cervical spine. However, whether cervical vertigo is an independent entity still remains controversial. There are different hypotheses explaining vertigo of a cervical origin, including Barré-Lieou syndrome, proprioceptive cervical vertigo, RVAO, and migraine-associated cervicogenic vertigo. Each has a different pathophysiological mechanism, diagnostic characteristics, and optimal treatment. Diagnosis is dependent upon correlating symptoms of imbalance and vertigo with neck pain and excluding other vestibular disorders based on history, examination, and vestibular function tests. Treatment is challenging. Manual therapy is recommended for treatment of proprioceptive cervical vertigo. Anterior cervical surgery and percutaneous laser disc decompression are effective for the cervical spondylosis patients accompanied with Barré-Liéou syndrome. As to RVAO, a rare entity, when the exact area of the arterial com- pression is identified through appropriate tests such as MRA, CTA or DSA, decompressive surgery should be the chosen treatment. Via integration of the best available evidence, we wish to make the most appropriate clinical decisions for patients with cervical vertigo.

Conflict of Interest
All authors declare no conflict of interest. There was no external funding in the preparation of this manuscript.

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