

# Cervicogenic vertigo: a report of three cases\*

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*Cervicogenic vertigo is defined as a sensation of rotation, resulting from an alteration of the neck proprioceptive afferents of the upper cervical spine. As a consequence of their association with the vestibular nucleus, patients frequently experience a sensation of rotation or falling when they turn or flex and extend their head. Nystagmus may be present but no other neurological deficits are typically found. Restrictions in joint play are commonly palpated in the upper cervical spine motion segments, in particular C1-C3. Although spinal manipulation to the involved segments has provided relief, consideration of other etiological factors is essential. This paper discusses the etiology, diagnosis, and management of cervicogenic vertigo. Three cases illustrating the typical presentation and management are included.*  
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**KEY WORDS:** vertigo, cervical spine, mechanoreceptors, manipulation, chiropractic.

*Les vertiges d'origine cervicale sont définis comme étant une sensation de rotation, résultat d'un dérangement proprioceptif afférent dans la région des hautes cervicales. Ces propriocepteurs étant en contact continu avec le noyau vestibulaire, un mouvement de la tête créera chez les patients une sensation de chute ou de rotation. Le patient peut aussi souffrir de nystagmus, mais aucune autre manifestation neurologique n'est typique de ce désordre. Sur palpation, on retrouve souvent des restrictions articulaires dans la région des hautes cervicales, principalement les segments C1-C3. Même si des manipulations vertébrales des segments impliqués apportent un soulagement, il est important de considérer les autres sources étiologiques possibles. Cette étude couvrira l'origine, le diagnostic et l'approche thérapeutique face aux vertiges d'origine cervicale. Trois cas typiques, avec manifestations et traitement sont inclus.*  
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**MOTS-CLÉS :** vertige, colonne cervicale, mécanorécepteurs, manipulation, chiropratique.

## Introduction

Vertigo is defined as a sensation of rotation, characterized as subjective (patient perceives he/she is rotating) or objective (patient perceives that the object is rotating).<sup>1,2</sup> Although this definition appears simplistic, in reality it presents a diagnostic challenge to chiropractors. This challenge comes from the multifaceted etiology of vertigo that may involve several anatomical mechanisms. The structures subserving these mechanisms may be located centrally in the central nervous system or peripherally in distant connecting structures.<sup>3,4,5</sup>

The peripheral and central causes of vertigo are multiple, yet its etiology is often enigmatic. One peripheral cause of vertigo, namely cervicogenic vertigo, is associated with a disturbance of

the cervical somatic sensory nerve afferents. This latter etiology reflects a disturbance of the cervical spinal afferents resulting from a dysfunction of the zygapophyseal joints and the musculature of the upper cervical motion segments.<sup>6-10</sup>

Chiropractors frequently see patients who present with neck pain that may be associated with vertiginous symptoms. The purpose of this article is to familiarize the clinician with the etiology, clinical presentation, diagnosis and management of cervicogenic vertigo. Three case reports are presented to illustrate the typical features.

## Case reports

**Case one** – A 65-year-old retired man presented with a 20 year history of vertigo and lower cervical spine stiffness. The onset was insidious with no history of trauma. During the attacks of vertigo, he described a side to side movement of the room lasting about 10 seconds. The vertigo was triggered by extension and/or quick rotation of his neck and by trunk flexion.

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Aspirin had a relieving effect on the neck pain but not on the vertigo. The patient consulted his medical doctor for the vertigo and was prescribed Tranxene (a sedative), which gave him minimal relief. The patient was otherwise healthy.

On examination, the ranges of motion of the cervical spine were normal and pain free. Upon active and passive extension of the cervical spine, the patient felt unsteady and perceived a side to side movement of the room. On palpation, there was tenderness and segmental restriction of the suboccipital articulations on the left. Joint play challenge of the left C1-C2 articulation reproduced the vertigo. The Hallpike manoeuvre was performed, i.e. with the patient sitting, his head was rotated 45 degrees laterally and he was then briskly moved to the head hanging position, with the head about 30 degrees in hyperextension, over the edge of the table. In the classical response, vertigo appears after a latency of approximately 2 to 4 seconds.<sup>17</sup> This manoeuvre reproduced the vertigo but the intensity of the attack diminished when it was performed 5-6 times. Neck torsion testing produced slight vertigo when his head was kept immobile. With the patient in the supine position, neck extension with rotation reproduced the symptoms, but no nystagmus was observed. Neurological examination of the cranial nerves, cerebellum and peripheral nervous system was unremarkable.

X-ray examination of the cervical spine consisting of antero-posterior, lateral and flexion/extension views revealed mild spondylosis of the mid to lower cervical spine. They were otherwise unremarkable.

A diagnosis of benign paroxysmal positional vertigo associated with cervicogenic vertigo was made on the basis of the history and physical examination. The plan of management consisted of vestibular habituation exercises to correct the vestibular dysfunction, gentle mobilization directed to the suboccipital restriction on the left, and soft tissue therapy to the cervical musculature.

The patient reported a 100% improvement after eight treatments over a three week period. Follow-up at 18 months, revealed that he was still symptom free.

**Case two** – A 62-year-old male presented with a 10 year history of insidious neck pain with associated headaches and dizziness. The neck pain was described as a dull ache, localized to the right side of the neck extending from the suboccipital region to the medial border of the scapula. The pain was aggravated by movements, and relieved by rest.

The associated dizziness, which he was unable to attribute to a specific cause, was described as an unsteady, 'woozy' sensation of his body swaying rather than his surroundings. This sensation was aggravated by forward flexion and extension of the head, leaning forward with his head flexed, and if he applied digital pressure to the upper and mid cervical regions while lying supine. Although, there was no history of loss of consciousness, confusion, inco-ordination or seizures, he did report feeling nauseous but had not vomited. He reported no history of tinnitus nor deafness. He had been investigated at a local hospital,

including routine skull films, CT scans and caloric testing, which were found to be unremarkable. Aside from a chronic history of low back pain, he reported to be in good general health.

Examination revealed an alert and healthy male in no apparent distress. His blood pressure was 132/84 and pulse was 72 and regular. The ranges of motion of his cervical spine were full and painless except in right rotation and lateral flexion, which were reduced by pain by about 50% (compared to the left). Palpation revealed tenderness and restriction in the joint play at the level of C1-C2 on the right, with associated tenderness of the right suboccipital and cervical paraspinal muscles. The subjective vertigo was reproduced by applying pressure to the right C1-C2 level, as well as quickly rotating his head to either side. This was also noted with the neck-torsion test. The cranial nerves were intact. Further orthopaedic testing was unremarkable.

The patient was treated for a C1-C2 posterior joint dysfunction with associated cervicogenic vertigo. Treatment included the use of specific cervical rotational manipulation and soft tissue massage to the surrounding muscles. After the first treatment, the patient no longer complained of vertigo but the neck pain persisted for another week. At the completion of a regime of manipulation, the patient reported to be asymptomatic. He was given a series of exercises and followed.

To date, the patient has been followed for about six years. He continues to experience the occasional episode of vertigo about once a year, which is readily relieved by manipulation.

**Case three** – A 30-year-old female presented with a six month history of 'dizziness', neck pain and headaches. The neck pain and headaches began the day following a car accident; while the 'dizziness' appeared two weeks later. The headaches were constant and of a dull nature with bilateral retro-orbital radiations. The neck pain was severely aggravated by flexion-extension of the head. Associated symptoms included a mention that her eyes tired quickly and she would then have difficulty focusing.

On physical examination, the ranges of motion of the cervical spine were painfully limited in rotation bilaterally and flexion-extension. Assessment of vertigo was positive both for the vestibular and cervical areas. Cerebellar testing revealed co-ordination deficits both in the upper and lower limbs. Deep palpation of the upper cervical spine musculature and joints, especially at the level of C1-C2 and the inferior obliques, reproduced the vertigo. Opening the mouth and testing these areas again, increased the vertigo.

X-rays revealed an alordotic cervical spine. On the neutral lateral view the occiput appeared to be in a position of extension on C1, and C1 appeared to be in extension on C2. They were otherwise unremarkable.

The patient was referred to a local hospital's ENT department for further testing, which proved unremarkable.

The patient was treated for C0-C1-C2 dysfunction with associated cervicogenic vertigo. Treatment included the use of



spinal manipulative therapy and soft tissue therapy to the cervical and upper thoracic spine. The temporomandibular joint and the jaw musculature were treated with soft tissue therapy and mobilization. Ranges of motion exercises were recommended.

After one month of treatment, the patient no longer complained of headaches or vertigo, although the cervical spine ranges of motion were painful at the extremes. The patient was given specific strengthening and stretching exercises at this time. Follow up approximately three years later revealed that the patient noted episodic headaches and had one episode of vertigo that was successfully treated with manipulation.

## Discussion

### Etiology

The reflexes responsible for maintaining balance are regulated through the stimulation of the afferent nerve fibres of the semicircular canals, the proprioceptive input from the eye muscles and the afferents from the cervical spine. A disturbance in any one of these systems is believed to reinforce the symptomatology seen in the 'vertigo syndrome'.<sup>9</sup> For example, one possible mechanism consists of the facilitation of the neck proprioceptive afferents at the level of the vestibular nucleus causing a secondary dysfunction of the balancing reflexes.<sup>6-12</sup>

The implication that there is a relationship between the cervical nerve roots and vertigo was first described by Biemond in 1939.<sup>6</sup> Later, in 1961, after sectioning C2 and C3 posterior nerve roots in patients suffering from torticollis, he observed a strong postural nystagmus oriented to the side of the surgery.<sup>6</sup> Confirming this influence of the cervical spine, Philipszoon provoked nystagmus by neck torsion in patients with unexcitable labyrinths.<sup>7</sup> Norré postulated that if the neck proprioceptors are neurophysiologically equivalent to the vestibular organs, any disturbance of the cervical sensory input would cause dysfunction in the vestibular nucleus in a way similar to that described for vestibular vertigo.<sup>8</sup>

Recent theory has proposed that such dysfunction is a component rather than a distant entity in the etiology of this 'vertigo syndrome'.<sup>8</sup> The contribution of the upper cervical spine to this etiology, lies in the intricate neurological connection between the dorsal roots of the spinal nerves C1-C3 and their synapses with the vestibular nucleus.<sup>2,6-13</sup> The cervical zygapophyseal joints are the most densely innervated of all the spinal joints.<sup>14</sup> Hülse reports that 50% of the proprioceptors are found in the joint capsules of the cervical vertebrae C1-C3.<sup>15</sup>

The above neurological characteristic may help explain why traumatic, degenerative, inflammatory or mechanical derangements of the cervical spine can affect the mechanoreceptor system and give rise to vertigo.<sup>14,16</sup> Hülse, reviewing the files of those suffering from cervical disequilibrium, found that in one-third of the patients the onset was attributed to trauma, usually whiplash; in one-third the onset was insidious; and in the remaining third several causes were reported, including manual

therapy to the upper cervical spine.<sup>15</sup>

In addition to patients experiencing vertigo following trauma, they may also exhibit an associated positional nystagmus. Positional nystagmus is defined as nystagmus occurring in the immediate aftermath of movements of the whole body or between the head and trunk.<sup>6-8</sup> It is frequently a consequence of traumatic lesions to the cervical posterior nerve roots.

Neurophysiological studies in rabbits have shown a relationship between the effect of deafferentation of the cervical nerve roots<sup>6</sup> or the effect of section of the spinal cord<sup>8</sup> and the production of nystagmus. Studies in asymptomatic men comparing the balance reflexes elicited by the cervical proprioceptors to that obtained from labyrinthine stimulation, further adds support to this postulate.<sup>11</sup> However, others caution that the presence of nystagmus is often indicative of more serious neurological or vascular conditions and careful assessment is imperative.<sup>2,15,17</sup>

Although the supportive evidence is mostly empirical, the pathogenesis of cervicogenic vertigo can be seen as a summation of stimulations from cervical spine and labyrinth. Barnes has concluded from his study in man, that "the cervical afferent information serves to modulate the activity of second order neurons, facilitating the action of the vestibulo-ocular reflex".<sup>11</sup>

### Diagnosis

The most constant symptom in cervicogenic vertigo is the report of the sensation of rotating or falling elicited by the rotation or the flexion and extension of the neck. Neck pain and suboccipital headaches are often present. Patients often report a history of trauma.<sup>15</sup>

Physical examination is helpful in confirming the diagnosis. The neck torsion test is first performed without stabilization of the head, thereby stimulating the labyrinthine afferents. (see Figure 1) It is then performed by sitting a patient on a turning stool, immobilizing the head whilst rotating the body. (see Figure 2). Reproduction of the vertigo and nystagmus when the head is immobilized suggests a cervicogenic origin for the symptoms.<sup>7,8,9,18</sup>

The associated positional nystagmus which may be present, is found to beat in the horizontal plane.<sup>8</sup> Comparing the nystagmus present in normals to that present in subjects with cervicogenic vertigo, Norré found no difference in its frequency but did note that it appeared more intense in the vertigo group.<sup>9</sup>

Hülse is more cautious, however, and warns the clinician to exercise care in examining patients with nystagmus. He reports that nystagmus of cervicogenic origin often appears immediately during head movements in rotation and the intensity decreases as a result of adaptive responses. In contrast, nystagmus resulting from vertebrobasilar insufficiency appears at the extreme of head rotation after a latency of about 50 seconds and the intensity is found to increase.<sup>15</sup>

Tenderness to deep palpation is usually noted in the suboccipital region and may reproduce the vertigo as seen in the three cases presented. Motion palpation of the cervical spine fre-





Figure 1

Patient rotating head from side to side



Figure 2

Patient rotates body while doctor holds head steady.

(Reprinted by permission. Fitz-Ritson D. Assessing cervicogenic vertigo. JMPT)

quently reveals restriction in intersegmental joint play in the upper cervical spine. Norré found a statistically significant difference in the frequency of appearance of upper cervical 'blockage' in subjects with neck-torsion nystagmus compared to those without nystagmus.<sup>9</sup>

Caloric testing of the inner ear is often performed to rule out labyrinthine involvement. However, in cervicogenic vertigo this test has a normal response.

As a rule, neurological deficits are not observed.<sup>15</sup>

Radiographic examination of the cervical spine fails to reveal any characteristic features. Degenerative joint disease may be present if the patient is older or it can be secondary to previous trauma to the cervical spine. These changes have not yet been shown to play a predictive role in the diagnosis of cervicogenic vertigo.

### Differential diagnosis

The difficulty in attributing the vertiginous symptoms to the cervical spine reflects the potential of the interaction of more than one physiological system. The clinician must first categorize the etiology of vertigo. One method is to divide the etiologies into three basic categories: peripheral, central and systemic.<sup>3,4,5</sup> (See Table 1)

The peripheral causes of vertigo include the dysfunction of the organs of the inner ear; namely the semicircular canals, the utricle and the saccule. It is here that cervicogenic vertigo is listed due to the contribution of the mechanoreceptors. The central causes of vertigo, on the other hand, deal with the spinal cord, brain and its vascular supply.

The systemic causes of vertigo are often the result of a side effect of a drug or the consequence of an underlying systemic pathology. Thus, an awareness of the secondary complaints or the taking of drugs should be considered when eliciting a patient's history.

Although categorization of the above etiologies is presented, their differentiation may not be so readily accomplished. For the chiropractor, this is especially true when differentiating between cervicogenic vertigo, benign paroxysmal positional vertigo (BPPV) and vertebrobasilar insufficiency (VBI). Distinguishing between these three etiologies may be puzzling because of their association with cervical structures and their similar presentation.

BPPV is a benign inner ear disorder, associated with characteristic torsional positional nystagmus induced by a rapid position change from sitting to the head-hanging right or left position (Hallpike manoeuvre).<sup>10</sup> Bourgeois et al. reported that



TABLE 1  
CATEGORIZATION OF THE ETIOLOGIES  
OF VERTIGO<sup>3,4,5</sup>

<b>Peripheral causes</b>	Peripheral vestibulopathy/labyrinthitis Benign paroxysmal positional vertigo (BPPV) Post-traumatic vertigo Menieres syndrome Bacterial infection Cervicogenic vertigo
<b>Central causes</b>	Demyelinating disease Acoustic tumours Seizures Cerebral haemorrhage Vertebro basilar artery dysfunction
<b>Systemic causes</b>	Drugs (anticonvulsants, hypnotics, antihypertensives, alcohol, analgesics, tranquilizers) Vestibulotoxic drugs (Streptomycin, Gentamycin, Aminoglycosides) Hypotension Endocrine disease (diabetes and hypothyroidism)

BPPV is also induced by lying down and getting up, turning to one side in the supine position and by looking upward or bending down.<sup>20</sup> The above provocative mechanisms all involve cervical spine motion, thereby possibly misleading the clinician.

Similarly, confusion may be seen in vertigo arising from VBI. The blood supply to the vestibular nucleus is derived from the basilar and vertebral arteries. If the blood flow to the vestibular nucleus is reduced, the vestibular function will be impaired and vertigo will result.<sup>17,21</sup> Since the blood supply from the basilar and vertebral arteries is more extensive than just to the vestibular nucleus, vertigo is rarely an isolated symptom in vertebrobasilar insufficiency syndrome. Other symptoms of VBI include disturbances in vision, diplopia, nausea, ataxia, impairment of trigeminal sensation, dysarthria, hemianesthesia and hemiplegia.<sup>17,21</sup>

The diagnosis of cervicogenic vertigo is, therefore, deduced from a history implicating a mechanical disorder of the neck associated with vertigo. The clinical provocative manoeuvres, namely the neck torsion test, palpation and joint play challenge of the cervical spine motion segments, help differentiate the underlying etiology.

However, due to the potential complication from a mis-

diagnosis, if any doubt exists the patient should be referred for further testing and imaging.

### Management

The management of vertigo depends of its etiology. Although this is based on empirical data, conservative therapy is often effective in the treatment of cervicogenic vertigo.

It is appreciated that the natural course of the disease has not been established, however, the three cases reported herein, responded well to conservative therapy. Short amplitude, high velocity spinal manipulative therapy to the levels of joint fixation, frequently found in the cervical segments C1-C3, has been reported to be beneficial.<sup>2,15,22</sup> Henderson and Cassidy caution that the patient be carefully assessed if manipulation is to be attempted.<sup>17</sup>

In addition to manipulation, soft tissue therapy should also be directed to the suboccipital musculature, which is frequently hypertonic.<sup>2,15</sup> Stretching and strengthening exercises may prove to be effective in the short- and long-term care of these patients. A carefully planned program that involves exercises progressing from flexibility to strengthening to endurance, emphasizing the suboccipital muscles, should be given.<sup>22</sup>

When cervicogenic vertigo is associated with benign positional vertigo, vestibular habituation training should be part of the plan of management.<sup>20,23</sup> With this technique, the patient sits down on the bedside with the eyes closed, moves in the position that elicits the vertigo for 30 seconds (e.g. forward flexion or lateral flexion) and then returns to the upright position for 30 seconds; this is repeated five times. These exercises are repeated five to 10 times daily. The therapy is usually successful after three weeks.<sup>20</sup>

### Conclusion

In conclusion, this paper reported three cases of cervicogenic vertigo and their conservative management. Studies have postulated that altered cervical spine mechanoreceptor activity may contribute to the aberrant modulation of the vestibular nucleus, resulting in the subjective sensation of 'vertigo'.

Empirically, spinal manipulative therapy (adjustments) directed to areas of restricted intersegmental joint play, commonly in the upper cervical spine, may be successful. However, an awareness of other more sinister etiological conditions is imperative and if suspected, then appropriate investigations should be pursued prior to recommending conservative care.

### References

- 1 Fitz-Ritson D. Assessment of cervicogenic vertigo. Accepted for publication. JMPT.
- 2 Brunarski D. Autonomic nervous system disturbances of cervical origin including disorders of equilibrium. In: Vernon H, ed. Upper Cervical Syndrome. Baltimore: Williams and Wilkins, 1988: 189-193.
- 3 Slater R. Vertigo, how serious are recurrent and single attacks? Postgrad Med 1988; 84(5):58-67.



- 4 Troost BT. Dizziness and vertigo in vertebrobasilar disease: part I. *Stroke* 1980; 11(4): 301-303.
- 5 Troost BT. Dizziness and vertigo in vertebrobasilar disease: part II. *Stroke* 1980; 11(4): 413-415.
- 6 Biemond A, De Jong JMBV. On cervical nystagmus and related disorders. *Brain* 1969; 92:437-458.
- 7 Philipszoon AJ. Vertigo d'origine cervicale. *Acta Oto-Rhino-Laryngologica Belgica* 1970; Tome 24: Fasc. 3.
- 8 Norré ME, Stevens A. Cervical vertigo. *Acta Oto-Rhino-Laryngologica Belgica* 1987; Tome 41: Fasc. 3.
- 9 Norré M, Stevens A. Le nystagmus cervical et les troubles fonctionnels de la colonne cervicale. *Acta Oto-Rhino-Laryngologica Belgica* 1976; Tome 30: Fasc. 5.
- 10 Baloh RW. Dizziness, hearing loss and tinnitus: the essentials of neurotology. Philadelphia: FA Davis Company, 1984: 152.
- 11 Barnes GR, Forbat LN. Cervical and afferent control of oculomotor response in man. *Acta Otolaryngol* 1979; 88:79-87.
- 12 Rubin AM, Young JH, Milne DH, Schwartz DW, Fredrickson JM. Vestibular-neck integration in the vestibular nuclei. *Brain Research* 1975; 96:99-102.
- 13 Fitz-Ritson D. The direct connections of the C2 dorsal root ganglia in the Macaca Iru monkey: relevance to the chiropractic profession. *J Manipulative Physiol Ther* 1985; 8(3):147-156.
- 14 Wyke B. Neurology of the cervical joints. *Physiotherapy* 1979; 65(3):72-76.
- 15 Hülse M. Disequilibrium caused by a functional disturbance of the upper cervical spine, clinical aspects and differential diagnosis. *Manual Medicine* 1983; 1(1):18-23.
- 16 Wyke B. Articular neurology and manipulative therapy. In: *Aspects of manipulative therapy. Proceedings of a multi-disciplinary International Conference on Manipulative Therapy*. Melbourne: Lincoln Institute of Health Sciences; 1979: 67-71.
- 17 Henderson DJ, Cassidy JD. Vertebrobasilar vascular accidents associated with cervical manipulation. In: Vernon H, ed., *Upper cervical syndrome*. Baltimore: Williams and Wilkins, 1988: 194-206.
- 18 Jongkees LBW. Cervical vertigo. *Laryngoscope* 1969; 79:1473-1484.
- 19 Baloh WB, Honrubia V, Jacobson K. Benign positional vertigo: clinical and oculographic features in 240 cases. *Neurology* 1987; 37:371-377.
- 20 Bourgeois PM, Dehaene I. Benign paroxysmal positional vertigo. *Acta Neurol Belg* 1988; 88:65-74.
- 21 Bogduk N. Cervical causes of headache and dizziness. In: Grieve GP, ed, *Modern manual therapy of the vertebral column*. New York: Churchill Livingstone, 1986: 289-302.
- 22 Fitz-Ritson D. The chiropractic management and rehabilitation of cervical trauma. *J Manipulative Physiol Ther* 1990; 13(1):17-25.
- 23 Norré ME, Beckers AM. Vestibular habituation training. *Arch Otolaryngol Head Neck Surg* 1988; 114:883-886.