

NOSOLOGICAL ENTITIES?

Cervical vertigo

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Proprioceptive input from the neck participates in the coordination of eye, head, and body posture as well as spatial orientation. On this basis it has been argued that a syndrome of cervical vertigo might exist. However, cervical vertigo is a controversial clinical entity and patients with suspected disease often have alternative bases for their symptoms.¹

The neck contains mechanisms directly involved in balance control (neck afferents), cardiovascular control (carotid bodies), and purely vascular structures (carotid and vertebral arteries). Neck movements are also invariably associated with head movements. Thus, experiencing unsteadiness or vertigo associated with neck movements could be due to a disorder in vestibular, visual, vascular, neurovascular, or cervicoproprioceptive mechanisms. Table 1 summarises the possible differential diagnoses.

Without further specification, however, the term cervical vertigo is reserved for cases where the suspected mechanism is proprioceptive. The reasoning is as follows. 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. Degenerative or traumatic changes of the spine could induce distorted sensations of head motion (vertigo). This line of thought is, however, not as straightforward as it seems. Clearly, vestibular lesions or experimental vestibular activation (for exam-

ple, a caloric test) produce powerful illusions of self motion. Similarly, visually induced illusion of self motion is readily experienced—for example, the feeling that the train we are in has started to move off when, in fact, it is the train next to ours which has done so.

Evoking a clear sensation of head turning by cervical stimulation is, by contrast, not so straightforward. To start with, to investigate neck proprioception, the head must be immobilised while the trunk is rotated. Unless this technical precaution is taken, any normal or abnormal sensations of head turning can always be due to vestibular stimulation. With this approach, under special conditions of perceptual uncertainty (for example, both the subject's head and trunk can be independently rotated in the dark), trunk rotation can induce sensations of head turning.² However, when the subject's head is unambiguously fixed, or in normal viewing conditions, trunk rotation does not induce sensations of head turning. This is so, even in certain pathological conditions when trunk rotation is capable of inducing strong nystagmus—for example, bilateral absence of vestibular function,³ or occasionally, in cerebellar lesions.⁴

Questions relevant for the discussion of cervical vertigo are: What is the functional relevance of neck afferent input and how does the lack of or distortion of such input lead to vertigo or disequilibrium? Ataxia and unsteadiness occurring with sensory polyneuropathy

Table 1 Differential diagnosis of cervical vertigo: vertigo, unsteadiness, or oscillopsia triggered/aggravated by head-neck movements

Disorder	Assumed mechanism
Labyrinthine:	
Benign paroxysmal positional vertigo	Canalolithiasis, cupulolithiasis
Post-traumatic otolith vertigo	Dislodged otoconia, causing unequal heavy load on macula
Perilymph fistula	Floating labyrinth
Vestibular nerve:	
Unilateral vestibular failure (eg, vestibular neuritis)	Cross coupling effects with acute vestibular tone imbalance
Bilateral vestibular failure	Defective vestibulo-ocular reflex
Vestibular paroxysmia	Neurovascular cross compression
Nerve compression by cerebellopontine angle mass	Conduction block or ectopic discharges
Ocular motor:	
Extraocular eye muscle or gaze paresis	Inappropriate vestibulo-ocular reflex
Central vestibular:	
Central positional nystagmus/vertigo	Cerebellar disinhibition
Migraine without aura	Motion sickness due to sensory hyperexcitability
Migraine with aura (basilar migraine, vestibular migraine)	Spreading depression involving vestibular structures
Vestibulocerebellar ataxia	Vestibulocerebellar dysfunction
Vascular:	
Rotational vertebral artery occlusion	Ischaemic depolarisation
Carotid sinus syndrome	Global cerebral ischaemia
Intoxication:	
Positional alcohol nystagmus/vertigo	Cerebellar and specific gravity differential between cupula and endolymph (buoyancy mechanism)
	Cerebellar and ocular motor
Drugs (eg, antiepileptics)	

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are readily recognised and explained by a deficient sense of position of the lower limb joint.⁵ Dizziness and unsteadiness suspected to be of cervical origin could be due either to loss or inadequate stimulation of neck receptors in cervical pain syndromes. Thus far this has never been shown.

Functional relevance of neck afferents

Proprioception is mostly dependent on the deep short intervertebral neck muscles, which are extensively supplied with muscle spindles.^{6,7} The neck input participates in perceptual functions and reflex responses—namely, cervicopostural and cervico-ocular reflexes.

PERCEPTUAL FUNCTIONS

The perception of head or trunk rotation in space would be erroneous if only vestibular stimulation or only neck stimulation were involved. When vestibular and cervical stimuli are combined (head rotation relative to the trunk), the perception of both trunk and head rotation in space reflects the true position.^{2,8}

Vestibular and visual cues produce postural corrections but these responses change direction with changes in head position.⁹ When the head is rotated horizontally by 90° to the right or left—for example, horizontal head movements and horizontal retinal slip of the visual scene (right-left in head coordinates) no longer indicate lateral body sway; instead, they represent fore and aft movements.¹⁰ Consequently, the compensatory postural adjustments must be corrected and this function is also mediated by neck afferences.¹¹

The perceived “straight ahead” and the “subjective visual vertical” can also be modified by cervical stimulation. Unilateral electrical stimulation of the neck¹² causes deviation of the subjective vertical. Vibration of neck muscles, which stimulates the primary endings of the muscle spindles as if the muscle were being stretched¹³ elicits an illusion of head tilt and apparent movement of a visual target.¹⁴ Accordingly, subjective “straight ahead” shifts toward the side of the vibrated dorsal neck muscle.¹⁵ It has now been clarified that changes in the subjective straight ahead¹⁵ and the illusory motion of a target light during neck vibration¹⁶ are due to minute slow phase eye movements. This vibration induced cervico-ocular reflex is, in agreement with head/trunk rotation studies,³ significantly enhanced in patients with bilateral absence of vestibular function.¹⁶ In unilateral vestibular lesions, the increase in muscle spindle input (as tested by vibration) is asymmetric, restricted to the affected side, and gradually builds up over weeks.¹⁵ Of note, however, both visual illusions and postural responses are comparatively stronger than any illusion of head movement. It would seem that neck input is important for the generation of automatic reflexes but less important for generating conscious perceptions of head turning.

REFLEX RESPONSES

Two reflexes are mediated by neck proprioceptors: the postural neck reflexes and the cervico-

ocular reflex. Tonic neck reflexes, studied by Magnus,¹⁷ innervate limb muscles asymmetrically. In humans, tonic postural neck reflexes can be elicited only in the newborn^{18,19}—for example, ipsilateral flexion and contralateral extension of the limbs with head rotation (“fencing posture”). Neck input not only modulates body posture, but also stabilises the head with respect to the trunk by cervicocollic reflexes, interacting with vestibulocollic reflexes which stabilise the head in space.^{20,21} In healthy human beings, neck reflexes form a part of the multisensory intersegmental postural control mechanism thus making it virtually impossible for the clinician to carry out a selective test of neck function by simple postural manoeuvres.

It was Bárány²² who first demonstrated tonic cervico-ocular reactions in rabbits, elicited by motion of the trunk relative to the head. In humans, this tonic neck-eye reflex can only be seen in the newborn¹⁸ or in rare patients with gross CNS lesions.²³ Bikeles and Rutin²⁴ were the first to report nystagmus during head rotation in patients with complete vestibular loss, which they ascribed to sensory input from neck joints. More recent studies of the cervico-ocular reflex, elicited by rotating the trunk about the stationary head, have shown that this reflex is weak in normal subjects.³ A low velocity nystagmus, however, can be elicited in some normal subjects.^{3,25,26} This reflex is adaptively enhanced in acquired vestibular loss, thereby partially substituting the vestibulo-ocular reflex deficit in the monkey²⁷ and in humans.^{3,28-30} Thus, attempts to define cervical vertigo on the basis of cervical nystagmus³¹ are impractical: cervical nystagmus occurs in healthy subjects and can be particularly strong in patients with no cervical vertigo.^{3,4}

Experimental cervical vertigo

In animals, transverse section of suboccipital muscles, surgical deafferentation of C1-C3, or suboccipital anaesthesia results in locomotor ataxia.³²⁻³⁵ Local anaesthesia of deep posterolateral neck tissue in humans^{35,36} usually elicits a transiently increased ipsilateral and decreased contralateral extensor muscle tone with a tendency to fall, gait deviation, and past-pointing towards the injected side. Dieterich *et al.*³⁷ confirmed this in patients with cervicogenic headache investigated before and after bilateral therapeutic anaesthetic C2 blockades; however, they found no specific abnormality with static posturography, or subjective visual vertical or routine electronystagmography. The weak horizontal spontaneous nystagmus, directed away from the injected side as seen by Barré,³⁶ is not a typical feature in humans.^{35,37} Biémond³⁸ reported positional nystagmus due to upper cervical root section in the rabbit but Cohen³⁹ showed that this positional nystagmus is species specific: most pronounced in rabbits, less in the cat, and subtle in the rhesus monkey. Positional nystagmus cannot be attributed to a disturbance of the cervical sympathetic chain³⁶; animal experiments^{35,40} indicate that it most probably represents a tone imbalance of upper cervical roots.

Clinical evidence for cervical vertigo?

Because section or anaesthesia of cervical roots or muscles causes an asymmetry in somatosensory input, unilateral irritation or deficit of neck afferents could create a cervical tone imbalance, thus disturbing integration of vestibular and neck inputs. However, it has not been shown that whiplash injuries or cervical pain syndromes produce such a tone imbalance with ataxia and vertigo. Rotational vertigo and nystagmus associated with pain arising from the cervical spine with tenderness and limitation of neck movement should not be called cervical vertigo; indeed, when post-traumatic, vertebral artery dissection should be ruled out.⁴¹

Symptoms of cervical vertigo, if it exists, would be a sensation of lightheadedness or floating unsteadiness and slight ataxia of stance and gait, perhaps more on head turns. This can be inferred from the experimental unsteadiness induced in humans by unilateral suboccipital local anaesthesia.³⁵⁻³⁷ As somatosensory cervical input converges with vestibular input to mediate multisensory control of orientation, gaze in space, and posture, the clinical syndrome of cervical vertigo could theoretically include perceptual symptoms of disorientation, postural imbalance, and ocular motor signs although the last looks particularly unlikely. Consequently, further clinical studies seeking to define cervical vertigo should focus on establishing reliable measures for it. These could include psychophysics, oculography, posturography, and measurements of cervicospinal reflexes but, as assessment under static conditions has so far proved inconclusive, further investigations should focus on dynamic somatosensory studies. 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. Complaints of vertigo or unsteadiness on turning the head are much more likely to imply vestibular rather than cervical dysfunction.

Some of the more conscientious studies have been based on posturography data.⁴²⁻⁴⁵ Patients with chronic cervicobrachial pain (not selected for complaints of vertigo) had poorer postural control, based on vibration induced and galvanically induced body sway, than normal controls.⁴² It was also noted that physiotherapy was of value in reducing neck pain as well as dizziness and postural balance.⁴⁴ The findings, however, are not specific enough to establish a diagnosis of cervical vertigo.

Hypothetical mechanisms

Firstly, it is not known how traumatic, degenerative, inflammatory, or rheumatic diseases affect neck sensory input. In such uncharted regions, various hypotheses thrive—for example, the hypothesis of cervical vertigo after whiplash injury. Suggestions have included neuromuscular⁴⁶ and neurovascular mechanisms⁴⁷ and mechanical obstruction of the vertebral artery.⁴⁸ Longet³² very early made the incidental finding that post-traumatic vertigo and ataxia improve with the use of a neck

collar.⁴⁹ But head trauma and whiplash injury affect not only neck structures. Whiplash injuries often damage the brain,⁵⁰⁻⁵¹ making the interpretation of abnormal vestibulo-ocular tests difficult.⁵²⁻⁵³ The otoliths are highly vulnerable to accelerations; damage to them may cause otolith vertigo,⁵⁴ characterised by a benign course similar to that of neck pain. In addition, dislodged otoconia often also enter the lumen of the semicircular canals, resulting in canal lithiasis and post-traumatic benign paroxysmal positional vertigo (BPPV).⁵⁵ It must be borne in mind that canal lithiasis can involve not only the posterior canal (“classic” BPPV) but also the horizontal and anterior canals, thus explaining atypical presentations which would have been previously confused with cervical vertigo or nystagmus.

A convincing mechanism of cervical vertigo would have to be based on altered upper cervical somatosensory input associated with neck tenderness and limitation of movement. Interstitial inflammatory mediators have been postulated to sensitise muscle spindles,⁵⁶ and myofascial trigger points exhibit spontaneous EMG activity, which is compatible with hyperactive muscle spindles.⁵⁷ 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. Particularly, this would occur during active head movements, when expected and actual reafferent input would not match.

Differential diagnosis

Differential diagnosis of vertigo associated with cervical symptoms or head-neck movement avoidance is broad (table 1). If it is post-traumatic or follows cervical whiplash injuries, then post-traumatic otolith vertigo,⁵⁴ or benign paroxysmal positioning vertigo,⁵⁸ central vestibular dysfunction secondary to brainstem concussion, vertebral artery dissection, and perilymph fistulas should be considered.⁵⁵ In non-traumatic cases, psychogenic vertigo⁵⁹⁻⁶⁰ can have similar symptomatology but cerebellar or spinal ataxia, vestibular paroxysmia,⁶¹ and bilateral vestibulopathy⁶²⁻⁶³ should be considered first, before psychogenic or cervical origin is assumed. Not uncommonly, cervical pain is actually secondary to a genuine vestibular disorder, such as vestibular neuritis, and probably develops as patients stiffen up their neck muscles to avoid head movements. This feature is well recognised by physiotherapists working with vestibular patients.⁶⁴ Lesions of the extracranial portion of the vertebral arteries, as it travels the cervical column within the transverse foramina (portion V2) and loops from C2 into the foramen magnum (portion V3), have to be considered in the differential diagnosis. As this part of the vertebral artery is relatively free from atherosclerosis, occlusions due to neck movements/positions, neck manipulations, osteophyte compression, trauma, and “spontaneous” dissection can occur.⁶⁵ More widespread posterior circulation features should be the norm but some cases with fairly selective vestibular symptoms have been well

documented.⁶⁶ By contrast, there seems to be no scientific basis for the syndrome of cervical-sympathetic irritation.³⁶ It is likely that most cases of this syndrome are pure vestibular disease, vertebral artery compression, vestibular paroxysmia, or basilar migraine.

In summary, vertigo can be accompanied by cervical pain, associated with head injury, whiplash injury, or cervical spine disease. In some cases it can improve dramatically with physiotherapy. None of these instances provides convincing evidence of a cervical mechanism, and alternative explanations are almost always possible. All clinical studies on cervical vertigo to date have three weak points: (1) the inability to confirm the diagnosis, (2) the lack of a specific laboratory test, and (3) the unexplained discrepancy between patients with severe neck pain without vertigo and patients complaining of disabling vertigo with moderate neck pain. If cervical vertigo exists, appropriate management is the same as that for the cervical pain syndrome, and this management should not be denied any patient. As such therapy is carried out in any case, the debate on the relevance and mechanism of cervical vertigo is more of theoretical interest than of practical relevance.

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