Vertebrobasilar insufficiency: an insufficient term in need of retirement

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The concept of vertebrobasilar insufficiency (VBI) was introduced by Silversides in 1954. Milliken and Siekert coined the term in 1955 when describing eight patients with symptoms suggesting episodic “insufficiency” of blood flow in the vertebrobasilar arterial territory supplying the brainstem with—in four cases—basilar artery thrombosis at necropsy. This followed Denny-Brown’s descriptions of ‘cerebrovascular insufficiency’—referring to focal cerebral ischaemia in any of the cerebral arterial territories—and Miller Fisher’s use of ‘carotid insufficiency’ to describe transient ischemic attacks (TIA) in the carotid artery territory.

VBI remains a controversial clinical entity lacking clear diagnostic criteria. The term is used to describe transient neurological deficits in the vertebrobasilar territory and thus denotes hypoperfusion of the cerebral tissue within the posterior circulation. VBI therefore is no different from what is more commonly described as posterior circulation TIA—usually caused by athero-thromboembolism related to vertebrobasilar stenosis (Figure 1A), cardiac embolism, or small vessel occlusion—and we therefore argue that the term VBI is therefore redundant.

So why do some clinicians continue to use the term? Some use it as a shorthand for presumed vertebrobasilar ischaemia relating to direct vertebral artery compression induced by movement of the head (usually lateral rotation). Indeed, the vertebral

arteries are particularly vulnerable to mechanical stress between the atlas and axis (Figure 1B). Occlusion at this level during neck and head rotation is termed Bow hunter's syndrome—named after the archery a patient was practising when developing lateral medullary (Wallenberg) syndrome (Figure 1C shows the typical infarct). The incidence of postural- or neck-movement related neurological symptoms in patients with TIA of the posterior circulation is unknown but is likely to be very low. CT or MR angiography are the imaging modalities of choice in people with suspected vertebrobasilar stenosis. Dynamic angiography has confirmed occlusion of the vertebral artery in a symptomatic patient during 90° head rotation, but is not a routine investigation. In our experience, patients with a single occluded or significantly stenotic vertebral artery do not develop neurological events on head turning, presumably because head rotation is usually insufficient to cause vascular compression, or due to adequate collateral arterial supply. Thus, neck-movement related vertigo is only exceptionally due to direct vascular compression, and nearly always attributable to another mechanism (e.g. vestibular, visual, or cervico-proprioceptive). Patients with vertigo triggered by head (and neck!) movement are most likely to have benign paroxysmal positional vertigo (BPPV), this being the most common cause of head-movement related vertigo worldwide [1]. Vestibular migraine may also present with head-movement related dizziness or vertigo [2] and clinicians should seek other migrainous features when assessing these patients. Rarer causes include Menière's syndrome, with hearing loss and tinnitus, and vestibular paroxysmia, with very brief and recurrent attacks of vertigo triggered by head movement, often also with 'typewriter' tinnitus.

Nevertheless, patients with symptomatic vertebrobasilar ischaemic events have 22% risk of stroke within 90 days of presentation (similar to anterior circulation TIA) [3], highlighting the need for their prompt identification and treatment. Symptoms attributable to vertebrobasilar ischaemia include dizziness and syncope (47%), unilateral limb weakness (41%), dysarthria (31%), headache (28%), and nausea or vomiting (27%) [4]. However, patients with posterior circulation ischaemia can present with transient neurological attacks that may be non-focal (e.g. transient generalised weakness, "dizziness" or "wooziness", or even syncope), leading to delayed diagnosis of brainstem ischaemia. Usually there are multiple symptoms due

to the close proximity of relevant brainstem structures, but diplopia, dysarthria or vertigo can be the sole presenting complaint. The differential diagnosis of acute *isolated* vertigo includes vestibular neuritis, vestibular migraine, and BPPV. Thus, while patients with cardiovascular risk factors and neurological symptoms that are potentially due to posterior circulation ischaemia—particularly vertigo, visual disturbance, or syncope—may have had a posterior circulation TIA, clinicians should first consider commoner causes.

In contrast with the anterior circulation, there is as yet no established clear interventional strategy for symptomatic large artery stenosis of the posterior circulation. Treatment may include endovascular approaches, but the current standard of care is medical therapy (short-term dual anti-platelet and long-term single agent), high intensity statins and careful blood pressure control. The two randomised studies comparing medical therapy with vertebrobasilar revascularisation were not sufficiently powered to demonstrate statistically significant differences in efficacy [5, 6]. Nevertheless, patients with persistent symptoms despite maximal medical therapy with dual antiplatelet therapy should be considered for endovascular revascularisation, particularly if there is >70% bilateral vertebral artery stenosis.

Whilst VBI describes neurological events secondary to stenosis of the vertebrobasilar system, it is an unnecessary term for posterior circulation TIA and should no longer be used. In our experience, brainstem ischaemia due to vertebral artery compression induced by neck movement—often called VBI—is exceptionally rare (though still awaits systematic study). The message for clinical practice is clear: neurological symptoms related to head (and neck) movement (e.g. isolated vertigo) are far more likely to have a cause other than VBI.

Key points:

1. Vertebrobasilar insufficiency is more appropriately termed posterior circulation transient ischaemic attack (TIA).
2. Posterior circulation TIA typically present with transient neurological symptoms that may not be focal, such as vertigo, diplopia, or unsteadiness.

3. Isolated vertigo on head movement/rotation is usually caused by a peripheral vestibular disorder (e.g. BPPV) rather than vascular compromise to the neck vessels.

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Competing interests

AC has nothing to declare

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Figure 1 A. Time of flight MR angiography of the posterior circulation showing right vertebral artery narrowing (white arrow) secondary to atherosclerosis; B. Anatomy of the vertebral system in relation to the vertebral column at the cervical level, highlighting the tortuous course through the atlanto-axial structures (filled circle), where compression of the vessel may occur; C. Diffusion-weighted image (DWI) sequence of an MR scan of brain in a patient with vertebral artery stenosis leading to a lateral medullary infarct (white arrow).