Haemodynamic transient ischaemic attack

Sir,

To the comprehensive review of transient ischaemic attack and its underlying causes (vol 72(7), 2011, p. 372) I would like to add a brief mention of so-called 'haemodynamic transient ischaemic attack', a subtype of transient focal neurological deficit caused by hypoperfusion resulting from a drop in blood pressure which is either systemic or regional (Klijn and Kappelle, 2010).

According to the authors of one review 'The feature that distinguishes haemodynamic transient ischaemic attack… from other stroke subtypes is that blood flow towards part of the brain is too low and results in ischaemia in an area of the brain or retina that is at a distance from the blocked vessel' (Klijn and Kappelle, 2010). When systemic hypotension is aetiologically implicated in haemodynamic transient ischaemic attack, this can be triggered by factors such as rising from sitting or supine posture, exercise, transition from a cold to a warm environment, consuming a meal, coughing, and antihypertensive medication (or its uptitration) (Klijn and Kappelle, 2010). In one example the precipitating cause was a leak from an abdominal aortic aneurysm (Bollu et al, 2009).

A feature of haemodynamic transient ischaemic attack which should raise suspicion for this diagnosis is a brief period (less than 5 minutes) of unilateral limb shaking after rising or after exercise, especially if this is followed by transient paresis of the involved limb (Klijn et al, 2000; Klijn and Kappelle, 2010). Postprandial stroke is another example of haemodynamic transient ischaemic attack (Kamata et al, 1994), attributable to postprandial hypotension.

Where the focal neurological episode is attributable to a regional drop in blood pressure the mechanism is 'increased metabolic demand… that cannot be met because of already marginal perfusion' (Klijn and Kappelle, 2010), as exemplified by retinal claudication (Klijn and Kappelle, 2010). Retinal claudication is characterized by transient monocular blindness after exposure to bright light, and is highly suggestive of carotid artery occlusive disease (Klijn et al, 2000).

Minimum diagnostic studies for patients with suspected haemodynamic stroke include evaluation of postural changes in blood pressure, brain imaging for infarcts between the deep and superficial arterial system of the middle cerebral artery or between the supply territories of the anterior cerebral artery and middle cerebral artery, in a so-called 'rosary-like' pattern in the white matter along or above the lateral ventricle (Klijn and Kappelle, 2010), and imaging of the internal carotid artery.

Patients who have internal carotid artery stenosis in the presence of haemodynamic compromise (characterized by limb shaking) have significantly higher risk of recurrent ischaemic stroke than counterparts who do not have haemodynamic compromise (odds ratio 8.2, 95% confidence interval 2.3–29.3) (Persoon et al, 2010). Other studies that might be considered include contrast angiography to assess collateral blood flow pathways, and ophthalmological examination to check for venous stasis retinopathy and chronic ocular ischaemic syndrome.

Oscar Jolobe
Retired Geriatrician
c/o John Rylands University Library
Manchester M13 9PP

Bollu M, Marte-Grau A, George SM, Arsura EL, Bobba RK (2009) Transient ischemic attack in an elderly patient: What to expect? *Am J Med Sci* **338**: 155

Kamata T, Yokota T, Furukawa T, Tsukagoshi H (1994) Cerebral ischemic attack caused by postprandial hypotension. *Stroke* **25**: 511–13

Klijn CJ, Kappelle J (2010) Haemodynamic stroke: Clinical features, prognosis, and management. *Lancet Neurol* **9**: 1008–17

Klijn CJM, Kappelle LJ, van Huffelen AC et al (2000) Recurrent ischemia in symptomatic carotid occlusion. Prognostic value of hemodynamic factors. *Neurology* **55**: 1806–12

Persoon S, Keppelle LJ, Klijn CJM (2010) Limb-shaking transient ischaemic attacks in patients with internal carotid artery occlusion: a case control study. *Brain* **133**: 915–22