Original articles

Transient ischaemic attacks related to carotid stenosis precipitated by straining, bending, and sneezing

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Summary

Three patients are described in whom one or more carotid territory transient ischaemic attacks (TIAs) were preceded by sneezing, straining, or bending over. It is argued that the mechanism involved dislodgment of embolic material from the site of carotid atheroma. This mechanism should be considered as an alternative to paradoxical embolism when TIAs are precipitated by such physiological manoeuvres. Furthermore, TIAs should be added to the list of medical hazards associated with such events.

Keywords: transient ischaemic attacks; carotid stenosis; sneezing; straining

Clinical evidence suggests that most carotid territory transient ischaemic attacks (TIAs) are due to thromboembolism from the carotid bifurcation, heart, or aorta.¹⁻² Recently, there has been increased interest in the frequency of paradoxical embolism through a patent foramen ovale.³⁻⁴ Straining with elevation of right heart pressures at the time of a TIA or stroke is usually taken as evidence in favour of such a diagnosis. I have encountered three patients, however, in whom the circumstantial evidence strongly suggests that friable mural thrombus was discharged from the carotid bifurcation by the cardiorespiratory accompaniments of sneezing, straining, and doubling over.

Case reports

Case 1

A 53-year-old man developed loss of vision in the left eye followed by right-sided tingling in the arm and leg for 10 minutes whilst straining on the toilet. Over the next 3 days he had two episodes of word-finding difficulties lasting 10 minutes on each occasion. He then suffered a grand-mal seizure. Examination revealed no deficit apart from a mild naming difficulty, but angiography showed bilateral carotid stenosis. On the left this was almost total, due to the presence of obvious thrombus in the lumen. Intracranial filling on both sides followed injection of the right carotid artery. It was concluded that straining had caused embolisation. The patient had a successful endarterectomy with no further TIAs in the following year. He remained well 3 years later.

Case 2

Another man aged 50 years recorded a facial palsy lasting one week. A further week later while sitting he sneezed and immediately fell to the left with paralysis of his left arm which was also numb. He got up and found that he was unstable on his legs. His speech was slightly slurred. He was able to move his arm again within minutes but full recovery had still not occurred when he was seen 2 weeks later. When reviewed, he admitted to several episodes of numbness at the left corner of his mouth lasting a few minutes only over the previous 2 years. Carotid angiography revealed 50% stenosis on the left and 90% stenosis on the right. At right carotid endarterectomy 2 weeks later the lumen was occluded by loose thrombus.

Case 3

A 45-year-old man described a series of attacks of brief right-sided amaurosis fugax lasting 10-20 seconds. Each time visual loss began inferiorly and recovery commenced superiorly. On some occasions the loss described was obviously an altitudinal hemianopia. These attacks occurred weekly until he was prescribed aspirin, whereupon they became less frequent (less than monthly). He pointed out that one predictable trigger to individual attacks had been bending over suddenly. He described one different episode when straining on the toilet. He found he could not locate his zipper, and then noticed that his left arm and leg were numb. The sensory change persisted for 48 hours during which he noticed an inability to appreciate the temperature of the ventilation system in his car with his left hand. Examination 3 weeks after this episode revealed a right-sided carotid bruit and soft sensory signs in the left limbs. Cranial computed tomography showed a mature infarct in the right temporo-parietal area. Angiography showed a tight carotid stenosis on the right which was confirmed at endarterectomy which was complicated by a hemiparesis. He made a partial recovery and had no further episodes in the following year.

Discussion

The cardiorespiratory effects of raised intrathoracic pressure, such as that occurring during straining, have been extensively studied during the Valsalva manoeuvre.⁵ As intrathoracic

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racic pressure rises there is a matching rise in arterial pressure which may amount to 80–90 mmHg. This is maximal within a few cardiac cycles, perhaps four or five. A second rise occurs during the overshoot after release of the pressure as the glottis is opened. The pressures reached in the thorax during coughing are even higher, reaching 100–200 mmHg in spontaneous coughing. Consciousness may be preserved during ventricular fibrillation by the expedient of coughing every 1–3 seconds. The cardiac compression caused by the changes in intrathoracic pressure is enough to maintain carotid blood flow.⁶ Cerebrospinal fluid (CSF) pressure also passively follows intrathoracic pressure and venous pressure rises. Nasal stimulation, for example by capsaicin, elevates blood pressure in the experimental animal by 70 mmHg, even at doses below those that cause actual sneezing.⁷ Rarely, sneezing has caused a surge in plasma catecholamine levels associated with a phaeochromocytoma.⁸ These mechanical and circulatory changes underlie the well-known medical hazards of sneezing, straining, and coughing. These include retinal detachment, conjunctival haemorrhage, mediastinal haemorrhage, syncope,⁹ surgical emphysema, vertigo and deafness from membrane rupture,¹⁰ rib fracture,¹¹ prolapsed intervertebral discs, coronary artery spasm,¹² subarachnoid haemorrhage, headache,¹³ drop attacks in the presence of the Arnold-Chiari malformation,¹⁴ CSF rhinorrhoea,¹⁵ and pneumocephalus.¹⁶ The only benefit of sneezing, apart from defence of the airway,¹⁷ appears to be the cure of hiccups which was known to Hippocrates.¹⁸

The present series of cases suggest that cerebral embolism should be added to this list of

hazards. The patients each developed signs of cerebral ischaemia at the moment that intrathoracic pressure was elevated by sneezing, straining, or bending over suddenly. In two of them, loose thrombus was visible in the parent carotid artery at angiography, and confirmed at surgery. In another, classical amaurosis fugax attacks with altitudinal field loss were strongly supportive of an embolic mechanism associated with a tight carotid stenosis. The sneeze is unlikely to have been part of the symptomatology of the TIA since all were carotid territory events, and sneezing is thought to be mediated through the brainstem.¹⁹ No case showed evidence of arterial dissection, as may be triggered by sneezing, in the vertebral artery for example.²⁰ Fischbeck, Bradley, and Bank²¹ described a patient who developed a hemiparesis on sneezing in whom angiography revealed three aneurysms on the middle cerebral artery. They suggested that the Valsalva manoeuvre had produced hypoperfusion during the phase of impaired cardiac filling. It is also possible that embolism from an aneurysm²² was triggered during the phase 1 increase in forward systolic flow as intrathoracic pressure rose. That sneezing may also dislodge cardiac emboli is suggested by the case of a patient with a central retinal artery occlusion due to a calcific embolus in the presence of calcific aortic valve disease immediately after two violent sneezes (LA Wilson, personal communication).

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