is difficult to understand how cerebrovascular disease alone can adequately explain these patients' orthostatic changes.

Cerebral perfusion pressure at any point in the brain is the arithmetic difference between the local arterial pressure and the pressure of the surrounding CSF. Arterial blood and CSF are each continuous fluid media subject to the same hydrostatic principles. Their densities are approximately equivalent. On rising to upright posture, the arterial pressure drops in the brain, but the adjacent CSF pressure normally drops by more or less the same amount. Thus, there is little or no change in the difference between the two pressures (that is, the cerebral perfusion pressure) and circulation to the brain is maintained.

In the four patients of Stark and Wodak something has failed in this mechanism by which we ordinarily maintain our cerebral perfusion during postural change. It is simplest to suggest that their CSF might not have been an unobstructed fluid medium. Perhaps adhesions or some form of spinal stenosis prevented the CSF pressure in the head from dropping as rapidly as the intracranial arterial pressure fell when they assumed erect posture. Thus, as they rose to the upright position, the difference between their arterial and CSF pressures became narrower, and their cerebral perfusion pressure declined. Their neurological symptoms became manifest, of course, in the areas served by the most severely diseased arteries.

There is no information in the four case reports to confirm or refute this hypothesis. Even if it is correct, it may still be true that the patients were well served by the endarterectomies which they received. By removing the local arterial obstructions, the surgeons improved the delivery of blood to those areas which had previously been the most vulnerable to reduction in cerebral perfusion. Nevertheless, it is interesting to speculate about alternative approaches to the physiology and therapy of "primary orthostatic cerebral ischaemia".

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Wodak and Stark reply:

We agree with Dr Kahn that one may encounter asymptomatic patients with cerebrovascular disease of a severity comparable to the patients with "primary orthostatic cerebral ischaemia" reported by Stark and Wodak (*J Neuro Neurosurg Psychiatry* 1983;46:883-91). We fail to see however why this should suggest that vessel narrowing cannot adequately account for the patients' posturally dependent symptoms. Some patients seem to be clinically unaffected by carotid occlusion but that does not mean all patients will tolerate it. Dr Kahn accepts that "primary orthostatic cerebral ischaemia" developed because the four patients reported could not maintain adequate cerebral perfusion when standing. He suggests this is best explained by postulating a disturbance of CSF flow even though he concedes there is no evidence for or against this suggestion. One could also surmise that the syndrome arose from a failure of the jugular venous pressure to fall on assuming an erect posture though once again we have no evidence to confirm or deny this. Perhaps we were mistaken in regarding the occlusion, or near occlusion, of all of the neck vessels supplying the brain as the most obvious cause of a disturbance of cerebral perfusion. We must admit that we felt our patients' response to endarterectomy misled us into believing their vessel narrowing was germane to their symptoms. We attribute responsibility for our error to William of Occam and humbly confess that there is no logical reason to suppose that the simplest explanation will invariably prove correct. But in the 600 years since Occam formulated his maxim it has, as Bertrand Russell observed, proven "a most fruitful principle in logical analysis".

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