Matters arising

Isolated facial palsy in chickenpox

Sir: We report a case of isolated facial palsy during the course of chickenpox. This is with reference to Jamal and Husaini's short report on Bell's palsy.¹ Its occurrence during the course of chickenpox is rare, although facial palsy has been described with Ramsay-Hunt syndrome and occult infection with varicella-zoster virus with raised antibody titres.^{2,3} Manning and Adour⁴ found varicella to be the cause in only one out of 58 cases of isolated facial palsy in children.

A 22-year-old female, admitted to the antenatal ward with antepartum haemorrhage in December, 1983, developed chickenpox during the hospital stay. She awoke with a right-sided infranuclear facial palsy on the 16th day of chickenpox while the rash was abating. There was loss of taste sensation in the anterior two thirds of tongue on the right side. There was no other neurological deficit. Nerve conduction studies performed on 14th day of facial palsy showed motor unit potential of 30 μ V and 10.2 ms in right orbicularis oculi. On the left side the amplitude of motor unit potential was 3.5 μ V and latency of 3 ms. There was partial recovery of the facial palsy one month after its onset when the patient was discharged from hospital. This patient thus had an isolated facial palsy during the course of chickenpox with evidence of demyelination as suggested by delayed nerve conduction. The occurrence of facial palsy during the course of chickenpox should probably be attributed to the varicella-zoster virus.

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⁴ Manning JJ, Adour KK. Facial paralysis in children. *Pediatrics* 1972;49:102-9.

Hypoperfusion in the aetiology of subcortical arteriosclerotic encephalopathy (Binswanger type).

Sir: The advent of CT scanning has permitted a redefinition of the clinical and neuroradiological features of Binswangers subcortical arteriosclerotic encephalopathy.¹ The demyelination of the white matter seen on CT scans and at necropsy has been considered to be due to the effects of ischaemia, particularly in the watershed between cortical and deep vessels affected by arteriosclerosis and hypertension.² The possibility that poor perfusion might contribute to its pathogenesis³ prompted us to review a series of cases to see if there was any clinical evidence that the patients had suffered from any kind of haemodynamic crisis.

Thirty-seven cases diagnosed on CT scan as having white matter changes attributable to vascular disease were reviewed. Evidence was sought from the case notes as to the presence of hypotension or cardiac dysrhythmia, postural symptoms or other events that might be indicative of periods of poor cerebral perfusion.

Twenty of the patients had already been known to be hypertensive and a further 11 had a pressure over 160/90mm Hg at the time of presentation. Treatment in one patient had brought his mean arterial pressure down from 186 to 93mm Hg, a level which might well have been below the lower limit of autoregulation of blood flow in a hypertensive subject.⁴ Documented postural hypotension was found in one patient, the pressure falling from 150/90 lying to 110/70 standing. Four other patients described postural symptoms. For 2-3 years one man had developed dizziness and light headedness when walking and two patients had blackouts related to the adoption of the upright posture. The fourth case concerned a man who developed focal neurological problems on getting up out of a hot bath. Two further patients had diabetic neuropathy, one with overt autonomic involvement suggesting that postural hypotension might have been a problem. Three more patients described drop attacks although it was not possible to judge whether they were likely to be due to hypoperfusion. One patient had a severe anaemia (Hb 8 g/dl) and another had deteriorated after a drug overdose when he may well have been hypotensive. Two patients had frequent palpitations, in one

due to documented paroxysmal atrial tachycardia. Two others had a carotid stenosis, which was thought to have been haemodynamically significant from the angiographic appearance. In one instance the changes were bilateral. Finally two patients had specific causes for an encephalopathy. One man had pulmonary and cerebral emboli and an elevated ESR and was thought to have an arteritis. The other had hyponatremia.

Thus of these 37 patients 17 had some clinical evidence possibly incriminating episodic hypoperfusion in the development of their encephalopathy. While these are uncontrolled observations their frequency suggest that a more rigorous prospective study of such factors is warranted.

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Primary orthostatic cerebral ischaemia

Sir: The four cases of "primary orthostatic cerebral ischaemia" presented by Stark and Wodak (*J Neurol Neurosurg Psychiatry* 1983;46:883-891) offer an opportunity to re-examine the mechanism by which humans maintain cerebral perfusion when rising to the upright position. Many other elderly or diabetic patients have comparable degrees of vessel narrowing without having posturally dependent symptoms. None of the four patients had a significant fall in systemic blood pressure upon assuming erect posture. Thus, it

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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