

## 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.<sup>1</sup> 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.<sup>2,3</sup> Manning and Adour<sup>4</sup> 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  $\mu$ V and 10.2 ms in right orbicularis oculi. On the left side the amplitude of motor unit potential was 3.5  $\mu$ 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.

VK MURTHY,  
IMS SAWHNEY,  
S PRABHAKAR,  
JS CHOPRA

Department of Neurology  
Postgraduate Institute of Medical Education  
and Research,  
Chandigarh. 160012, India

### References

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<sup>4</sup> 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.<sup>1</sup> 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.<sup>2</sup> The possibility that poor perfusion might contribute to its pathogenesis<sup>3</sup> 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.<sup>4</sup> 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.

MJG HARRISON  
The Middlesex Hospital,  
Mortimer Street,  
London, W1N 8AA

JOHN MARSHALL,  
The National Hospital for Nervous  
Diseases,  
Queen Square,  
London, WC1 3BG, UK

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- 4 Strandgaard S, Olesen J, Skinhøj E, Lassen NA. Autoregulation of brain circulation in severe arterial hypertension. *Br Med J* 1973;1:507-10.

### 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