Hyperventilation during migraine attacks

We have recently seen four patients who hyperventilated during the headache phase of some of their migraine attacks. The symptoms frightened the patients and raised diagnostic difficulties for their family practitioners.

Case report

A 24-year-old film projectionist (case 1) had had common migraine from the age of 10. During the past two years he had had four severe attacks with additional features, in two of which he was taken to hospital by ambulance. During these recent episodes he had numbness round the mouth "like a dental anaesthetic," paraesthesiae ascending the limbs, a tightness round the mouth that interfered with his speech, a constricting sensation round his chest that made him "fight for breath," and a stiffening of the hands that he imitated by flexing the wrists and metacarpophalangeal joints and extending the interphalangeal joints. These added symptoms, associated with clouding of memory, occurred one hour after the headache started and lasted one to two hours, though the headache persisted for a further four hours. He found these symptoms frightening and thought that he was having a heart attack. Furthermore, at one hospital the doctor seemed puzzled and called in three others for advice.

Examination showed a tall, thin, tense young man without abnormal neurological signs. Deep breathing for three minutes produced his circumoral symptoms—his mouth looked pursed and he had dysarthria, he looked pale and anxious, and he wanted to get up and walk around the room. He said that the symptoms were characteristic of his recent severe episodes but without limb involvement.

Details of this patient and three others who hyperventilated with their attacks are shown in the table.

Details of four patients with hyperventilation during headache phase of migraine

Case No	Age (years)	Sex	Age of onset and type of migraine	Features of hyperventilation
1	24	M	10 years common	Circumoral and limb paraesthesiae, tightness in chest, dysarthria, carpodigital spasm, clouding of memory. Deep breathing for three minutes reproduced symptoms
2	40	M	Teens common	Took big gulps of air to prevent vomiting, hands became stiff and tingled
3	14	M	8 years classical	Previous attacks occurred during day. Recent severe migraine woke him from sleep. Mother noted boy was "panting" and patient complained of pins and needles in hands
4	27	F	11 years classical	Frightened to lie down in darkened room where she had difficulty in breathing. Developed numbness of tongue and mouth as well as cold, numb hands and feet

Comment

Migraine and hyperventilation occurring at different times in the same patient, although uncommon, presents no diagnostic challenge. When circumoral and limb paraesthesiae occur simultaneously with a migraine headache basilar migraine would seem at first sight to be a reasonable diagnosis. Yet in Bickerstaff's[1] original series vertigo, dysarthria, and gait ataxia were the usual symptoms, though paraesthesiae of the lips, tongue, and peripheries featured less often. But timing is the main difference. In basilar migraine brainstem symptoms are part of the aura preceding the headache, whereas the patients described here noted their sensory disturbance during and often at the height of the headache phase. Two patients also had stiff hands with the characteristic posture of the *main d'accoucheur*, which is not a feature of basilar migraine. We have found no mention of carpopedal spasm in current or older migraine case reports, even in the more severe cases.[2] The "experiments of nature" recorded here could explain the loss of consciousness reported in severe migraine attacks, although hypotension seems an alternative mechanism. More observations during such attacks are required.

A positive diagnosis is essential for treatment and to reassure the patient, who is advised not to take too deep or rapid breaths or to rebreathe expired air from a paper bag. The significance of carbon dioxide did not escape Wolff,[3] who administered 10% carbon dioxide in 90% oxygen or air to 15 patients but obtained inconsistent responses. We think that the relation between carbon dioxide pressure (Pco_2) and migraine has not been adequately explored. This is surprising in view of the potent vasomotor effects of carbon dioxide on the general and cerebral circulations and the fact that anaesthetists regularly hyperventilate patients to reduce brain volume during neurosurgery. If the idea of two types of migraine sufferers, dilators and constrictors, is correct[4] carbon dioxide retention during sleep[5] could induce migraine by vasodilatation; whereas hyperventilation provoked by excitement, anxiety, or expectation of physical or sexual activity[5] could precipitate vasoconstrictive migraine. Further, patients often "sleep an attack off," when a rise of Pco_2 could affect blood vessels, among other autonomic changes.

Our novel observation is that hyperventilation occurred at the height of a migraine, making the attack seem worse to the patient. We speculate that the migraine not only seemed worse but was exacerbated by the vasoconstrictor effect of hyperventilation. If this is true Pco_2 may play a part in the pathophysiology of migraine.

1 Bickerstaff ER. Basilar artery migraine. *Lancet* 1961;i:15-7.
2 Klee A. *A clinical study of migraine with particular reference to the most severe cases.* Copenhagen: Munksgaard, 1968.
3 Wolff HG. *Headache and other head pain*, 2nd ed. New York: Oxford University Press, 1963:237.
4 Blau JN. Migraine: a vasomotor instability of the meningeal circulation. *Lancet* 1978;ii:1136-9.
5 Slonim BN, Hamilton LH. *Respiratory physiology*, 3rd ed. St Louis: Mosby, 1976:142.

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Combined carbon haemoperfusion and haemodialysis in treatment of penicillin intoxication

Penicillin is well tolerated in most patients with normal renal function. Severe penicillin intoxication with neurological manifestations may occur, however, when large doses are given to a patient with impaired renal function.[1] Haemodialysis is beneficial in penicillin intoxication, although the clearance is low.[2] Little penicillin is cleared by peritoneal dialysis.[2] We present a case of penicillin intoxication with severe central nervous system symptoms in which combined haemodialysis and haemoperfusion was successfully applied.

Case report

A 60-year-old woman with analgesic nephropathy who had been on regular four-hour haemodialysis three times weekly for 15 months developed pleuropneumonia. Treatment with phenoxymethylpenicillin 0·8 g thrice daily was begun. Ten days later septicaemia was suspected and the treatment was changed to benzylpenicillin 5 MIU intravenously four times a day. This resulted in marked clinical improvement. The patient became drowsy after four days of intravenous penicillin treatment, with involuntary twitching of the facial muscles and jerking hand movements. She deteriorated progressively; became comatose with generalised, continuous muscle jerking; and appeared moribund. Penicillin intoxication was suspected, and treatment was discontinued nine days after it began. Before haemodialysis on the same day the plasma concentration of penicillin, measured by the agar diffusion method,[3] was 100 μg/ml. After 5·5 hours' haemodialysis the concentration fell to 85 μg/ml. A haemoperfusion column with 300 g activated carbon (Adsorba 300 C, Gambro) was inserted in series after the dialyser. In plasma samples from the inlet and the outlet of the column 15 minutes after the start of haemoperfusion the penicillin concentrations were 56 and 16 μg/ml. Clearance of penicillin was 143 ml/min at a blood flow of 200 ml/min. Combined haemodialysis and haemoperfusion for three hours reduced the plasma concentration to 21 μg/ml. The symptoms from the central nervous system receded, but the patient was still drowsy at the end of the treatment. The next day she was fully awake. She recovered without residual neurological abnormalities.