socioeconomic benefits of early diagnosis and management are manifest.

W HARKNESS Senior Registrar in Neurosurgery Atkinson Morley's Hospital, Copse Hill, London SW20 0NE

#### References

- 1 Asari S et al. Delineation of unruptured cerebral aneurysms by computerised angiotomography. J Neurosurg 1982;57:527-34
- 2 Newell DW et al. CT infusion scanning for the detection of cerebral aneurysms. J Neurosurg 1989;71:175-9
- 3 Pickard JD et al. Effect of oral nimodipine on cerebral infarction and outcome after subarachnoid haemorrhage: British aneurysm nimodipine trial. Br Med J 1989;298: 636-42
- 4 McKenna P, Willison JR, Lowe D, Neil-Dwyer G. Recovery after subarachnoid haemorrhage. Br Med J 1989;299:485-7

# Seizure induction by alcohol in patients with epilepsy

The article on seizure induction by alcohol by Heckmatt et al. (January 1990 JRSM, p 6) does not necessarily address the problem of alcohol, but rather of drinks which happen to contain alcohol. The difference may appear to be trivial, but may in fact be quite significant as shown by the following remarks concerning my own observations on one patient, namely my son who suffers from idiopathic epilepsy.

Some 8 years ago I noticed that he was regularly having fits on Saturday mornings after a week in which he had been virtually fit-free. I attributed this to drinking a little wine from a metal goblet on the Friday evenings and assumed that the alcohol was the responsible agent. However, switching to grape juice did not cure the problem and it was necessary to omit both the wine and the grape juice to avoid the Saturday morning fits. Since the drinks acquired a metallic taste when drunk from the goblet it seemed possible that the dissolved metals were triggering the epilepsy, although it could have been some other chemicals. More recently I found that 50 mg of pure tartaric acid, or 40 mg of pure citric acid, both of which are present in wine, will induce fits some 18-24 hours later. The same result was obtained with 40 mg of pure malic acid (which occurs in apples, tomatoes and plums). It seems inconceivable that these organic acids themselves are toxic, since three of them contribute to the citric acid cycle. Moreover, gas-liquid chromatography of the plasma and urinary organic acids showed no abnormality (Dr Chalmers, Northwick Park Hospital). However, it is now well established<sup>1-3</sup> that citric acid can promote absorption of aluminium, and the same is probably true for other metals and for other hydroxy-organic acids. It therefore seems reasonable to postulate that the epileptic fits were triggered by the organic acids in the wine, possibly by promoting absorption of metals.

In the article by Heckmatt et al., patient 8 in Table 2 had fits 24 h after one unit of alcohol, and it seems possible that he too was suffering from intoxication with some chemical other than ethanol. It would seem desirable to test the effect of pure ethanol on induction of fits before assuming that it is this chemical that is responsible in sensitive patients.

G A ROSE Department of Pathology St Paul's Hospital, London WC2

#### References

- 1 Slanina P, Frech W, Ekstrom L-G, et al. Dietary citric acid enhances absorption of aluminium in antacids. Clin Chem 1986;32:539-41
- 2 Hewitt CD, Poole CL, Westervelt FB, et al. Risk of simultaneous therapy with aluminium and citrate compounds. Lancet 1988;ii:849.
- 3 Hyperaluminemia in renal failure: the influence of age and citrate intake. J Clin Nephrol 1989;31:40-4

### The death of Mozart

I read with interest the book review by Sakula on the health of Mozart (February 1990 JRSM, p 131). In the book the author has no doubt deliberated on the mode of Mozart's death a subject which has long been of interest to both Mozart lovers and medical historians. Richard Wagner wrote in 1830 that most musicians believed that Mozart had been poisoned by Salieri. In 1984 a new theory was put forward by Dr Peter Davies [the author of the reviewed book] suggesting that Mozart's death was due to Henoch Schonlein purpura<sup>1</sup>. Two recent papers however may be of great interest to readers as they shed a different light on this old debate.

Puech et al. have recently studied Mozart's skull in the Salzburg Mozarteum. In one article they describe a mild craniofacial dysmorphism secondary to premature synostosis of the metopic suture which is confirmed by observing Mozart's portraits2. In addition they found a linear fracture in the temporoparietal area on the outer left side of the skull radiating towards the base<sup>3</sup>. Several bony modifications were noted on the underside of the visible portion of the fracture which the authors suggest is due to the presence of a chronic extradural haematoma which shrank and calcified as a secondary phenomenon. This may well have accounted for Mozart's headaches which started in the spring of 1790 and his weakness, faints or possible fits and his eventual paralysis on the 20 November 1791 culminating in his eventual death on the 5 December. ARPAN K BANERJEE Westminster Hospital

Deane Ryle Street, Horseferry Road, London SW1

## References

- 1 Davies PJ. Mozart's Illness and Death Musical Times 1984:CXXV
- 2 Puech B, Puech PF, Tichy G, et al. Craniofacial dysmorphism in Mozart's skull. J Forensic Sci 1989;34:487
- 3 Puech B, Puech PF, Dhellemmes P, et al. Did Mozart have a chronic extradural haematoma? *Injury* 1989;20:327-30

## Medical student selection

Horton writes (February 1990 JRSM, p 125) that 'Roberts and Porter argue that selection should be based upon psychometric tests rather than scientific achievement in A level'. We do not believe and did not write this (May 1989 JRSM, p 288). Horton does not seem to understand the nature of psychometric tests which provide a profile of a candidate's personality. Thus it is meaningless to talk about low scores. In fact, we wrote that 'there is no real alternative to prolonged assessment'. We believe that psychometric tests are potentially useful but they should be only one of several components in the selection process.

G D ROBERTS A PORTER 37 Upper Gordon Road, Camberley, Surrey GU15 2HJ