Matters arising

Papilloedema with extramedullary erythropoiesis and Cushing's syndrome.

Sir: It is possible that the severe papilloedema which appeared after the use of metapyrone in the patient described by Griffith and colleagues¹ was caused by a sudden reduction in circulating levels of cortisol.²

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References

- Griffith DNW, Crockard HA, Ross EJ, Huehns ER. Papilloedema with extramedullary erythropoiesis and Cushing's syndrome. *J Neurol Neurosurg Psychiatry* 1984; **47**:752-3.
- Neville BGR, Wilson J. Benign intracranial hypertension following corticosteroid withdrawal in childhood. *Br Med J* 1970; **3**:554-6.

Griffith replies:

Dr Neville makes a reasonable point but we are grateful for the opportunity to present further information which makes this suggestion appear unlikely to be the correct explanation. Not only was the patient commenced on hydrocortisone (30 mg/day) at the time of starting metyrapone, thus preventing a hypocortisolaemic state in absolute terms, but neither was a rapid fall in cortisol levels induced as no overall reduction had been caused by the metyrapone when papilloedema developed, as shown by the following values:

Metyrapone was commenced on 22nd September and papilloedema noted on 7th October.

Plasma cortisols (nmol/L) - 9.00 a.m. values:

6th August	630
25th September	925
3rd October	605

Midnight values:

4th August	775
25th September	640
2nd October	950

Urinary free cortisols (nmol/24 hours) (Normal range 220-1030)

19th September	4550
4th October	9255.

Syringomyelia, an hypothesis and proposed method of treatment

Sir: I have hesitated to reply to Mr Williams' comment on my views about syringomyelia (*J Neurol Neurosurg Psychiatry* 1983; **46**:365-7), because of my respect for his great knowledge, originality and expertise in the matter, in case it appears that I am in controversy with him. In fact, I think our views are complementary.

I have described the wedge and Mr Williams has described the hammer.

I suggested that the reason that the syrinx in the spinal cord continued to extend at its ends, was that it had an oval shape imposed upon it by the covering of the spinal cord and longitudinal arrangement of its tissue planes. The ends of the syrinx were the sites of maximal stress in the capsule in accordance with well understood physical laws (that is, that the tension in the wall, T, of a cavity is inversely proportional to the sum of reciprocals of the principal radii of curvature of the wall, R₁ and R₂, and proportional to the pressure difference ΔP across the wall).

$$T = \Delta P / \left(\frac{1}{R_1} + \frac{1}{R_2} \right)$$

That is, where the curvature is greatest, so is the tension in the walls, and hence a cavity is most likely to rupture its capsule at its sharpest point—this is in accord with everyday experience, and Laplace's theorem is a special case of this general formula.

The phenomenon of syringomyelia occurs only in the spinal cord because in other areas of the central nervous system, cavities tend to assume a more spherical shape in compliance with these same laws. In a sphere the tension is evenly distributed over the whole wall so there is no point of excessive tension where rupture is likely. It is similar to the situation of a bubble which assumes a spherical shape as this is the only stable shape. In the spinal cord, however, the covering and tissue planes prevent the formation of a sphere and a cavity assumes an ovoid shape with relatively sharp ends. Hence the shape and mechanics of the syrinx necessarily form a potential fluid wedge in the spinal cord.

Mr Williams' careful work has described how cough impulses and other causes of a rise in CSF pressure may produce a hammer which drives the wedge along the cord. The rises of pressure in the fluid of the syrinx, whether open or closed, will produce stresses in the wall of the syrinx which will be most likely to rupture it at the sharp ends of the syrinx.

Thus, the two views are not incompatible but complementary. The structure of the spinal cord makes any fluid filled cavity, open or closed, ovoid and thus a potential wedge which may be hammered along the grain of the spinal cord by those sudden rises in pressure which Mr Williams has demonstrated with coughing or Valsalva's manoeuvres.

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Sir: Edwards and Glen-Bott (*J Neurol Neurosurg Psychiatry* 1984; **47**:960-4) consider the potential of viloxazine to precipitate seizures in patients, and concentrate on the evidence from the Committee of Safety of Medicines (CSM). They correctly note that this compound is generally thought to have less epileptogenic properties than many other antidepressants, but in their summary conclude with the statement that this drug "is not contraindicated in epileptic patients requiring antidepressant medication".

Although the seizurogenic properties of antidepressants are clearly important when prescribing psychotropic drugs to epileptic patients, another important factor considered by Edwards and Glen-Bott only on the very last line of their article is the pharmacokinetic interaction between the antidepressants and the anticonvulsants. Although there are few investigations of this subject there is now growing evidence that viloxazine may interact adversely with several commonly used anticonvulsant preparations. Thus, early trials¹ provided some evidence that viloxazine may precipitate phenytoin intoxication and more recent data reported by Pisani et al² refer to an interaction with carbamazepine. In the latter study, five out of seven patients developed symptoms of carbamazepine intoxication following the administration of viloxazine, and discontinuation of the antidepressant resulted in remission of symptoms, suggestive of toxicity, and a return of elevated serum carbamazepine levels to acceptable values. In one patient viloxazine was associated with a rise in phenobarbitone levels.

Clearly further information is required on the interaction between other psychotropic and antiepileptic drugs as it is estimated that some 18% of epileptic patients