More on portal hypertension

From Dr Saeed Ahmad Senior Physician and Vice Chairman Department of Medicine, Fairmont General Hospital

Fairmont, West Virgina, USA

Dear Sir. The recent editorial by Dr Blendis in the August 1979 issue (p. 549), warrants further comment.

The relationship of polycystic kidney and polycystic liver is well documented (Comfort et al. 1952). Campbell et al. (1958) reported three adult cases of bleeding oesophageal varices and portal hypertension with polycystic liver and kidney disease.

Recently, DelGuercio et al. (1973) recorded two middle-aged patients with bleeding oesophageal varices attributed to portal hypertension secondary to polycystic liver and kidney disease. These authors concluded: 'one patient died after haemorrhage which might have been better treated if the index of suspicion for bleeding oesophageal varices had been greater'.

As more patients' lives are prolonged by maintenance haemodialysis, the possibility of bleeding oesophageal varices as the source of upper gastrointestinal haemorrhage in patients with polycystic kidney disease must be emphasized.

Chronic vitamin A intoxication can lead to portal hypertension in the absence of cirrhosis (Russell et al. 1974). This centrizonal hepatic fibrosis has been revealed in about half the reported cases. The prognosis is quite good and the abnormalities tend to recede on withdrawal of vitamin A. But, once portal hypertension and ascites are present, this syndrome may persist even after withdrawal of vitamin A (Russell et al. 1974).

Dr Blendis questions the true association of hepatic vein thrombosis in women on oral contraceptives. There can be little doubt that the Budd-Chiari syndrome can be caused by use of oral contraceptive steroids (Hoyumpa et al. 1971, Zimmerman 1978). A number of young women taking contraceptive steroids have been found to develop this syndrome as a result of thrombotic occlusion of the hepatic vein induced by the thrombogenic effect of the oestrogenic steroid (Hoyumpa et al. 1971, Zimmerman 1978). In these patients, the classical Budd-Chiari syndrome has developed, usually with a fatal outcome. Finally, radiation hepatitis causing severe congestive hepatopathy of the Budd-Chiari syndrome should be borne in mind (Zimmerman 1979).

Interruption of the rising morbidity and mortality from liver disorders could only occur when available information is utilized to develop preventive and therapeutic measures.

Yours faithfully SAEED AHMAD 19 September 1979

References

Campbell G S, Bick H D, Paulsen E P, Lober T H, Watson C J & Vanco R L (1958) New England Journal of Medicine 259, 904-910

Comfort M W, Gray H K, Dahlin D C & Whitesell F B (1952) Gastroenterology 20, 60-78 DelGuercio E, Greco J, Kim K E, Chinitz J & Swartz C

(1973) New England Journal of Medicine 289, 678-679 Hoyumpa A M, Schiff L & Helfman E L (1971) American Journal of Medicine 50, 137

Russell R M, Boyer J L, Bagheri S A & Hruban Z (1974) New England Journal of Medicine 291, 435-440 Zimmerman H J

(1978) Hepatotoxicity. Appleton-Century Crofts, New York Zimmerman H J

(1979) Medical Clinics of North America 63, 567-582

Intelligence and prenatal progesterone

From Dr Katharina Dalton 86 Harley Street, London W1N 1AE

Sir, Thank you for the opportunity to comment on the letter from Dr Meyer-Bahlburg (November Journal, p 878). His suggestion that my data may not stand up to scientific scrutiny could turn out to be a boomerang, for the true scientist does not confuse progesterone with progestogen. Dr Meyer-Bahlburg either does not know, or fails to understand, the differences between natural progesterone and the unnatural, man-made progestogen medroxyprogesterone acetate (Provera), which has a formula unlike any naturally-circulating steroid. Johanssen (1971) has shown that the administration of medroxyprogesterone acetate lowers the level of circulating progesterone. Medroxyprogesterone acetate is metabolized by a route different from that of natural progesterone, which is excreted in the urine and faeces as pregnanediol. Examination of the distribution of titrated pregnanediol in the pre-viable fetus shows that, after the liver, the brain and spinal cord are the areas of highest concentrations of pregnanediol (Cooke et al. 1967). Thus, regardless of his negative findings of the effect of antenatal medroxyprogesterone acetate on the intelligence of the offspring (Meyer-Bahlburg et al. 1977), this has no bearing on the effect of the natural progesterone on the offspring.

In the opening paragraph of Dr Meyer-Bahlburg's letter, he expresses the fear that my optimistic conclusions . . . may stimulate unnecessary and possibly unwarranted hormone treatment during pregnancy' even though, in the final paragraph of my paper (June, p 397), I stated: 'It would seem that progesterone administered in early pregnancy is beneficial not only for the relief of pregnancy symptoms and the prophylaxis of pre-eclamptic toxaemia but also to enhance intelligence. It will not produce a race of geniuses but merely ensure full brain development'. Are the relief of pregnancy symptoms and the prophylaxis of pre-eclamptic toxaemia unwarranted and un-

necessary hormonal treatments?

However, I do agree with Dr Meyer-Bahlburg that further studies are required and would reiterate the closing sentence in my paper: 'This is also seen as an example of clinical observation in general practice and demonstrates how the general practitioner can open the eyes of research workers in other disciplines, who can then probe deeper into the possibilities which have been offered to them'. No one has yet sought to probe deeper into the prophylaxis of pre-eclamptic toxaemia with natural progesterone, nor into the effect of natural progesterone on the intelligence of the offspring. Yours faithfully

KATHARINA DALTON 4 October 1979

References

Cooke I D, Wiqvist N & Diczfalusy E (1967) Acta endocrinologica 56, 43 55 Johanssen E D B (1971) Acta endocrinologica 68, 779 Meyer-Bahlburg H F L, Grisanti G C & Ehrhardt A A (1977) Psychoneuroendocrinology 2, 383

East Anglian bladder stone

From Dr A Batty Shaw Norfolk & Norwich Hospital, Norwich

Sir, I was interested in Dr J B Penfold's figures (September *Journal*, p 710) of the admission rate for bladder stone to the Essex County Hospital, Colchester from 1820–30, for such figures are hard to come by. In my paper on the Norwich School of Lithotomy (Batty Shaw 1970) I gave reference to all the figures I could trace on hospital admissions and the prevalence of endemic bladder stone in Great Britain, but if Dr Penfold's letter brings further figures to light I will be among those most interested.

Among the papers I quoted was a remarkably detailed study by Cadge of Norwich (Cadge 1874), who published the mortality rates for bladder stone from different areas and gave the admission rate for bladder stone cases between 1868-73 for 83 hospitals in England, Wales, Scotland and Ireland to which he had written. The highest number of bladder stone cases proportionate to the total number of cases admitted was at the Jenny Lind Hospital for Children, Norwich, followed by Great Yarmouth, Peterborough and the Norfolk & Norwich Hospitals. Cadge's figures also show the proportion at Colchester, Bury St Edmunds and Ipswich Hospitals where the total number of bladder stone cases admitted were respectively 4, 19 and 15. Thus there are some reliable figures, albeit for a short period, for the hospitals in which Dr Penfold expressed an interest.

Cadge's study, in my view, provides the most convincing evidence that endemic bladder stone was commoner in Norfolk, not only than in any other East Anglian county but, for a reason not yet

explained, than in any other county in England. This conclusion I stated in my paper 'East Anglian bladder stone' (March Journal, p 222), so I agree with Dr Penfold that my sentence elsewhere in the paper, if taken in isolation, that 'bladder stones were common in its [East Anglian] counties for three centuries' requires qualification.

I used the title 'East Anglian bladder stone' for two reasons that I stated when I delivered my paper. First, because bladder stone undoubtedly occurred in parts of East Anglia other than Norfolk and I wished to encompass some of the important contributions to the subject that such areas had made. Second, as a tribute to Mr J F R Withycombe of Cambridge as the first East Anglian President of the Section of Urology, who had kindly invited me to give my paper. When I delivered this I also defined my use of 'East Anglia'. In common with other East Anglians I have great affection for Colchester but I used the historical definition of East Anglia's southern boundary, and one usually favoured by others, that does not extend south of the Stour.

Yours sincerely A BATTY SHAW 27 September 1979

References

Batty Shaw A (1970) Medical History 14, 221 Cadge W (1874) British Medical Journal ii, 212

Otolaryngology in the curriculum

From Mr Norman A Punt London SW3

Sir, The points made by Mr J F Neil in his Presidential Address (August *Journal*, p 551) are extremely valuable, and it is to be hoped that notice will be taken of them by the appropriate authorities.

I would add that in my view all medical students should be trained in the use of the forehead mirror. The reason why they do not learn this skill is that firstly it is time-consuming to teach, and secondly the students know that they will not be examined in this proficiency in the final surgical examination. It should be possible for every student attempting this examination to be made to put on a forehead mirror and examine at least one patient. If students know that this will be expected of them and that they may fail the examination if they are incompetent in this respect, then they will take the trouble to learn it. It is not until they turn up in a casualty department or as an ENT house surgeon that they realize their lack of competence in so many procedures which they will need to use both at that time and probably later in general practice. Yours faithfully

NORMAN A PUNT 18 September 1979