demonstrated persistent elevation of the ST segments in the precordial leads, and a change to coronary sinus rhythm with flattening of T waves in leads V₃ to V₆. These abnormalities had reverted by day 7.

The only drugs administered over this period were vitamin K and phenoxymethyl penicillin for a sore throat. The fauces were injected on day 2, without exudates, and later atypical mononuclear cells appeared in the peripheral blood. It was not possible to exclude a viral myocarditis but complement fixation tests for *R. burneti* gave negative results. The Paul-Bunnell was also negative.

Two cases of cardiac abnormality in addition to Dr. Sanerkin's have been described with paracetamol overdose.^{2,3} At necropsy subendocardial haemorrhages and muscle necroses were demonstrated in one of these. E.C.G. evidence of myocardial and pericardial damage has also been reported.^{2,3} The present case was complicated by cardiac arrhythmia. Although this has been described with overdose of other drugs⁴ it has not hitherto been reported with paracetamol.

It is well established that hepatic damage appears several days after admission, when the patient may appear otherwise well. We suggest that the occurrence of arrhythmia is a further indication for continued inpatient observation after paracetamol overdose. In particular, the effect of the drug on a degenerate or ischaemic myocardium is not known.

We are indebted to the Poison Reference Laboratory, New Cross Hospital, and to Dr. Arnold Bloom for permission to report this case. —We are, etc.,

E. J. WILL
A. M. TOMKINS

Whittington Hospital,
London N.19

- 1 Prescott, L. F., Wright, N., Roscoe, P., and Brown, S. S., *Lancet*, 1971, 1, 519.
- 2 Maclean, D., et al., *Lancet*, 1968, 2, 849.
- 3 Pimstone, B. L., and Uys, C. J., *South African Medical Journal*, 1968, 42, 259.
- 4 Coull, D. C., Crooks, J., Dingwall-Fordyce, I., Scott, A. M., and Weir, R. D., *Lancet*, 1970, 2, 590.

Vasodilators in Menière's Syndrome

SIR,—I was interested to read of Dr. N. M. Piercy's experience (30 October, p. 303) of the use of betahistine hydrochloride in Menière's syndrome.

I observed a similar dramatic improvement in auditory function, with a marked diminution in the frequency of attacks of acute vertigo, which occurred in a 46-year-old male patient with Menière's syndrome shortly after he had commenced treatment with betahistine hydrochloride (Serc). This patient, who had undergone a right stapedectomy in 1963, was first seen by me six years later when he complained of tinnitus in his left ear, associated with recurrent attacks of giddiness and nausea. The attacks lasted for four to five days at a time with about a fortnight's interval between each one.

On examination he exhibited a marked conductive hearing loss, and on referral to an E.N.T. consultant the diagnosis of Menière's disease in his left ear was confirmed. Audiometry performed at the same time showed a perceptive deafness in both ears, more severe in the left ear. After a multiplicity of treatment his condition showed no improvement, and after about nine months he became obviously mentally depressed. He was then put on a regime of betahistine hydrochloride, 8 mg three times a day, and an improvement in his perceptive hearing, which was confirmed by audiometry, soon became apparent. His

attacks of vertigo came less often and, to date, he has only experienced one such attack since March this year.

Incidentally, I wonder if your heading to Dr. Piercy's letter "Vasodilators in Menière's Syndrome" is strictly accurate. I understand that betahistine hydrochloride, like histamine, is considered to act by its relaxant effect on the precapillary sphincters in the labyrinthine microcirculation, in which case it could not simply be described as a vasodilator.—I am, etc.,

J. U. TUKE

Sherborne, Dorset

Hepatic Vein Occlusion

SIR,—Your leading article on hepatic vein occlusion (4 September, p. 550) pointing out the poor prognosis of the condition conveys the impression that surgical treatment is rarely worthwhile.

There does, however, appear to be one type of Budd-Chiari syndrome where associated membranous obstruction of the hepatic veins and abnormalities of the inferior vena cava may be seen.¹ This type of lesion is potentially treatable by surgery though both diagnosis and surgery may be difficult. The details of such a case are given briefly below.

A white female aged 19 presented with pain under the right costal margin on sitting or standing. An abdominal swelling developed over the next 3-4 days. On examination she was slightly dyspnoeic with gross ascites and liver palms. There were no oedema or vascular spiders.

Investigations: Haemoglobin 13 g/100 ml. Normal peripheral blood and marrow, electrolytes, urea, and E.S.R. Bilirubin 1.7 mg/100 ml. Alkaline phosphatase 5 K.A. units. Albumin 3.5 g/100 ml.

Inferior vena cavogram showed narrowing of the inferior vena cava anteriorly with retrograde filling of vertebral, sacroiliac, and right azygos veins. Splenic venogram showed anatomically normal hepatic veins. The inferior mesenteric vein filled, and this, the splenic vein, and the hepatic veins emptied, slowly.

She was treated with diuretics and sodium restriction but an initial favourable response was maintained for a few months only and she was readmitted. Catheter studies showed a 7-8 mm pressure gradient across the narrowed portion of the inferior vena cava. The catheter entered the stump of the left hepatic vein, the lumen of which appeared totally obstructed just beyond its mouth. On the right side the catheter could be introduced into a major vein but only two short thin channels filled.

At operation it was considered that the inferior vena cava lesion was the most significant factor, and a pericardial patch was inserted in the inferior vena cava, but post-operatively the patient continued to deteriorate and died two weeks later.

At necropsy she was jaundiced. There was thrombus in the right atrial appendage, and portal vein thrombosis was present. The feature of main interest was the right and left hepatic veins, which showed occlusion approximately 2 cm from their point of entry into the inferior vena cava by oblique relatively thin and translucent fenestrated membranes (Fig.). The middle hepatic vein appeared abnormal with several narrow channels entering a short wide mouth. Microscopically the membranes had no

characteristic features to suggest an origin and were devoid of haemosiderin and elastic tissue.

Macroscopic appearance of terminal few cm of opened left hepatic vein. The lumen is obstructed by a delicate fenestrated membrane, through which is passed a probe.

The genesis of this lesion is not known: it has been suggested that it is congenital and related to closure of the ductus venosus.² Though the presentation is invariably in adult life a case without Budd-Chiari syndrome has been documented,¹ which confirms that the lesion may be clinically silent. The dense fibrosis sometimes observed around the inferior vena cava does not seem to favour a congenital lesion.

Successful results have been reported with varying surgical procedures,^{1,3} and though these remain difficult they probably offer the best chance of a good long term result in these cases.—We are, etc.,

D. J. EVANS
M. P. G. LITTLE

Department of Pathology,
Hammersmith Hospital,
London W.12

- 1 Yamamoto, S., Yokoyama, Y., Takeshige, K., and Iwatsuki, S., *Gastroenterology*, 1968, 54, 1070.
- 2 Kimura, C., Shirohane, H., Hirooka, M., Iwahashi, K., and Maetani, S., *Journal of Cardiovascular Surgery*, 1963, 4, 87.
- 3 Hepner, G. W., Steiner, R. E., and Bentall, H. K., *British Journal of Surgery*, 1969, 56, 245.

Geriatric Patients

SIR,—I would like, through your columns, to be allowed to congratulate Dr. B. Isaacs on his excellent paper (30 October, p. 282). Any geriatrician will recognize the impressive amount of work involved. The conclusions and suggestions arrived at appeal immediately to the experience of geriatricians. He shows that almost two-thirds of the cases accepted had not been referred until care and help had broken down, or threatened to do so. He also suggests that illness and disability in the elderly must exhaust any conventional system of domiciliary hospital care.

He says that he was not in a position to offer day hospital care, but this itself has a great impact on the situations described by Dr. Isaacs. However, there is a further approach to this difficulty of exhaustion of relatives, which Dr. Isaacs rightly recognizes and identifies as a powerful influence. For some years, locally, careful preparation had preceded the inauguration of a domiciliary psychogeriatric nursing service. This was the research project of a psychiatric team. My experience of this project has been small but it leads me to wish to see a further service of this sort in my own working area. Such a service seems to offer great hope and prospect of meeting the needs which "exhaust