a departure from this which I hope will be all for the better.

I have only recently returned from a tour of Denmark and Sweden where I was invited to go, through kindness of the Welsh Hospital Board. I visited a considerable number of hospitals in both countries and I can honestly say that, apart from providing really fantastic residential accommodation for the patients in every direction, they really have not solved the problem of what to do with the mentally subnormal person. We were told time and again that there is not enough room for children who become adults, and for adults to leave hospital to go into the community. So far as the former case is concerned the reasons given us were that by and large parents would not accept their relatives home. Even after careful clinical assessment done by multidisciplinary teams including doctors, psychologists, welfare workers, etc., patients were still not being removed. This really did surprise us because the social services in Sweden are really extremely well organized and have sufficient numbers to carry out just about everything that the social services department should be capable of doing.

It left no doubts in our minds that in spite of what I considered to be perfection in the care of the subnormal the majority of parents and relatives of patients still favour hospital care.—I am, etc.,

D. C. JONES

Hensol Hospital,
Glamorgan

Seat-belt Fasteners

SIR,—If seat belts are to be compulsory on all cars should not the standardization in type and operation of their fastening clasps also be made compulsory? More and more different devices are making their appearance, each with its own opening puzzle. Among them we have the clasp and bar type, the tongue in the slot edge-pressure release type, which may be in one case placed half way across the pelvis and in another slotted into the floor between the seats, and then there is the side-on knob-in-the-hole type. In the event of an accident a would-be rescuer could well waste valuable time discovering how to operate a particular release mechanism before an injured or unconscious occupant could be got clear of danger or free for treatment.

One of the reasons why some people will not wear seat belts lies in the fear that they will not be able to free themselves in an emergency, especially if the car turns over. If we go on producing more and more variations in this particular piece of apparatus it will come to pass that what were safety belts at an early stage in an accident could moments later become danger belts. A standard fastening would quickly become universally recognizable, especially if it were of the simple clasp and bar flick release type. —I am, etc.,

C. W. A. EMERY

St. Annes-on-Sea, Lancs

Diagnosis of Appendicitis

SIR,—Every diagnostician will agree that the diagnosis between an acute gastroenteritis and acute appendicitis when the appendix is lying in the pelvis and accompanied by diarrhoea and vomiting is a most difficult one, more so when there are cases of food-poisoning in the district. There is a simple test that is rarely used, and one which I have failed to see in print, that will make clear the diagnosis.

It is to get the patient to cough. If this hurts the abdomen (excluding spinal lesions) the examiner is dealing with an acute inflammatory lesion of the peritoneum such as acute appendicitis, diverticulitis, acute salpingitis, etc. The test is specially valuable because it can be applied without a hand being placed on the abdomen. It is exceptionally useful in dealing with small, nervous, and fretful children, who may scream when the doctor arrives and cry whenever the abdomen is touched. In these cases the mother can ask the child to cough, and even a very small child will try and do what she asks. If the child says that this hurts its "tummy," then an acute peritoneal inflammation is present—acute appendicitis. No stranger, doctor, or nurse need be present when this test is applied. Coughing will not cause abdominal pain in gastroenteritis, colitis, or acute mesenteric adenitis.

A further test, rarely reported in recent articles on acute appendicitis, is to inquire if there is any pain on passing water, more so towards the end of micturition. This symptom is rarely volunteered; it must be inquired for. As the bladder is an abdominal organ in children, the symptom is more common in this age group. It occurs in over 40% of children with acute appendicitis. In adults it is a valuable symptom when the appendix is buried in the pelvis.

Percussion, in the manner in which one would percuss a chest, is a valuable method of examination that is rarely employed. It is a much more sensitive and delicate test than the rebound phenomenon, which is sometimes done so roughly as to be harmful. Percussion has the advantage that in an inflammatory lesion the most sensitive spot is over the lesion and is a help to the surgeon in placing his incision.—I am, etc.,

G. D. F. MCFADDEN

Sunningdale,
Berks

Teratogenic Effects of Anticonvulsants

SIR,—Evidence regarding the teratogenicity of anticonvulsants seems to be accumulating rapidly. The evidence that anticonvulsants predispose to the development of cleft lip and cleft palate is now both clinical and experimental.[1 2] The evidence that anticonvulsants can produce chromosomal aberrations in the fetus is referred to and added to in the letter from Drs. I. C. Roman and A. Caratzali (23 October, p. 234).

There is experimental evidence to suggest that anticonvulsants may interfere with sexual maturation and differentiation,[3] but hitherto the only clinical suggestion that this may be so is the brief reference to a case of intersex occurring in the infant of a mother taking barbiturates during pregnancy.[4] I should like to report one further case, which in fact was phenotypically identical to the case mentioned by Nelson and Forfar.[4]

The infant in question was born at term to a woman suffering from post-traumatic epilepsy and taking phenobarbitone, primidone, phenytoin, phensuccimide, and carbamazepine (equivalent to 760 mg phenobarbitone per day using the scale of equivalence proposed by Hunter *et al.*[5]). The infant had ambiguous external genitalia (a large clitoris with rudimentary prepuce and common urogenital opening). Radiography and laparotomy revealed a hemiuterus, Fallopian tube, and streak gonad on the left, with a normal testis and seminal vesicle on the right. Chromosome analysis was performed on peripheral lymphocytes and both gonads. All were reported to be homogeneously 46/XY.

This infant differs from that mentioned by Nelson and Forfar in that the karyotype was normal male, whereas in their case there was mosaicism, most of the cells being XO but some being apparently XY with an imperfectly formed Y chromosome.[6]

It may of course be that the occurrence of the syndrome of mixed gonadal dysgenesis in these two infants together with the history of exposure in utero to anticonvulsants is coincidental. However, there is now considerable experimental evidence to suggest that just such a syndrome might be due to anticonvulsants. I should be most interested to know whether other readers have had experience of any other cases of failure of sexual differentiation in infants exposed to anticonvulsants in utero.—I am, etc.,

G. P. MCMULLIN

Rvecate Centre,
Sheffield

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Acute Myocardial Necrosis in Paracetamol Poisoning

SIR,—The report by Dr. N. G. Sanerkin (21 August, p. 478) describing myocardial necroses after self-poisoning with paracetamol prompts us to give the following account.

A 19-year-old girl, 20 weeks pregnant, was admitted having taken 30-50 paracetamol tablets with half a bottle of whisky. The blood level of paracetamol (estimated by gas chromatography) was 18·4 mg/100 ml 10 hours after ingestion and 1 mg/100 ml at 24 hours. From the data of Prescott *et al.*[1] hepatic damage was expected and the Table shows the abnormalities that occurred confirming this.

In view of the evidence that myocardial necrosis might also result, serial E.C.G.s were recorded. Those on days 2 and 4

Day	1	2	3	4	5	6	7	8
Prothrombin Time (in secs.) Control 12 ..		22	35	24	15	15	16	12
Serum Bilirubin (mg/100 ml)	0·1			1·0	1·5	1·6	1·2	1·3
S.G.O.T. (IU/l.) Normal 24	24	169		1350	437	185	94	53

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 V_3 to V_6. 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 drugs[4] 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

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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.

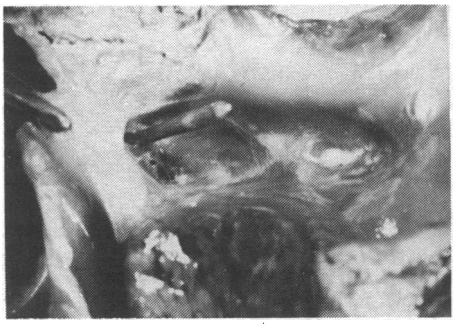
Investigations: Haemoglobin 13 g/100 ml. Normal peripheral blood and marrow, electrocytes, urea, and E.S.R. Bilirubin 1·7 mg/100 ml. Alkaline phosphatase 5 K.A. units. Albumin 3·5 g/100 ml.

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 to 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 postoperatively 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

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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