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.,

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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.,

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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.,

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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 anti-

convulsants 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, phensuccinimide, 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.,

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- 1 Meadow, S. R., *Proceedings of the Royal Society of Medicine*, 1970, 63, 48.
- 2 Elshove, J., *Lancet*, 1969, 2, 1074.
- 3 King, T. M., Fahim, M. S., and Bolt, D. J., *American Journal of Obstetrics and Gynecology*, 1968, 101, 850.
- 4 Nelson, M. M., and Forfar, J. O., *British Medical Journal*, 1971, 1, 523.
- 5 Hunter, J., Maxwell, J. D., Carrella, M., Stewart, D. A., and Williams, R., *Lancet*, 1971, 1, 572.
- 6 Keay, A. J., personal communication, 1971.

Acute Myocardial Necrosis in Paracetamol Poisoning

SIR,—The report by Dr. N. G. Sanerkin (21 August, p. 478) describing myocardial necrosis 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