Uterus in a Male

SIR,—A man aged 40 was admitted in July 1967 with a reducible right inguinal hernia and an undescended left testis. The right testis, normal in size and consistency, was present in the scrotum.

Exploration of the right inguinal canal revealed a completely patent processus vaginalis with the testis inside the sac. On applying traction to the testis, an oval mass, apparently intra-abdominal, was felt in continuity. At laparotomy the oval mass proved to be a fairly well-formed uterus, with gonads on either side. The right gonad was the testis situated in the scrotum, with epididymis and vas deferens attached to it. The left gonad was entirely intra-abdominal and relatively smaller; a fallopian tube attached to the uterus was present in close proximity, but there was no spermatic cord.

The left gonad was removed to avoid future complications and examined histologically. No ovarian or testicular tissue could be made out; there was marked interstitial cell hyperplasia. Biopsy specimen from the right gonad showed atrophic seminiferous tubules with large islands of interstitial cells. The nuclear sex was male. The chromosomal pattern could not be ascertained.

The patient described here was male in all respects except for the persistence of Müllerian duct on the left side, which had developed into uterus and tube. Sex differentiation in utero, particularly development of the genital ducts, is controlled by the organizing power of the gonads, especially the testis. In the male the Müllerian duct is suppressed by the inhibitory influence of the fetal testis. Also, when aberrations in the genital duct development accompany unilateral cryptorchism, they usually occur on the affected side.¹ In this case, on the left side, weak androgenic influence of the undescended gonad (if not absence of a testis) was probably responsible for the persistence of Müllerian duct, while on the right side the presence of a descended testis influenced the suppression of Müllerian duct and development of Wolffian duct.—We are, etc.,

ARABINDA MITRA.

Taore Hospital,
Kondagaon,
Madhya Pradesh, India.

KAMAL DAS.

Damodar Valley Corporation Hospital,
Chandrapura, Bihar, India.

REFERENCE

- 1 Jeffcoate, T. N. A., *Principles of Gynaecology*, 3rd edn., p. 211. London, Butterworths, 1967.

Vagotomy and Peptic Ulcer

SIR,—Mr. H. Burge and his colleagues (15 August, p. 372) are to be congratulated on at least two counts. Firstly, they have pioneered selective vagotomy in this country. This operation is emerging from controlled trials by other groups as superior to truncal vagotomy in respect of completeness of gastric vagotomy¹⁻³ and of post-vagotomy diarrhoea.⁴ Secondly, their series is the largest published in the world literature. It is all the more disappointing, therefore, that more precise data upon the

follow-up of these patients are not forthcoming in support of their statements.

Mr. Burge and his colleagues state that their "long-term study confirms Dragstedt's very early opinion that recurrent duodenal ulcer after vagotomy is due always to incomplete nerve section." Many authors would agree with this provided that the Zollinger-Ellison syndrome is excluded. The authors go on to say "it can therefore be prevented by the correct use of the electrical stimulation test." This may be true, but evidence for the accuracy of the statement is not provided by the authors nor are we told their own success rate with the test. This defect would be remedied by figures for the incidence of incomplete vagotomy and of recurrent ulcer in their series. It is not clear whether the ten reported cases represent the only recurrent ulcers that have actually occurred.

The experience of the gastric follow-up clinic at this hospital is that the reported incidence of recurrent ulcer is proportional to the diligence with which it is sought. The reviewed incidence of recurrent ulcer after vagotomy with drainage is approximately 5% while the incidence of incomplete vagotomy averages over 30%.⁵ Some workers with a special interest in the insulin test would regard the latter as a very considerable underestimate.⁶ Thus the ratio of incomplete vagotomy to recurrent ulcer is around 6 to 1. By inference, in the event of Mr. Burge's series yielding no more than the ten reported cases of recurrent ulcer, a minimum of 60 patients must have had unsatisfactory electrical stimulation tests. For the benefit of those who have also had limited success with this test which is so attractive in principle some more definite figures would be of great value.

The leucomethylene blue test of Lee⁷ is summarily dismissed. While it is true that in our experience, as in that of the authors quoted, this dye stains blood vessels and connective tissue in addition to nerve, it is also true that if after dealing with main vagal trunks the surgeon clears the lower oesophagus of all dye-staining tissue the result is likely to be a complete gastric vagotomy. Thus Lee has done a great service in focusing attention on careful oesophageal clearance. The dye should be more widely used.

The authors go on to remark, when discussing insulin tests, that "gastric acid levels can vary spontaneously to a marked degree before insulin was given." Since, in the absence of an abnormal non-gastric source of gastrin, basal or spontaneous acid output is probably dependent on vagal pathways, such an observation is only likely to be made in the presence of incomplete vagotomy. Spurious elevations of the basal acid can be minimized by delaying at least three months after operation before performing the insulin test and by careful attention to technique. Recognition that Hollander's criterion of a positive response to insulin, depending as it does upon basal acid concentration, can be unsatisfactory has led many workers to modify both the performance and interpretation of the test.⁶ It is regrettable therefore that Mr. Burge and his colleagues did not describe their methods.

Finally, their statement that "no case can be made for preoperative acid studies in planning the surgical treatment of duodenal

disease" would only be true if all patients merited the same operation, if gastric surgery were free of complications, and if deaths did not occur from the Zollinger-Ellison syndrome.—I am, etc.,

C. V. RUCKLEY.

Gastrointestinal Unit,
Western General Hospital,
Edinburgh 4.

REFERENCES

- 1 Sawyers, J. L., Scott, H. W., Edwards, W. H., Shull, H. J., and Law, D. H., *American Journal of Surgery*, 1968, **115**, 165.
- 2 Mason, M. C., Giles, G. R., Graham, N. G., Clark, C. G., and Goligher, J. C., *British Journal of Surgery*, 1968, **55**, 677.
- 3 Kennedy, T., and Connell, A. M., *British Journal of Surgery*, 1969, **56**, 385.
- 4 Kennedy, T., and Connell, A. M., personal communication, 1970.
- 5 Kay, A. W., in *After Vagotomy*, ed. J. A. Williams, and A. G. Cox, p. 1. London, Butterworths, 1969.
- 6 Ruckley, C. V., and Circus, W., in *Progress in Gastroenterology*, ed. G. B. J. Glass, Vol. 2, p. 73. New York, Grune and Stratton, 1970.
- 7 Lee, M., *British Journal of Surgery*, 1969, **56**, 10.

Continuous Ventilation and Oedema

SIR,—I read with interest the letter about continuous ventilation and oedema (29 August, p. 522). Patients with total respiratory paralysis have been artificially ventilated for even longer periods without developing oedema, but sometimes oedema can occur if the ventilation is inadequate or if a supra-atmospheric pressure persists in the trachea during the expiratory phase. Either of these might be put right by adding subatmospheric pressure during the expiratory phase. It would be instructive if Dr. J. Styles and his colleagues can give information whether under-ventilation may have occurred (perhaps in the patient's unsuccessful attempts to wean herself off assisted ventilation), or whether airway pressure may have been unusually high.—I am, etc.,

JOHN SPALDING.

Department of Neurology,
The Radcliffe Infirmary,
Oxford.

Pulmonary Tuberculosis Follow-up

SIR,—A by-product of mass chest radiography that cannot be overlooked and which has not been mentioned in the discussion of the report on the future of mass radiography (Circular H.M. (69) 97)¹ is the subsequent breakdown of old tuberculous lesions identified as a result of a mass survey.

In 1963 we completed a community chest x-ray survey of the adult population of East Suffolk, in which 80% of adults aged fourteen and older were x-rayed. A register was made of all those whose x-rays showed significant abnormalities considered after careful clinical, bacteriological, and radiological assessment to be due to inactive tuberculosis. Each year since 1965 these patients have been invited to attend the M.M.R. unit when it has visited the district in which they live. Completed figures of the subsequent findings are available from 1966 (Table). These show that from 1,372 obser-

Year	Community Survey Follow-up		Healed Tuberculosis Register Follow-up	
	No. examined each year	No. with Tuberculosis	No. examined each year	No. with Tuberculosis
1966	320	4	1,272	1
1967	332	6	1,328	2
1968	331	2	1,325	3
1969	389	1	1,556	0
Total Observation Years	1,372	13	5,481	6

vation years 13 patients (1.0%) have developed active tuberculosis. For comparison, patients who have been treated previously for pulmonary tuberculosis, the majority having had a full standard course of anti-tuberculous drugs and five years' follow up at the clinic, have been similarly followed. In 5,481 observation years there have been six reactivations of tuberculous disease (0.01%).

These figures show clearly the great value of following untreated inactive tuberculous lesions and confirm the excellent control achieved by adequate anti-tuberculous drug treatment. It could be argued that the community survey patients should have been given drug treatment. It is doubtful if it would have been possible to enlist the co-operation and adequately supervise such prophylaxis in persons who consider themselves fit.

The procedure for managing patients with inactive untreated pulmonary tuberculosis varies greatly between clinics. In some the patients are assessed and dismissed from follow-up, others are diligently followed by regular clinic attendance, and

some few receive antituberculous drugs. Our results show that both groups of patients may be followed up by 70 mm. M.M.R. without serious risk of the disease breaking down between the annual visit. In only one instance did a patient, who had had inadequate drug therapy, break down in the intervening period and infect her husband and child. In two recent community chest x-ray surveys in the Borough of Ipswich, in which 7,372 adults over 14 years were x-rayed, over 1.2% showed just such lesions.

The lesson to be learnt from this and other similar experiences² is that all apparently healed tuberculous lesions must be followed, no matter from where they originate, and mass chest x-ray is a good way of finding and following them.—I am, etc.,

CHARLES J. STEWART.

St. Helen's Hospital,
Ipswich.

REFERENCES

- 1 Department of Health and Social Security. *Mass Miniature Radiography Service*, H.M. (69) 97. London, 1969.
- 2 Styblo, K., et al., *Bulletin of the World Health Organization*, 1967, 37, 819.

Driving and Epilepsy

SIR,—You report (15 August, p. 362) that from 1 June 1970 new regulations for the issue of driving licences allow some people suffering from epilepsy to hold a driving licence. Such a person who suffers from epilepsy and who wants to drive must satisfy three conditions, one of which is that he has been free of an attack while awake for at least three years. No distinction is made between idiopathic and symptomatic epilepsy. The limited interval of three years may well be hazardous as the following patients in my practice during the past 15 years amply illustrate.

A man aged 40 years was discharged from the R.A.F. in 1949 suffering from epileptiform attacks. Phenobarbitone was started and he continued to take this drug until 1954, when it was finally stopped. He had been fit-free during this time. In November 1955 he had a major fit while golfing. Phenobarbitone was restarted and continued until 1962. He applied for and was granted a driving licence. He has driven daily since. In November 1966 he collapsed in status while walking to his car, his only fit during the preceding 12 years. Phenobarbitone has been restarted and he remains fit-free.

A man aged 23 suffered frequent major fits during childhood and until 1960. He was then controlled with phenobarbitone and phenytoin and he remained fit-free until he left school and started to work. By 1968 his phenobarbitone had been reduced to a nightly dose. He applied for a driving licence and was referred to a neurologist, who found no clinical evidence of epilepsy but subsequent E.E.G., though for the greater part normal, showed occasional bursts of instability which became suggestive of epileptiform pattern on overbreathing. He was advised to re-apply in a further two years and meanwhile to

continue phenobarbitone at night. In 1969 he developed several major fits while travelling to work, his first for nine years.

A man aged 44 years suffered from occasional major fits until 1953, when primidone was prescribed. He apparently had kept his driving licence because in 1962 after being free of major fits for six years he developed a convulsive attack while driving and an accident resulted. His driving licence was withdrawn and E.E.G. the following year was normal. He continues to take primidone and has remained free of attacks since, but repeat E.E.G. in 1966 showed some epileptic features.

A man aged 43 was injured in 1951, when a cylinder exploded, damaging his skull and left temporal area of brain. After elevation of skull fragments and excision of damaged brain he made an unexpectedly good recovery. He remained aphasic for some months but had no other residual neurological deficit. A few minor fits occurred in 1952 and he was started on phenobarbitone, which by 1955 he discontinued. He applied for a driving licence in 1959, and was examined by a neurologist, who found no evidence of disease and his licence was granted. He has driven daily since. After some prolonged work in 1969 he collapsed in status epilepticus as he walked from his car. He had been free of major epileptic attacks for 14 years.

A man aged 52 years had occasional major fits and took phenobarbitone until 1953, when he discontinued the drug. He remained well until in 1958 he was crushed by a bus and his attacks returned. Phenobarbitone was restarted and apart from a doubtful minor episode in 1963 he remained well and phenobarbitone had been reduced to grain $\frac{1}{2}$ (30 mg.) at night. He applied for a driving licence and did not disclose to the neurologist who examined him that he was still taking phenobarbitone. The E.E.G. was stated to be normal and he had no other abnormal C.N.S.

signs. The neurologist considered him to be fit to hold a driving licence. In 1968 he had a major fit after an interval of at least five and probably eight years.

Clearly, the statutory requirement that an epileptic who wishes to apply for a driving licence "be free from any major epileptic attack while awake during the preceding three years" is not necessarily adequate to ensure the safety of other road users.—I am, etc.,

K. T. FARN.

Atherstone,
Warwicks.

Diabetes and the Driving Licence

SIR,—Arising out of the recent regulations and the correspondence concerning epilepsy and the driving licence, one wonders what advice to give to diabetics when they apply for a licence.

The question to be answered is: "Are you suffering from any other disease or disability likely to cause the driving of a motor vehicle by you to be a source of danger to the public? If you are in doubt about your answers you should get professional advice."

My advice to well-controlled diabetics, who are not subject to hyperglycaemic or hypoglycaemic attacks, has been to answer: "No." Also I have not advised them to disclose that they suffer from diabetes to their motor insurance societies, unless specifically asked.

I would be grateful for the experience and opinions of other doctors on what advice they give their patients. Obviously there must be some diabetics who should answer "Yes," but I do not feel that all should be obliged to declare their disability. The most potentially dangerous cases must be the undiagnosed and untreated diabetics.—I am, etc.,

PATRICK J. O'CONNELL.

Wallington, Surrey.

Paraquat Toxicity

SIR,—It was sad that in paying tribute at the passing of Dr. Otto Warburg Drs. D. M. Stokes and D. A. Walker (22 August, p. 462) should draw attention to his theory on the origin of cancer cells which has little experimental support. The many inaccuracies of fact in the "evidence" they put forward confirm indeed their statement that they are not qualified to comment.

Reference to the literature would have shown that diquat does not cause "proliferation of the epithelium of the lung." After ingestion of paraquat (for which they evidently did not realize methyl viologen is merely another name) it is proliferation of fibroblasts, not epithelium in the lung, which is the striking pathological feature of the lesion. Proliferation of epithelium only occurs in terminal bronchioles and is probably a secondary phenomenon since it is found in other types of fibrosis involving this region of the lung. The fibroblastic reaction has none of the features of a cancerous process, and prolonged experiments in several animal species have failed to show any carcinogenic action of paraquat. This is accepted by the medical authorities in more