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 connexion with the processus vaginalis and 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 was deferens attached to it. The left gonad was entirely intraperitoneal 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 fusion of a complete undescended testis. Histological examination showed an undescended ovary with a lining of germinal cells, tubules, and a corpus luteum. It was felt that the patient might have a state of Müllerian duct syndrome, as there was no congenital anomaly except for the undescended testis.

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REFERENCES


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 vagotomy and of post-vagotomy diarrhoea. 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 Drahnetz's very early opinion that recurrent duodenal ulcer after vagotomy is due always to incomplete nerve sequence." 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 has usually been reported in previous papers only. Nevertheless, we told our 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 proportionate to the diligence with which it is sought. The reviewed incidence of recurrent ulcer after vagotomy with drainage is approximately 5% rarely while the incidence of incomplete vagotomy averages over 30%. Some workers with a special interest in the insulin test would regard the latter as a very considerable underestimate. 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 vagotomy 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 leucocytomethylene blue test of Lee is summariy 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 nerves, 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; 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 stimulated acid is probably dependent upon vagal pathways, such an observation is only likely to be made in the presence of incomplete vagotomy. Spurios elevations of the basal acid can be minimised by securing 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 upon suppression of basal acid concentration, can be unsatisfactory has led many workers to modify both the performance and interpretation of the test. 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.,

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REFERENCES


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.,

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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) 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 worthy of careful clinical, bacteriological, and radiological assessment to be due to inactive tuberculosis. Each year since 1965 these patients have been asked 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-
viation years 13 patients (1-0%) have developed active tuberculous. For comparison, patients who have been treated previously for tuberculosis, the risk of tuberculosis after 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 whether it would have been possible to
enlist the co-operation and adequately supervise such prophylaxis in persons who con-
sider themselves fit.

The procedure for managing patients with
inactive untreated pulmonary tuberculosis varies greatly between clinics.
In some the patients are assessed and dis-
missed 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.
measurements only. The breaking down of the
between the annual visit. In only one instance did a patient, who had had inade-
quate 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 experiences is that is 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,
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Ipswich.

Driving and Epilepsy

Sir,—You report (15 August, p. 362) that
from 1 January 1970 new regulations for the
issue of driving licences allow some people
suffering from epilepsy to hold a driving
licence. Such persons suffer 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 two years. No diagnosis 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 epileptic
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. Ph-n-barbitone 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 and marked slowing 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. to
that time 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 pheno-
obarbitone, which by 1955 he discontinued. He
applied for a driving licence in 1959, and was
examined by a neurologist, who found no evi-
dence 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 12 years. A man aged 52 years had occasional major fits and took phenobarbitone until 1953, when
he discontinued the drug. He remained well until
1958 when 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 arain 1 (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.,

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Diabetes and the Driving Licence

Sir,—A arising out of the recent regulations
and the correspondence concerning epilepsy
and the driving licence, I wonder what advice
to give to diabetics when they apply
for a licence.

The question to be answered is: “Are you
suffering from another disease or disabil-
ity 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 diabe-
tics.—I am, etc.,

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Paraquat Toxicity

Sir,—It was sad that in paying tribute at the
passing of Dr. Olilex 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
tories and pathologists who have followed
him in this line, which is the striking pathological feature of the
lesion. Proliferation of epithelium only occurs in terminal bronchioles and is prob-
ably a secondary phenomenon since it is
found in other types of fibrosis involving
this region of the lung. The fibroblastic re-
action has none of the features of a cancer-
ous 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