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.,

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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.,

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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)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-