Correspondence

Preperitoneal Prosthetic Herniography

SIR,—For some time it has been my view that the operative treatment of inguinal hernia as most commonly performed is illogical, and has certain disadvantages which would be best avoided if acceptable alternatives were available. The object of the operation of separating the general peritoneal cavity from the hernial sac at the level of its neck is achieved by an approach through the inguinal canal which disrupts its anatomy unnecessarily, and a repair effected by approximating the conjoint tendon to Poupart's ligament seeks to unite muscle to ligament, utilizing and distorting tissues which by the very presence of the hernia have demonstrated their incompetence.

The first layer of the abdominal parietes to be transgressed in the development of a hernia is obviously the deepest layer, allegedly the fascia transversalis. It would seem logical, therefore, to reinforce the abdominal parietes at this level in order to prevent recurrent herniation; to do so in the plane of the conjoint tendon savours of "closing the stable door after the horse has bolted." I have not, however, had any confidence in a repair based on suturing the fascia transversalis, as this tissue has always seemed to me to be insubstantial, and I have as an alternative taken to reinforcing this layer with a prosthesis. In order to overcome the aforementioned disadvantages of conventional hernia operations the following operative sequence has been evolved which I designate "preperitoneal prosthetic herniography."

A midline suprapubic incision1 carried out under muscle-relaxant anaesthesia and an extra-peritoneal approach to the inguinalfemoral region enables a bilateral operation to be carried out if necessary and allows easy direct access to the hernial sac at its source. The hernial sac is either withdrawn into the abdominal cavity or divided at its neck and the defect in the peritoneum closed. A prosthesis of Marlex mesh2 is sutured in position to the following points: the pubic tubercle, the peritoneum of the superior pubic ramus, the ilio-pubic tract lateral to the internal inguinal ring, the anterior rectus sheath in the midline, and the deep aspect of the rectus muscle. By so doing the postoperative incidence of recurrence in my hands has been only 1.5 per cent., the repair being effective in 98.5 per cent. of cases, with no recurrence during an average follow-up of 10 years.

L. F. TINCKLER.
Royal Gwent Hospital,
Newport, Mon.

REFERENCES

Diabetes Mellitus and Prostatic Hyperplasia

SIR,—I have read with interest the paper by Mr. J. B. Bourke and Dr. J. P. Griffin (23 November, p. 492). In a recent morbidity survey of 713 men aged 70 years and over,1 the incidence of benign prostatic hyperplasia was 20.9%, of diabetes mellitus 1.9%, of old

years who were multiparous and under 30 years of age.—I am, etc.,
J. ELIZABETH MACGREGOR.
Department of Obstetrics and
Gynaecology
University of Aberdeen.

REFERENCE

SIR,—We think we are bound to draw attention to Dr. R. W. Lacey's letter (7 December, p. 642). We are aware of the shortcomings in the method used in our study (23 November, p. 492), and the final paragraph of the discussion stated that "it is possible that the high incidence of diabetes found in this study might in part be due to the stress of operation and glucose-tolerance testing between the seventh and twelfth postoperative day when the patient was ambulant and eating a normal ward diet may have been insufficient precaution. Reduction of glucose tolerance with increasing age may also be a factor (Streeten et al., 1965; W.H.O. Expert Committee on Diabetes Mellitus, 1965; Butterfield, 1966). Further work is being undertaken to study these points.

The significance of the last sentence seems to have been overlooked. Despite the small series of 51 patients, it has confirmed the earlier findings of a retrospective study of 432 patients, who were submitted to operation for benign prostatic hyperplasia during a five-year period. An increased incidence of known cases of diabetes mellitus compared with normal populations was reported and substantiated by statistical analysis.

We are surprised that Dr. Lacey is unaware of the hypothesis that benign prostatic hyperplasia is one manifestation of a relative increase of oestrogen with advancing age; for it is not novel. The data for this were fully reviewed by both Teilmann2 and Scott.3 Evidence of an increased oestrogen-androgen ratio has been provided by Marmorson et al.4 who reported that this oestrogen-androgen ratio in 24-hour urinary collections from men with benign prostatic hyperplasia was increased compared with normal controls. It was this finding of abnormal oestrogen secretion which we quoted as further support for the hypothesis that benign prostatic hyperplasia is one manifestation of a relative increase of oestrogen secretion with advancing age. All these references were discussed in our paper.

We should, however, like to include the age distribution of our patients, which we apologize for omitting, together with the incidence of diabetes in each 10-year age group.

<table>
<thead>
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<th>Age Group in Years</th>
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<td>80-84</td>
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