prostate down on to the triangular ligament. The catheter itself is not sufficient to do this, and if one relies on the catheter alone there is usually a long gap between the two ends which has to heal by fibrous tissue and this inevitably leads to an extremely difficult stricture. I know that it is impossible to stitch the two ends of the urethra itself as they are inaccessible, but, though difficult, it is not impossible for those used to operating in the retropubic space to fix the prostate and therefore the prostatic urethra down to the triangular ligament and therefore the membranous urethra with the stitches I have described.

It is a great pity that this point is not stressed more in the routine textbooks of surgery for I think most surgeons and many urologists rely on the indwelling catheter to approximate the two ends. I am quite sure that if this amount of tension is put on a Foley catheter further damage can occur and in any event, it would be unlikely to bring the two ends anywhere near together.—I am, etc.,

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Surgery for Rectal Prolapse

SIR,—In reply to Mr. R. S. Lawson (24 April, p. 224) I think it is important to provide some background to the paper in which I reported the results of rectosigmoidectomy for rectal prolapse at St. Mark's Hospital.

The staff of this hospital were unhappy about the results of a number of operations carried out for this distressing complaint, and I was given the task of reviewing all cases of rectal prolapse admitted to St. Mark's Hospital between the years 1948 and 1960 inclusive. The total number of cases reviewed was 536. Of these, 142 were treated by rectosigmoidectomy. Thirty-two ((22%) were untraced. Full details were available on the remaining 110 cases. Of these, 50% developed a complete recurrent prolapse postoperatively and 8% a mucosal recurrence. Half of the complete recurrences occurred within three years of the operation. Admittedly the criterion adopted for assessment of incontinence in this study is exacting in that we define it as the uncontrolled passage of solid or liquid faeces. Attempts to quantify it in any other way make nonsense of statistical evaluation. A knowledge of anorectal physiology makes the poor continence rate understandable in that this operation anastomoses a highly active segment of colon—namely, the sigmoid—to the top of the anal canal, which in rectal prolapse has defective sphincters. Add to this the loss of rectal sensation concomitant upon excision of the rectum and these patients are left with very little of the normal physiological requirements for faecal continence, so that when they do develop recurrent prolapse they present a very difficult salvage problem indeed. Further amputation is not a proper solution.

The follow-up and postoperative management of these cases was thorough, and they were all taught sphincter exercises and a number of them were given courses of pelvic faradism, which does not appear in itself to improve muscle function but it does help to re-educate the patient in the use of his pelvic muscles. This is important as investigation by electromyography shows that many of these patients have lost their pelvic postural reflex and the faculty of conscious proprioception where their pelvic muscles are concerned.

In spite of these measures this very large number of recurrences occurred—surely because the operation of rectosigmoidectomy is not based on either sound physiological or aetiological principles. One has great respect for Ernest Miles and his works but at the time he developed this operation for rectal prolapse very little was known about the physiology of the anus and rectum and, in all fairness, one should recognize Aufret's original contribution of first performing the operation for a gangrenous rectal prolapse in 1886.

With regard to Mr. Lawson's other points, we did not find the obesity had any significant bearing on success or failure with this operation.

His final point in regard to avoidance of alcohol and heavy beer drinking puzzles me. The predominant incidence of complete rectal prolapse in this country occurs in women from the age of 40 upwards with a peak in the seventh decade. I can only conclude that the incidence of rectal prolapse in Melbourne with regard to sex and age differs from that in this country, or maybe the drinking habits do.—I am, etc.,

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Hormones in Advanced Cancer

SIR,—In the article on hormones in advanced cancer (26 June, p. 760) the following statement occurs. "Larger doses of L-thyroxine (0·4 mg daily) and tri-iodothyronine (200 mg daily) may control recurrent disease and should be tried after other methods of treatment have failed, regardless of the histological type of the tumour."

I know of no evidence that this treatment has any beneficial effect in undifferentiated tumours of the thyroid and to prescribe these hormones "regardless of the histological type of the tumour" is incorrect, unless the patient is hypothyroid.—I am, etc.,

RAYMOND GREENE

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Iatrogenic Raynaud's Phenomenon

SIR,—We would like to report the development of Raynaud's phenomenon in a patient treated with the recently introduced antihypertensive agent clonidine.

The patient is a 28-year-old man with severe hypertension owing to chronic proliferative glomerulonephritis of five years' duration. His renal function has remained relatively stable over this period (creatinine clearance 56 ml/min/1·73 m^2) but on recent hospital admission his blood pressure was found to be 200/140 mm Hg, despite methyldopa and bethanidine therapy. On this occasion clonidine was gradually substituted for the above therapy to a dosage of 0·9 mg daily with initial good control of his hypertension. At the end of a four-week period on this dosage he presented with dry mouth, drowsiness, complete impotence, a dry scaly rash of the dorsal aspect of the hands beginning with bulla formation, and arthralgia. More striking was his complaint of recent onset of Raynaud's phenomenon de novo of one week's duration, which had become so troublesome that he requested withdrawal of the drug. Withdrawal resulted in gradual cessation of his symptoms over a period of three days, with no recurrence over the last four months. There was no evidence of cervical rib; lupus erythematosus, latex, and antinuclear factors were negative; erythrocyte sedimentation rate and immunoglobulin levels showed no change from previous figures; there was no evidence of eosinophilia nor other haematological abnormality.

The symptoms suggested a syndrome resembling hydrallazine-induced systemic lupus erythematosus but evidence for this was not forthcoming. Skin eruptions have been reported,[1] and five patients (one of whom had transient Raynaud's phenomenon which disappeared during continuation of the drug) were reported in discussion at a symposium on clonidine.[1] The drug is a peripheral vasoconstrictor due to its alpha-adrenergic action when given intravenously,[2] but when given orally its hypotensive action is thought to be mediated by reduced vascular smooth muscle response to catecholamines and angiotensin.[3]

It is interesting that Raynaud's phenomenon can develop with such diverse peripheral actions of the drug and we wonder if other workers have knowledge of similar phenomena.—We are, etc.,

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1 *Carapres in Hypertension:* A Symposium held at the Royal College of Surgeons of England, March, 1968, Ed. M. E. Connolly, p. 213-215, London, Butterworths.
2 Barnett, A. J., and Cantor, S., *Medical Journal of Australia*, 1968, **1**, 87.
3 Zaimis, E., and Hanington, E., *Lancet*, 1969, **2**, 298.

Sniffing Syndrome

SIR,—The correspondence (19 June, p. 708) on the use of medicated aerosols by asthmatics in response to your leading article on the "Sniffing Syndrome" (24 April, p. 183) has prompted us to give this brief account of experiments we have undertaken to assess the risks that may be associated with using isoprenaline aerosols propelled by fluorocarbons.

In common with other workers, such as Reinhardt *et al*,[1] we have found that serious cardiac arrhythmias can be produced under certain severe experimental conditions by the rapid intravenous injection of a large dose of adrenaline during the inhalation of the fluorocarbon propellants by conscious dogs. In contrast to Taylor and Harris,[2] however, we found this cardiac sensitization to be only temporary, since an injection of adrenaline a few minutes after cessation of exposure never resulted in arrhythmias.

In similar experiments, using isoprenaline in place of adrenaline, we were unable to produce cardiac arrhythmias during fluorocarbon inhalation. Furthermore, when we tried to simulate the use of a pressurized