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.,

N. H. PORTER

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Sussex

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 *Catapres 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

isoprenaline inhaler during an acute asthmatic attack in man, by giving 0·5 μg/kg isoprenaline intravenously every 30 seconds to hypoxic dogs during a five-minute exposure to fluorocarbon 11, we again failed to induce cardiac sensitization. The Pao_2 values of approximately 50 mm Hg recorded in these experiments are comparable to those found by Tai and Reed[3] in patients with clinically severe asthma, but the blood concentration of fluorocarbon and the dose of isoprenaline were far in excess of those likely to result in man from the use of pressurized inhalers. The concentrations of fluorocarbon 11 in the blood were at least twenty times greater than those found in man by Dollery *et al.*[4] following a massive overdose of 30 puffs of an inhaler in two minutes, and the dose of isoprenaline was equivalent to at least 50 puffs of an inhaler in the space of five minutes.

Our results, therefore, do not support the view that the unexplained deaths among asthma sufferers could have been due to cardiac sensitization resulting from the use of pressurized aerosols containing isoprenaline.

These results were presented in detail to the European Society for the Study of Drug Toxicity in Berlin, June 1971, and are to be published in the Proceedings.

—We are, etc.,

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1 Reinhardt, C. F., Azar A., Mayfield, M. E., Smith, P. E. jun., and Mullin, L. S., *Archives of Environmental Health* 1971, **22**, 265.
2 Taylor, G. J., and Harris, W. S., *Journal of the American Medical Association*, 1970, **214**, 81.
3 Tai, E., and Read, J., *Lancet*, 1967, **1**, 644.
4 Dollery, C. T., Draffan, G. M., Davies, D. S., Williams, F., and Connolly, M. E., *Lancet*, 1970, **2**, 1164.

Infant Cot Deaths

SIR,—Among the many hypotheses put forward to account for these tragic infant deaths the suggestion of the possibility of airway obstruction of the larynx by closure of the epiglottis has not been mentioned.

The most common postmortem findings reported have been those of a minor upper respiratory tract infection. This would suggest that the infant would have a cough with some tacky mucus. If one would postulate that the sequence of events that lead to death would be that the infant coughs up a little mucus, and, as all infants do, attempts to swallow the mucus that reaches the pharynx. This results in closure of the laryngeal airway by the epiglottis, which becomes "gummed" down by some residual mucus, effectively blocking the next step, which would be an inspiration, thus forcing the epiglottis down further over the larynx. As the tidal air has been expelled in the process of coughing, there is no way for the infant, or even an adult, to make a forced expiration to force the epiglottis open. Death is rapid and silent—without a struggle from asphyxiation or vagal inhibition.

The rapid death leaves little time for morbid pathological changes to be found and in the process of handling the dead infant some residual air is forced from the lungs, opening the epiglottis and destroying the only evidence of cause of death.

If this hypothesis, which would account for the lack of any significant postmortem changes, is accepted, and it would appear to be a reasonably valid one, we have come to the reluctant conclusion that there is no solution to avert this tragic problem.—I am, etc.,

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Severe Malarial Infection

SIR,—The report from Ibadan entitled "Severe Malarial Infection in a Patient with Sickle-cell Anaemia" (22 May, p. 445) is by no means as unusual as the authors would appear to suggest. When working at Harari Hospital, Rhodesia, I encountered several cases of acute malaria (due to *P. falciparum*) in patients with sickle-cell disease. The diagnosis of sickling was confirmed by haemoglobin electrophoresis, and though I do not have the records at hand I can remember at least one child with sickle-cell anaemia who died in an acute attack of malaria. I know that other members of the medical staff there had the same experience.

We knew of course from the original observation of Beet,[1] and the work of Brain,[2] Raper,[3] [4] and others, of the increased resistance to malaria conferred by the sickling trait. However, it was our impression that malarial infections occurred in the homozygous state which were quite often severe, which could precipitate a crisis, and which could rapidly lead to death through dehydration, sequestration, and lysis of red cells. When presented with a "sickle-cell crisis," then, it was always our practice to disregard theoretical considerations of increased resistance to malaria and to make a search for the parasite.—I am, etc.,

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1 Beet, E. A., *East African Medical Journal*, 1946, **23**, 75.
2 Brain, P., *British Medical Journal*, 1952, **2**, 880.
3 Raper, A. B., *Journal of Tropical Medicine and Hygiene*, 1950, **53**, 49.
4 Raper, A. B., *British Medical Journal*, 1956, **1**, 965.

Humidity in Hospital

SIR,—Dr. J. R. R. Wray's letter (19 June, p. 708) drawing attention to excessively dry atmospheres in new hospitals and houses is timely. There is now much advertising of the latest heating methods, which produce even hotter and drier atmosphere, so that the relative humidity in both hospitals and houses is commonly down to 20-25% in cold weather. This low humidity at a room temperature of 70°F (22°C) causes severe drying of naturally moist surfaces. This is unpleasant for the healthy, and dangerous for the diseased, respiratory tract. Dr. Wray very rightly warns that cheap hygrometers are often unreliable. I suggest that *any* air hygrometer needs constant checking; the whirling wet bulb instrument is the one of choice.—I am, etc.,

DOUGLAS NEEDHAM

Aberdeen

Terminology

SIR,—What is the purpose of the word "disease" in the phrase "chronic obstructive airways disease" (5 June, p. 561)? Presumably this phrase is intended to refer to a group of patients with a specified disorder of function in the respiratory system, chronic airways obstruction. We have become accustomed to the use of phrases indicative of disorders of function in other systems (for example, systemic hypertension, intestinal obstruction, raised intracranial pressure, congestive heart failure) or even on the vascular side of the lungs themselves (pulmonary hypertension), without thinking it necessary to add "disease."

Why is it so generally thought necessary to add this word in referring to abnormally high resistance to gas-flow in the lungs? To my mind, in this context it is worse than useless; its presence distracts attention from the main purpose of the use of this sort of term, namely to refer to groups of patients characterized by a common disorder of function, without commitment to implications about aetiology, structural changes, or other characteristics.—I am, etc.,

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Multiple Eponyms

SIR,—I wonder whether Dr. T. J. David (12 June, p. 655) would accept for his record the description of craniofaciomandibular dyostosis syndrome by the Troquart-Apert-Franceschetti - Greig - Helmholz - Harrington - Marie - Sainton - Crouzon - Treacher - Collins syndrome. If not, perhaps he will allow me to add Böök-Hesselvik-Buckley-Yakovlev - Park - Powers - Jackson - Scott - Banks - Brown - Harper - Meisenbach - *et al.*—I am, etc.,

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Infantile Subdural Haematoma

SIR,—The interesting paper by Mr. A. N. Guthkelch (22 May, p. 430) is of particular concern to Home Office pathologists, for whom certain cases of fatal subdural haematoma in infancy have been a problem for some time. Such fatalities are best considered in the three main distinguishable categories.

(1) Those with obviously related head injury such as bruising or fractures or both.

(2) Those with injuries elsewhere on the body, but no significant injury to the head.

(3) Those with no significant injury anywhere.

The first category presents no problem in that the injuries clearly lead to death. The second category is more difficult, particularly if the other injuries are of long standing, but there should be no hesitation in attributing the fatal subdural haematoma to the general violence implicit when there are injuries of appropriate severity and duration to other parts of the body.

In the third category I have for some