fill this seat, the choice lies between a career grade officer, a general practitioner, or a hospital junior doctor with a number of house appointments behind him.

If staffing is to be entirely by career grade officers, then the minimum number is five and not four as suggested by Mr. F. C. Durbin and Mr. J. S. Batchelor (14 August, p. 432). To staff with only four would mean a 42-hour week without taking into account time for handing over between shifts, holidays, study leave, or sickness.

For these career grade officers, there is need for consultant pay without specialist connotation. A new name is needed and I suggest that of "senior emergency officer."—I am, etc.,

G. RIGBY-JONES

London W.1

### Prognosis in Industrial Dermatitis

SIR,—I was most interested in your leading article entitled "Prognosis in Industrial Dermatitis" (21 August, p. 445), but the comment that "the payment or not of 'compensation,' which it is assumed includes injury benefit, did not affect the prognosis" really surprised me, and recalled the results of my own investigation.[1] Having been involved with the (then) Ministry of National Insurance in London in matters concerning dermatology since its inception, I had been able to institute a long-term follow-up of a fair number of my cases of occupational dermatitis.

The investigation covered a series of 250 consecutive claimants, involving a wide variety of occupations, and in a follow-up of 139 of these cases (83 males and 56 females) my conclusions read "In every one of 139 cases followed-up from 1-4 years compensation for occupational dermatitis is being paid. Not one has recovered fully and been able to resume his original occupation without further trouble, and 'hardening' has not been in evidence. The vast majority of the females has never worked again but continues to draw compensation." "Compensitis" (acute, sub-acute, and chronic), was to me one of the most important factors in the prognosis at that time. Perhaps the great change in social security since those days has altered the attitude of the British working man, as, judging from your leading article, "fings ain't wot they used t'be."—I am, etc.,

B. BENTLEY-PHILLIPS

Faculty of Medicine,
University of Natal,
Durban, South Africa

1 Bentley-Phillips, B., *Practitioner*, 1954, **172**, 531. 1960, **75**, 766.

### Malignant Hyperpyrexia

SIR,—Your leading article "Malignant Hyperpyrexia" (21 August, p. 441) does not present the most recent information on this confused subject. We feel the most important question has yet to be answered—namely, the relevance of the muscle contracture which occurs in at least 70% of cases.[1] If the syndrome can develop without muscle contracture, is the contracture either a secondary phenomenon or an associated (but not causative) phenomenon?

We were surprised that your leader writer was unaware of the report[2] that halothane (and methoxyflurane) induced a reversible muscle contracture in specimens of myopathic muscle. It is interesting to note that Kalow and associates[1] failed to demonstrate a halothane-induced contracture in muscle taken from patients who had recovered from malignant hyperpyrexia, and we attribute this to the use of unphysiological temperatures (25°C) in their experiments.

More recent work in our department on a member of a known myopathic family has shown that procaine hydrochloride both reverses established halothane-induced muscle contracture and prevents its initiation. Thus Dr. G. G. Harrison's findings in pigs (21 August, p. 454) and our own with human muscle are complementary, and the rationale of the use of procaine in patients with malignant hyperpyrexia is confirmed. Although procaine is used clinically for its stabilizing action on cell membranes, the reason for proposing this drug for hyperpyrexic muscle contracture was because of its ability to block caffeine-induced muscle contracture,[3] probably by inhibiting calcium release by the sarcoplasmic reticulum. Calcium can also be accumulated by mitochondria[4] and the therapeutic value of procaine in malignant hyperpyrexia may be due to an intracellular action, stabilizing the mitochondrial membrane.

The use of isoprenaline in the treatment of hyperpyrexic patients, as mentioned in your leading article, is controversial. It is well known that isoprenaline increases cyclic AMP by stimulating adenyl cyclase, and Pollock and Watson,[5] quoted by you, have proposed a theory of the pathogenesis of malignant hyperpyrexia based on a rise in the intracellular concentration of cyclic AMP.

Finally, although it is impossible to define adequately the condition or possibly conditions of malignant hyperpyrexia before its aetiology is known, we regret the failure to attempt a "working definition" in your leading article. Many of the published cases do not appear to us to have been true examples of this syndrome, having developed only mild pyrexia during or even after anaesthesia. We believe that *malignant hyperpyrexia is a specific potentially fatal condition occurring during anaesthesia in which heat production exceeds physiological heat loss to an extent that causes a progressive rise of body temperature at a rate of at least 2°C per hour.* All other manifestations of the syndrome such as muscle contracture, hypoxia, acidosis, lactacidaemia, and cardiovascular collapse are not initially invariable and are probably secondary effects.—We are, etc.,

N. P. KEANEY
F. R. ELLIS

Department of Anaesthesia,
University of Leeds

1 Kalow, W., Britt, B. A., Terreau, M. E., and Haist, C., *Lancet*, 1970, **2**, 895.
2 Ellis, F. R., Keaney, N. P., Harriman, D. G. F., Kyei-Mensah, K., and Tyrrell, J. H., *British Journal of Anaesthesia*, 1971, **43**, 721.
3 Feinstein, M. B., *Journal of General Physiology*, 1963, **47**, 151.
4 Lehninger, A. L., *Biochemical Journal*, 1970, **119**, 129.
5 Pollock, R. A., and Watson, R. L., *Anaesthesiology*, 1971, **34**, 188.

### The New F.F.R.

SIR,—Dr. F. Pygott (28 August, p. 533) and Dr. C. G. Whiteside (11 September, p. 639) express the concern felt by radiologists at the new F.F.R. examination. The detrimental effect likely to be experienced as a result is described by Dr. Pygott. As radiology is today anything but a popular specialty with some 60 unfilled posts, this move seems likely to cause a further deterioration.

Is it not time that both the D.M.R.D. and F.F.R. were abandoned and replaced by a board qualification, such as in Canada, where recently the Fellowship has been abolished?

The British love of multiple diplomas is surely an expression of academic vulgarity and should be discouraged. A further satisfactory alternative is to use the Edinburgh membership or the M.D. with radiology as the qualification.—I am, etc.,

GEOFFREY MEDHURST

Kettering,
Northants

### Cyclophosphamide and the Bladder

SIR,—Another, quite different, aspect of your leading article on "Cyclophosphamide and the Bladder" (26 June, p. 726) may be of interest to those faced with a similar problem—namely, the direct intravesical instillation of cyclophosphamide in the palliative treatment of carcinoma of the bladder. At a recent follow-up examination of a patient with inoperable carcinoma of the bladder treated by regular instillation of cyclophosphamide there was no evidence of advance of the tumour during the 2½ years' treatment with the drug, and during this time the patient has been leading an active worthwhile life free of symptoms in his own home.

A 58-year-old man with inoperable carcinoma of the bladder was admitted for terminal care in May 1969. He was bleeding profusely from the bladder and had severe frequency and dysuria. There was no evidence of secondary spread of the tumour. He was grossly anaemic with a haemoglobin of 3 g/100 ml. On admission he was given 4 pints (2·3 l.) of blood. At the same time twice daily instillations of 1 g of cyclophosphamide in 50 ml of saline were started.

The haematuria lessened within a few days and soon ceased altogether. Within three months all symptoms of dysuria and frequency disappeared. The interval between the instillation was increased to one week. For the past two years the patient has been looking after himself at home, cycling to hospital once a week for treatment. Six months after treatment a cystoscopy showed absence of cystitis and bleeding, and a little sloughing of the tumour, which the surgeon thought was probably a little smaller in size.

At follow up in June 1971 his haemoglobin was 14·3 g/100 ml and white cell count 8,500 mm³. He was feeling very well and had no urinary symptoms. At cystoscopy the appearances of the tumour were almost identical with those found in February 1968 with an extensive carcinoma, involving the neck and whole of the left side of the bladder. Bladder capacity in June 1971 was 4-5 oz (110-140 ml).

Though there had been no regression of the tumour in this period of over three years the patient has had complete remission of his symptoms for the last 2½ years, and remains in ignorance of the true nature of his complaint. The only other treatment he