the experience of a sighted person. Blind-folding will give some idea of the physical disabilities of the blind but no idea of the heightening of his other senses which he may eventually employ. It cannot reproduce the emotional background. We are so used to "eye to eye" relationships with our fellows that we inevitably feel insecure at our first meeting with someone with whom this is impossible.

As for teaching, I do not foresee any improvement in the situation as less and less time becomes available for specialized teaching of this sort. The task of equipping the family doctor to anticipate the odd ophthalmic disaster is bound to take precedence over training him for all but the most rudimentary aspects of care and rehabilitation of the blind and partially-sighted. Nevertheless, the family practitioner must have confident recourse to others who can supply this expertise, and such others will be grateful to Dr. Shaw for helping their understanding.—I am, etc.,

W. J. DINNING

London N.6

Vitamin D and Myocardial Infarction

SIR,—It was most interesting to read in Professor V. Lindén's article (14 September, p. 647) that according to his investigations the consumption of vitamin D was significantly higher in infarction patients than in control subjects.

As Professor Lindén points out, fish liver is the natural source of vitamin D in Norway. In Britain the main source is egg yolk, one of the few natural foods in the diet containing vitamin D in quantity. My research on the intake of eggs in the United Kingdom¹ shows a rapid rise in consumption since 1920, apart from short periods of relative deprivation during the 1939-45 war. I have demonstrated that the rise in egg consumption closely follows the rise in mortality from coronary heart disease, and though egg yolk is also a rich source of cholesterol it may be that the presence of vitamin D in the yolk is an added potent factor associated with coronary heart disease as highlighted by my investigations.

While the aetiology of coronary heart disease is considered to be multifactorial, the potential danger of consuming eggs in quantity should not be overlooked.—I am, etc.,

F. ESKIN

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Barnsley Area Health Authority,
Barnsley

¹ Eskin, F., *Community Health*, 1971, 2, 179.

Glucagon in Management of Perforated Oesophagus

SIR,—I read with interest the paper by Mr. O. Daniel and his colleagues (21 September, p. 720) on the use of glucagon in the treatment of acute diverticulitis. I have used it in the management of iatrogenic rupture of the oesophagus in elderly, bad risk patients. Many of these patients are unacceptable candidates for major surgery and any agent that may assist in the conservative management of the case is of interest.

Glucagon is a potent suppressor of gastric secretion.¹ Since the initial mediastinitis after

perforation of the gullet must be largely due to chemical irritation, any agent that reduces both the quantity and acidity of gastric secretion might be expected to be of benefit. Furthermore, there is evidence that glucagon diminishes gastric motility and also has an antispasmodic effect on the lower oesophageal sphincter.² Reducing motility in the upper gastrointestinal tract should diminish the amount of gastro-oesophageal contents expelled through a perforation.

On this theoretical basis I gave glucagon to three elderly patients with perforated oesophagus. A subclavian central venous line was inserted immediately the diagnosis was made and oral fluids were withheld. Cephalothin was administered in bolus doses of 3 g every six hours and glucagon was given in an initial dose of 1 mg over five minutes and subsequently at a rate of 2 mg every six hours by continuous infusion. Glucagon was continued until a Gastrografin swallow indicated that the perforation was sealed off. This occurred within four days in one case and within five days in the other two cases. A noteworthy feature of these three cases was that at no time was it necessary to drain the chest. Only minimal amounts of fluid were detected in the pleural cavities.

In the light of these results I suggest that when it has been decided to treat a case of lower oesophageal perforation conservatively glucagon may well contribute to therapeutic success.—I am, etc.,

ROBERT PICKARD

St. George's Hospital,
London S.W.17

¹ Dotevall, G., and Kock, N. G., *Gastroenterology*, 1963, 45, 364.

² Jaffer, S. S., et al., *Gastroenterology*, 1974, 67, 42.

Periodic Fever

SIR,—At what stage does one stop investigating a patient with recurring bouts of pyrexia and persuade him instead to accept a "diagnosis" of "Periodic Fever"?

A 65-year-old man has suffered from recurring bouts of fever for four years. The temperature rises to 38°-39°C for 24 hours every five days, with aching in the loins and varying degrees of prostration. All investigations have been negative and there has been no response to antibiotics or to steroids. A chance finding was that the expected spike of fever failed to appear on three occasions following administration of tetanus toxoid. Careful monitoring of temperature has shown accentuation of fever every 31 days, suggesting that the underlying rhythm is 124 hours, coinciding every 31 days with the normal circadian temperature rise. A pint (0.6 l.) of blood, removed at the height of the fever, produced a fever when retransfused on the third day, suggesting the presence of a circulating pyrogen.

Reimann and McCloskey,¹ in a recent report of a case of 23-day fever and mentioning a further instance of 14-day fever, make a plea for the recognition of periodic disorders due to unknown biorhythms and the avoidance thereby of the unnecessary expense and suffering of prolonged and fruitless investigation.

The purpose of this letter is twofold. Firstly, to seek help: I still feel the need to be aware of the possibility of a microbial cause for fever of five-day rhythm and, if there is one, it may be that some of your erudite readers have knowledge of it. Secondly, to bring to more general notice this extremely trying recurring disorder.

To give the term periodic fever a capital P and a capital F does not of course elevate

it to the status of a diagnosis. It may, however, draw attention to a problem still very much in need of elucidation.—I am, etc.,

T. H. BARRIE

Sherborne, Dorset

¹ Reimann, H. A., and McCloskey, R. V., *Journal of the American Medical Association*, 1974, 228, 1662.

Non-union of the Scaphoid

SIR,—Your leading article (5 October, p. 2) presents a well-balanced and thoughtful appraisal of the problems associated with non-union of the scaphoid. There are, however, two points which merit comment.

While grafting for non-union by the anterior approach has many adherents, it has the disadvantages of not achieving as rigid fixation as is possible by a carefully placed peg graft insertion via the posterolateral approach. This approach is objected to on the grounds of causing possible damage to the blood supply but is there any evidence to indicate that avascular necrosis or other complications have resulted from the use of this approach for the treatment of established non-union? The complication of avascular necrosis of the proximal fragment and semilunar, which is associated very particularly with varying degrees of perilunar transscaphoid dislocation, is not greater following open than closed reduction.

The second comment is that a careful excision of the whole scaphoid has proved a satisfactory procedure in the treatment of late un-united fractures, particularly those associated with osteoarthritic change. It should be emphasized, however, that if through some technical difficulty it is possible to remove only one fragment this should most certainly be the distal fragment, which is the portion affected by secondary arthritic change, the proximal fragment being in no danger in this respect. In all these operations it is certainly highly desirable to remove the distal $\frac{1}{2}$ in (8.5 mm) or so of the styloid process of the radius, but wide excision of the styloid process can affect stability.—I am, etc.,

F. C. DWYER

Liverpool

Management of Lithium Treatment

SIR,—I feel that in their paper on "Blood Levels and Management of Lithium Treatment" (4 September, p. 650) Dr. J. L. Crammer and his colleagues misrepresent the case as regards the diurnal variations in serum lithium levels. The evidence we have acquired from 23 experiments on patients stabilized for a minimum of 10 weeks on each of the three lithium carbonate preparations on the market is that following each dose as lithium is absorbed from the gastrointestinal tract there is a corresponding rise in the serum lithium level lasting for about two to four hours. Thereafter the level gradually declines over the interdose interval as lithium is eliminated through the kidneys. In the stabilized patient lithium equilibrium is maintained through the daily dose making good the renal lithium loss. The highest serum level found in 200 serum samples was 1.8 mmol/l. and in the vast majority of subjects the level did not fall outside the range 0.5-1.5 mmol/l. at any time during

the 24 hours. We did not find the variation in serum lithium level to be "quite outside the therapeutic range" and I feel that statements such as that "the lithium level fluctuates greatly during the 24 hours" are misleading and alarmist. I agree that it is important to realize that serum lithium is not at a constant level throughout but, rises and declines as described, and that it is necessary to know the relationship between the time of the blood sample and the last medication when interpreting the biochemistry result. The usual practice is to take the blood sample prior to the next dose.

Finally, I would dispute the statement that "a basal level less than 2.0 mmol/l. lithium remains an empirical safety rule." Schou¹ has stated that levels over 1.3 mmol/l. should be reported as soon as possible. As the authors point out, the post-absorption level can be 2.5 times the basal level. Thus to accept a resting level of less than 2.0 mmol/l. means that for part of the day the serum lithium level can be in excess of 4.0 mmol/l. The risk of lithium accumulation and consequent severe toxicity is great at these serum levels and we would advise that the basal lithium level should not exceed 1.3 mmol/l.—I am, etc.,

ERNEST H. BENNIE

Leverdale Hospital,
Glasgow

¹ Schou, M., Amdisen, A., and Baastrup, P. C. *British Journal of Hospital Medicine*, 1971, 6, 53.

Side Effects of Lithium Carbonate

SIR,—Dr. J. E. Duffield (24 February 1973, p. 491) will be interested to know that we have made precisely the same observations regarding lithium's effect on the taste of butterfat.

In a group of over 450 people treated with lithium carbonate approximately one out of 20 patients complains that butter and other dairy products with butterfat taste spoiled and that the spoiled taste disappears three days after they stop taking lithium. Even more patients note an alteration in the taste of celery and many in our clinic have sworn off celery sticks, chow mein, chop suey, etc.

We have been so impressed by lithium-related alterations in gustation that we have begun a large clinical and laboratory study. One of us (I.H.) has already shown that in rats lithium is concentrated in the olfactory bulbs, caudate, and pituitary to a greater extent than that observed in other parts of the brain. This accumulation in the olfactory bulbs could conceivably be responsible for taste distortion in our patients.—We are, etc.,

JONATHAN M. HIMMELHOCH
ISRAEL HANIN

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Western Psychiatric Institute and Clinic,
Pittsburgh, Pennsylvania

Oedema of Mountains

SIR,—Recent experience of mine and of others shows that the term pulmonary oedema of mountains¹ is far too narrow and that oedema of mountains affects other organs as much as the lungs. I was in a

recent expedition to the Hindu Kush mountains, Afghanistan. Including two other expeditions at the same base, whom I helped, there were some 30 climbers attempting mountains up to a height of 24,500 ft (7,448 m). Two cases of oedema of mountains occurred.

In the first a fit man aged 45 climbed to 18,000 ft (5,472 m) on the third day of arriving at the base camp, which was at 16,000 ft (4,864 m). He returned with some malaise and dyspnoea and with definite swelling of the face, hands, and ankles accompanied by severe oliguria. The signs disappeared quickly after an oral dose of bumetanide 2 mg but recurred on two further occasions, including one ascent to 22,000 ft (6,688 m), accompanied by much more severe malaise. After another week's rest at base camp he managed to climb slowly to 22,000 ft without incident. This was perhaps a renal form of oedema.

A second man aged 41 remained six days at 22,000 ft because of storms. On the sixth day he appeared drowsy and complained of headache. He remained in bed for a few hours, when it became obvious to his friends that he was severely ill and in coma. I met him at 20,000 ft (6,080 m), when he had already begun to improve a little. He was stuporose and had definite signs of paraplegia, with very brisk reflexes and bilateral extensor plantar responses. There was slight oedema of the face and hands, and apparently he had passed hardly any urine during the previous 24 hours. He improved quickly with an intravenous dose of 1 mg bumetanide and was brought down to base camp the same day. Over the next few days he rapidly improved and the findings on physical examination became quite normal. Unfortunately, no ophthalmoscope was available. This was certainly a cerebral form.

In both patients a few late inspiratory crackles were present without other cardiorespiratory signs. No other case of oedema of mountains occurred.

Thus the proper term for this singular illness of high altitudes is "oedema of mountains." Others have also drawn attention to involvement of the brain or eye in this illness as well as the lungs.^{2,4} Climbers and doctors on expeditions should be aware of this, while theories of pathogenesis directed entirely to pulmonary explanations are far too narrow. The oliguria and the swelling of the face and hands others and I^{5,6} noted suggest that the kidney may be an important factor, and this is being investigated.—I am, etc.,

A. PINES

East Herts Hospital,
Hertford, Herts

¹ *British Medical Journal*, 1972, 3, 65.

² Singh, I., et al., *New England Journal of Medicine*, 1969, 280, 175.

³ Wilson, R., *Annals of Internal Medicine*, 1973, 78, 421.

⁴ Radford, P., *British Medical Journal*, 1973, 3, 294.

⁵ Sheridan, J. W., and Sheridan, R., *Lancet*, 1970, 1, 242.

⁶ Pines, A., *British Medical Journal*, 1972, 3, 642.

Screening for Hypertension

SIR,—Dr. D. S. Short (12 October, p. 103) points out that in recording the diastolic arterial pressure the fourth phase represents the correct reading. I suggest, however, that in practice the recognition of this phase may be difficult. I have seen a number of cases of healthy persons penalized for life assurance on account of an allegedly high diastolic pressure (and apparently abnormal pulse pressure) erroneously recorded at the fourth phase. The fifth phase is unmistakable and therefore more reliable, provided one realizes that it is 4 or 5 mm Hg below the true diastolic pressure.—I am, etc.,

T. W. PRESTON

Reigate, Surrey

Economies in the N.H.S.

SIR,—I agree entirely with the comments of Dr. H. P. Hughes concerning unnecessary procedures and investigations (5 October, p. 41). Apart from x-ray investigations, there are multitudes of unnecessary pathological tests requested daily. The result of this blunderbuss approach is certainly a gross wastage of staff time and millions of pounds annually.

The medicolegal aspect is one side of the matter. The other side is simply a question of conscience with regard to a discriminatory approach to all investigations. It is a pity that the cost of individual investigations does not receive more publicity. I feel that a greater sense of awareness by the medical profession on this matter would produce tremendous savings—savings which could be used to modernize equipment generally and thus to help the very people who are investigating the unnecessary procedures.

If only they realized they were cutting their own throats!—I am, etc.,

A. R. MANNERS

Stockton-on-Tees

N.H.S. Contraceptive Services

SIR,—It was with extreme disquiet that I read the letter from Dr. R. W. Green (12 October, p. 109) on the subject of fees for contraceptive care. He considered it a fee of £1.56 was "money for old rope." This I find completely incredible.

Many general practitioners would not share his view that pelvic, blood pressure, breast examinations, etc. should be undertaken by a doctor only on the initial visit by the patient and that subsequent follow-up can, in most cases, be satisfactorily carried out by a practice nurse. I for one do not consider that a practice nurse is competent to decide the significance of a "pill erosion" and what advice this entails, nor the significance of glycosuria, nor what early signs of excessive oestrogen build-up are to be looked for, to name but a few complications commonly seen at follow-up.

To suggest that £1.56 is "money for old rope" for this kind of care and supervision is, to my mind, utterly contemptible and insulting to his colleagues, and plays into the hands of politicians who are only too happy to get medical care on the cheap.—I am, etc.,

MICHAEL J. EMSLIE

Eastbourne

SIR,—I read with increasing incredulity Dr. R. W. Green's personal views on the recommended rate for the yearly Pill prescription (12 October, p. 109); in fact I had to re-read his letter to believe my eyes.

Needless to say I am not among those who believe that £1.56 a year is "money for old rope" and sincerely hope that this type of divisive opinion, coming at a time when the profession as a whole is surely fighting not to improve its lot, but merely to maintain it, will not deter our negotiators from demanding a sensible fee for yet another item of service.

Let those who accept a fee of £1.56 take it. I will reject it and give family planning a miss.—I am, etc.,

JOHN P. LEWIN

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