ted in the first few days of the disease in adequate doses four-hourly, is accompanied in the first 4-6 hours by a characteristic reaction which we termed the diagnostic penicillin-leptospirosis response.²

The highest concentration of leptospirosis is in Malaysia and Indonesia.⁵ Nevertheless, the presence of leptospirosis in Malaya, now Western Malaysia, was first noted by Fletcher in 1928.⁶ Yet in the 1950s it was reported that in the British armed Forces in Malaya 35% of all fevers, excluding malaria, were due to leptospirosis.⁷

I suggest that the number of cases of leptospirosis now being diagnosed in the United Kingdom at present represents only the tip of the iceberg if 85% or so of all cases are mild to moderately severe, and misdiagnosed as they were in Malaya in Fletcher's time and much later. The many confusing manifestations of this disease, or leptospirosis syndromes as they have been called, have been repeatedly stressed.^{1,2,8,9}

I believe that leptospirosis in the United Kingdom and the Republic of Ireland will be diagnosed more frequently in the future when, in febrile illnesses of apparently indeterminate origin, the overall history includes, in every case, not only the occupational history but a history of contact with cattle, pigs, dogs, rats, mice, and voles as well as with soil and water polluted with their infected urine. When such a history becomes routine there is bound to be an increasing awareness of leptospirosis in the United Kingdom. With that there will be earlier provisional diagnosis of leptospirosis, with the immediate early institution of adequate parenteral penicillin four-hourly for the first 24 hours and thereafter six-hourly. When such treatment begins in the first few days of the illness there is the inevitable appearance of the diagnostic penicillin-leptospirosis response; such is my experience and that of my colleagues.—I am, etc.,

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- 1 Mackay-Dick, J., and Watts, R. W. E., *Lancet*, 1919, 1, 907.
- 2 Mackay-Dick, J., and Robinson, J. F., *Journal of the Royal Army Medical Corps*, 1957, 103, 186.
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- 6 Fletcher, W., *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 1928, 21, 265.
- 7 McCrumb, F. R., Jun. et al., *American Journal of Tropical Medicine and Hygiene*, 1957, 6, 238.
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Adrenal Failure in Bronchial Asthma

SIR,—We appreciate that there are problems associated with oral corticosteroid reduction and the substitution of beclomethasone in the treatment of asthma, but we think that the dangers have been misrepresented and hope that this will not prevent the continued use of a promising form of treatment, whose main advantage is that it permits reduction of the dose of oral corticosteroids.

The real danger is that beclomethasone may not be so effective as oral corticosteroids and that the asthma may relapse, a risk that arises whenever effective treatment is withdrawn in any disease. This risk is present

regardless of hypothalamo-pituitary-adrenal (H.P.A.) function.

Drs. Ruth M. Clayton and P. Howard (2 June, p. 547) suggest that corticosteroid reduction exposes the patient to the possibility of failure of the adrenal to respond to stress, but this risk is already present in any patient who has H.P.A. suppression, and could be guarded against only by continually providing corticosteroids in a quantity equivalent to the maximal output of the normal adrenal—300-400 mg of hydrocortisone daily—which is obviously absurd. If the actual dose is reduced from 15 to 7.5 mg prednisolone daily and severe stress should occur either from status asthmaticus or some unrelated condition, high-dose replacement would be necessary and it would make little difference whether the patient was receiving 15 mg or 7.5 mg, as both of these are inadequate for stress replacement. The higher dose, however, will perpetuate H.P.A. suppression, whereas the lower dose may allow some function to return. If gradual reduction and withdrawal is possible, H.P.A. function will return to normal, often by the time the steroid is completely withdrawn or within a short time afterwards.^{1,2} Thus continuation of the higher dose actually increases the risk of adrenal failure.

It is difficult to draw any conclusion from the case quoted by Drs. Clayton and Howard in which the patient died within six hours of onset of a mysterious illness. Adrenal failure does not usually lead to such rapid death, and we do not believe that an extra 7.5 mg of prednisolone would have significantly altered the outcome.

We agree with Dr. A. O. Robson (30 June, p. 781) that the response to tetracosactrin has a loose correlation with the response to stress, but failure of the upper end of the H.P.A. axis is present in a significant proportion of corticosteroid-treated patients in whom the response to tetracosactrin is normal.^{2,3} The insulin hypoglycaemia test is the only adequate way of testing the integrity of the whole axis, and should be used if worthwhile conclusions are to be drawn.⁴—We are, etc.,

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- 1 Plumpton, F. S., Besser, G. M., and Cole, P. V., *Anaesthesia*, 1969, 24, 3.
- 2 Daly, J. R., Myles, A. B., Bacon, P. A., Beardwell, C. G., and Savage, O., *Annals of the Rheumatic Diseases*, 1967, 26, 18.
- 3 Jasan, M. K., et al., *Quarterly Journal of Medicine*, 1967, 36, 261.
- 4 Jacobs, H. S., and Nabarro, J. D. N., *Quarterly Journal of Medicine*, 1969, 38, 475.

Mediterranean Anaemia in Antiquity

SIR,—Dr. W. T. Menke's interesting case from the Hippocratic collection (26 May, p. 489) was not, I think, one of sickle-cell thalassaemia. Lehmann and Cutbush¹ suggested 20 years ago that the sickle cell had an Indian origin and came west comparatively recently, and I² put forward the view that its distribution in Africa might be explained by a migration during the Christian era which also left behind the shorthorn zebu breed of cattle. If this is so, there were not many sickle-cell genes in the Aegean in

the period during which the Hippocratic corpus was composed. The case may well have been one simply of thalassaemia, in which the spleen is characteristically large and leg ulcers, though not the rule, do sometimes occur. But the Greeks, I am sure, would have had a different word for it; the term thalassaemia ought surely to apply to the dilution of the blood with sea-water, such as occurs—for all I know—in those drowned at sea.—I am, etc.,

PETER BRAIN

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- 1 Lehmann, H., and Cutbush, M., *British Medical Journal*, 1952, 1, 404.
- 2 Brain, P., *Man*, 1953, 53, 154.

Hazards of Laparoscopy

SIR,—I was interested to read Mr. M. McD. Usherwood's account of the breakage of Palmer's diathermy forceps (30 June, p. 773). I have had the same problem during a laparoscopic sterilization. Another pair of Palmer's forceps was not immediately available, but fortunately it was possible to remove the broken claw of the forceps by means of sigmoidoscopy biopsy forceps through the cannula, and after a short interval the sterilization was completed by the laparoscopic method.

The forceps were almost new, and I wonder if these were isolated incidents or whether some of the current instruments available are not strong enough for this purpose.—I am, etc.,

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The Suspended Retractor

SIR,—The deep-chested patient who needs a vagotomy and the patient with a high, small liver and adherent gall bladder present exposure problems which are very much helped by the suspended retractor. With the aid of the normal lithotomy poles and cross-bar, a Doyen retractor lifts up the chest cage and is suspended by tape from the cross-bar with a small Durham retractor.

This technique was developed by Mr. G. Dimopolous, of Durban. I have found it extremely valuable but have failed to secure the interest of manufacturers. I am therefore writing in the hope that surgeons will try it out—their assistants will be grateful and perhaps for the first time will be able to see for themselves what is going on, while the anaesthetist will raise no objection at all.—I am, etc.,

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High-altitude Oedema Presenting as Coma

SIR,—The syndrome of high-altitude oedema has been increasingly documented over the past 10 years.¹⁻⁶ Typically the disease presents with respiratory symptoms following on from severe mountain sickness. Cerebral forms are recorded, but according to Singh et al.,¹ unconsciousness takes days to appear. The patient recorded by Foster⁵ was unconscious, but this was in terminal pulmonary oedema. Pine's patient⁶ became comatose, but details of timing are not recorded.