Unquiet Rest

SIR,—Chauvinism in medical research is to be deplored, but your leading article on unquiet rest (23 September, p. 715) does less than justice to the pioneering British work of Masterton[1] on sleep patterns of junior medical staff. As early as 1965 he drew attention to both the lack of sleep obtained by busy residents on a surgical service and the effects this could have on the feelings they had about themselves and their work. The experiments of Friedman and his colleagues[2] referred to in your leading article now confirm objectively for medical performance what has been known for some time in other fields—namely, that in prolonged intellectual tasks ability declines with acute sleep lack.[3] However, doctors are not yet sufficiently impressed with this to order their lives properly and, as Masterton showed, the pressure of external affairs in a hospital environment may be the overriding factor which produces the sleep lack state.

The lack of sleep in intensive care units has also been recognized since 1964, when Edgerton and Kay[4] and Kornfield, Zimberg, and Malm[5] first postulated that it might be of importance in the acute delirium-like states that can occur during incarceration in the intensive care environment. This hypothesis, which was extended by one of us (Dudley[6]) to include other problems of sensory barrage and deprivation, has proved somewhat too simplistic.

While by definition we can regard sleep lack as bad and from observation recognize that it may contribute to the patient's disturbed convalescence, its aetiological importance in delirium and its influence on the physiology of recovery require much further study.—We are, etc.,

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1 Masterton, J. P., *Lancet*, 1965, **1**, 41.
2 Friedman, R. C., Bigger, J. T., and Kornfeld, D. S., *New England Journal of Medicine*, 1971, **285**, 201.
3 Wilkinson, R. T., *Proceedings of the Royal Society of Medicine*, 1969, **62**, 903.
4 Egerton, N., and Kay, J. H., *British Journal of Psychiatry*, 1964, **110**, 433.
5 Kornfeld, D. S., Zimberg, S., and Malm. J. R., *New England Journal of Medicine*, 1965, **273**, 287.
6 Dudley H., *Journal of the Royal College of Surgeons of Elinburgh*, 1968, **13**, 1.

Success of Adoption

SIR,—Dr. J. E. G. Vincenzi (30 September, 831) states that 51 out of 1,000 children referred to a local authority child guidance clinic were adopted. This incidence of 5·1% is notably higher than that of 2·9% in a survey of about 1,600 referrals to a children's neuropsychiatric unit.[1] Moreover, he gives no information on the proportion of these children adopted after the age of 6 months. Preadoptive trauma or lack of opportunity to form normal social attachments can lead to maladjustment, which cannot then be attributed to the child's adoptive status.

It is possible indeed that *Growing Up Adopted*[2] gives an over-optimistic view of adoption, and it is certain that the situation of the illegitimate child kept by his unmarried mother would be improved by better social support. However, Dr. Kellmer Pringle and her colleagues have been careful to point out, both in this report and elsewhere,[3] that evidence from biased samples of the kind collected by psychiatrists is inherently suspect.—I am, etc.,

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1 Humphrey, M., and Ounsted, C., *British Journal of Psychiatry*, 1963, **109**, 599.
2 Seglow, J., Kellmer-Pringle, M., and Wedge, P., *Growing Up Adopted*. National Foundation for Educational Research in England and Wales, 1972.
3 Pringle, M. L. K., *Adoption—Facts and Fallacies*. London, Longmans, 1967.

Sir,—Dr J. E. G. Vincenzi's warning (30 September, p. 831) against an overoptimistic view of the success rate in adoption is not borne out by our own national findings,[1] even by the criterion of referral to child guidance clinics. There is no statistically significant difference between the adopted and the rest of the national cohort of children in the proportion who attended such clinics.

If some clinical studies find a higher proportion of adopted children among their referrals does this really warrant Dr. Vincenzi's assumption "that there is in these children a genetically determined maturational defect"? I would suggest that there are simpler explanations for any such phenomenon. Perhaps I could mention just a few. Adopted parents have already had some dealings with the social and medical services in connexion with their adopted child and will usually be conscious of any preplacement handicaps, so may well be more ready to seek specialist advice. Because the myth of "bad blood" and of the adverse effects of poor heredity is so prevalent adoptive parents may be more prone than parents generally to think they can see signs of instability. Also adopters may be less reluctant to admit to behaviour difficulties in their adopted children, feeling that "heredity" rather than they themselves may be responsible. And a similar process of rationalization may make teachers, doctors, and social workers more ready to refer the adopted child for psychiatric advice.

Thus it seems to me that whatever arguments there might be for or against adoption it is unwise to cite the referral rate to psychiatric services as a "warning." All studies of adopted children have shown that the kind of home the parents provide and the kind of care they bestow are the most important preconditions for a satisfactory outcome for all concerned.— I am, etc.,

MIA KELLMER PRINGLE

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1 Seglow, J., Kellmer Pringle, M., and Wedge, P., *Growing Up Adopted*. National Foundation for Educational Research in England and Wales, 1972.

Scrotal Cancer Continues

SIR,—Your leading article on scrotal cancer (7 October, p. 3) stands in need of correction on one point. Percivall Pott died in 1788, so it was not he who described the case of epithelioma on the wrist of a gardener in 1808. This case was added in a footnote to Pott's original description of cancer scroti by James Earle, who edited the posthumous editions of Pott's book of surgery.[1] It was one of two cases which Earle mentioned in the note, both designed to illustrate the dangerous nature of soot. In addition to the account of the gardener Earle recounts a case of scrotal cancer in a man who was not a sweep but who had lodged for some years in a sweep's house in the room where the bags of soot were stored. He developed signs of the disease only after he had moved out into more pleasant lodgings.

Earle's reason for adding these cases was, he said, because "Mr. Pott seemed to suppose that this species of cancer was peculiar to chimney-sweepers; but I have strong grounds for thinking that he was mistaken in that idea." And so he was.—I am, etc.,

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1 *The Chirurgical Works of Percival Pott*, a new edition to which are added occasional notes and observations by Sir James Earle, Vol. 3, p. 182. London, J. Johnson, 1808.

Malaria in the U.K.

SIR,—I refer to the most unsatisfactory comments made by your correspondents from the Public Health Laboratory Service on the subject of Dr. M. J. Colbourne's letter (14 October, p. 112).

The original statement (9 September, p. 652) baldly states that "so far as is known there are no drug-resistant stains [*P. falciparum*] in Africa." This is an obviously incorrect statement so why not admit it and explain that "when discussing resistance we were referring to treatment and not prophylaxis"? Dr. Colbourne was perfectly justified in drawing attention to this erroneous and even misleading statement which could well have led to wrong advice being given about malarial prophylactic drugs in Africa.

The report by Sagnat and others[1] refers to a series of 45 comatose children suffering from malaria and admitted to a paediatric hospital in Brazzaville. It can hardly be accepted as epidemiological evidence on which to base a "figure of 35% for the death rate in *P. falciparum* infections in West Africa"—in my experience this is far too high.—I am, etc.,

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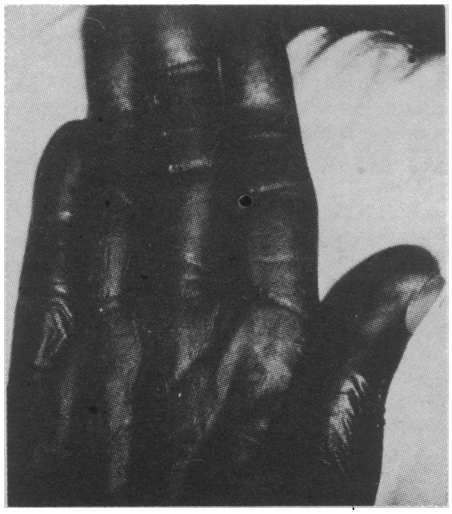
1 Sagnat, H., Morineaud, J. P., Revil, H., Thomas, J., and Masart, Y., *Médecine Tropicale*, 1967, **27**, 606.

Pigmentation in Megaloblastic Anaemia

SIR,—Hyperpigmentation of the skin of the palms, over the interphalangeal joints and terminal phalanges of the soles of the feet, and of the buccal mucous membranes has been described in vitamin B_{12}-deficient Indian adults and infants[1] and Nigerian adults.[2] Less pronounced but similar changes were reported in folate-deficient South African women in the postnatal period,[3] but in a previous note[4] it was denied that hyperpigmentation was ever seen in folate-deficient pregnant Nigerians. It has been suggested that this is a physical sign specific to vitamin B_{12} deficiency.[1] We wish to report a Nigerian woman with striking hyperpigmentation of the soles and palms (Figure), though not of the buccal mucosa, associated with megaloblastic anaemia and folate deficiency.

The patient was a Hausa woman, aged 50, living in a village in the savanna zone near Mallumfashi, North Central State, Nigeria. She complained of fever and dizziness for six weeks, weakness so that she was unable to walk more than about half a mile (805 m), and swelling of the feet for one week. She admitted to two pregnancies, with both children alive, the younger being about 12 years old. She gave no history of any previous illness. She appeared well nourished (weight 37·5 kg) but severely anaemic, with oedema of the feet and hyperpigmentation of both palms and soles (Fig. 1). Her haemoglobin



(Hb) concentration was 4·5 g/100 ml, with anisocytosis, both macrocytosis and microcytosis; polychromasia; hypochromia; and nucleated red cells in the peripheral blood smear. Bone marrow smears showed megaloblastic erythropoiesis, giant metamyelocytes, and stainable intracellular iron plentiful in the reticuloendothelial cells but not in the erythroid precursors. The total white cell count was 6,900/μl, with a normal differential count but hypersegmentation of neutrophil polymorphs; Hb electrophoretic pattern was AA, the serum vitamin B_{12} concentration was 1,000 pg/ml (Professor B. O. Osuntokun, University of Ibadan), but the serum and red-cell folate activities were not estimated as the patient had received oral folic acid before specimens could be collected. A histamine test meal showed no free acid and the total acid was 3·0 mEq/l. Skin tissue for biopsy was not taken until the eleventh day after admission, when the patient was showing a haematological remission following folic acid therapy. The skin colour had not faded by this time and the section showed hyperkeratotic squamous epithelium with numerous pigment-containing melanophores in the basal layer. There was no other histological difference from normal pigmented skin.

The patient received oral folic acid 5 mg/day for two days before the possibility of vitamin B_{12} deficiency was considered and this therapy stopped. She had a reticulocyte response reaching a maximum of 15% on the fifth day; the Hb began to rise from the eighth day to reach 10·5 g/100 ml on day 21 and 11·5 g/100 ml on day 27. Cyanocobalamin 1μg/day was given intramuscularly from day 21 to 29, but there was no further reticulocyte response. She received ferrous sulphate 200 mg three times a day for 29 days; she took the normal ward diet. She was discharged from hospital on day 29 with a supply of oral folic acid but was not seen again. The pigmentation had faded and her palms and soles were normal.

Investigations were far from complete in this patient, but vitamin B_{12} deficiency was excluded by bioassay and she showed a complete haematological remission after oral folic acid. It may be concluded that her megaloblastic anaemia and skin hyperpigmentation were both the result of folate deficiency. No cause of the deficiency was discovered, but the presence of hypochromic microcytes and intracellular iron in the histiocytes but not in the red cell precursors suggested that there was an underlying chronic inflammatory process.

Vitamin B_{12} deficiency is usually more long-standing than folate deficiency at the time of diagnosis. It may be that skin pigmentation is a reflection of chronicity of megaloblastosis rather than specific to vitamin B_{12} deficiency, and this would explain why folate-deficient South Africans show the sign in the late postnatal period while Nigerians do not when they are rapidly depleted by the demands of malarial haemolysis and pregnancy. The present patient was likely to have had a long-standing deficiency. Patients with sickle cell anaemia are sometimes deficient in folate for long periods before diagnosis, and it will be interesting to hear whether this sign has been noticed in these individuals.

The mechanism of hyperpigmentation remains obscure. Attempts to demonstrate a disturbance of melanin synthesis have not been helpful.[5-7] Alternatively, it could be a manifestation of altered cell division, which had reverted to normal by the time the skin was examined histologically in the present subject.

We thank Professor E. H. O. Parry and Dr. N. McD. Davidson for permission to publish details of this patient, who was under their care.—We are, etc.,

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1 Baker, S. J., Ignatius, M., Johnson, S., and Vaish, S. K., *British Medical Journal*, 1963, 1, 1713.
2 Watson-Williams, E. J., and Fleming, A. F., *Blood*, 1966, 28, 770.
3 Baumslag, N., and Metz, J., *British Medical Journal*, 1969, 2, 737.
4 Fleming, A. F., and Ive, F. A., *British Medical Journal*, 1969, 3, 238.
5 Fleming, A. F., and Broquist, H. P., *American Journal of Clinical Nutrition*, 1967, 20, 613.
6 Satwekar, K., Radhakrishnan, A. N., and Baker, S. J., *Clinica Chimica Acta*, 1968, 20, 53.
7 Baker, S. J., Mathan, V. I., and Abe, K., *Blood*, 1970, 35, 83.

Dissecting Aneurysm and Autoimmune Thyroiditis

SIR,—Hashimoto's disease is reported with lymphomas[1] and thymomas,[2] and also after sarcoid thyroiditis.[3] In this syndrome the chronic irritation of prolonged cellular immunity sometimes seems to alter and accelerate cell division so that neoplasms develop.[4] The Hashimoto's thyroiditis in the 75-year-old woman with hypertension, reported by Drs. Angela Hilton and R. S. Whittaker (30 September, p. 827), was perhaps related more closely to the earlier bladder carcinoma than to the degenerative dissecting aneurysm.—I am, etc.,

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1 Cox, M. T., *Journal of Clinical Pathology*, 1964, 17, 591.
2 Dawson, M. A., *American Journal of Medicine*, 1972, 52, 406.
3 Karlish, A. J., and MacGregor, G. A., *Lancet*, 1970, 2, 330.
4 MacGregor, G. A., *British Medical Journal*, 1972, 1, 375.

Foley-catheter Induction of Labour

SIR,—During the discussion time of a recent Upjohn symposium on prostaglandins I mentioned a method of inducing labour with the help of a Foley catheter. Since then I have had a number of inquiries about the technique. It was introduced to the Bedford maternity department in 1967 after I had read a paper by Embrey and Mollison,[1] and it is used for induction of labour only when the cervix is found to be unripe and unfavourable. Like the original authors, I have found it to be effective and sepsis has not been a problem.

The patient is given Pamergan P100 (pethidine hydrochloride 100 mg and promethazine hydrochloride 50 mg) as premedication. She is then asked to pass urine and catheterization is avoided if possible. After placing her in the lithotomy position and carrying out the usual aseptic preparation the pelvis and cervix are assessed. If the cervix is found to be ripe and the presenting part favourable a low amniotomy is performed using Smyth's forceps or Dumoulin's instrument. If the cervix is unripe a 26-gauge Foley catheter is used. The tip beyond the balloon is cut off with sterile scissors before insertion. The catheter is then passed so that the balloon lies just inside the internal os and 50 ml of sterile water is inserted. Occasionally the os is so tightly closed that it is necessary to expose the cervix with a speculum and to hold its anterior lip with a pair of sponge-holding forceps.

If the catheter has not been passed spontaneously after 12 to 24 hours it is removed and the cervix reassessed. Almost always the cervix is found to have become effaced and partially dilated. A low amniotomy is then performed and usually labour progresses soon afterwards. Occasionally further stimulation of the uterus is required with intravenous oxytocin. Prophylactic antibiotics are not given.—I am, etc.,

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1 Embrey, M. P., and Mollison, B. G., *Journal of Obstetrics and Gynaecology British Commonwealth*, 1967, 74, 44.

General Practice Observed

SIR,—Your leading article, "General Practice Observed" (30 September, p. 781), drew attention to the recent flood of reports on general practice. After reading these documents it is perhaps worth making one or two points before we all get overwhelmed by a plethora of verbal nonsense.

Firstly. it is now being stated as if it were a brand-new discovery that general practitioners need to understand human behaviour and learn about the patients' social and cultural background and their influence on disease. Secondly, the move accelerates to involve general practitioners in hospital work, including the revival of that once oft-maligned institution the G.P. hospital.

While most of us accept the need to try to improve general practice, especially with the advent of the new larger impersonal health centres, and the increasing use of deputizing services, these two points are but a return to the old pattern of general practice obtaining 50 or more years ago and carried on successfully by family doctors who had little if any formalized vocational training. All of which makes one question whether too much emphasis is being placed on vocational training schemes, many in-