knowledge of the height and nude weight of the child and ready access to the nomogram itself. These surface area formulae are themselves subject to considerable error and discrepancy. To relate the dose of nalorphine to 1.73 m² surface area entails further complicated mathematics, since not even the most precocious child is likely to have a surface area approaching that figure.

Let us leave mathematics to the mathematicians and allow clinicians faced with respiratory depression due to Lomotil to ventilate with oxygen and to give the antidote based on the less complicated measurement of body weight.—I am, etc.,

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¹ Starling, E. H., and Evans, C. A. L., *Principles of Human Physiology*, 14th edn., ed. H. Dawson and M. G. Eggleston, p. 543. London, Churchill, 1968.

Prevention of Hyaline Membrane Disease

SIR,—Further to your leading article (14 April, p. 65) delayed clamping of the cord after delivery may also be important in the prevention of the respiratory distress syndrome. It is estimated that (particularly if ergometrine is given to the mother and the cord is not clamped) a baby can receive a volume of blood from the placenta equivalent to as much as half its entire blood volume after delivery.¹ Hypovolaemia seems a logical cause for failure of pulmonary expansion which this simple precaution helps to prevent. The baby (especially if premature or dysmature) should be held at or beneath the level of the vulva, and if the cord is pulsating well it is important not to cut it off from this significant, warm, oxygenated blood transfusion.

Though rarely practised, this is not a new idea, and Erasmus Darwin wrote in 1803 that "another thing very injurious to the child is the tying and the cutting of the navel string too soon. . . ."—I am, etc.,

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¹ Dawes, G. S., *Foetal and Neonatal Physiology*, Chicago, Year Book Medical Publishers, 1968.

Intrathecal Streptomycin and Deafness

SIR,—The article by Dr. J. Stevenson (19 May, p. 411) on bacterial and tuberculous meningitis has deservedly drawn much attention (23 June, p. 716).

As a person who became totally deafened at the age of 50 years I cannot allow to pass unchallenged the statement by Dr. Stevenson that "many experienced clinicians still favour the use of intrathecal streptomycin in the earliest stages" of tuberculous meningitis. Mention is also made of drug resistance and hypersensitivity necessitating a change to one of the newer drugs.

There would seem no essential difference between the management of childhood tuberculous meningitis and that in the adult—in both the prognosis is more favourable when early diagnosis is made and adequate chemotherapy given (with the tubercle bacillus in mind) while awaiting the results of culture in order to establish the diagnosis.

The ototoxicity of the neomycin group of drugs, including streptomycin, is well documented in the literature—for example, by Berg.¹ The sequel of deafness, particularly when acquired early in life before auditory memories are established, is a tragedy of the first degree. I doubt whether this risk is worth taking. I am persuaded from my comparatively wide reading in the field of sensory neural deafness that streptomycin should not be used intrathecally. This I understand, is the opinion of most of the experts since newer drugs have become available for the treatment of this critical illness. The fact that deafness as a result of intrathecal streptomycin may occur in only a minority of cases is no justification for its use even as a life-saving measure when these newer drugs may be tried.—I am, etc.,

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¹ Berg, K., *Annals of Otolaryngology and Rhinology*, 1949, 58, 448. Berg K. *Am. Otol.* 1949, 58, 448-455.

Behçet's Syndrome and Venous Thrombosis

SIR,—Since reading the two case reports by Drs. T. Chajek and M. Fainaru (31 March, p. 782) we have treated a 21-year-old man who presented with a deep vein thrombosis and features suggesting Behçet's syndrome—recurrent mouth ulcers, acne, pyoderma, and intermittent epididymitis but without genital ulceration. The plasma fibrinogen titre was 1/128 and plasma fibrin-fibrinogen related antigen was 12 µg/ml.

Ten days before admission the patient had jolted his left hip slightly while playing with a child. Two days later the whole limb began to swell. On admission ascending venography revealed an extensive femoral vein thrombosis. Intravenous streptokinase was given, 600,000 U in the first 30 minutes followed by 100,000 U hourly for 96 hours. Treatment was accompanied by an unusually severe febrile response (temperature up to 41°C). Intravenous infusion sites had to be changed frequently because local phlebitis developed rapidly. Warfarin was given after 60 hours' streptokinase infusion. Owren's one-stage prothrombin time was 14% of normal at 96 hours. Clinically there was a dramatic reduction in size of the leg associated with the development of dilated superficial veins over the thigh. However, a second venogram showed extension of the femoral thrombosis and presence of thrombus in several tibial veins that had been clear previously.

The improvement in Drs. Chajek and Fainaru's cases of superior vena caval occlusion treated with fibrinolytic agents was assessed solely by clinical examination. While appreciating that failure of recanalization of an occluded superior vena cava is less likely to be accompanied by a marked clinical improvement, we would emphasize that physical signs of improvement may not indicate disappearance of thrombus. Our patient's leg probably improved because of dilatation of collateral veins at a time when thrombus was actually extending. In Behçet's syndrome thrombophlebitis frequently develops at venepuncture sites¹ and this too was a feature in our patient. Caution is needed, therefore, in the absence of convincing evidence of thrombolysis, before advocating the use of intravenous fibrinolytic agents. The treatment of venous thrombosis in this rare syndrome may require a different approach from that considered appropriate in other circumstances. Our understanding of the

problem would be improved by further case reports, especially since enough cases are unlikely to be available for the purposes of a clinical trial.—We are, etc.,

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¹ Pitney, W. R., *Clinical Aspects of Thromboembolism*, p. 26. Edinburgh and London, Churchill Livingstone, 1973.

Smallpox

SIR,—I must congratulate Dr. A. B. Christie on his recent article on smallpox (23 June, p. 539).

As rightly brought out in the article, it is the variola sine eruptione which proves a challenge in diagnosis, but equally important are the very mild modified cases of smallpox which if not detected early keep the torch of a smallpox epidemic burning. While instituting the "expanding ring technique"¹ for the control of an epidemic of smallpox, it is most important to keep a vigilant watch on the most immediate contacts who,

Langer's lines, after Dixon.² Reproduced by courtesy of Messrs. Churchill Ltd.

despite vaccination and a good "take," can develop modified smallpox.

With regard to the question of severe chickenpox versus smallpox, I think it is sensible and wise to err on the wrong side and regard all severe cases of chickenpox as potential cases of smallpox until proved otherwise. In such situations, in my experience, mapping of the lesions on a chart and comparing it with Langer's lines² as shown in the accompanying figure has been found very useful, as the distribution of lesions in chickenpox follows the Langer's lines very closely, whereas those of smallpox do not.

Lastly, with regard to laboratory diagnosis I think agar-gel diffusion based on antigen and antibody reaction, a test devised by Dumbell and Nizamuddin,³ has proved outstandingly successful, giving reliable, rapid results in about six hours.—I am, etc.,

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¹ Ramsay, A. M., and Emond, R. T. D., *Infectious Diseases*. London, Heinemann Medical Books, 1967.

² Dixon, C. W., *Smallpox*. London, Churchill, 1962.

³ Dumbell, K. R., and Nizamuddin, M., *Lancet*, 1959, 1, 916.

Treatment of Systemic Lupus Erythematosus with Renal Insufficiency

SIR,—I was interested to read the papers by Dr. M. L. Snaith and others (28 April, p. 197) and Dr. P. H. Feng and others (26 May, p. 450) on the use of chlorambucil and cyclophosphamide respectively in the therapy of systemic lupus erythematosus (S.L.E.). I would like to record a case of S.L.E. with renal insufficiency which responded to a combination of cyclophosphamide and corticosteroids.

An Indian girl aged 13 years presented in January 1972 with weakness, chest pain, arthralgia, and ulcers in the mouth. She was emaciated and pyrexial. Lymphadenopathy was present in the right axillary region and the left posterior triangle of the neck. The haemoglobin level was 7.5 g/100 ml, leucocyte count 5,000/mm³, and platelet count 50,000 c/mm³. L.E. cells were present in the blood. The urine was normal and blood urea 40 mg/100 ml. Chest x-ray showed a honeycomb picture at the bases compatible with S.L.E. She was treated with prednisone 60 mg daily, and though she was on it on an intermittent basis in that follow-up was not regular she deteriorated clinically. The patient was readmitted in March 1973 complaining of weakness, fever, backache, and chest pain. She was markedly emaciated, pale, and ill-looking and weighed only 55 lb (25 kg). Investigations showed that her renal condition had deteriorated: the urine contained albumin 1+ and 5 leucocytes per high-power field, the blood urea was 105 mg/100 ml, and the glomerular filtration rate 18 ml/min. Other investigations showed: serum complement 43 mg/100 ml, haemoglobin 6.5 g/100 ml, leucocyte count 6,000/mm³, platelet count 30,000/mm³, Coombs test positive, and mild haemolytic anaemia present. Renal biopsy showed focal tuft adhesions and cellularity of the glomeruli with marked focal thickening of the basement membrane ("wire loop" lesion). An ammonium chloride test confirmed that the patient could not acidify her urine in that the pH of the urine remained at 5.8 in spite of systemic acidosis.

The patient was treated with prednisone 30 mg daily and cyclophosphamide 50 mg on alternate days. She gradually improved, her weight gain was 100 lb (45 kg), and there was no temperature. The glomerular filtration rate rose to 31 ml/min, the blood urea dropped to 40 mg/100 ml, and the serum complement rose to 66 mg/100 ml. Chloram-

bucil 4 mg daily was then substituted for the cyclophosphamide, but this produced a relapse in her condition in that she became febrile. On reintroducing cyclophosphamide the patient became afebrile. Efforts to stop cyclophosphamide failed as she became febrile and began to lose weight. She was finally discharged on prednisone 30 mg daily and cyclophosphamide 50 mg on alternate days. The only side effect from cyclophosphamide was mild alopecia.

This case illustrates the following features: (1) Patients with S.L.E. may improve in a combination of prednisone and cyclophosphamide even in renal insufficiency. (2) The combination of prednisone and cyclophosphamide in this case was shown to be more effective than chlorambucil and steroid therapy. (3) Some patients with S.L.E. may become dependent on cyclophosphamide and stopping it may produce a relapse.

In a study of 14 patients suffering from S.L.E. who had evidence of renal involvement shown on renal biopsy it was found that the combination of steroids and cyclophosphamide was more effective and produced fewer side effects than steroids alone. Details of this work are being prepared for publication.

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Infantile Overnutrition

SIR,—Your leading articles (30 June, p. 727) and the article by Dr. R. K. Oates in the same edition (p. 762) have once more drawn attention to the dangers of obesity in infants. The danger stressed in this condition is the probability of obese infants remaining obese children and becoming obese adults. In 1965¹ and again, with a colleague, in 1971² I drew attention to another danger of obesity in infants—namely, the increased morbidity rate in the first year of life. We suggested that this obesity might be due to increased retention of minerals in tissue fluids subsequent to the greater protein content of cow's milk, and this now seems to be confirmed by Dr. Oates's work.

Since we have become aware of this problem, my partners and I have tackled it energetically from the early months of infancy, and our health visitors, who look after our 150 new babies each year, are now well alerted to spot those babies who are gaining weight excessively in the early weeks and give the appropriate advice to the mothers on the question of feeding. It is probably true to say that since about 1968 infant obesity has been virtually eliminated from this practice. There is no doubt in our minds that if general practitioners, doctors working in well-baby clinics, and health visitors were adequately alerted to this problem and dealt with it energetically by early advice and close supervision the problem could be almost completely overcome in a very short period of time. I would like to pay tribute to the work which our health visitors have done in combating this problem; our success has been due entirely to their vigilance and competence.—I am, etc.,

P. D. HOOPER

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¹ Hooper, P. D., *Practitioner*, 1965, 194, 391.

² Hooper, P. D., Alexander, E. L., *Practitioner*, 1971, 207, 221.

Infant-feeding Practices

SIR,—Dr. R. K. Oates (30 June, p. 762) and your leading article (p. 727) outline the many hazards of the artificial feeding of infants. It is time that the medical and nursing professions gave the lead in encouraging the natural feeding—that is, breast-feeding—of infants and educating mothers to choose it.

Dr. Oates says: "Clearly the problem is one of education of mothers beginning in the maternity unit and continuing through the advice of health visitors and family doctors"—yes, but education in breast-feeding, with artificial feeding only a second choice. It should be stressed that breast-feeding is the best start for infants.—I am, etc.,

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Psychosomatic Disease

SIR,—In the introduction to his Charles West Lecture Dr. John Apley (30 June, p. 756) discusses briefly the history of the notion of psychosomatic illness, stating: "I cannot find that he (Charles West) used the word psychosomatic, though it was known in his time. Like Willis and Sydenham before him, he did, however, describe patients with what today would be called psychosomatic disorders."

In the original Arabic textbook of medicine *al-Qanoon*, which was written early in the eleventh century, Avicenna (979-1037) gave a good detailed clinical description of what nowadays would be regarded as a psychosomatic manifestation of a reactive depression. He described the emotional and physical changes in the frustrated lover. He was aware of the relation between emotional excitation and the appearance of extrasystoles in the pulse. Moreover he was able to make use of this relation to detect the identity of the love object through the appearance of extrasystoles as the names of various towns, streets, families, persons, etc., were mentioned to the patient.—I am, etc.,

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Women Doctors' Pensions

SIR,—It is clear from recent correspondence that there remains a good deal of misunderstanding in regard to the pensions of women doctors. The absurd example of the gigolo husband referred to by Dr. Mary Duguid (14 July, p. 111) was intended to be so ridiculous as to bring home to women doctors the fact that such a situation would never arise, and I hope most people understood this.

If I might perhaps once again endeavour to clarify the position in regard to the lack of a widower's pension for women doctors:

(1) This is not the control of the Department of Health, nor of the Superannuation Committee. It is a firm ruling by the Inland Revenue and it applies to several million women workers in this country. This situation can be changed only by the Inland Revenue rules; a concerted effort by many of the women's organizations is