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 m^2 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. Eggleton, 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 Otology, Rhinology and Laryngology*, 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 sites[1] 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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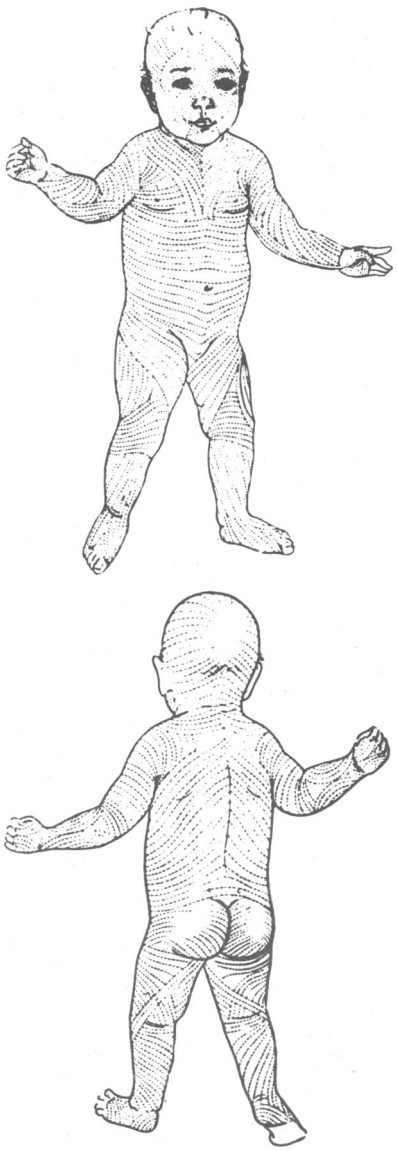
1 Pitney, W. R., *Clinical Aspects of Thromboembolism*, p. 26. Edinburgh and London, Churchill Livngstone, 1973.

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.