and such data would obviously be worth having.—I am, etc.,

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Intramuscular Injection and Coagulation Defects

SIR,—I read with interest the remarks of Dr. P. M. Jones (24 June, p. 770) on the danger of intramuscular injections in haemophiliacs. The danger also exists—and to a greater extent—in patients receiving anticoagulant therapy, especially heparin. In haemophiliacs only one system of coagulation is disturbed—that is, the intrinsic—whereas anticoagulant therapy influences both the intrinsic and the extrinsic systems. Heparin has also, through its antithrombin action, some effect on platelet aggregation. I have seen several patients treated with heparin by intravenous drip and one receiving an indanedione preparation in whom, after intramuscular injections, large intragluteal haematomata developed, extending into the muscles of the thigh. The haematoma formation was associated with acute exsanguinating anaemia and hypotension, necessitating prompt blood transfusion. One elderly patient—in another hospital—died because of rapidly progressive exsanguination and shock. Intramuscular injections should be avoided in every patient actually on anticoagulant treatment as well as in patients with other coagulation defects.

On the other hand, in my experience, the danger of intramuscular injections is far less in various thrombocytopenic states (idiopathic thrombocytopenic purpura, leukaemias, pancytopenias); it is known that platelets are not necessary for extrinsic coagulation. In such situations I have observed only transient extravasations (but without permanent crippling) and never large exsanguinating haematomas in the gluteal musculature. This was especially evident in several patients with severe thrombocytopenia due to marrow aplasia treated for many months with large intramuscular doses of testosterone propionate.[1]—I am, etc.,

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1 Dyk, T., Piotrowski, M., and Kostecka, W., *Polski Tygodnik Lekarski*. In press.

Asthma Deaths

SIR,—In suggesting in your leading article (28 November, p. 443) an answer to the question of the cause of the recent rise in mortality from asthma you seem to rely entirely on the paper by the epidemiologist Stolley.[1] He found that asthma mortality had increased decisively only in those countries in which aerosol bottles containing five times the usual concentration of isoprenaline were available. Unfortunately, there are too many exceptions to this rule. Thus in Denmark and Sweden, with modest but definite increases in asthma mortality, no strong-isoprenaline aerosols were available. Japan also had none of these preparations but had a high mortality throughout. Norway had, as you mention, a definite rise in asthma mortality, but it had only one-quarter to one-tenth of the consumption of strong-isoprenaline aerosols compared with the United Kingdom. The Netherlands, on the other hand, had some of these aerosols, but no rise in mortality at all. These incongruities make the assumption of Stolley most doubtful.

Much worse is that there is no evidence that isoprenaline is cardiotoxic. It has been used for 30 years, and if it had caused cardiac deaths this should have been noticed. Asthmatics who die suddenly in an attack (and often have used catecholamines a short time beforehand) usually show at necropsy as the most prominent change many viscous mucous plugs in the bronchi, which obstruct breathing. There is nothing to contradict the assumption that they die from lack of oxygen, and indeed the arterial oxygen tension is found to be dangerously low in such cases. This is much more likely to be the cause of death than the isoprenaline. Moreover, in those patients who inhale isoprenaline or other catecholamines incessantly tolerance to them quickly develops. Every practitioner who sees many asthmatics (but not the epidemiologist) knows patients who, at a sudden deterioration of their complaint, start inhaling every hour or half-hour and soon complain that "it does not help any longer." This is the first sign of tolerance, a danger signal not to be overlooked. It occurs with a concentrated aerosol as well as with a dilute one. I first described it 20 years ago[2] and have mentioned it repeatedly since.[3,4] The tolerance has also been shown experimentally to develop in animals and in man.[5]

It follows that abuse of isoprenaline can have, at most, played an indirect part in the increase of asthma mortality by creating tolerance to itself. The patient with severe asthma who has no other treatment is then left with an isoprenaline nebulizer that no longer helps him, and he may become an easy victim of a renewed attack unless he has other efficient remedies—aminophylline, potassium iodide, or corticosteroids. It has been well documented that in most fatal cases the patient had little or no corticosteroid to protect him.

I believe this answer to your question should be considered.—I am, etc.,

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London N.3

1 Stolley, P. D., *American Review of Respiratory Disease*, 1972, **105**, 883.
2 Herxheimer, H., *The Management of Bronchial Asthma*, London, Butterworth, 1952.
3 Herxheimer, H., *Lancet*, 1968, **2**, 216.
4 Herxheimer, H., *Lancet*, 1972, **1**. 98.
5 Connolly, M., Davies, D. S., Dollery, C. T., and George, C. F., *British Journal of Pharmacology*, 1971, **43**, 389.

Fracture of Lippes Loop

SIR,—Last[1] has recently reported 15 cases of fracture of the Lippes loop in utero; the average time the loop had been retained before fracture was 38 months, with a range of 22-62 months.

At this hospital we have seen a number of women whose loop has fractured apparently while being removed (though possibly it had broken spontaneously in utero in at least some cases). In virtually all these cases the loop had been inserted more than two years previously. Since every patient with a fractured loop has had to be admitted to hospital for removal of the remnant under anaesthetic, we have adopted the practice of removing loops after two years (that is, at the second annual check-up) and inserting a fresh loop at the same clinic attendance. Many of the loops removed more than two years after insertion have had a rough, pitted surface and have lost their natural elasticity. Could this be due to a chronic "foreign body reaction" on the part of the endometrium?—I am, etc.,

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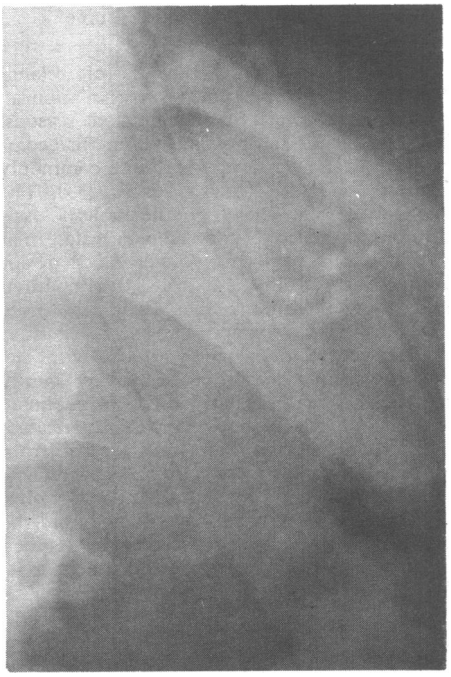
1 Last, P. A., *Journal of Obstetrics and Gynaecology of the British Commonwealth*. 1972, **79**, 190.

Radiology of Swallowed Earthworm

SIR,—It is well known[1] that roundworms (*Ascaris lumbricoides*) may readily be demonstrated on contrast examination of the small gut. They may show both as an intraluminal filling defect and because the worm's alimentary canal is outlined with ingested barium suspension. So far as I can ascertain, no one has previously reported the radiological findings with earthworms (*Lumbricus terrestris*) in the human alimentary tract.

The accompanying plain abdominal radiograph of a 3-year-old child shows material which is pretty obviously "dirt," yet it is arranged in an orderly fashion in a coil. Accordingly there was no hesitation in identi-



fying the opaque parts as ingested soil in the alimentary tract of an earthworm lying in the stomach. This was confirmed when the history was obtained. The child had indeed swallowed an earthworm and the worried parents had brought him up for reassurance. —I am, etc.,

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1 Shanks, S. C., in *A Textbook of X-Ray Diagnosis*, ed. S. C. Shanks, and P. Kerley, Vol III, 3rd edn. London, H. K. Lewis, 1958.

Trichuris trichiura Infestation

SIR,—It should be emphasized that the *Trichuris trichiura* infestations reported by Dr. D. M. Lynch and others (14 October,