revealed four deaths, all from the physical conditions the absence from which of clinical confirmation led to their symptoms being interpreted as neurotic.

A young male ataxic was found post mortem to have a glioma tracking selectively down the mid-brain. A woman with unexplained diarrhoea and weakness had a single small secondary in the liver, no primary discovered post mortem. An anxious man with recurrent invaliding chest pain died of a massive infarct. An intractable asthmatic died in an attack, no more helped by psychological than by physical methods.

However one looks at this dip from the barrel it is a reminder of how all too easy it is to allow universally latent psychopathology to fill a gap. The pragmatic actuary's target may be not the psychoneurotic patient but the tenuous nosology.

H A ANDERSON

Lyme Regis

Local corticosteroids in asthma

SIR,—Dr H M Brown (28 May, p 1408) blames you, to my mind unjustly, for altering your earlier, somewhat overoptimistic view of the advantages of steroid aerosols in asthma. Effective local steroid treatment of asthma started as early as 1955¹⁻⁴ with the insufflation of micronised corticosteroid from a powder blower, but it became popular only much later when given as an aerosol from a metered bottle. Replacing systemic by aerosolised steroids depends (a) on the severity of the case, as documented by the systemic maintenance dose, (b) on the accessibility of the surface of the bronchial mucosa. Most published studies show that patients on small maintenance doses (5.0-7.5 mg/day) can often be maintained on aerosol alone. If they require 10 mg/day or more and were given about 800 µg steroid aerosol in addition,⁵⁻⁸ this made it possible to reduce the oral dose by 5-8 mg/day. However, if the bronchial mucosa is covered, as is often the case, by tough mucus, this seems to obstruct the absorption of the aerosol, as might be expected.⁹ Unfortunately many authors have not reported how many of their patients had hypersecretion. Also, the prompt dilator effect of beta stimulants from aerosol bottles used by many asthmatics often creates typical conditional reflexes, which may also work after the steroid aerosol although it has no dilator action.

Three practical conclusions seem to follow: (1) Patients with bronchial hypersecretion (particularly those with persistent cough or regular nocturnal cough) are unlikely to benefit from steroid aerosols. (2) Patients with very mild dry asthma, mostly of allergic origin, who inhale bronchodilators not more often than twice daily, will probably benefit from steroid aerosols about 800 µg per day, or, alternatively, from an oral maintenance dose of 5-7.5 mg/day. If they are reasonably fit without either, neither systemic nor inhaled steroids are indicated. (3) Patients who have life-threatening attacks of asthma or are in danger of becoming invalids through asthma should in no circumstances be given a steroid aerosol but require high doses of a systemic steroid, beginning usually with 30 mg prednisone daily in the morning and tapering very slowly off as outlined elsewhere¹⁰ until the daily maintenance dose is found. Only then, and only if this maintenance dose is higher

than 7.5 mg/day, should gradual partial replacement of the systemic maintenance dose by steroid aerosol be attempted. During this process and afterwards continuous observation is necessary for spontaneous increases of the asthmatic obstruction by intercurrent infection or other factors, best noticeable from the increasing number of bronchodilator inhalations required. In such a case the systemic steroid must be doubled at once regardless of the steroid aerosol and, if necessary, other measures taken. In no case should the decision whether to increase the systemic steroid be left to the patient or, in the case of a child, to the parents. It is here that the danger of sudden death threatens, and it is here that I profoundly disagree with Dr H M Brown. I agree, however, in so far as the family doctor often is too reluctant to give high corticosteroid doses. This reluctance has caused, as some of our most experienced observers have shown,^{11,12} unnecessary deaths from asthma. I and my co-workers have observed during 25 years more than 2000 asthmatics requiring long-term steroids, but have seen surprisingly few deaths during this time, mostly in the aged and through non-asthmatic complications; none in children, of whom we have seen fewer.

In my experience steroid aerosols offer an advantage only to patients with moderately severe asthma who need systemic corticosteroids in a dosage of more than 7.5 mg per day. They will often be able to lower the maintenance dose by adding some steroid aerosol, thus perhaps reducing the risk of adrenal suppression.

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Reliable measuring of blood pressure

SIR,—We now have several effective anti-hypertensive drugs. We have proof that blood pressure reduction is beneficial in those with moderate or severe hypertension. What is the next goal? It would be interesting to know if drug treatment of mild hypertension is worth while, but I share the doubt expressed in your leading article (4 June, p 1429) about the likely result of the MRC trial. I do not think it will be decisive.

In my view probably the greatest need today is for some simple means of determining an individual's usual blood pressure as distinct from that which develops when he is confronted by a doctor (or nurse or technician) and subjected to the discomfort of sphygmomanometry. There is no doubt that this artificial environment adds a variable, unknown, and often large supplement to the ordinary pressure—a supplement which is considerably increased if the medical examina-

tion is considered to carry a threat of loss of livelihood. Under such circumstances I have recorded a pressure as high as 220/120 mm Hg in an otherwise normotensive subject.

It is frequently argued in favour of casual readings that in spite of their deficiencies they are the best guide we possess to prognosis and thus to the need for treatment. Although this is true in relation to large groups of people, casual readings are a poor guide to prognosis in individuals and inferior to basal readings.¹ Others argue in favour of casual readings that a rise in blood pressure with excitement indicates the existence of a state of "labile hypertension" and that this carries an adverse prognosis. Such an inference is completely unproved.

No, it would be an immense advantage if we could eliminate the artefact caused by sphygmomanometry. Blood pressure can be recorded accurately over long periods without the presence of a doctor or other medical worker, by means of an intra-arterial catheter, but so far no reliable means of doing this non-invasively has been devised. The next major advance in the management of hypertension will be the development of a reliable, inexpensive, non-invasive piece of equipment capable of recording the blood pressure repeatedly over a period of an hour or more with the patient in a state of relaxation. The first manufacturer to produce such equipment could expect to make a fortune.

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¹ Smirk, F H, in *Antihypertensive Therapy: Principles and Practice*, ed F Gorss, p 355. Berlin, Springer, 1966.

Bleeding in renal failure: a possible cause

SIR,—Following the report of Kazatchkine *et al*¹ that factor VIII—von Willebrand factor activity (VWFR) is decreased in the plasma of patients with chronic renal failure and may partly explain the haemorrhagic tendency in that condition, we have measured factor-VIII-related antigen (VIIIa), factor-VIII-procoagulant activity (VIIIc), and VWFR in a group of 20 patients undergoing long-term haemodialysis.

VIIIc was estimated by a standard two-stage assay,² VIIIa by unidirectional immunoelectrophoresis using the Laurell technique,³ and plasma VWFR by aggregation of formalin-fixed platelets in the presence of ristocetin.⁴

The results are as follows:

	Mean %	Range %	Reference range %
VIIIc	267	122-404	50-200
VIIIa	219	93-400	50-170
VWFR	180	83-291	50-200

We confirmed the elevated levels of VIIIc and VIIIa found by Kazatchkine *et al* and others, but none of our patients had a depressed VWFR level. Indeed, the mean level was towards the upper limit of normal. VWFR levels did not differ significantly from VIIIa levels, but the difference between mean VIIIc and VWFR is highly significant (using two-way variance analysis, $t=4.48$, $P<0.001$) as is the difference between VIIIc and VIIIa ($t=2.66$, $P<0.05$).

We conclude, therefore, that our patients'