present trend in vocational training. Talking to others at postgraduate meetings, I suspect most of us feel much the same.

Since I became a GP 20 years ago I have kept a record of all those problems and conditions that could not have been experienced as well, or better, elsewhere. I have yet to cover both sides of a sheet of foolscap. The new jargon of general practice may cause some to feel a pride in their own importance, but for those who prefer to take a pride in the excellence of their work the place to acquire ability is in the crowded outpatient departments of any general hospital.

As for the remedy—it is for the young to recognise the conceit contained in all that dressed-up, pretentious verbiage.

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An open-access morbid pathology facility for GPs?

SIR,—Your correspondents' claim that there is a need for an open-access morbid pathology facility for GPs (7 July, p 51) is well made, but I would question the desire for such a service in the eyes of the general public.

The vast majority of patients and many doctors find the process of necropsy distressing when applied to a friend or close relative, and in the days when I was a preregistration house officer it always seemed to me incredibly callous to have to announce a death to the relatives with one hand and request a necropsy with the other; that this is regarded as the norm in many hospitals even when the cause of death is not in doubt reveals an unthinking attitude on the part of senior staff and a lack of appreciation of personal emotions. That many relatives accede to this sort of unnecessary request is, I believe, an indication of the status enjoyed by the profession rather than of any public feeling for absolute diagnosis.

It is, of course, true that there are occasions in general practice when a necropsy outside the coroner-police axis could be desirable, but the proposed procedure might well be just as distasteful for many relatives, and I feel sure that there would in fact be little demand for it by humane GPs who would not wish to make this sort of request of bereaved friends and patients.

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SIR,—Dr A J Ferris and Dr A P Glanville (7 July, p 51) plead convincingly for GPs to be able to have postmortem examinations on their patients. I agree. Unfortunately, where this facility already exists, experience shows that it is only a small minority of GPs who take advantage of it. Shortly after my appointment as pathologist to North Teesside District in 1960 I made it known that I was prepared to do postmortem examinations at the request of any local GP. After some years of campaigning on my part, the then North Tees Hospital Management Committee agreed in 1965 to pay the costs incurred by funeral directors in transferring patients from their home to the hospital for necropsy.

Though the availability of this service has been notified to all GPs in the district on at least two occasions, it has been used relatively infrequently. Of the 80 or so GPs who practise in the area served by this hospital, only 11 ever

requested a necropsy. Moreover, in the 10 years 1969 to 1978 inclusive only 23 such requests were made, an average of just over two per year, and 12 of these requests came from one three-man practice.

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Coronary artery spasm

SIR,—Your leading article (14 April, p 969) suggests that coronary artery spasm may be the cause of variant (Prinzmetal's) angina and that, in turn, the spasm may be produced by "some intrinsic factor related to autonomic control."

This factor may be cholesterol. We have good evidence that in the renal, cerebral, and myocardial vascular beds resistance vessels are sensitised to the effect of vasoactive substances such as noradrenaline and 5-hydroxytryptamine by increased levels of circulating low-density lipoprotein. For example, 5-hydroxytryptamine normally has no effect on cerebral blood flow; in the presence of raised plasma cholesterol concentrations, however, 5-hydroxytryptamine is vasoconstrictor.¹ Also, in dogs fed on a cholesterol-rich diet sufficient to produce a doubling of the serum cholesterol level there is a tenfold increase in the sensitivity of both beta-receptor mediated vasodilatation (in anaesthetised dogs)² and alpha-receptor mediated vasoconstriction (in conscious, instrumented dogs).

We have similar data for other steroids, particularly corticosterone, oestrogen, and progesterone, in the cerebral circulation of the baboon.^{3,4} These steroids also sensitise cerebral resistance vessels to vasoactive amines.

What is the mechanism of this potentiation? β -Oestradiol is well known to inhibit noradrenaline uptake-2 (the uptake of noradrenaline released from adrenergic nerve terminals into heart muscle⁵) and may act in the same way to block uptake-2 into vascular smooth muscle cells. Inhibition of this uptake will result in an increased noradrenaline concentration at or close to the receptors. This may be a property of other steroids such as cholesterol, oestrogen, and progesterone. Another possibility, which we are investigating, is that chronic hypercholesterolaemia or hyperoestrogenaemia will modify the noradrenaline-adrenergic receptor binding characteristics and may even affect the ratio of alpha- to beta-receptors on the vascular smooth muscle membrane.

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¹ Bloom, D S, *et al*, *British Journal of Pharmacology*, 1975, **54**, 421.

² Rosendorff, C, *et al*, *Proceedings of the VIII World Congress of Cardiology*, 1978, **157**.

³ Mendelow, A D, *et al*, *Stroke*, 1977, **8**, 322.

⁴ Mendelow, A D, *et al*, *Stroke*, 1977, **8**, 326.

⁵ Iversen, L L, and Salt, P J, *British Journal of Pharmacology*, 1970, **40**, 528.

Saving asthmatics

SIR,—In your leading article (9 June, p 1520) it was stated that there is a clear case for establishing confidential inquiries into the circumstances in which young asthmatic patients die from their disease.

The research committee of the British Thoracic Association first carried out a pilot study and found that 70-80% of these patients

die outside hospital or within one hour of reaching hospital. A subcommittee of the research committee is carrying out during this year, 1979, a confidential inquiry into all asthma deaths in persons aged 15 to 64 living in the Merseyside and West Midland Regional Health Authority areas. It is estimated that 120 to 150 deaths will occur. The co-ordinator is visiting GPs and relatives to obtain information and we are extremely grateful for the helpful way in which he is being received. A report about the confidential inquiry should be complete by mid 1980.

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SIR,—Your leading article (9 June, p 1520) and subsequent letters made a valuable contribution to an important subject.

There is, however, one dangerous manifestation of asthma which has perhaps not received the attention which it deserves. This is the severe breathlessness, sudden in onset and frightening in its severity, coming on without obvious cause, and within minutes turning ordinary asthma into an acute struggle for survival, with the patient on his feet using all available auxiliary muscles. Such paroxysms are completely unexpected, though they are grafted on persistent asthma which so often is underrated in its severity. In this real emergency inhalers are useless and intravenous therapy is virtually impossible. In my experience a full dose of adrenaline, 1 mg subcutaneously, can then be lifesaving. Would it not be possible to make adrenaline injections easily available to asthmatics for self-administration as an automatic gun which could be fired immediately if required? It is reasonable to hope that such a gun, if used by the patient early at the onset of sudden and severe asthma, will make a real contribution to saving asthmatics. Observation also makes it reasonable to believe that in sudden severe attacks it is the asthma that kills and not the inhalant, which in such a state is unlikely to travel far beyond the pharynx.

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Long-acting nitrates in the prophylaxis of angina pectoris

SIR,—Dr Andrew Smith and Professor M D Rawlins' statement that "long-acting nitrates are doubtful value in the prophylaxis of angina" (21 April, p 1073) if taken at face value would deprive many patients of the benefit of these extremely useful drugs.

Although it was previously thought that long-acting nitrates were ineffective in the therapy of angina pectoris owing to their inactivation in the liver,¹ this view has recently been refuted by well designed double-blind studies in which sorbide nitrate has been shown to increase exercise tolerance effectively.^{2,3} Many patients cannot use beta-blockers because of obstructive airways disease or severe congestive heart failure. In this group sorbide nitrate will often be the only treatment available and the added benefit of pre- and after-load reduction for those in cardiac failure will be obtained. The mode of action is through pre-load reduction with a drop in filling pressures and consequent