CORRESPONDENCE

We may return unduly long letters to the author for shortening so that we can offer readers as wide a selection as possible. We receive so many letters each week that we have to omit some of them. Letters must be signed personally by all their authors. We cannot acknowledge their receipt unless a stamped addressed envelope or an international reply coupon is enclosed.

Correspondents should present their references in the Vancouver style (see examples in these columns). In particular, the names and initials of all authors must be given unless there are more than six, when only the first three should be given, followed by et al; and the first and last page numbers of articles and chapters should be included. Titles of papers are not, however, included in the correspondence section.

Suicide in doctors and their wives

SIR,—Why is the medical profession at such high risk for suicide? In male doctors this is thrice that in the general public, ten times that in members of Parliament and senior civil servants, and six times that in university teachers and the clergy. In unmarried women doctors the frequency is more than twice that in unmarried women in general, but is comparable to that in selected groups of professional women.[1] The data for married women doctors are, unfortunately, unreliable.

The special knowledge that doctors have of toxicology and surface anatomy may mean that impulsive suicide attempts are usually fatal, whereas many more are survived in the general public. The very high risk of suicide in pharmacists (who share some of this knowledge) supports this view. What is controversial is whether stresses related to the practice of medicine, possibly interacting with personality flaws in vulnerable doctors, play a part.

The finding of a standardised mortality ratio (SMR) for suicide of 458 in doctors' wives arouses concern that the families of doctors are also drawn into the net. This figure (based on 31 deaths during 1970-2) exceeds by far that found in the wives of other professionals including pharmacists. (There are no comparable figures at present for suicides in the children of doctors.) Searching for aetiological factors, the popular belief that some doctors attempt to cope with their frustrations by recourse to alcohol and other drugs is supported by the SMR of 311 in male doctors aged 15-64 dying of cirrhosis. (In addition to the 59 male doctors in England and Wales who took their lives during 1970-2 a further 18 died of cirrhosis.) Unmarried women doctors do not share this latter risk, but doctors' wives do so to a certain extent.

In order to elucidate the role of marital strain in the suicides of doctors and their wives I have looked at data[2] on the civil states of the 55 male doctors aged 25-64 who committed suicide during 1970-2 (see table). There is an excess of unmarried and of divorced doctors, which, on further analysis, is concentrated in the age group 35-54. Twice as many doctors among the suicides have remained unmarried later than among their living colleagues, and there are also disproportionately more divorces. The numbers, however, are small. The majority were married at the time of their deaths, and some of these marriages may have been under strain, but it is obviously impossible to determine the extent on these data. Among male doctors as a whole the distribution of civil states was identical to that seen in social class I, which does not support either the notion that marital strain is more common in the medical profession or that male doctors as a group tend either to delay marriage or not to marry at all. Nevertheless, one wonders about the 12 bachelor suicides and whether, exclusively dedicated to their careers, they met with ultimate disappointment in their professional aspirations only to find a cushion of family support absent during their hour of need. Equally, they may have failed to marry on account of those very idiosyncracies which led eventually to their suicides. It is quite likely that a number of the total group were psychiatrically ill and suffered from depression, complicated or otherwise.

However we may speculate, there is clear evidence of an occupational hazard of suicide in doctors and their wives. The envisaged health committee of the General Medical Council is a step taken at the tertiary level. Primary prevention should begin with career counselling prior to medical school so as to ensure congruency between the expectations of the aspirant medical student and the realities of life in the profession. Admission procedures

Marital states of 55 suicides among male doctors aged 25-64 (England and Wales, 1970-2)

	Single	Married	Widowed	Divorced
Suicides	12 (21·8%)	40 (72·7%)	1 (1·8%)	2 (3·6%)
Living doctors*	476 (11·2%)	3708 (87·3%)	29 (0·7%)	36 (0·9%)
Social Class I*	7332 (11·6%)	53 778 (86·7%)	454 (0·7%)	467 (0·8%)

$\chi^2 = 14·84$, df 6; $p = 0·02$.
*Ten per cent samples.

to medical schools should include measures to detect vulnerability, and the curriculum should embody programmes which will help the student to cope with later professional stresses. Faculty advisors, tutors, and organised peer groups should be aware of their duty to recognise and intervene at an early stage rather than to wait for the GMC to take action when problems may be ineradicably established.

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[1] Sakinofsky I. Suicide in doctors and wives of doctors. *Can Fam Physician* 1980;**26**:837-47.
[2] Office of Population Censuses and Surveys, England and Wales.

Beta-blockers in the treatment of myocardial infarction

SIR,—Professor Geoffrey Rose in his letter (19 April, p 1099) rightly drew attention to the need to give confidence limits in "negative" trials such as that recently reported with beta-blockers in the secondary prevention of myocardial infarction (29 March, p 885), and Dr N S Baber and Mr J A Lewis (5 July, p 59) have performed a useful service in drawing further attention to this point. They would have done better, however, to have quoted the references and omitted the names of the beta-blockers, since no comparative conclusions can possibly be drawn from the data. Their illustrative examples tend to confuse rather than clarify the situation; since their list is incomplete, it mixes studies of entirely different design and duration, and the results given arise from different types of analysis.

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***The omission of the full reference list from the letter by Dr Baber and Mr Lewis was an editorial suggestion, made regretfully to save space. The authors do, however, say in their letter that the references will be supplied on request.—ED, *BMJ*.

Hypertensive response to labetalol in phaeochromocytoma

SIR,—Dr G Reach and others (31 May, p 1300), reporting the effects of labetalol in three patients with phaeochromocytoma, conclude that the drug "could be useful in the preoperative management of patients with phaeochromocytoma provided the blood pressure was carefully monitored." Careful blood pressure recording is required since a paradoxical rise in blood pressure has been described in two patients with phaeochromocytoma following the oral administration of labetalol.[1 2] We have learnt caution with this drug by experiencing a similar case.

A 46-year-old man, who was admitted (day 1) with a supine blood pressure of 150/100 mm Hg rising to 260/150 mm Hg on standing, was shown preoperatively to have a phaeochromocytoma (24-h urinary conjugated metanephrine excretion 130 μmol; normal $< 3\ \mu$mol) that was subsequently confirmed at laparotomy. Supine blood pressure readings over the first four days of admission ranged from 110/70 mm Hg to 150/100 mm Hg with the exception of an isolated reading of 220/140 mm Hg. Oral labetalol was commenced on day 5 in a dose of 200 mg 8 hourly (600 mg daily) resulting in sustained hypertension (210/150 mm Hg), which was not abolished by increasing the dose of labetalol to 400 mg 8 hourly (1200 mg daily) on day 6 (280/160 mm Hg). Satisfactory blood pressure control was achieved by withdrawal of labetalol and the oral administration of phenoxybenzamine 10 mg 6 hourly (40 mg daily) from day 7 onwards (110/80 mm Hg). In addition propranolol was commenced from day 9 with no adverse effect on blood pressure control (120/80 mm Hg).

Hypertension in phaeochromocytoma may be aggravated by the administration of a β-adrenoreceptor blocker before adequate α-adrenoreceptor blockade is established.[3] While labetalol has both α- and β-adrenoreceptor blocking actions it has predominantly β-adrenoreceptor blocking activity.[4] Thus the administration of this drug may provoke a paradoxical rise in blood pressure in patients with phaeochromocytoma, particularly if the tumour is secreting adrenaline as its predominant catecholamine.[1] Labetalol should therefore be used with caution in patients with phaeochromocytoma and may have no advantage over conventional preparations with α-adrenoreceptor blockade followed by additional β-adenoreceptor blockade.

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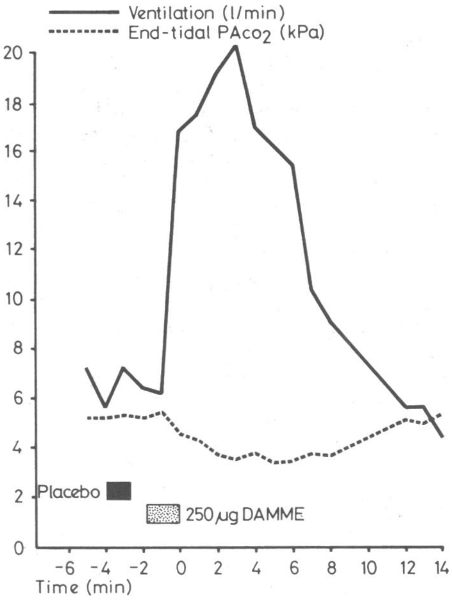
[1] Rosei EA, Brown JJ, Lever AF, *et al*. *Br J Clin Pharmacol (Suppl)* 1976;**3**:809-15.
[2] Briggs RSJ, Birtwell AJ, Pohl JEF. *Lancet* 1978;i:1045-6.
[3] Ross EJ, Prichard BNC, Kaufman L, Robertson AIG, Harris BJ. *Br Med J* 1967;i:191-8.
[4] Brittain RJ, Levey GP. *Br J Clin Pharmacol (Suppl)* 1976;**3**:681-94.

Asthma and enkephalin

SIR,—Dr R D G Leslie and others (5 January, p 16) reported five patients with asthma induced by chlorpropramide alcohol (CPA). This was partially blocked in one patient by a high dose of naloxone but in another there was no definite effect. In the former patient the enkephalin analogue DAMME (250 μg) produced tightness of the chest, breathlessness, and a fall in forced expiratory volume in one second (FEV_1), which had largely resolved by 20 minutes. They did not attempt to block this effect with naloxone. The one asthmatic patient tested experienced no change in FEV_1 with CPA or DAMME. They concluded that CPA asthma is mediated by enkephalins.

We have studied minute ventilation, FEV_1, and forced vital capacity (FVC) in six normal (non-asthmatic) subjects infused with 250 μg of DAMME. No change in FEV_1 or FVC was recorded in any subject. In one subject hyperventilation occurred in association with a feeling of oppression in the chest, breathlessness, and anxiety (fig). Hyperventilation may precipitate an asthmatic attack[1] and it is possible that DAMME-induced hyperventilation may be responsible for the fall in FEV_1 in the asthmatic patient of Dr Leslie and his colleagues. Enkephalin-induced asthma may therefore be no more than a non-specific consequence of anxiety and hyperventilation.

A feeling of oppression in the chest lasting 20-30 minutes in about 20% of subjects given DAMME 250 μg has previously been reported.[2 3] The facial flushing, analgesia, and hormonal effects which occur in nearly all subjects last about two hours. The time course of the fall in FEV_1 in the asthmatic patient reported therefore approximates better



Hyperventilation in a normal subject given the enkephalin analogue DAMME.
Conversion: SI to traditional units $PACO_2$:—1 kPa = 7·5 mm Hg.

to that of the feeling of chest oppression, anxiety, and hyperventilation. The case for CPA-induced asthma mediated by enkephalin-like substances is thus not proved. Nevertheless care should be exercised in giving DAMME to asthmatic subjects; but our initial results suggest that it is safe in normal subjects.

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[1] McFadden ER, Ingram RH. *N Engl J Med* 1979;**301**:763-9.
[2] Krebs E, Roubicek J. *Pharmakopsychiat* 1979;**12**:86-93.
[3] Graffenried B, Dol Pozo E, Roubicek J, *et al*. *Nature* 1978;**272**:729-30.

Effect of plasma exchange in rhesus immunisation

SIR,—Regarding the article by Mr G R Barclay and others (28 June, p 1569) on the apparent adverse effect of plasma exchange on anti-D production in rhesus immunisation, we do feel that their conclusions are unduly pessimistic. We have treated 18 similar cases with a 64% success rate (report on 14 in press).

It has been shown in animal experiments by Branda and his co-workers[1] that plasma exchange following secondary immunisation results in a permanent lowering of the antibody level. However, in a recently published article Branda[2] now states that when antigen is continuously present only a temporary lowering of antibody can be achieved, with a prompt return to high levels. This finding is significant with regard to the case in question.

The circumstances outlined by Mr Barclay and colleagues would strongly suggest that their patient was being regularly subjected to the Rh antigen and therefore the immunological response was "switched on." The Rh-positive antigen could well have got into the mother's circulation in the replacement plasma, as a result of the amniocentesis, or during intra-uterine transfusion.