prevent some of the life endangering problems highlighted in Dr Bion's original paper.¹

We frequently transport critically ill patients, intubated and ventilated, some 40 miles over winding country roads to a regional neurosurgical centre. The Lifestat 100 (Physiocal) non-invasive blood pressure monitor is designed specifically for use in the field or in a moving vehicle. In our experience this monitor has given reliable recordings when used under such adverse conditions. Since the most important role of the attending anaesthetist is to ensure an adequate supply of oxygen to the tissues, pulse oximetry is used routinely in the ambulance. Both the Biox 3700 (Ohmeda) and the Criticare 502 (Criticare Systems Inc) that we possess provide a continuous display of both oxygen and pulse rate.

The use of this equipment is such a practical means of monitoring during transfer that we would not consider undertaking transport of a critically ill patient without it.

T M CRAFT
P H CHAMBERS

Department of Anaesthesia,
Milton Keynes General Hospital,
Milton Keynes MK6 5LD

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Aspirin all round?

SIR,—Two reports have been published on the efficacy of prophylactic aspirin in preventing cardiovascular disease in British (30 January, p 313) and American doctors.¹ The results of these two studies conflict, and Professor Michael Orme discussed the possible reasons (30 January, p 307). We think, however, that a further aspect should be considered. The two studies differ in one major respect—the dosage interval. The American study used intermittent dosing—325 mg every second day—whereas the British study used 500 mg every day.

We think that the dosing interval is of major importance because of our findings concerning the synthesis of the two major cardiovascular prostanooids, thromboxane and prostacyclin. Both in healthy individuals and in patients with acute cardiovascular disease, such as acute myocardial infarction, a dose of 500 mg aspirin suppresses thromboxane production for at least three days.^{2,4} Prostacyclin production, however, is suppressed for only a few hours. This means that intermittent dosing with aspirin preserves the desired effect on thromboxane formation with minimal disturbance of prostacyclin production. The reason for this is aspirin's well known irreversible acetylation of cyclo-oxygenase, which inhibits thromboxane formation throughout the platelet's whole life span.⁵ In contrast, the endothelium can re-synthesise cyclo-oxygenase and could thereby quickly reverse the effects of aspirin.^{2,3}

Intermittent dosing of aspirin therefore has an important and probably beneficial effect on the cardiovascular system through suppressing thromboxane production with only minimal influence on prostacyclin production. This might have been important in the clinical outcome of the two studies.

PETER HENRIKSSON
KRISTER GREEN
OLE VESTERQVIST
OLOF EDHAG
GUNDARS RASMANIS

Cardiac Division,
Karolinska Institutet, Huddinge University Hospital,
S-141 86 Huddinge, Sweden

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Home visiting by consultants

SIR,—Dr Graham Mulley's leading article (20 February, p 515) reminded me that when I was appointed as a consultant physician five years ago I found domiciliary visits of value as they introduced me to the small number of general practitioners who attended with me, made me aware of the physical nature of the district where I work, and kept me in touch with the surroundings in which my patients live. It quickly became apparent that making domiciliary visits was less efficient than seeing patients in the hospital and I found that if I offered an appointment to patients who needed to be seen urgently within one to two days I cut my domiciliary visits from two a week to one a fortnight.

As the article hints, there are some savings for the health service from this but a financial loss to me. To be more specific, if consultant geriatricians average 211 visits in a year they will earn an extra £7068.50 and if electrocardiography is performed at half of these an extra £1767.12 is added. If the average is 211 per year some must be doing the permitted maximum of 300, which will earn £10 050 a year plus the rate for electrocardiography if appropriate. This will be added to pension rights if performed in the last year of service. These are large sums by any standards and those sitting in large hospital clinics will wonder if they should be out on the road, driving to what is often a relaxed interview at the patient's home. This financial incentive to see patients at home appears excessive and possibly unnecessary. Many of the points picked up by a home visit could be assessed by a district nurse or general practitioner and forwarded to the consultant.

PETER WILKINSON

Ashford Hospital,
Ashford, Middlesex TW15 3AA

SIR,—The excellent leading article by Dr Graham Mulley (20 February, p 515) points out that domiciliary visits by consultant geriatricians may reduce the need for hospital admission but are time consuming and expensive in terms of consultation fees. In west Newcastle the consultant geriatricians perform few domiciliary visits, and most home assessment visits are performed by a team of junior hospital doctors attached to the geriatric unit at Newcastle General Hospital. The team comprises two senior registrars and two registrars spending equal time training in geriatric and general medicine and two senior house officers in geriatric medicine. Requests for home visits from general practitioners are processed by a geriatric services coordinator, and most visits are made within 72 hours.

During 1985 the team carried out 272 visits, with the senior house officers making 136 visits, the registrars 59, and the senior registrars 77. The

outcome of the home visits was as follows: 47 patients required emergency admission, 33 needed routine admission, 15 were offered respite care, 106 were referred to day hospital, 25 were subsequently reviewed as outpatients, eight were referred to other agencies, and eight required a further home visit. Surprisingly, there were 30 patients who required no further follow up. The outcome of the visits by senior house officers did not differ significantly from that of the registrars or senior registrars (χ^2 test, $p > 0.05$).

Home visiting is an important element of training for junior medical staff attached to the geriatric unit, not only providing opportunities for the primary assessment of elderly patients and their carers but also increasing awareness of social, psychological, and physical interactions in the elderly. Home visiting by junior staff may be a cost effective alternative means of extending geriatric services into the community.

J M GRIMSHAW
R M FRANCIS
D M BEAUMONT

Department of Medicine (Geriatrics),
Queen Elizabeth Hospital,
Gateshead NE9 6SX

Health care in a deprived community

SIR,—Having received many laudatory letters and comments in recent weeks from colleagues in other primary health care teams about our strenuous efforts, and to some extent success, in improving the preventive health care of a deprived community in Cleveland (16 January, p 173) we were extremely interested to read the critical letter from colleagues serving similar deprived communities in Wales (20 February, p 569). They felt uncomfortable about some aspects of our paper, and their discomfiture seemed to orientate around four major points.

Firstly, Dr Jonathan Richards and colleagues imply that our work would be welcomed by supporters of the government's white paper on primary care. That our servants at Westminster have chosen to promote (and we hope adequately fund) preventive aspects of primary care gives us cause for celebration since they are merely furthering concepts and trends that developed primary health care teams are already instituting and attempting to make them universal.

Secondly, Dr Richards and colleagues say that they cannot claim our success. We can offer little immediate comfort here since the work needed from the whole team (but especially the practice nurses and health visitors) to improve preventive care will be enormous, as ours has been. There is relief on a more distant horizon, however, when team members are no longer visiting patients with morbidity inducing whooping cough and measles in deprived settings or shouldering the burden of care of young hemiplegics or the ghastly preterminal and terminal care of patients with carcinoma of the cervix.

Thirdly, Dr Richards and colleagues claim that we have pressurised our deprived community into conforming to the higher standards of preventive care of their more endowed counterparts, even denying these families the "freedom . . . to decline the professional advice of the primary health care team." If the Merthyr Tydfil doctors study the seven "methods" that we used to improve the uptake of preventive care they will see that five of them were operational changes and implementation of new systems that involved change by, and even pressure on, team members rather than our patients. The more we familiarised ourselves with the gaps in preventive care the more it appeared that it was our inappropriate system of providing

care that was responsible for the poor uptake. It was not due to massive apathy or recalcitrance, which we must confess, probably with other GPs, had been our mistaken impression in earlier years. Of the other two methods, one was the postal provision of data about their preventive care (much appreciated by the families incidentally), and only the other, the visiting of families in their homes, could be interpreted as "pressure." But all such visits had been carefully prearranged after much counselling by sensitive health visitors using non-judgmental negotiating skills and always respecting the right of the patient to decline. That some did is evidenced by our total lack of 100% scores.

Finally, the authors accuse us of making the less compliant families unpopular because they upset the statistics. Nothing can be further from the truth. The small number of non-compliers—a few of whom are persistently "apathetic" and "recalcitrant" despite all our efforts (as indeed are some of their endowed controls)—are accepted by us as they are: we will not stop counselling them, we will hope to become more adept at it, and ultimately more successful, but in the mean time they will be no less popular; if anything the very reverse in that they provide a continuing challenge to us.

Dr Neil Colman (20 February, p 574) was worried about confidentiality with regard to contraception in our preventive care letters to households. We gave this much thought, and in fact did not mention contraception in the letters but almost always raised this topic when a patient came for other preventive care measures. We were also extremely cautious and carefully scrutinised records before mentioning cervical cytology in teenagers.

We hope these comments relieve the anxieties of our critics.

G N MARSH
D M CHANNING

Norton Medical Centre,
Stockton on Tees,
Cleveland TS20 1AN

Tardive dyskinesia

SIR,—In my article on tardive dyskinesia (16 January, p 150) I did not address the complex implications of the existence of similar abnormal movements in untreated psychotic patients.^{1,2} Dr Trevor Turner raises this issue (5 March, p 719), apparently rehearsing the argument that tardive dyskinesia is not drug related but a feature of psychotic illness.

He offers descriptions of abnormal movements in psychiatric patients before the advent of antipsychotic drugs as evidence of tardive dyskinesia, but such historical reports require careful interpretation. Mackay³ and Marsden⁴ considered that the movements generally described were different in character from those of tardive dyskinesia. Furthermore, the terminology is confused, reflecting various notions of the cause of the motor phenomena observed.^{2,4} In addition, the patient population almost certainly included patients with organic disease such as encephalitis lethargica and syphilis, which can present with both psychotic features and motor disorder.^{2,5}

An element of the historical perspective Dr Turner may have neglected is the number of reports that appeared soon after the introduction of antipsychotic drugs clearly describing tardive dyskinesia as a novel syndrome.⁶ The following decades have seen an increase in the prevalence of the condition accompanying the widespread use of these drugs,⁷ a finding not easily dismissed as simply a manifestation of increasing awareness of tardive dyskinesia.

Neither of the two studies cited by Dr Turner refers to the abnormal movements identified as tardive dyskinesia. One of the investigations compared inpatients with chronic schizophrenia with and without a history of antipsychotic drug treatment and found a similar prevalence of abnormal involuntary movements.⁸ While this finding confirms the occurrence of spontaneous movement disorder in schizophrenia, the contribution of drug treatment is acknowledged in that "exposure to neuroleptics produced a striking increase in both the prevalence and severity of abnormal movements."⁹ Grouping movements into clinically recognisable syndromes showed a particular susceptibility to orofacial dyskinesia in the drug treated patients.

My article referred to advancing age and particular features of the schizophrenic illness as risk factors for the emergence of tardive dyskinesia. Antipsychotic drug treatment may interact with the disease process and age related cerebral deterioration to hasten or provoke the appearance of abnormal movements.¹⁰ Similarly, Dr Turner states that tardive dyskinesia results "primarily from the intrinsic disease process itself, mediated by dopamine antagonists." The specificity of such theories for schizophrenia, however, is challenged by the emerging evidence of increased susceptibility to early onset tardive dyskinesia in patients with affective disorder receiving antipsychotic drugs and the occurrence of tardive dyskinesia in non-psychotic patients treated with these drugs.¹⁰

In addition to tardive dyskinesia, schizophrenic patients taking antipsychotic medication may show other drug related movement disorders such as dystonia and akathisia. They can also exhibit stereotypies and mannerisms; a host of other abnormalities of posture, tone, and motor activity⁴; and possibly more subtle disturbances of motor function,¹¹ all thought to be related to the illness. The distinction between these types of movements, and, more fundamentally, the dissociation of the effects of drug treatment from any specific contribution of the schizophrenic illness, is relevant to the important research and legal issues mentioned by Dr Turner. Progress in this subject, however, is unlikely to be advanced by adopting his rather indiscriminate application of the term tardive dyskinesia. The term should be confined to orofacial dyskinesia and choreiform trunk and limb dyskinesia where this has developed in association with the administration of dopamine antagonist drugs.

THOMAS R E BARNES

Department of Psychiatry,
Charing Cross and Westminster Medical School,
London W6 8RP

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Dyskinesia—research and treatment. Berlin: Springer Verlag, 1985:79-87.

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SIR,—In commenting on the leading article by Dr Thomas Barnes (16 January, p 150) Dr Trevor Turner (5 March, p 719) criticises its statement that tardive dyskinesia is a disorder of movement caused by neuroleptic drugs. He buttresses his dissent by referring to historical and more recent studies indicating involuntary movements in psychotic patients never exposed to such treatment. Curiously, both authors cite my own work in support of their contradictory analyses.¹ Neither appears to have appreciated its fundamental point.

Research in tardive dyskinesia is one of the few areas of scientific inquiry where experimental studies are entertained when they fail to include what is usually considered to be an essential, but here virtually unobtainable, control group—that is, in this case, one of indistinguishable patients in whom psychotic illness has run its course in the absence of treatment with the presumed offending neuroleptic. The available evidence indicates that the baseline level of involuntary movement disorder in untreated psychotic illness has been underestimated, particularly in patients with severe chronic disease. Evidence also indicates, however, that this unappreciated baseline can be further raised by long term exposure to neuroleptic drugs. The likelihood of involuntary movements appears to increase with the extent of cognitive impairment, both in treated and in untreated populations, suggesting some common association with abnormalities in cerebral structure.^{1,2}

Our view is that neuroleptics may not be the "cause" of tardive dyskinesia. Rather, they seem to hasten and exacerbate the appearance of involuntary movements among patients who would probably manifest such movements spontaneously.

JOHN L WADDINGTON

Department of Clinical Pharmacology,
Royal College of Surgeons in Ireland,
Dublin 2, Eire

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Microvascular fragility at high altitude

SIR,—During a recent expedition to the Himalayas we investigated microvascular fragility by the technique of mucosal petechiometry, as used by Hunter *et al*¹ and Dr J S Milledge and others (27 February, p 610). A 1 cm suction cup was applied at a negative pressure of 200 mm Hg to the buccal mucosa of the lower lip for one minute, and the petechiae produced were counted. In 23 subjects (21 men, two women, age 23-60) there was a significant increase in petechiae after a five day ascent from 3800 m to 4900 m (mean 1.8 v 6.5, $p < 0.02$ Wilcoxon's signed rank test). The number of petechiae increased in 14 subjects, decreased in five, and was unchanged in four.

There was no association between the number of petechiae and symptoms of altitude sickness or arterial oxygen tension. One person in whom the number of petechiae increased from one to 20 had a single retinal haemorrhage; there were no other retinal haemorrhages. The presence of nail