haemorrhages at high altitude⁷ was not related to microvascular fragility; at 4900 m splinter haemorrhages were seen in four climbers with less than 15 petechiae and in none of four with more than 20 petechiae. Nail and retinal haemorrhages were not confined to those suffering from acute mountain sickness.

In our study microvascular fragility did appear to be a feature of high altitude exposure, supporting the view of Hunter *et al.*¹ The slow ascent and greater altitude attained by our expedition members may account in part for the differences between our results and those of Dr Milledge and others. In eight of our climbers tested at sea level the mean petechiae count was one and no increase in petechiae was observed after ascent to 3800 m. In the authors' group a mean petechiae count of 11.5 was seen at 1300 m altitude; no sea level data were presented.

In the absence of an association with altitude sickness symptoms or arterial oxygen tension it is unlikely that increased microvascular fragility is implicated in the pathogenesis of acute mountain sickness. Microvascular fragility may be a characteristic of certain people at high altitude rather than characteristic of high altitude sickness.

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1 Hunter DJ, Smart JR, Whitton L. Increased capillary fragility at high altitude. *Br Med J* 1986;292:98.

2 Heath D, Williams DR. *Man at high altitude*. Edinburgh: Churchill Livingstone, 1977.

Crohn's disease presenting as anorexia nervosa

SIR,—The title of the paper by Drs A P Jenkins and others (5 March, p 699) is misleading, because the patients they describe did not suffer from anorexia nervosa and did not present with symptoms of anorexia nervosa. They point out that the American Psychiatric Association's *Diagnostic and Statistical Manual* includes five criteria for the diagnosis of anorexia nervosa but did not add that all of these are required. The four cases reported satisfied only one of these criteria—weight loss, which is a symptom of very low diagnostic specificity. It can occur in a wide range of physical disorders which may present in young women, and in addition to Crohn's disease these include, for example, malignant disease, diabetes, and pulmonary tuberculosis. It is also a common symptom in other mental illnesses, particularly depressive disorders.

The other criteria for diagnosis, particularly disturbance of body image and refusal to maintain body weight, which involves extreme voluntary measures to lose weight, have much more diagnostic specificity and are not symptomatic of any physical disease or other psychiatric disorder. They can usually be elicited by taking a careful history from the patient and also close family members and, if doubt remains, by careful observation of the patient's behaviour.

In the first two cases reported there is no mention of these cardinal features and presumably they were not looked for. In the third case it is said that there were "no food phobias and she saw herself as thin," and in the fourth case it is said that she "did not have a distorted body image." Thus there is no reason to suppose that any of these patients had symptoms of anorexia nervosa. Three of them are said to have presented with a history of "anorexia": despite the name anorexia nervosa is

not characterised by poor appetite, although this may develop in advanced stages, and about half of all patients exhibit binges of overeating.

As doctors we tend to fear that the occasional physical disorder may be missed, even when there is no evidence for it on the basis of history and examination, and overinvestigation facilitates the process of somatisation of mental disorders, which is only now beginning to be recognised as a widespread problem in medical practice. The search for primary organic diseases in patients with anorexia nervosa delays recognition of the disorder and helps to endorse the views of patient and family that the main problem is physical. By contrast we do not appear to show equal concern about the possibility that mental illnesses may be missed when patients present with somatic complaints.

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The need to make rugby safer

SIR,—While spinal injuries from rugby are a cause for concern (16 January, p 149; 13 February, p 498; and 5 March, p 719), particularly when there are avoidable factors, these injuries should be viewed in perspective. Other sporting pursuits can be much more hazardous.

In our spinal injuries unit there have been only two admissions in the past 10 years because of injuries from rugby. There have, however, been 12 admissions after recreational diving accidents, five of which occurred while the victim was abroad on holiday. Most of these injuries were at unsupervised pools, beaches, or open water. Nine of these patients suffered complete spinal injuries, and three incomplete injuries, but all were at a high (C4-6) level. Our experience in this respect is similar to that of Young and Northup,¹ who also indicate that diving injuries are much more common than rugby injuries.

A public education campaign is urgently required to emphasise the dangers of diving in unknown territory, particularly abroad and on holiday, when alcohol may play an important part, to try to reduce the incidence of these tragic injuries.

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1 Young JS, Northup NE. Statistical information pertaining to some of the most commonly asked questions about spinal cord injury. *Science Digest* 1979;1:17.

Diabetes and pancreatic cancer

SIR,—Professor R C N Williams (13 February, p 445) suggests that the recent onset of diabetes or a sudden increase in the requirement for insulin in a diabetic should raise the suspicion of cancer of the pancreas and may offer a clue to early diagnosis. That last claim, however, is unfortunately not supported by our experience.

A retrospective examination of the files of all patients with proved pancreatic carcinoma diagnosed at our hospital in 1973-83 showed diabetes (two or more sequential fasting venous blood glucose concentrations of ≥ 7.2 mmol/l) in 45 out of 74 patients compared with only eight out of 70 matched patients with carcinoma of the colon ($p < 0.005$). In 10 patients diabetes had been diagnosed more than two years before the pancreatic tumour (range 64-120 months, mean

64 months), in 11 less than a year before (range 0.5-12 months, mean 3.3 months, median 3 months), in 15 concomitantly, and in nine after the tumour was found (mean 5.1 months). All patients had locally unresectable or metastatic disease at the time of diagnosis. Most of the patients with recent, possibly tumour related, diabetes had only mild to moderate hyperglycaemia (less than 11.1 mmol/l in 23 of 35 patients) and many were treated by diet alone. No relation was found between the development of diabetes and either the site of the tumour in the pancreas or its estimated size.

Thus the well known association between pancreatic cancer and the development of diabetes¹ is even more striking in an Israeli population, but the belief that awareness of this association can not only serve as a diagnostic clue in patients with unexplained pain, weight loss, or jaundice but also aid an "early" diagnosis of potentially resectable disease seems overoptimistic, at least for the population we examined. The appearance of diabetes associated with pancreatic cancer usually heralds the more common and ominous signs by too short a time (median three months) to have any real impact on the patient's prognosis.

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1 Schwartz SS, Ziedler A, Moossa AR, Kuku SF, Rubenstein AH. A prospective study of glucose tolerance, insulin, C-peptide and glucagon responses in patients with pancreatic carcinoma. *Am J Dig Dis* 1978;23:1107-10.

Early growth delay in diabetic pregnancy

SIR,—Dr Minna Bloch Petersen and others (27 February, p 598) suggest that children with a history of a first trimester crown-rump length smaller than expected from the menstrual history in diabetic pregnancy have an increased risk of developmental delay, although their results were significant only for language and speech ($p = 0.009$). It has previously been suggested that these fetuses may have experienced growth delay in very early gestation and have an increased risk of congenital malformation.¹

The existence of retarded fetal growth in very early gestation is difficult to prove unless the time of ovulation is known. Other studies^{2,3} have shown that "early fetal growth delay" occurring in four of 11 diabetic pregnancies was explained by delayed ovulation (as judged from salivary progesterone levels) and that ultrasound measurement (of biparietal diameter between 12 and 18 weeks' gestation) is significantly better than an optimal menstrual history in predicting the date of delivery.^{2,3} We have found that in 56 insulin dependent diabetic mothers with certain menstrual data, regular 28-30 day cycles, and no recent contraception 11 fetuses had a crown-rump length (measured in the first trimester) which was six or more days less than expected from the menstrual data. Among these 11 fetuses the discrepancy between ultrasound and menstrual ages was explained in two by anencephaly; the other nine were normal and healthy at delivery with birth weights > 2500 g. Of the 41 fetuses whose ultrasonic and menstrual ages agreed, two had major cardiac malformations, three aborted spontaneously, and the remainder were normal and healthy at delivery with birth weights > 2500 g. Our nine fetuses with "early growth delay" continued to grow at the rate expected had their ultrasonic ages been correct, as did those in the study of Dr Bloch Petersen and others, suggesting that, despite the rigorous inclusion criteria, ovulation in these pregnancies

occurred later than assumed. Would their present findings have been the same if the ultrasonic age had been accepted and prematurity allowed for?

A crown-rump length which is smaller than expected in diabetic pregnancy is more likely to be due to delayed ovulation than to a temporary delay in fetal growth in very early pregnancy. Such a finding, however, should be followed up by expert ultrasonic assessment in the second trimester to establish the true gestational age and exclude fetal abnormality, particularly anencephaly.

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- 1 Pedersen JF, Mølsted-Pedersen L. Early fetal growth delay detected by ultrasound marks increased risk of congenital malformation in diabetic pregnancy. *Br Med J* 1981;283:269-71.
- 2 Steel JM, Johnstone SD, Corrie JET. Early assessment of gestation in diabetics. *Lancet* 1984;ii:975-6.
- 3 Warsof SL, Pearce JM, Campbell S. The present place of routine ultrasound screening. *Clin Obstet Gynecol* 1983;10:445-57.

Achieving a Balance—a time for action

SIR,—My old friend Professor John Salaman rightly points out (5 March, p 717) the disgraceful bottleneck that exists among our surgical registrars, who, in spite of higher qualifications and immense experience, cannot obtain promotion to senior registrar status. However, I cannot understand his solution, which is to curtail considerably the numbers of registrars. Certainly this would indeed cut down further entry of trainees into the arena but surely it would do nothing for the current stockpile of senior registrars and registrars in general surgery. There is only one solution for that—a substantial increase in the number of surgical consultant posts. This has been promised by our masters as a reward for cutting our numbers of senior registrars, but scanning the advertisement pages of the *British Medical Journal* each week fails to show the slightest evidence that this is taking place.

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SIR,—At a recent meeting in Bristol to discuss with officials from the Department of Health and Social Security the “plan for action” on *Achieving a Balance* it was made quite obvious that the government has no plans to cope with the financial implications of the 2% consultant expansion. The old myth was produced again that consultants, by increasing throughput, will be so cost effective that the scheme will be self financing. The government seems unable, or unwilling, to grasp the message that treating more patients costs more money even though the cost per case falls.

Many districts will probably not be able to fund the increased number of consultants, and it is absolutely vital that the reduction in junior staff levels is not allowed to occur unless and until consultant expansion occurs. It was pointed out at our last medical staff committee meeting that since registrars work an average of 80 hours a week each one needs to be replaced not by one consultant but by two. At a time when junior staff hours are being reduced it is ludicrous to expect consultants to

double their working hours. It is ludicrous also to expect consultants to operate all night and still turn up to do a full day's work in the operating theatre or outpatient clinic. It is not only junior doctors who may make mistakes when tired.

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The Shaw report

SIR,—Dr Eric MacDonald's letter (12 March, p 791) on the Scottish report *Staffing the Service* makes a number of suggestions which fall outwith the remit of the report's steering committee but some of which deserve comment.

Dr MacDonald clearly dislikes the variety of intermediate grades which exist at present, but he should realise that, with all their faults, they were achieved after long periods of negotiation and bargaining between the profession and its employers. Nor is there today strong evidence that large numbers of our professional colleagues would accept his alternative proposals for such grades; indeed, he is in cloud cuckoo land if he believes either that registrars would be happy or well trained without a service contribution or that his proposed general practitioner/intermediate grade doctors would readily accept “out of hours resident sessions.”

I must remind Dr Macdonald, as I have had to do with many of my colleagues, that our report differs from its English counterparts in that it is a document drawn from evidence obtained from seniors and juniors of all specialties in all hospitals. This makes it a consensus view, with all the weaknesses which that implies. But it is also its strength and I would hazard a guess that this makes its implementation more likely than Dr MacDonald believes.

As to his comments on consultants, the steering committee would have agreed with him on one point at least, that they would have wished to recommend more than the final numbers suggested by the report. But he can argue that with his consultant colleagues, and nowhere more than in his own specialty.

Nevertheless, while I disagree with Dr MacDonald on many points in his letter, he presents several thought provoking ideas and I would be the last to discourage anyone who believes he has a better solution than ours.

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Points

How are head lice transmitted?

Dr AIDAN MACFARLANE (Community Health Offices, Radcliffe Infirmary, Oxford OX2 6HE) writes: Dr P K Buxton says that “head lice are transmitted via combs, brushes, and hats” (13 February, p 489). All the teaching at the moment is that head lice are transmitted only from head to head contact. I would be most grateful if Dr Buxton could give us his source of information because—if what he says is true—it has considerable implications for what is undoubtedly a common and perplexing problem.

Dr E R M SAUNDERS (Gwent Health Authority, Newport NP9 4PA) writes: Dr P K Buxton perpetuates the idea that head lice are transmitted by combs, brushes, and hats. A head louse off a human head is either dying or doomed and the only method of spread is by head to head contact.

Dr P K BUXTON (Department of Dermatology, Royal Infirmary, Edinburgh) replies: Arthur Rook states that pediculosis capitis is transmitted by “shared hats, caps, brushes and combs.”¹ In his definitive treatise *The Louse*, Professor P A Buxton does not actually state how head lice are transmitted but in the exhaustive study of their life cycle he found that head lice died of old age after living for 34 days, for 31 of which they lay nine eggs a day? The lice he obtained from the scalp hairs were very much alive and were able to live for some weeks in a suitable environment in the laboratory—so they were not “dying or doomed.” There is no doubt that Professor Buxton considered head wear to be implicated in transmission; he states, “In treating an infective head do not forget to sterilize the hat.”² Likewise, Orkin *et al* clearly state, “Pediculosis capitis may be communicated by shared hats, combs and brushes.”³ Experimental data to settle this point would clearly be of value.

- 1 Rook A, Wilkinson DS, Ebling FJG, Champion RH, Burton JL. *Textbook of dermatology*. 4th ed. Oxford: Blackwell, 1986:1046.
- 2 Buxton PA. *The louse*. London: Edward Arnold, 1939.
- 3 Orkin M, Maibach HF, Parish LC, Schwartzmann RM. *Scabies and pediculosis*. Philadelphia: J P Lippincott, 1977.

Postviral fatigue syndrome and mobility allowances

Dr STEPHEN T GREEN (Clinical Department of Infectious Diseases, Ruchill Hospital, Glasgow G20 9NB) writes: Dr Anthony David and others (5 March, p 696) conclude that there are major problems with the definition and understanding of postviral fatigue syndrome. As profound fatigue is a prime feature of the condition mobility is often impaired, occasionally to a disabling degree, and in such circumstances many of the patients I have been concerned in counselling and treating have applied to the DHSS for mobility allowances, with an almost universal lack of success. One reason for this appears to be a lack of understanding or knowledge within the DHSS about the postviral fatigue syndrome. While many problems wait to be resolved, particularly regarding the development of firm diagnostic criteria, those of us who see patients who have been assigned to the “postviral fatigue syndrome compartment” are in little doubt that at least some of them suffer from very real illness. Thus I await the establishment of hard diagnostic criteria for the postviral fatigue syndrome which could be applied to everyone, including the DHSS.

Aspirin and prostacyclin

Dr GEOFFREY P WALSH (Blackburn) writes: The discrepancy between the results of the American low dose (one tablet per two days) aspirin trial and the higher dose (two tablets per day) British trial may, as Professor Michael Orme suggests (30 January, p 307), be due to the low dose aspirin's saving of prostacyclin (PGI₂). Could this explain the worldwide largescale differences in coronary heart disease rates, as shown particularly by Japan, where essential fatty acid consumption, mainly in the shape of eicosapentaenoic acid in oily fish, enhances prostacyclin at the expense of platelet thromboxane and may add its own rather weak prostacyclin (PGI₃)? Furthermore, because prostacyclin seems to guard against excessive platelet deposition in the vessel wall it may have a role in preventing atheroma that is denied to aspirin. Duguid suggested that atheroma might be initiated via the incorporation of thrombi into the intima rather than via intimal permeability,¹ and animal experiments have shown an 80% reduction in the extent of coronary atheroma using cod liver oil (B H Weiner *et al*, unpublished). Because the eicosapentaenoic acid in oily fish forms only a weak substrate for cyclo-oxygenase, however, whereas aspirin is so effective, might aspirin and eicosapentaenoic acid used together be more efficient and provide protection against both thrombosis and atheroma formation?

- 1 Duguid JB. Thrombosis as a factor in the pathogenesis of coronary atherosclerosis. (*Journal of Pathological Bacteriology* 1946;58:207-12.