machine which she peddled with her right foot using her left foot to work the brake. This machine was installed in her home during the first world war and she continued to produce wrist bands in this manner until the early 1950's.

The absence of Paget's disease in her left leg and, indeed, the rest of her skeleton strongly suggests that the recurrent physical trauma associated with some 40 years of peddling her treadle machine has predisposed the right leg and right side of the pelvis to this disease. Subsequently she has developed severe osteoarthritis of the right hip necessitating a total hip replacement and a degree of high-output heart failure secondary to the Paget's disease.

We therefore concur with the hypothesis that repeated physical trauma predisposes to the onset of Paget's disease in the affected limbs.

My thanks are due to Dr C Romer for drawing my attention to this case.

T M GASPER

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Epilepsy

SIR,—I was interested to read the article on epilepsy (14 April, p 1000) and especially interested to note the tragic fate of the unfortunate patient—suffocation in his pillow during a major seizure.

For some years I have been advocating the use of a special pillow in cases of grand mal epilepsy—especially in teenage and young epileptics.¹ Such tragic deaths are not uncommon. This is the tenth case of which I have personal knowledge, adding one more to the eight reported in July 1978 and another about whom I learned in August 1978.

The only manufacturers of such pillows are Melco Products Ltd, of Tottington, Near Bury, Lancashire.

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¹ Wilson, J B, *British Medical Journal*, 1978, 2, 200.

Mercury battery ingestion

SIR,—The ingestion of mercury batteries probably occurs more frequently than is documented and Mr D T Reilly in his report (31 March, p 859) recommended early operation to avert potentially fatal poisoning. Our experience with one such case raises several questions.

A 1-year-old girl swallowed a mercury battery (Mallory Px 625 dry cell, contents similar to those described in the report). She was observed, appeared to develop an upper respiratory tract infection, and vomited 12 hours later. She began passing dark grey stools 24 hours later and at this stage an x-ray revealed an intact battery lying within the stomach. A laxative was given to hasten the passage of likely intoxicants released from the battery. Reassurance from the makers was obtained by telephone that the battery was inert and ought to resist dissolution but that, should this occur, the contents were unlikely to form toxic components. Vomiting was episodic and at 48 hours a second x-ray showed the battery had separated into two discs, one still in the stomach, the other having progressed to the ascending colon. Maxolon was administered and Isogel was given to increase bulk and hopefully to "encase" the now sharp-edged discs, which could have lacerated the pylorus and gut wall. The baby ceased vomiting, developed colic, and continued to pass dark grey stools; and by 72 hours one half of the battery appeared, followed shortly by the second. Neither serum nor urine levels of mercury had been checked. After a week of anorexia and some lassitude an apparently

total recovery followed, and now at the age of 6 she is normally developed, active, and intelligent.

Several points deserve comment. Firstly, it must be emphasised that these batteries are in increasing use in cameras, fancy key-ring flashlights, etc, and must be kept well out of reach of children. Secondly, the nickel-plated casing would not have corroded sufficiently quickly to account for stool discolouration, which simply suggests release of battery contents, as confirmed by the in-vitro test described in the report. Radiological evidence of an "intact" battery is therefore unreliable. According to information received, the amount of mercuric chloride formed would decrease with increasing usage of the battery, as discharge of the dry cell reduces the amount of mercuric oxide available to form the chloride.

Absorption of mercury was not monitored in the case described here and, although all is well, one is left with an abiding concern that toxic damage to various organs might have occurred over the three-day period. If this is so, can it be confirmed, quantified, and the likely long-term consequences predicted? The possibility of poisoning and the mechanical difficulty of negotiating a tiny pylorus would certainly indicate operative removal. However, in Mr Reilly's report the absence of toxicity over a period exceeding 24 hours and in our case the clinical normality six years later, despite ample opportunity for intoxication, may in fact permit a conservative approach to be adopted.

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Differences between Leeds fractures and London fractures?

SIR,—Drs R Wootton and J Reeve (14 April, p 1017) have questioned the statistical validity of the difference in plasma 25-hydroxy vitamin D (25-OHD) level between our femoral neck fracture patients and controls and they suggested that we should apply a logarithmic transformation to our data (3 March, p 589). Unfortunately, this is impossible because of the zero values in both our groups. The non-parametric Wilcoxon sum rank test is appropriate, however, and its application to the data yields a P value of less than 0.00001. This compares with P values of <0.001 with the Student's *t* test and <0.0001 by the χ^2 test. Your correspondents can therefore rest assured that the difference between the two groups, which is so apparent to the naked eye, is statistically valid.

As to why the data from Northwick Park are so unlike those from Leeds, the most likely explanation is in the choice of controls. We are not given details of the elderly subjects admitted for cold orthopaedic surgery to Northwick Park Hospital but they are likely to include a high proportion of relatively immobile subjects with joint disease whose exposure to sunlight is restricted. Our controls, on the other hand, were randomly selected from a random sample of the population of Leeds living in their own homes. As over 90% of our fracture patients were also admitted from their homes, the two groups are comparable.

In this connection, it may be relevant that, whereas our control data are almost normally distributed, the Northwick Park data are, in your correspondents' own words, "skewed." This in itself suggests that their control series

is not in fact a normal group but, like their and our fracture groups, pathological.

As Drs Wootton and Reeve say, our data are compatible with the hypothesis that vitamin D deficiency contributes to fractured neck of femur in the elderly in Leeds; we cannot accept that they have provided evidence to the contrary in London.

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Microscopic words

SIR,—Dr B J Freedman in his letter (14 April, p 1021) reveals himself as the erudite author of your enjoyable feature "Words." Unfortunately, however, he perpetuates a common error in restricting Robert Hooke's use of the word "cell" to the empty variety that makes up cork. As I pointed out in the original version of my letter, which was published abridged in "Points" (24 February, p 559), Hooke¹ described not only the dead cells of cork, but also the living entities, "for, in several of those Vegetables, whilst green, I have with my *Microscope*, plainly enough discover'd these Cells or Poles (sic) fill'd with juices, and by degrees sweating them out."

Clearly Hooke's "cells" fulfilled Dr Freedman's stringent criteria for the use of the word in its modern biological sense.

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¹ Hooke, R, *Micrographia: or some Physiological Descriptions of Minute Bodies made by Magnifying Glasses*, p 116. London, The Royal Society, 1665.

Unusual reaction to monoamine oxidase inhibitor

SIR,—I had an outpatient who was an industrial chemist involved in devising new dressings or fillers for paper, and was therefore exposed to vapour, mist, or spray from his reagents. He had become despondent about his progress, and I prescribed phenelzine, 15 mg three times a day. He understood the dietary restrictions, and disclosed that cheese did not agree with him anyway. This may have been an unheeded warning to me. When seen again he was very well, his research being successful.

He had been so drug-compliant that he had observed dietary restrictions for a full 24 hours before his first and only dose, but within minutes he felt malaise and nausea. Half an hour later he must have been shocked, his wife remarking how grey and pale he looked; four hours later he had a "blinding headache," which passed off. He did not contact any doctor, and so the opportunity of checking on his clinical state, urinary metabolites, and serum enzymes was lost.

Reflecting that this reaction suggested that all his metabolic amine detoxification systems must be saturated, and that his research might have utilised higher amines for their known antiseptic and surface tension activity, I asked about his reagents. He wrote that one of them was a substituted propane diamine acetate, the second a tertiary amine, and the third a substituted imidazoline acetate.

I suggest that in unusual circumstances

when prescribing monoamine oxidase inhibitors we should not only consider dietary amine intake.

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Air embolism after removal of intravenous catheter

SIR,—I was interested to read the report from Dr Sheena M Ross and others (14 April, p 987) concerning a case of fatal air embolism occurring after the accidental removal of a central venous catheter. I suggested that air embolism might occur via the introducing cannula last year¹ and unfortunately my prediction has proved correct. There have now been about 34 reported cases of serious air embolism occurring when central venous catheters are used and the common cause at the present time is accidental disconnection of the administration sets from the catheter hub. The complication has been noted during dressing changes and also the insertion procedure. Ordway² has shown in animals that fatal quantities of air can rapidly enter the venous circulation through fine-bore cannulas.

I think that the introducing cannula should be withdrawn clear of the skin in order to remove the hazard of air embolism and to minimise the risk of infection from the sequestered blood lying between the central venous catheter and the introducing cannula. Finally, air embolism has been reported to occur via the track of a recently removed catheter³ and an occlusive dressing should be placed over the wound when catheters are removed.

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¹ Peters, J L, and Armstrong, R, *Annals of Surgery*, 1978, 187, 375.

² Ordway, C B, *Annals of Surgery*, 1974, 179, 479.

³ Paskin, D L, Hoffman, W S, and Tuddenham, W J, *Annals of Surgery*, 1974, 179, 266.

Motorcycle accidents

SIR,—I wish to take issue with Dr J G Avery (10 March, p 686) and his interpretation of motorcycle accident figures.

I submit that it is meaningless to apply the figures for the United States to Britain as driving conditions and habits there are very different from those in this country (for example, there is no limitation on the size of a motorcycle purchased by a novice save his resources, and he may buy an enormously powerful, unstable, and almost frighteningly vulnerable machine with little or no experience). It is not therefore surprising that accidents of primary effect, such as loss of control while cornering, are comparatively common. In England, however, there is a limitation on the capacity of the motorcycle one may buy until one has passed the test, providing a small benefit in terms of safety; far greater safety is inculcated by taking the RAC/ACU (Royal Automobile Club/Automobile Union) training course; this, sadly, is not compulsory.

A fairly recent survey¹ indicated that, of all accidents involving motorcyclists aged 17 and over, blame of any sort could be attributed to the motorcyclist in less than 33%. In another survey,² reported in this journal (6 January, p 39), one-third of the motorists actually

failed to see the motorcycle. What is more, of the 120 accidents examined in detail, 31 were caused by another vehicle leaving a minor road into the path of the motorcyclist and another 13 were caused by another vehicle turning across the path of an oncoming motorcyclist—in neither case, patently, could any blame be attached to the unfortunate rider. These two types of accident contributed almost three times as many to the total as the next major cause, loss of control while cornering, and were six times more frequent than the number of crashes caused by a motorcyclist turning into the path of another vehicle. This sheds a different light on Mr Eugene Hoffman's "menace on the roads" (p 686); and Dr Avery's comment that there seems to be no other method than helmet compulsion for "significantly reducing the carnage" seems positively pathetic in its inadequacy.

The fault does not lie with the poor, much-maligned, and all-too-often dead or seriously maimed motorcyclist, and it does not lie with the legislator who believes that polyurethane and fibreglass cocoons immunise against C3-4 dislocations (the commonest cause of death in instantly fatal accidents is cervical cord rupture). It lies with the multitudes of motorists of questionable visual ability who notice nothing less than five feet across. I'm not surprised that the poor motorcyclist has had enough. He was been monumentally long-suffering, he has donned his helmet, turned a tasteful day-glo orange (new colour seen recently—nausea green), dutifully put his headlamp on permanently, and swathed himself in reflective tape. All to no noticeable avail.

Legislation that compels people to do things must be bad legislation—if I wish to take up an NHS bed being rescued from my own Bacchanalian or nicotinic excesses that is "socially acceptable," but should I choose a motorcycle (primarily a fuel-and-resources-efficient way of transport, let us not forget) parliamentarians obediently rear up like Bateman's horrified guardsmen. I hope that it is clear from the foregoing that the answer lies in re-educating the car driver by making the driving test more stringent, so that people are more aware of the smaller vehicles on the road. To be fair, I would like to see the RAC/ACU test made compulsory since the Government seems unlikely to set up a sensible training scheme.

So the answer? Train everybody better; and, incidentally, let us have some action on drunken driving—a motorcyclist friend of mine was in an orthopaedic ward for three months after being mown down by a drunk driver travelling the wrong way round a roundabout.

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¹ Transport and Road Research Laboratory, *Motorcycle Accidents and Injuries*. Crowthorne, TRRL, 1973.

² Transport and Road Research Laboratory, leaflet LF 576. Crowthorne, TRRL, 1975.

Coronary artery spasm

SIR,—A clue to the nature of the "perverted internal secretion which favours spasm of the arteries" mentioned in your leading article (14 April, p 969) may lie in a relationship between coronary artery spasm and migraine.

Twelve patients who suffered from both

migraine and chest pain of an anginal nature were investigated by Leon-Sotomayor.¹ In six of them coronary arteriograms were carried out, four showing segmental spasm accompanied by anginal pain, which was promptly relieved by nitroglycerine. In four of the six the angiogram precipitated migraine. These and other observations and investigations led him to suggest that the underlying mechanism was an exaggeration of sympathetic activity. A similar but opposite alteration in vasomotor tone affecting both cranial and cardiac vessels is suggested by Ekblom² to explain the remission of anginal pain during periods when his patient suffered from cluster headaches. If migraine and coronary spasm share a common pathology this association is unlikely to be always innocent. Leviton³ has shown that the risk of a fatal heart attack is apparently increased in people with migraine, particularly in the younger age groups.

If there is such an association it is surprising that there are so few observations in the literature, for if it exists it should be fairly common. These pilot studies suggest that it is worth looking for. Much migraine can be avoided by appropriate management. Perhaps the same can be done for cardiac spasm.

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¹ Leon-Sotomayor, L A, *Angiology*, 1974, 25, 161.

² Ekblom, K, and Lindahl, J, *Headache*, July 1971, 157.

³ Leviton, A, Malvea, B, and Graham, J R, *Neurology*, 1974, 24, 669.

Proteinuria at high altitude

SIR,—It will be fascinating to read the account by Drs A R Bradwell and J Delamere of 17 trekker-doctor-scientists collecting 24-hour urine samples (2-3 litres each?) for 14 days in the Himalayas, which they refer to in their letter (21 April, p 1083). Presumably aliquots were preserved and analysed back in Birmingham and details of the methods used will be vital.

Preservation and transport present immense difficulties in the tropics. Aliquots I took at heights of up to 7500 metres on a technically easy climb in the Hindu Kush showed strong protein reactions to Bili-Labstix but variable results in laboratory analysis in London, because of deterioration during transport despite keeping them as cold as possible.¹ Aliquots I took at 6000 metres in the Andes were ruined by defective refrigeration in a British Airways plane. Most other "field" investigators have similar experience. So, though Bili-Labstix estimations are crude they compensate by allowing freshly taken samples of urine to be assessed on the spot, by contrast with the inevitable deterioration in samples brought back to the UK for analysis.

Obviously ordinary trekking to moderate heights is very different from difficult high-altitude mountaineering in activity, exposure, and ease of collection of samples. Mountaineers would give a very "blue" answer to requests to collect 24-hour specimens of urine during technically difficult, dangerous, long, and exhausting climbs. My data do give a fair picture of physiological trends in the particular circumstances, though with the small numbers of climbers differences could not be valid statistically.

Rennie and Joseph's paper² will still be considered a model study by most until