Causes of osteoporosis in men with vertebral crush fractures in study by Francis et al¹ and at osteoporosis clinic at King's College Hospital (figures are numbers of patients)

Cause of osteoporosis	Francis et al's study, 1989 ²	Osteoporosis clinic, King's College Hospital, 1989-90
Primary	40	10
Steroid treatment	15	16
Neoplastic	7	1
Gastric surgery	5	1
Hypogonadism	5	1
Anticonvulsant treatment	4	
Osteogenesis imperfecta	4	4
Homocystinuria	2	
Immobility	1	1
Steatorrhoea	1	
Combined (including alcohol plus other risks)	10	
Alcohol (sole risk)		8
Diabetes mellitus		2
Total	94	44

treatable conditions such as hyperthyroidism and multiple myeloma which may present as osteoporosis.

In addition to the tests that Anderson suggests we include a full blood count, erythrocyte sedimentation rate, thyroid function tests, liver function tests, paraprotein studies, and measurement of urinary calcium concentration in our initial investigation of men presenting with osteoporosis. We do not routinely measure the serum prolactin concentration unless initial investigations or the clinical picture suggest hypogonadism, and, although steroid treatment is a common cause of osteoporosis, endogenous hypercortisolism is rare so we do not screen for Cushing's syndrome unless there are other clinical reasons to suspect the diagnosis.

Osteoporosis in men is often neglected. There has been little evaluation of treatments in men, and we hope that recognition of the clinical problem will lead to properly conducted studies.

T ALLAIN
P PITT
C MONIZ

Osteoporosis Clinic,
King's College Hospital,
London SE5 9PJ

1 Anderson DC. Osteoporosis in men. *BMJ* 1992;305:489-90. (29 August.)
2 Francis RM, Peacock M, Marshall DH, Horsman A, Aaron JE. Spinal osteoporosis in men. *Bone Miner* 1989;5:347-57.

Faecal urge incontinence caused by exercise

EDITOR,—E S Kiff does not mention faecal urge incontinence caused by exercise in the article on faecal incontinence.¹ This may reflect underrecognition of the well documented condition "runner's trots," which three of my patients have.

The patients (two women aged 28 and 42 and a man aged 39) all have well controlled distal ulcerative colitis. As Kiff mentions, reduced rectal capacity as a consequence of proctitis is a recognised cause of faecal urge incontinence. My patients, however, do not have any problems until they engage in long distance road running. Each trains on average 40-60 km a week and frequently participates in races of up to marathon distance. Their training schedules are so disrupted by extreme urgency or actual incontinence that they have to plan routes through wooded countryside or past public conveniences. They find that a combination of a low residue diet and antidiarrhoeal drugs, although handicapping, may allow them to compete in races. All patients know apparently healthy club colleagues who suffer similar symptoms.

Oettlé has studied the effect of moderate exercise (50% maximum oxygen consumption) on the gastrointestinal system of healthy subjects.² He found that transit time was dramatically accelerated both by running and by cycling on a bicycle ergometer. Frequency of defecation was not, however, increased. A colon transit study was performed on one of my patients. The patient ingested 10 radio-opaque beads, which were evident in the right colon on abdominal radiography immediately before exercise. After running for eight minutes at 12 km/h on a treadmill the patient had to stop and visit the toilet with considerable urgency. A repeat radiograph immediately afterwards showed one residual bead in the rectum.

Halvorsen et al have reported the effects of more strenuous exercise: they found that 20% of a group who ran marathons in their leisure time experienced symptoms that seriously affected their performance.³ Some evidence suggests that this may partly be due to an ischaemic enteropathy.⁴ In summary, exercise such as running may cause faecal urge incontinence, particularly if other factors such as proctitis are present.

P J MULLEN

Medical Division,
Princess Mary's RAF Hospital,
Halton,
Aylesbury HP22 5PG

1 Kiff ES. Faecal incontinence. *BMJ* 1992;305:702-4. (19 September.)
2 Oettlé GJ. Effect of moderate exercise on bowel habit. *Gut* 1991;32:941-4.
3 Halvorsen FA, Lyng J, Glomsaker T, Ritland S. Gastrointestinal disturbances in marathon runners. *Br J Sports Med* 1990;24:266-8.
4 Bounous G, McArdle AH. Marathon runners: the intestinal handicap. *Med Hypotheses* 1990;33:261-4.

Iatrogenic injuries in theatre

EDITOR,—T H Taylor mentions nerve damage, diathermy burns, and backache as examples of injury due to incorrect positioning on the operating table.¹ Another example is the ulcer that occurs in diabetic patients whose heels have not been adequately protected during their time on a hard theatre table or in the immediate postoperative period. At the very least such ulcers delay mobilisation—sometimes considerably in those who have had a joint replacement operation or an amputation; at worst they may lead to amputation.

Medical and nursing staff must not attribute these ulcers to misfortune or misadventure. They are due to carelessness and are preventable. Diabetic patients who have neuropathy or absent foot pulses must have their heels protected, either by foam leg troughs or by ample use of cotton wool padding, before they go to theatre and until they regain full consciousness and can move their legs freely. Sheepskin heel muffs are useless.

H CONNOR

County Hospital,
Hereford HR1 2ER

1 Taylor TH. Avoiding iatrogenic injuries in theatre. *BMJ* 1992; 305:595-6. (12 September.)

Use of live donors in renal transplantation

EDITOR,—The United Kingdom Transplant Support Service has recently produced its report covering transplant activity for 1991-2.¹ The report highlights the dramatic differences in clinical practice in Britain regarding the use of live related donors in renal transplantation.

Of the 37 transplant centres that reported using cadaveric transplants, only 26 also reported using live donors, which implies that in 11 units patients

receiving dialysis are not being offered this treatment option under any circumstances. The 26 units used 85 renal transplants from live donors compared with 1786 cadaveric transplants in the year, corresponding to a live donor transplant rate of about 5% overall. The use of live donors within these 26 units varied tremendously: in our unit and that at Charing Cross Hospital live donors contributed around 20% of all transplants while in other units they contributed only 1%. If all these 26 units achieved a 20% live donor transplant rate this would be equivalent to about another 270 grafts each year, representing an increase in total renal transplant activity of about 17%. If the 11 transplant units that do not use live donors at all changed their policy the potential increase in transplant activity would be substantially greater.

Extensive clinical experience of live donor renal transplantation, in Britain and the rest of the world and extending over a considerable period, shows it to be a highly satisfactory form of treatment. Even allowing for the continuing efforts to increase retrieval of cadaveric kidneys, this source of organs seems unlikely to be sufficient to keep up with the ever increasing number of patients joining the waiting list for a transplant. Further debate on live donor transplantation should take place so that this effective treatment can be offered to the greatest possible number of patients who are currently being maintained on long term dialysis programmes.

M A MANSELL
C J RUDGE

St Peter's Renal Unit,
Middlesex Hospital,
London W1N 8AA

1 United Kingdom Transplant Support Service Authority. *Annual report 1991-2*. Bristol: UKTSSA, 1992.

Change in drug treatment after discharge from hospital

EDITOR,—In Rachel Ann Cochrane and colleagues' study only five days of medication was provided when patients were discharged, yet the authors are surprised that medication was renewed without the patient necessarily having been reviewed by a doctor.¹ If patients are discharged on a Thursday there is little time in a busy practice for a non-urgent visit to be arranged before the medicine runs out. In addition, other NHS workers spend considerable time providing a renewed prescription and the drugs probably cost more from a community pharmacist than they would from the hospital pharmacy.

Perhaps one of the things that the working party being set up in Sunderland should consider is whether giving one month's worth of drugs on discharge would increase compliance.

N JOHN SHARVILL

Deal,
Kent CT14 7AU

1 Cochrane RA, Mandal AR, Ledger-Scott M, Walker R. Changes in drug treatment after discharge from hospital in geriatric patients. *BMJ* 1992;305:694-6. (19 September.)

EDITOR,—Rachel Ann Cochrane and colleagues investigated changes in elderly patients' drug treatment after discharge from hospital and found discontinuity of drug treatment and changes in both drugs and formulations.¹ We carried out a similar study in the east of Glasgow.² We visited 56 elderly patients on or after the fifth day after discharge from hospital (median day 8) and found that 15 had not had a new prescription issued. Fourteen drugs had been added to the new prescriptions issued and 17 had been omitted, from a possible total of 128 drugs. Inaccurately labelled containers or changed drug names, or both, were

found for 35 drugs. Contrary to hospital advice, 59 drugs were issued in childproof containers.

We had spent time counselling the patients on their treatment before discharge. We had attempted to improve communication by sending a copy of the discharge prescription to the general practitioner by first class post and alerting the practice surgery by telephone to the patient's discharge.

Closer communication between hospital and community care professionals is clearly required. Hospitals should endeavour to inform general practitioners of patients' discharge on the day of discharge or the next working day, and a subsequent home visit by either the doctor or a liaison health visitor would facilitate continuity of treatment. We agree that a more active role for community pharmacists would help iron out potential sources of confusion in nomenclature, timing of doses, and containers and would benefit this vulnerable group of patients.

JENNIFER BURNS
BRENDAN J MARTIN

Department of Geriatric Medicine,
Lightburn Hospital,
Glasgow G32 6ND

1 Cochrane RA, Mandal AR, Ledger-Scott M, Walker R. Changes in drug treatment after discharge from hospital in geriatric patients. *BMJ* 1992;305:694-6. (19 September.)
2 Burns JMA, Sneddon I, Lovell M, McLean A, Martin BJ. Elderly patients and their medication: a post-discharge follow-up study. *Age Ageing* 1992;21:178-81.

Human insulin and awareness of hypoglycaemia

EDITOR,—Mr R Kiln dismisses our study on insulin species and awareness of hypoglycaemia¹ as worthless.² Kiln's comments reflect a less than careful reading of our paper. Contrary to the assertion that "nobody . . . seems to have taken any account of what diabetic patients are saying," this was in fact the main selection criterion for entry into our study.

Kiln finds serious flaws in the design of our study. The first is that one month is not long enough for patients to detect a difference in insulin species. This comment shows a lack of understanding of the design of our study. The randomisation schedule resulted in 28 of 50 patients being treated for two consecutive months with porcine insulin. Not one of these patients was able to identify the species of insulin. Furthermore, other studies in which patients were treated for up to three months also failed to show a difference (A Stocks, personal communication).¹ Kiln suggests that improvement in control may have accounted for our finding of no significant differences between human and porcine insulins. There was no suggestion, however, that glycaemic control improved or changed during our study.

The second concern relates to the method by which patients were asked to identify the insulin species. Kiln's reference to "the questionnaires" again highlights a lack of understanding of our study methods. No questionnaire was used. Instead, patients were invited to identify the sequence of insulin species by whatever means they wished, which invariably required the help of relatives and friends. Participation in a study may alter behaviour. Since patients were aware that one of the main outcomes of our study was their ability to distinguish the insulin species, however, it is likely that any bias would have favoured the patient being able to pick a difference rather than the contrary. The fact that only two of 50 patients could do so is convincing evidence against a species effect on hypoglycaemic awareness.

We appreciate Kiln's disappointment at the accumulating evidence that overwhelmingly fails to support anecdotal impressions about human insulin. Anecdotal evidence is important in

medicine to alert scientists to possible associations. It must then, however, be tested by well designed, controlled studies. Our study tested this anecdotal evidence and did not show an association between human insulin and hypoglycaemic awareness in the very patients who thought that they were experiencing a problem.

Our study is one of five double blind crossover comparisons of human and porcine insulin in patients selected because they complained about hypoglycaemic unawareness developing after their transfer to human insulin (A Stocks, personal communication).^{1,4,5} Not one of these studies has found a significant difference between human and porcine insulin. The continued focus on insulin species diverts attention from the serious problem of hypoglycaemic unawareness in people with insulin treated diabetes.

STEPHEN COLAGIURI
JOHN J MILLER
PETER PETOCZ

Diabetes Centre, Prince of Wales Hospital,
Randwick,
New South Wales,
Australia 2031

1 Colagiuri S, Miller JJ, Petocz P. Double-blind crossover comparison of human and porcine insulins in patients reporting lack of hypoglycaemia awareness. *Lancet* 1992;339:1432-5.
2 Kiln MR. Human insulin and unawareness of hypoglycaemia. *BMJ* 1992;305:522.
3 Maran A, Macdonald IA, Gale EAM, Amiel SA. Human insulin does not affect the presentation of hypoglycaemia in type 1 diabetes: a clinical and laboratory study [abstract]. *Diabetic Med* 1992;9(suppl 1):11A.
4 Patrick AW, Bodmer CW, Tieszen K, White MC, Williams G. Human insulin and awareness of acute hypoglycaemic symptoms in insulin-dependent diabetes. *Lancet* 1991;338:528-32.
5 Ferrer JP, Esmatjes Z, Gonzalez-Clemente JM, Goday A, Conget I, Jimenez W, et al. Symptomatic and hormonal hypoglycaemic responses to human and porcine insulin patients with type 1 diabetes mellitus. *Diabetic Med* 1992;9:522-7.

Unexplained death in fit young people

EDITOR,—In the editorial on unexplained death in fit young people M J Davies states that there is an analogy with the sudden infant death syndrome.¹ Davies observes that in both instances necropsy does not yield a definitive answer, but the author focuses mainly on necropsy after sudden death. No mention is made of the fact that the National Institutes of Health in Bethesda, Maryland, updated its definition of the sudden infant death syndrome in 1989; this requires an investigation of the scene of death in addition to necropsy. A strong emphasis on such investigation may increase diagnostic reliability when the cause of death is unexplained by necropsy. My experience confirms that more thorough investigation of the death scene in sudden infant death confers a much greater chance of uncovering a cause of death other than the sudden infant death syndrome.²

In sudden unexplained death of a fit young person prompt investigation of the death scene by trained staff from the medical examiner's office, in addition to police investigation, may provide valuable information on the cause and manner of death not detected at the necropsy or by the police investigation. Police are trained and equipped to search for criminal activity, but public health problems, especially elusive environmental hazards, may escape their attention at the death scene.^{1,3}

Forensic pathologists might find it helpful to compile a checklist of basic procedures to use during an investigation of the scene of a sudden unexplained death of an infant or fit young person. Difficult diagnostic cases may require more than one visit to the scene, at different times of the day. Given adequate resources and professional collaboration, forensic pathologists who have restricted their practice to necropsies can easily

gain skill and conduct adequate investigations of the death scene. With an organised, systematic investigation of the death scene greater advances will be made in unravelling the mystery of sudden unexplained death.

MILLARD BASS

New York,
New York 11217,
USA

1 Davies MJ. Unexplained death in fit young people. *BMJ* 1992;305:538-9. (5 September.)
2 Bass M, Kravath RL, Glass L. Death-scene investigation in sudden infant death. *N Engl J Med* 1986;315:100-5.
3 Bass M, Hass R. Sudden infant death in Thailand and Alaska. *Lancet* 1991;337:304-5.
4 Bass M, Hass R. Sudden unexplained death in a Pacific archipelago (Mariana Islands). *Am J Forensic Med Pathol* 1991;12:182.
5 Bass M. The fallacy of the simultaneous sudden infant death syndrome in twins. *Am J Forensic Med Pathol* 1989;10:200-5.

Severity of imported falciparum malaria

EDITOR,—Stephen J Lewis and colleagues retrospectively investigated factors affecting the severity of imported falciparum malaria.¹ They found that prior antimalarial prophylaxis was associated with reduced severity of disease. In addition they concluded that ethnic origin (African versus white) was also associated with a favourable outcome: Africans (whether living in Africa or in Britain) were at significantly lower risk of developing severe malaria. The authors argue that this effect is due to acquired immunity against falciparum malaria. Immunity against subsequent infections with *Plasmodium falciparum* is, however, largely ineffectual, mainly because of the changing antigenic nature of the merozoites but also because most persisting anti-gametocyte IgG fails to offer immunological protection.²

The authors overlooked an important variable by failing to investigate the sickle cell status of their patients. In sub-Saharan Africa the rate of carriage of haemoglobin S is 10-30%.³ Heterozygotes for haemoglobin S are protected against falciparum malaria because the malarial parasites cause sickling of the cells containing the haemoglobin S and the affected cells are removed by the spleen and the reticuloendothelial system. The severity of falciparum malaria in those with sickle cell trait is therefore reduced.¹

Thus the observed effect of ethnic origin on the severity of falciparum malaria is probably due not to acquired immunity but to confounding variables, particularly sickle cell status.

DAVID BURGNER

Royal Hospital for Sick Children,
Glasgow G3 8SJ

MARTHA HICKEY

Western Infirmary,
Glasgow G11 6NT

1 Lewis SJ, Davidson RN, Ross EJ, Hall AP. Severity of imported falciparum malaria: effect of taking antimalarial prophylaxis. *BMJ* 1992;305:741-3. (26 September.)
2 Manson-Bahr PEC, Bell DR, eds. *Manson's tropical diseases*, 19th ed. Ballière Tindall, 1987.
3 Grendel D. Protection against Plasmodium falciparum infection in children with haemoglobin S. *Pediatric Infect Dis J* 1991;10:620-1.

Controlling deaths from volatile substance abuse

EDITOR,—A Esmail and colleagues suggest that butane gas lighter refills should be withdrawn from the market as abuse leads to many deaths, especially in the under 18s.¹ They acknowledge that withdrawal of these refills might "force young people into abusing other gaseous products." History suggests that this fear is well justified.

McBride, writing in 1910, devoted 17 pages of