most intradermal infiltrations, and the longer 27 gauge needles for infiltration along a dermal edge—for example, before lacerations are sutured—could be more widely adopted. Obviously these needles are less rigid than the 21 gauge (green) and 23 gauge (orange) needles used at present, and 21 gauge needles must be used for procedures such as deep nerve blocks and intracapsular joint injections. For giving most other local anaesthetics, however, the 27 gauge and 30 gauge needles would be adequate, and, I am sure, preferable for the patient. I am a dental surgeon, and during my current undergraduate medical training I have witnessed many patients being given unnecessarily painful injections of local anaesthetic by doctors who have received only rudimentary training in techniques. Little or no thought is being given to methods of reducing the discomfort of injection. This is at its worst in accident departments, with paediatric units giving most consideration, mainly by using topical anaesthetic creams.

Use of dental technology can facilitate Davidson and Boom's aim of providing warm sterile local anaesthetic for infiltration. Dental anaesthetics come in a variety of solutions and concentrations, with or without vasoconstrictors, and are supplied as sterilised, individually packed ampoules that fit directly into a standardised syringe, which are available in both aspirating and non-aspirating varieties. The ampoule is loaded into the syringe intact and the desired needle connected. This action punctures the ampoule so that there is no contact with the local anaesthetic before its infiltration. This system prevents the contamination of the local anaesthetic solution that is possible with repeated drawing from a stock bottle. Another bonus of using these ampoules is the ready availability of commercial thermostatic heaters, although a correctly adjusted baby's bottle warmer will do the same job at a much reduced cost. Their use also removes the need to overheat the solution to allow for cooling in the syringe, in turn reducing the dissociation of the local anaesthetic solution.

If I should need a skin laceration sutured I will visit my dentist first to have the local anaesthetic administered before attending casualty.

ALEXANDER J CRIGHTON

Edinburgh EH12 8BE

1 Davidson JAH, Boom SJ. Warming lignocaine to reduce pain associated with injection. *BMJ* 1992;305:617-8. (12 September.)

Dermatological causes of pruritus ani

EDITOR,—We believe that there are two important omissions from D J Jones's list of dermatological conditions that can cause pruritus ani.¹

Firstly, in female patients lichen sclerosus should be remembered. This disorder, in which characteristic white atrophic areas are found on the skin, particularly affects the genitalia but can extend to the perianal area. One of us (CIH) has observed that a fifth of women with vulval lichen sclerosus have pruritus ani, but the perianal symptoms may occur in isolation. The symptoms usually respond to potent topical steroids, but patients need to be followed up long term as there is a small risk of malignant change in the lesions.

Secondly, the importance of contact dermatitis as an aggravating factor in pruritus ani must be emphasised. In a recent study in the contact dermatitis clinic in Sheffield we patch tested 80 patients with pruritus ani. Results were positive in 55 patients. In 38 of these the reactions were to medicaments or their constituents. The commonest allergens were neomycin, fragrance mix, Peru balsam, and cinchocaine. Follow up in these patients showed an improvement or resolution of symptoms after advice in three quarters of the 55

with positive results. Patients with pruritus ani are at high risk of sensitisation from topical medicaments and toiletries, and we advise patch testing at an early stage in their management. We dispute the recommendation to use "wet wipes" as these may sensitise patients.

In conclusion, we emphasise the importance of examining the entire skin surface and the mucous membranes to obtain clues to the diagnosis and recommend that once a potent steroid has successfully alleviated symptoms the strength of steroid should be gradually reduced. In difficult cases skin biopsy may help with the diagnosis.

CHRISTINE I HARRINGTON

FIONA M LEWIS

ANDREW J G McDONAGH

DAVID J GAWKRODGER

Rupert Hallam Department of Dermatology,
Royal Hallamshire Hospital,
Sheffield S10 2JF

1 Jones DJ. Pruritus ani. *BMJ* 1992;305:575-6. (5 September.)

EDITOR,—D J Jones warns about the risks of contact sensitisation in the anal region and emphasises the importance of non-irritating, hygienic measures.¹ The advice to use "moist tissues normally used for babies' bottoms" is attractive, but moist toilet paper may contain preservatives that can be allergenic. In the Netherlands a relatively new preservative, methyl-dibromoglutaronitrile (in Euxyl K400), is sometimes used in these products.²

In the past 20 months I have seen 10 patients with a positive patch test reaction to methyl-dibromoglutaronitrile. In seven this was related to use of moist toilet paper in the anal region. Thus before advising patients with pruritus ani to use this type of tissue paper doctors should find out what preservatives it contains.

DERK P BRUYNZEEL

Department of Occupational Dermatology,
Free University Academic Hospital,
NL-1081 HV Amsterdam,
Netherlands

1 Jones DJ. Pruritus ani. *BMJ* 1992;305:575-7. (5 September.)

2 De Groot AC, Bruynzeel DP, Coenraads PJ, Crijns MB, Van Ginkel CJ, Van Joost T, et al. Frequency of allergic reactions to methyl-dibromoglutaronitrile (1,2-dibromo-2,4-dicyanobutane) in the Netherlands. *Contact Dermatitis* 1991;25:270-1.

Blood and breath alcohol concentrations

EDITOR,—Magne Nylenna and Richard Smith and subsequent correspondents discuss standardisation of units of measurement.^{1,2} Measurement of alcohol concentration is a case in point.

The concentration of alcohol (ethanol) permitted in a motorist's blood is different in different countries and also within regions of the same country—for example, the United States and Australia. Besides threshold limits of blood alcohol concentration many countries also have legislation referring to the concentration of alcohol in a specimen of breath. When reporting alcohol measurements for clinical and legal purposes investigators (and academic journals) use many concentration units. This practice has caused considerable confusion.

The first statutory alcohol limits for motorists were enacted in Norway in 1936 (0.50 mg/g) and then by Sweden in 1941 (0.80 mg/g). The legal limit in Sweden was lowered to 0.50 mg/g in 1957 and then to 0.20 mg/g in 1990. Note that the concentrations of alcohol are given here in terms of mass/mass. Britain introduced its legal alcohol limit for motorists in 1967 (80 mg/100 ml), which often appears in print as the ambiguous 80 mg%. Because 1 ml of whole blood weighs on average

1.055 g,³ 80 mg/100 ml corresponds to 76 mg/100 g.

In clinical chemistry laboratories plasma or serum is analysed more often than whole blood, and analyte concentrations are reported in accordance with the *Système International d'Unités* (SI). Here the amount of substance is the mole rather than mass and the preferred unit of volume is the litre. Accordingly, a blood alcohol concentration of 100 mg/100 ml is equivalent to 21.7 mmol/l (molecular weight of ethanol=46.06). Great care is needed when alcohol determinations made in hospital clinical laboratories are cited in legal proceedings dealing with road traffic offences. Plasma and serum contain more water than whole blood and therefore also more ethanol.⁴

Threshold limits of breath alcohol concentration have been derived from existing statutory limits for blood alcohol concentration by dividing by a blood to breath conversion factor. In Britain 80 mg/100 ml divided by 2300 gives 35 µg/100 ml, which is currently the statutory limit for breath alcohol concentration. Unfortunately, different countries opted for different factors when setting their limit for breath alcohol concentration. The table gives the legal alcohol limits (blood and breath) for motorists in various countries. When handheld breath alcohol devices are used in clinical and emergency medicine the results are often reported, without qualification, as if the blood alcohol concentration had been measured directly.^{5,6}

Statutory limits of alcohol in blood and breath in various countries

Country	Statutory blood alcohol	Statutory breath alcohol	Conversion (blood/breath)
Britain	80 mg/100ml	35 µg/100 ml	2300:1
Sweden	0.20 mg/g*	0.10 mg/l	2100:1
Norway	0.50 mg/g*	0.25 mg/l	2100:1
Austria	0.80 g/l	0.40 mg/l	2000:1
Holland	0.50 mg/ml	220 µg/l	2300:1
Germany	0.80 g/kg*†	‡	2100:1
France	0.80 mg/ml	0.40 mg/l	2000:1
US	0.10 g/100 ml§	0.10 g/210 l	2100:1

*1 ml of whole blood weighs 1.055 g.

†Concentrations of alcohol are determined in serum, and the result is converted into the presumed concentration in whole blood by dividing by 1.20.

‡Breath tests are used for roadside screening and not for evidential purposes.

§Applies in most US states.

The notion of reaching an international agreement about one common limit for blood or breath alcohol concentration for motorists or even the same unit of concentration (SI or otherwise) when reporting these measurements is attractive but hardly attainable.

AW JONES

Department of Alcohol Toxicology,
University Hospital,
581 85 Linköping,
Sweden

1 Nylenna M, Smith R. Americans retreat on SI units. *BMJ* 1992;305:268. (1 August.)

2 Correspondence. Americans retreat on SI units. *BMJ* 1992;305:585. (5 September.)

3 Lenter C. *Geigy scientific tables*. Vol 3. 8th ed. Basle: Ciba-Geigy, 1984.

4 Jones AW, Hahn R, Stalberg HP. Distribution of ethanol and water between plasma and whole blood; inter- and intra-individual variations after administration of ethanol by intravenous infusion. *Scand J Clin Lab Invest* 1990;50:775-80.

5 Marks V. Measurement of breath alcohol. *Lancet* 1992;339:187.

6 Hahn R. Measurement of breath alcohol. *Lancet* 1992;339:187-8.

Osteoporosis in men

EDITOR,—We believe that David C Anderson's guidelines for investigation for clinicians in doubt about the diagnosis of osteoporosis in men require some expansion.¹ The investigations suggested fail to screen for some of the commoner causes of secondary osteoporosis in men that we and others² have observed (table) as well as less common but

Causes of osteoporosis in men with vertebral crush fractures in study by Francis *et al*¹ and at osteoporosis clinic at King's College Hospital (figures are numbers of patients)

Cause of osteoporosis	Francis <i>et al</i> '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