enteritis."[1] The authors detail the oral therapy they used in terms of mmol/l.

Many years ago I was taught that the best oral rehydration solution was one that was made up of eight tablespoons of sugar and half a teaspoon of salt in one litre of water. Three glasses of this mixture were to be given after each stool. Does this simple, plain, and uncomplicated oral mixture, which could be understood by anyone, correspond to the glucose-electrolyte solution, measured in mmol/l, mentioned in the article?

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1 Mackenzie A, Barnes G. Randomised controlled trial comparing oral and intravenous rehydration therapy in children with diarrhoea. *BMJ* 1991;**303**:393-6. (17 August.)

AUTHORS' REPLY,—R K Edwards suggests making oral rehydration solution by adding eight tablespoons of sugar and half a teaspoon of salt to a litre of water; this would make a solution containing about 7% glucose (390 mmol/l) and sodium chloride 43 mmol/l. The glucose concentration of this solution would be dangerously high; glucose concentrations over 160 mmol/l (2·9%) are associated with less absorption of water and increased diarrhoea.[1] Most fruit juices and carbonated beverages (for example, lemonade, Coke, Pepsi, and Fanta) contain about 10% sugar,[1] so they should not be given undiluted to patients with diarrhoea. Edwards's recipe is probably a corruption of a World Health Organisation formula of eight level teaspoons of sugar and one level teaspoon of salt in a litre of water.[2]

For mild diarrhoea a suitable oral solution can be made at home by dissolving one heaped teaspoon of sugar (7 g sucrose, 3·5 g glucose) in a large cup of water. If the cup holds 200 ml this makes a solution containing about 1·8% glucose (100 mmol/l). The composition of home made solutions is very variable,[1] and we think that they should not contain salt because of the risk of salt poisoning. In areas with no cholera diarrhoeal stools usually have a low sodium concentration, and an appreciable sodium deficit is unlikely with mild diarrhoea (particularly if a solid diet is continued). For severe diarrhoea, particularly if there is dehydration, prepacked correctly formulated salts should be used to make a solution containing sodium 50-90 mmol/l, potassium 20-30 mmol/l, chloride 40-80 mmol/l, citrate 10 mmol/l, and glucose 83-111 mmol/l (1·5-2%).

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1 Dibley M, Phillips F, Mahoney TJ, Berry RJ. Oral rehydration fluids used in the treatment of diarrhoea. *Med J Aust* 1984;**1**:341-7.
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Auditing necropsies

SIR,—I Lauder's editorial[1] on the joint working party report *The Autopsy and Audit*[2] rightly concludes that necropsy is an excellent method of audit and needs to be encouraged. The report expresses concern about the fall in hospital necropsies, and Lauder suggests that this is because junior doctors find them distasteful and do not realise their value.

The report also suggests that the responsibility for obtaining a necropsy should lie with the consultant in charge of the case. The working party presumably think that this will result in more necropsies being performed, but I think they have misinterpreted the problem. Many consultants already instruct their junior staff to request a necropsy in all cases, unless the cause of death is in no doubt. Unfortunately, permission from the relatives is still refused more often than it is given, even when the consultant is directly involved. A similar situation exists with requesting organs for donation, and it seems to me that the general public, although becoming better informed on medical issues, is becoming increasingly squeamish about having its deceased relatives "cut up." How often have we heard the phrase, "He or she has been through enough already?" This phrase is often used in cases where there have been complications, and although the final cause of death may be clear much information can be gained from a necropsy that might improve our management in the future. Unfortunately, the argument of medical education is usually ignored by relatives.

Another problem is the reluctance of certain coroners and coroner's officers to request a necropsy when a case is reported to them, and I am sure Lauder is aware of this. One possible explanation is that the coroners are also trying to save money, and perhaps they too need to be educated about the educational value of a necropsy. Unfortunately, it is unlikely that district medical audit advisory committees will have any influence over them.

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1 Lauder I. Auditing necropies. *BMJ* 1991;**303**:1214-5. (16 November.)
2 Joint Working Party of the Royal College of Pathologists, Royal College of Physicians of London, and the Royal College of Surgeons of England. *The autopsy and audit.* London: RCPath, RCP, RCS, 1991.

Communicating necropsy results

SIR,—The findings of Paula Whitty and colleagues on the communication of necropsy results in North East Thames region cannot be accepted as they stand.[1] Although a delay in receiving the final report of 144 days is clearly unacceptable, 22 days may be a good performance, depending on the case mix at the particular mortuary. For example, many neurology cases require fixation of the brain for several weeks before even the macroscopic findings are available, let alone the histological results.

The authors do not mention how often members of the clinical team attended the necropsy. The doctor has to know the cause of death before requesting a hospital postmortem examination. The usual reason for a request is clinical interest, and it is therefore common for one or more of the clinical team to attend. Many pathologists would not consider it necessary to send out a preliminary report when the findings have been witnessed by members of the requesting firm.

The authors also imply that postmortem histology is somehow an optional extra rather than an integral part of the examination. I consider that a postmortem examination is as much an intellectual process as a physical one. It should start with a consideration of the history and should finish with the drawing of conclusions after consideration of the macroscopic findings, histological findings, and results of any microbiological or toxicological tests considered necessary. I am sure that the average pathologist's macroscopic diagnosis on a colectomy specimen is at least as accurate as most postmortem diagnoses, but would you expect a surgeon to be satisfied with a macroscopic diagnosis alone? The answer is not to try and restrict postmortem histological examination but to make sure it is done without unnecessary delays—something that many laboratories manage as a matter of course.

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Corticosteroids and tuberculosis

SIR,—Martin B Allen and Nigel J Cooke point out that lymph nodes may enlarge during antituberculous chemotherapy.[1] But lymph nodes may also enlarge in patients who have successfully completed chemotherapy.[2]

A 30 year old man started standard chemotherapy for cervical and supraclavicular tuberculosis adenitis (fully sensitive organisms). The lymph nodes decreased in size but after six months enlarged again. One was incised, but no acid fast bacilli were grown. Five months after he had finished 18 months' chemotherapy the lymph nodes again enlarged and became extremely uncomfortable. The lymphadenopathy resolved after two weeks' treatment with 20 mg prednisolone daily. He had no further problems in the subsequent two years.

It has been postulated that the mechanism for the enlargement of sterile nodes is a reaction to tuberculoprotein.[2] The enlargement is usually transient, but occasionally treatment with steroids may be necessary.[2]

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1 Allen MB, Cooke NJ. Corticosteroids and tuberculosis. *BMJ* 1991;**303**:871-2. (12 October.)
2 British Thoracic Society Research Committee. Short course chemotherapy for tuberculosis of lymph nodes: a controlled trial. *BMJ* 1985;**290**:1106-8.

Health of the nation

SIR,—Peter Anderson's article on alcohol and the health of the nation cannot be allowed to pass without comment.[1] From 1981 to 1982 alcohol consumption fell from 8·95 to 8·76 litres of pure alcohol per adult, not from 10·4 to 9·2 litres.[2] Convictions for drunkenness fell by 2%, not 11%, and drink-driving convictions increased by 3%—they did not fall by 8%.[3]

Anderson quotes a paper by Kendell *et al* in support of his belief that a substantial increase in the price of alcohol would affect heavy drinkers and more modest consumers alike.[4] In that paper only one factor (changes in excise) was selected as affecting patterns of consumption during 1978-81. Others could have been important—for example, employment and changes in income. Many of the heavy drinkers whose consumption declined during 1978-81 had become unemployed. Interestingly, too, during this period some light drinkers became heavy drinkers—a rather perverse result of changes in excise.

Anderson argues for a reduction in the population mean, which, in his opinion, predicts the number of heavy drinkers. But it is just as likely that the number of heavy drinkers predicts the population mean.

The claim that a 1% decrease in licensed outlets would result in a 2% decrease in consumption is contradicted by recent experience in the United Kingdom. During 1979-89 the number of licensed premises increased by 16% (from 170 100 to

199 500). Consumption fell from 9·81 litres of pure alcohol per adult to 9·47 litres.[3]

The categorical statement that "as affordability increases so does consumption and vice versa" is simply not true. The figure opposite this statement shows that during 1985-9 affordability increased sharply while consumption levelled off and then fell.

Anderson is rather scornful of current education campaigns that focus on sensible drinking because they may be counterproductive to his favoured population approach, which seems to entail elements of compulsion. In the *Health of the Nation* the government accepts that, in the end, people cannot be coerced into good health and that imposed strategies are valueless. The key seems to be education and not coercion that is based on flawed statistics and statistical reasoning.

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1 Anderson P. Alcohol as a key area. *BMJ* 1991;**303**:766-9. (28 September.)
2 *The drink pocket book*. Henley on Thames. NTC Publications, 1990:17.
3 *Statistical handbook 1991*. London: Brewers' Society, 1991.
4 Kendell RE, De Romanie M, Ritson B. Effect of economic changes on Scottish drinking habits, 1978-1982. *Br J Addict* 1983;**78**:365-79.

AUTHOR'S REPLY,—Communities with increasing aggregate levels of alcohol consumption tend to support increasing numbers of heavy drinkers and so increasing numbers of people with alcohol problems. This is a consistent finding across countries and over time and has been well reviewed and reported nationally and internationally.[1-3]

A persistent finding over time and across countries is that the most important determinant of alcohol consumption is affordability.[1 2 4] The relative stability of alcohol consumption in a period of increased affordability over the past five years in the United Kingdom does not refute this but argues for the effectiveness of health education programmes.

The relation between availability of alcohol and its consumption has been reviewed and confirmed by work by the Addiction Research Centre in York funded by the Economic and Social Research Council.[4]

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1 Bruun K, Edwards G, Lumie M, Makela K, Pan L, Popham R, *et al*. *Alcohol control policies* Vol 25. Helsinki: Finnish Foundation for Alcohol Studies, 1975.
2 Central Policy Review Staff. *Alcohol policies, 1979*. Stockholm: Sociologiska Institution, 1982.
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Zidovudine after occupational exposure to HIV

SIR,—I read D J Jeffries's editorial,[1] on prophylaxis with zidovudine after occupational exposure to HIV, and the following correspondence[2 3] with mixed feelings. Colleagues in Western countries are occasionally exposed to a patient in a so called high risk group. In our part of the world, however, we are all at high risk, doctors and patients alike.

I estimate—on good grounds—that 30% of my patients are HIV positive. I do over 100 operations a month, and from my personal experience I agree with A G Bird and colleagues that percutaneous injuries occur in about 15% of operations not counting occasions when patients' blood comes into contact with skin or splashes the eye without injury and disregarding the fact that we often operate while wearing used, resterilised gloves. On the basis of these figures I injure myself on average 4·5 times each month while operating on patients infected with HIV and would therefore be taking zidovudine continuously for the rest of my working life, if I followed Jeffries's recommendations. This would apply to all doctors, midwives, and surgeons in our part of Africa.

Zidovudine is not available in Kampala, but, even if it was, the side effects, dangers, and high cost of its long term use make the discussion about giving it after occupational exposure quite irrelevant and unrealistic in our situation.

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1 Jeffries DJ. Zidovudine after occupational exposure to HIV. *BMJ* 1991;**302**:1349-51. (8 June.)
2 Elkharrat D, Wautier JL, Caulin C, Bonnet N. Zidovudine after occupational exposure to HIV. *BMJ* 1991;**303**:309. (3 August.)
3 Brown EM, Caul EO, Roome APCG, Glover SC, Reeves DS, Harling CG. Zidovudine after occupational exposure to HIV. *BMJ* 1991;**303**:990. (19 October.)
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Coronary heart disease and elderly people

SIR,—Robert Beaglehole comments on the lack of evidence justifying the treatment of risk factors for cardiovascular disease in elderly people.[1] We have recently noted both an increasing number of inquiries from general practitioners about the justification for treating hyperlipidaemia in elderly people and an increased number of referrals of elderly patients with hyperlipidaemia to our lipid clinic.

The referral of two fit, slim, normotensive, and non-smoking women in their 70s with no family history of ischaemic heart disease prompted an inquiry into recent requests for estimations of cholesterol concentration made to our biochemistry department by general practitioners (table). This indicated that between the first six months of 1989 and 1991 requests had risen fourfold in patients aged under 65 and sixfold in patients aged 65 and over. The requests comprised 14·4% of all requests by general practitioners in the first half of 1991. Requests by hospitals remained static over this period. Requests from general practitioners arise mainly from the well person or lifestyle clinics that have flourished since the new general practitioner contract was introduced in 1990.

As Beaglehole comments, no clinical trials have assessed the efficacy of lipid lowering regimens in reducing cardiovascular morbidity or mortality in elderly people, and extrapolations from trials conducted in middle aged men may be unjustified.[2] Vigorous treatment of hyperlipidaemia is certainly indicated in younger subjects at high risk, but lack of evidence that treatment reduces total mortality and some concern that non-cardiovascular mortality may be increased[3] reinforce the case for a conservative approach to lipid lowering regimens in patients aged over 65.

Number of requests for serum cholesterol estimations to biochemistry department of Stobhill General Hospital, 1989-91

Age (years)	1989 Months 1-6	1989 Months 7-12	1990 Months 1-6	1990 Months 7-12	1991 Months 1-6
Requests from general practitioners					
16-	1307	1761	3046	4318	5231
65-	125	173	377	582	724
≥75	10	23	72	120	160
Requests from hospitals					
16-	2031	1922	2007	1977	2238
65-	281	293	318	266	361
≥75	64	76	44	65	59

Current guidelines do not state that systematic screening for hyperlipidaemia should *not* be carried out in elderly people,[4 5] perhaps on the grounds that a lipid lowering diet may do some good and cannot do harm. This ignores the anxiety and strain on limited budgets that may follow advice to modify lifelong dietary patterns. Several elderly patients with hyperlipidaemia referred to our lipid clinic have been upset by prior advice to follow a lipid lowering diet. They were delighted to be told to ignore their serum cholesterol concentration and resume their previous dietary pattern, which arrival at pensionable age might indicate had served them well.

Perhaps a citizen's charter for elderly people should guarantee that arrival at pensionable age (and certainly at age 70) provides immunity from lipid screening and lipid lowering diets and drugs. Immunity should remain in force until new evidence indicates that the introduction of lipid lowering regimens produces tangible benefits in old age.

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1 Beaglehole R. Coronary heart disease and elderly people. *BMJ* 1991;**303**:69-70. (13 July.)
2 Garber AM, Littenberg B, Sox HC, Gluck ME, Wagner JL, Duffy BM, *et al*. *Costs and effectiveness of cholesterol screening in the elderly*. Washington, DC: Office of Technology Assessment, United States Government Printing Office, 1989.
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4 Study Group, European Atherosclerosis Society. Strategies for the prevention of coronary heart disease: a policy statement of the European Atherosclerosis Society. *Eur Heart J* 1978;**9**: 77-88.
5 National Cholesterol Education Program. Report of expert panel on detection, evaluation and treatment of high blood cholesterol in adults. *Arch Intern Med* 1988;**148**:36-61.

Use of thalidomide in leprosy

SIR,—The use of thalidomide in the treatment of erythema nodosum leprosum has been discussed.[1-3] There is no doubt that such treatment is effective; but there is also no doubt that the side effects of thalidomide, particularly induced polyneuritis and teratogenicity (allergic vasculitis, thrombocytopenic purpura, and myxoedema have also been reported, and in animals toxicity depends on both the species and the sex[4]), are also of great concern to clinicians and others working with people with leprosy, particularly in developing countries. The lack of control over such a potentially dangerous drug both in clinics and when it is released to patients and the possible misuse of the drug for its known sedative properties are among the reasons for such concern.

The risk-benefit analysis of M F R Waters[3] would provide sufficient justification for using thalidomide if that was the only option for treatment. Though this may be the case now, important advances have been made in developing more suitable alternative compounds. The work of O'Sullivan is particularly important and is well known to those concerned in the chemotherapy of leprosy. He has prepared analogues of clofazimine that do not cause extensive pigmentation, have much improved pharmacokinetic properties, are active against drug resistant organisms, and retain, in some cases, anti-inflammatory properties.[5-7] The development of these compounds should be a priority of the world community, but sadly this is not the case because leprosy is regarded as an orphan disease.

Clofazimine and its analogues, however, have potential in the treatment of other diseases that