

enteritis.¹¹ 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.

2 World Health Organisation. *Treatment and prevention of acute diarrhoea. Guidelines for the trainers of health workers.* Geneva: World Health Organisation, 1985.

Auditing necropsies

SIR.—I Lauder's editorial¹ 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 necropsies. *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: RCP, 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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1 Whitty P, Parker C, Pietro-Ramos F, Al-Khanisi S. Communication of results of necropsies in North East Thames region. *BMJ* 1991;303:1244-6. (16 November.)

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 tuberculo-protein.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