who a year later introduced him into the Lunar Society. Later, however, Darwin seems to have become jealous of Withering's multifarious successes, and their friendship soured. Darwin's failure to mention Withering in either of his publications was reprehensible, but the omission of Withering's name from the other publications was probably due to ignorance of his contribution. Darwin ended his paper to the royal college by saying that digitalis wanted only "the stamp of some great name, to make it current amongst the faculty." Doubtless he hoped that that name would be his own, but posterity has accorded the accolade to its rightful recipient, William Withering.

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¹ Withering, W, *An Account of the Foxglove and some of its Medical Uses: with Practical Remarks on the Dropsy and other Diseases*. Birmingham, G G J and J Robinson, 1785.

² Darwin, C, *Experiments Establishing a Criterion between Mucaginous and Purgative Matter and an Account of the Retrograde Motions of the Absorbent Vessels of Animal Bodies in some Diseases*. Lichfield, J Jackson, 1780.

³ *London Medical Journal*, 1785, 6, 55.

⁴ Darwin, E, *Medical Transactions of the Royal College of Physicians of London*, 1785, 3, 255.

⁵ Baker, Sir G, *Medical Transactions of the Royal College of Physicians of London*, 1785, 3, 287.

⁶ Baker, Sir G, *Medical Transactions of the Royal College of Physicians of London*, 1772, 1, 175.

⁷ Warren, J, *London Medical Journal*, 1785, 6, 145.

⁸ Withering, W, *A Botanical Arrangement of British Plants; including the uses of such species, in medicine, diet, rural oeconomy and the arts. With an easy introduction to the study of botany*, 2nd edn. Edinburgh and London, G G J and J Robinson, J Robson, J Balfour and C Elliott, 1787.

Diet and the diabetic

SIR,—You rightly mention in your leading article on this subject (2 October, p 780) that "the composition of the ideal diet for insulin-treated patients is continually debated." The clinician in India and in other developing countries has so far been advocating a diabetic diet consisting of 45-50% of sugar-free carbohydrates similar to that prescribed for Western diabetics. But experience has taught us that such a diet is not practical in countries where the consumption of carbohydrates by the general population is usually not less than 70-80% of the total calories. Besides, a diabetic of average means can ill afford a diet composed mostly of proteins and fats.

Since only 8% of all the diabetic patients in our clinic stuck to the prescribed diet, largely because of the high cost, a study was undertaken to assess the effect of an isocaloric high-carbohydrate diet on (1) the control of hyperglycaemia, (2) the requirement of anti-diabetic agents, and (3) the serum lipid abnormalities. A total of 40 patients with diabetes, of the maturity-onset type in 31 and of the juvenile-onset type in 9, were studied, together with 20 aged-matched normal healthy controls. The diabetics were examined clinically and biochemically before and after hyperglycaemia had been controlled

on the standard diabetic diet containing 50% carbohydrate. They were then put on an isocaloric high-carbohydrate diet containing 70% carbohydrate for a period of four weeks and the tests were repeated.

In the normal subjects the fasting and 2-h blood glucose values and the sum of the glucose values in the glucose tolerance test (GTT) showed no significant change on the high-carbohydrate diet as compared with the standard diabetic diet ($P > 0.05$). Insulin kinetics also did not alter in these subjects as shown by the fasting, peak, and sum of insulin values after the GTT ($P > 0.05$) (see table). Serum cholesterol, triglyceride, and free fatty acid concentrations also showed no significant change.

In the maturity-onset group of diabetics the requirement of oral hypoglycaemic agents was increased in only one patient, who needed 2.5 mg more of glibenclamide daily when put on the high-carbohydrate diet, while in four the requirement was decreased. The fasting and 2-h glucose values and the sum of the glucose values in the GTT improved markedly in 19 patients, were unchanged in 8, and showed some deterioration in 4. In the juvenile-onset group the insulin requirement decreased in five patients and remained unchanged in the rest. Other workers in India, such as Vishwanathan¹ and Patel *et al*,² have reported similar experiences.

We therefore feel that hyperglycaemia can be controlled with a sugar-free isocaloric high-carbohydrate diet in the majority of diabetic patients. Besides, it is cheap and practical.

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¹ Viswanathan, M, *Madhumeha*, 1968, 8, 5.

² Patel, J C, *et al*, in *Proceedings of the World Congress on Diabetes in the Tropics*, 1966, p 527. Bombay, Diabetic Association of India, 1968.

Alcohol and road accidents

SIR,—Your leading article on this subject (12 March, p 665) calls for information about the blood alcohol concentrations in victims of road accidents. As you say, the taking of blood samples for this purpose from drivers would lead to considerable legal and ethical difficulties, but this does not seem to apply in our experience to breath sampling. For the past nine months breath samples have been taken for diagnostic purposes with an Alcolmeter¹ (made by Lion Laboratories Ltd, Pearl Street, Cardiff) from unconscious or drowsy patients, including victims of road or other accidents, admitted to the accident and emergency department of this hospital.

The Alcolmeter has the useful property of aspirating its sample, so that unconscious patients present no difficulty. The sample is

taken from a nostril at the end of expiration, which may be slightly accentuated by pressing on the chest. Conscious patients can easily be persuaded to blow through a mouthpiece attached to the instrument.

Although breath-alcohol analysis does not give such accurate and precise results as blood analysis² it is quite good enough for clinical purposes and has been found to be of considerable value in differential diagnosis. In one case a preliminary diagnosis of alcoholic coma was ruled out by a finding of a blood alcohol concentration (BAC) of only 40 mg/100 ml; further investigation showed that other drugs were involved. In other cases patients who claimed to have drunk very little alcohol were found to have BACs well over 100 mg, and in one road accident victim the level reached 200 mg. The information obtained in this way is used for clinical purposes only, but it is recorded in the patient's notes. If other accident and emergency departments adopted the same policy, the information you require would soon become available.

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¹ Medical Research Council, *Annual Report*, 1972-3, pp 104-105.

² Wright, B M, *et al*, *Medicine, Science and the Law*, 1975, 15, 205.

Potassium in heart failure

SIR,—Your leading article (19 February, p 469) states that potassium supplements are mandatory in some categories of patients with heart failure, one of these being "elderly patients on diuretics alone (because their intake is poor)." If, however, indiscriminate use of potassium supplements is to be avoided, the statement requires a positive answer to the following three questions in order to gain validity:

(1) Can potassium depletion be identified by generally available methods? Your article correctly points out that many patients in heart failure are depleted of total exchangeable potassium despite normal plasma concentrations and, one might add, the correlation between plasma potassium concentration and total body potassium is poor.¹ The answer to the first question is negative.

(2) Is correction of potassium depletion, once diagnosed, necessary? Many authors have failed to detect adverse symptoms or signs ascribable to potassium deficiency in patients on diuretics.^{2,3} The answer to the second question is equivocal at best.

(3) Are potassium supplements a reliable way of preventing or correcting potassium depletion? Down *et al*⁴ gave a negative answer to this question and Davidson *et al*⁵ found no relation between the dose of potassium supplements and either the plasma potassium concentration or total body potassium.

A study of 489 geriatric outpatients⁶ demonstrated that in 178 patients on diuretics potassium supplements did not influence the mean serum potassium concentration in either sex. Another important finding was that mean serum potassium in men on diuretics was only insignificantly decreased compared with men not on diuretics, whereas mean serum potassium in women on diuretics was significantly lower than in women not on diuretics. Only one of 44 men on diuretics had a reduced serum potassium concentration (3.3 mmol/l),

Serum insulin concentrations in mU/l (mean $\pm$ SD) during GTT in 20 normal subjects and 31 maturity-onset diabetics

	Standard diet (50% CHO)		High-carbohydrate diet (70% CHO)	
	Normals	Diabetics	Normals	Diabetics
Fasting	18.1 $\pm$ 2.6	28.8 $\pm$ 10.36	17.8 $\pm$ 2.23	22.35 $\pm$ 1.59
Peak	65.6 $\pm$ 10.25	90.85 $\pm$ 12.23	60.4 $\pm$ 6.0	87.7 $\pm$ 6.25
Sum of values at 0, 1, 2, 3 h	147.6 $\pm$ 18.18	295.0 $\pm$ 53.54	139.2 $\pm$ 12.53	263.55 $\pm$ 12.62

whereas 19% of the women on diuretics had hypokalaemia of varying degrees.

My conclusions are that the evidence in favour of a general recommendation of potassium supplementation in elderly, non-digitalised cardiac patients on diuretics is insufficient and that elderly women are probably more prone to diuretic-associated hypokalaemia than elderly men.

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¹ Wilkinson, P R, *et al*, *Lancet*, 1975, **1**, 759.

² Edmonds, C J, and Jasani, B, *Lancet*, 1973, **2**, 8.

³ Healy, J J, *et al*, *British Medical Journal*, 1970, **1**, 716.

⁴ Down, P F, *et al*, *Lancet*, 1972, **2**, 721.

⁵ Davidson, C, *et al*, *Lancet*, 1976, **2**, 1044.

⁶ Krakauer, R, and Lauritzen, M. To be published.

Beta-blockers and lipid metabolism

SIR,—Your leading article "Diet, energy balance, and genes—and serum cholesterol" (26 March, p 789) discusses the factors influencing lipid metabolism in relation to the genesis of ischaemic heart disease. The multifactorial basis of the common hyperlipidaemias is well demonstrated and you conclude by suggesting that further studies are needed to investigate the metabolic basis of the common hyperlipidaemias.

Adrenergic beta-blocking drugs have an established role in the treatment of ischaemic heart disease and hypertension and possibly exert a protective influence against myocardial infarction. These drugs have been shown to have many metabolic effects¹ and in particular there is evidence that they are capable of reducing free fatty acid levels during stress.^{2,3} However, little is known of the long-term effects of beta-blockade upon lipid metabolism. Recently Waal-Manning⁴ in a clinical study reported that the cardioselective drug metoprolol produced a significant rise in plasma triglyceride levels by as much as 42%. This observation is worrying since increased plasma triglyceride concentration is a recognised risk factor in ischaemic heart disease. It is possible that the chronic administration of adrenergic beta-blocking drugs to hypertensives, especially those with a pre-existing hyperlipaemic tendency, may not be beneficial in this respect.

I would suggest that there is a need for studies to investigate the long-term effects of beta-blockade upon lipid metabolism.

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¹ Himms-Hagen, J, *Pharmacological Reviews*, 1967, **19**, 367.

² Bjorntorp, P, *et al*, *Acta Pharmacologica et Toxicologica*, 1967, **25**, suppl 2, p 51.

³ Somerville, W, *et al*, in *New Perspectives in Beta-blockade*, ed D M Burley *et al*, p 275. Horsham, Ciba Laboratories, 1973.

⁴ Waal-Manning H J, *Drugs*, 1976, **11**, suppl 1, p 121.

The reprint game

SIR,—In recent issues there have been a number of letters expressing the attitudes of authors towards reprints. For some time now I have been making it a practice to enclose one American dollar in currency with each request for a reprint. It seems to me that this might constitute a mutually satisfactory general practice. I figure that if my interest is not that great I shouldn't be asking anyone else to go to any trouble. For the author

filling the request this will just about assure that his out-of-pocket expenses are met.

A A HOLZSCHUH

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Emetic response to salt

SIR,—I hasten to disagree with the suggestion of Drs C J C Roberts and H McNulty (26 March, p 840) that the sudden loss of consciousness of a 57-year-old woman following an overdose of oxprenolol (26 February, p 552) could have been caused by the salt emetic (which acted almost immediately) given by her husband. I wish to give an example of how the administration of a double-dose salt emetic which remained in a man's stomach for nearly an hour before being removed by another emetic did not have any dire consequences.

At 7 am in 1930 a young man, 21 years old, woke up with Monday morning depression and saw a green liniment bottle marked Poison on the mantle shelf. He decided to commit suicide, drank the contents, and threw the bottle out of the window, and stayed in bed to await results. At 7.45 am he felt ghastly, changed his mind, and called out to his brother to fetch the doctor at once. I came round by 8.15 am. He said that he did not know what the liniment was he had swallowed. He looked very pale and his lips were blue, but there was no corrosion around his lips or mouth.

I said, "I must go and find the bottle." He replied, "Don't leave me mate; save me!" I went through the first-aid book routine and gave him two tablespoons of salt in a tumbler of water with my compliments. He swallowed easily, but there was no emesis. After five minutes another two tablespoons of salt in a tumbler of water went down comfortably—still no result. I decided to go to the chemist's shop round the corner to fetch some apomorphine. He cried, "Don't leave me mate." I uttered the famous words from ITMA: "I go, I come back," and left. The chemist was raising his shutters at 9 am. I collected a tiny tube with 10 hypodermic tablets of apomorphine 1/20 grain (3 mg) each, and dissolved one tablet in a teaspoon of boiled water. I gave him a subcutaneous injection and three minutes later he brought up a bowlful of soapy looking water smelling of methyl salicylate linctus.

I went home to do the morning surgery and returned to the house to be told at 11 am that he had got out of bed, dressed and gone to work.

The emetic response to salt and water is usually under five minutes—as occurred in the oxprenolol case. In my case the salt and water remained for approximately an hour in this man's stomach. I believe that the methyl salicylate had an anaesthetic effect on the mucosa of the pharynx, oesophagus, and stomach preventing any sensation of nausea. The man was quite well after this suicidal attempt.

The first-aid handbook, 1929, says: "Repeat salt and water emetic every five minutes until vomiting occurs." This could be quite considerable if it was kept up for an hour—24 tablespoons of salt and 6 pints of water before emesis. For salt intoxication it all depends on how much is swallowed, how long it stays down, and how much is absorbed.

Apomorphine injections (or oral tablets) are no longer available. This stopped about 10 years ago because they were being over-used in so-called aversion therapy for alcoholism. Today ipecac syrup is recommended for use in the home. It is not readily available in all households but salt certainly is.

Beta-blockers are now being widely prescribed for angina and hypotension. The oxprenolol fatality does show that anyone who

swallows a month's supply in one go and seeks help *when staggering* is beyond the aid of emetics and pacemakers. Perhaps relays of attendants giving external cardiac massage for hours, without undue force, may be the solution.

N C HYPHER

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Allergy to diazepam

SIR,—My attention has been drawn to the letter of Dr S K Majumdar (12 February, p 444), in which I am quoted as saying that the active and common metabolite of all benzodiazepines is desmethyldiazepam. In fact I state¹ that desmethyldiazepam is the common metabolite of several benzodiazepines including medazepam, diazepam, and clorazepate. However, oxazepam is a metabolite of desmethyldiazepam and, like its chlorinated derivative, lorazepam, is fairly rapidly metabolised by conjugation with glucuronic acid and then excreted. Finally, both nitrazepam and flurazepam belong to different metabolic chains. Accordingly, cross-allergenicity between the various benzodiazepine groups cannot be automatically attributed to desmethyldiazepam.

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¹ Lader, M, in *Advanced Medicine—Topics in Therapeutics*, 2, ed P Turner, p 212. London, Pitman Medical, 1976.

Miscellaneous expense grants

SIR,—Many hospital doctors eligible for removal expenses are, I believe, experiencing difficulties in claiming reimbursement of unavoidable expenditure on moving. The BMA is trying to negotiate equality for doctors who, unlike any other NHS staff, cannot claim this grant as a lump sum. I would be grateful if any doctors who have examples of their hardship could write to me to support our claim to the Department of Health.

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* * * The Secretary writes: "The HJSC negotiating team is well aware of the problem. Negotiations have been going on for some time but have been thwarted by pay policy. The BMA secretariat has been and is in direct touch with the representatives of junior staff in the South-west Region. Some progress with the Health Departments is being made, but detailed figures would be most useful, particularly if copies of correspondence could be sent to the Secretary."—ED, *BMJ*.

Breath, alcohol, and the law

SIR,—The article by Dr T A A Alobaidi and others (18 December, p 1479) reported a wide range of values for the blood:breath partition coefficient for alcohol and concluded that "breath analysis is not an acceptable method for accurately determining blood alcohol concentrations." Referring to the claim that a