references on a disk and use the remaining space for the index and text files which I described in my paper. When I need to look for references which are stored on many floppy disks, I copy them on to the hard disk and use it to locate the data required more quickly.

I am delighted to state that well over 500 readers have sent requests for listings of the program. I am doing my best to dispatch them and to answer individual questions about hardware and software requirements. Detailed instructions on how to incorporate this program into other computer systems are also included.

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Drug points

Complete heart block induced by hyperkalaemia associated with treatment with a combination of captopril and spironolactone

Dr MAYUR LAKHANI (Department of Medicine, Addenbrooke's Hospital, Cambridge) writes: Further to the report by Drs T C N Lo and R J Cryer (21 June, p 1672) I would like to report a similar complication (which I observed at Perth Royal Infirmary, Scotland) associated with enalapril.

A man of 85 with severe congestive heart failure refractory to diuretic treatment was prescribed 5 mg of enalapril (as an inpatient) in another unit. Four weeks later he was admitted with transient loss of consciousness associated with bradycardia. On admission he was hyotensive (80/40 mm Hg) but no longer bradycardic (84 beats/min); a 12 lead electrocardiogram showed bifascicular heart block with atrial fibrillation (both previously present) but with widened QRS complexes (160 ms). He was taking frusemide 120 mg daily, spironolactone 100 mg daily, and enalapril 5 mg. Blood urea was 35·3 mmol/l (213 mg/100 ml) (9·7 mmol/l (58 mg/100 ml) before enalapril therapy) potassium 7·3 mmol(mEq)/l (previously 3·0 mmol/l), and sodium 129 mmol (mEq)/l. Hyperkalaemia was treated with intravenous insulin-dextrose, and enalapril and diuretics were discontinued. Ten hours after admission his cardiac rhythm changed to trifascicular heart block but with QRS complexes of normal duration. Shortly afterwards he developed complete heart block, which quickly progressed to asystole and death despite intravenous boluses of atropine. Pacing was considered inappropriate in view of his very poor physical condition and after careful discussion with his next of kin.

This case, along with that of Drs Lo and Cryer and those of others,[1] illustrates that serum potassium concentrations rise dangerously when angiotensin converting enzyme inhibitors are used in conjunction with potassium supplements or potassium sparing diuretics (aldosterone antagonists). This is not surprising as inhibition of aldosterone synthesis by converting enzyme inhibitors reduces the urinary losses of potassium, causing potassium conservation. Indeed, hypokalaemia does not occur when converting enzyme inhibitors are used together with loop or thiazide diuretics.[2 3]

There are two lessons from these cases. Firstly, it is generally not necessary to prescribe potassium supplements or potassium sparing diuretics when angiotensin converting enzyme inhibitors are used together with loop or thiazide diuretics. Secondly, blood urea and electrolyte values must be known before treatment is started and must be monitored during therapy, especially in congestive heart failure.

1 Packer M, Lee WH. Provocation of hyper- and hypokalaemic sudden death during treatment with and withdrawal of converting enzyme inhibition in severe chronic congestive heart failure. *Am J Cardiol* 1986;57:347-8.
2 Johnston CI, McGrath BP, Millar JA, Matthews PG. Long term effects of captopril (SQ14 225) on blood pressure and hormone levels in essential hypertension. *Lancet* 1979;ii:493-6.
3 McGrath BP, Arnolda L, Matthews PG, *et al.* Controlled trial of enalapril in congestive heart failure. *Br Heart J* 1985;**54**: 405-14.

Points

Diffuse peritonitis and chronic ascites due to infection with Chlamydia trachomatis

Drs CAROLINE BRADBEER and JAN WELCH (Departments of Genitourinary Medicine and Virology, St Thomas's Hospital, London SE1 7EH) write: Dr U A Marbet and others (5 July, p 5) describe an interesting manifestation of chlamydial infection, but no mention is made of the sexual partners of their two patients. Recent correspondence in the *Lancet* has underlined the need for treatment of sexual partners of women with pelvic inflammatory disease[1]; *C trachomatis* can be isolated from 29% of these men, many of whom are asymptomatic (personal communication, M Jacob). The authors' first patient had a relapse four months after treatment and two weeks after the insertion of a new intrauterine device. The relapse was probably caused by reinfection with chlamydia aggravated by the reintroduction into the uterine cavity of the intrauterine device. Many authorities believe that this method of contraception should not be used when there is a history of pelvic inflammatory disease. Infection with *C trachomatis* may present to physicians, surgeons, ophthalmologists, general practitioners, or genitourinary physicians. All doctors who manage sexually active people must be aware of the sexual mode of transmission of chlamydia; of the importance of tracing and treating all contacts; and of the risk of subsequent, sometimes silent, tubal disease and subfertility.

1 Barton SE, Greenhouse P, Atia W, Dutt TP. Chlamydia trachomatis infection in women: a case for more action? *Lancet* 1986;i:1215.

Non-steroidal anti-inflammatory drugs and the kidney

Dr J C DAVIDSON (Hamad General Hospital, Doha, Qatar) writes: I was surprised that Professor M L'E Orme did not mention patients with diabetes mellitus among those at special risk from the adverse effects of non-steroidal anti-inflammatory drugs on the kidney (21 June, p 1621). The association of diabetes (usually type II) with hyporeninaemic hypoaldosteronism manifested clinically as hyperkalaemia is well known.[1 2] Professor Orme points out that non-steroidal anti-inflammatory drugs produce hyporeninaemic hypoaldosteronism, and common sense dictates that these drugs should be given with great caution to diabetic patients; when they are used it may be wise to have the serum potassium concentration monitored, even in those patients whose glomerular filtration rate is more than 20 ml/min. Diabetic patients are six times as likely to develop renal papillary necrosis with urinary tract infections as non-diabetics, and it would seem prudent to avoid those drugs for long term use in diabetic patients. Diabetes mellitus is much more common than gout, cirrhosis, or the nephrotic syndrome so it should have been added to the list.

1 Leland OS, Matti PC. Joslin's diabetes mellitus. In: Marble A, Krall LP, Bradley RF, Christler AR, Soeldner JS, eds. *Joslin's diabetes mellitus*. Febiger, Philadelphia: Lea and Febiger, 1985:588.
2 Nadler JL, Lee FO, Hsueh W, Horton R. Evidence of prostacyclin deficiency in the syndrome of hyporeninaemic hypoaldosteronism. *N Engl J Med* 1986;**314**:1015-20.

Transcutaneous oxygen tension during exercise in patients with claudication

Messrs C P SHEARMAN, B R GWYNN, and M H SIMMS (Selly Oak Hospital, Birmingham B29 6JD) write: We were interested in the report from Dr T A H Holdich and others (21 June, p 1625) suggesting that transcutaneous oxygen tension ($TcPo_2$) measured on the calf during exercise may be a useful tool for quantitative assessment of patients with intermittent claudication. This concept is not new, however,[1 2] and we have been using the technique to study the effect of various drugs on such patients. Unlike the authors, we found the dorsum of the foot to be a more satisfactory site to monitor $TcPo_2$. Even in patients with mild symptoms a fall in $TcPo_2$ during walking occurred, while this was not always so when we recorded it from the calf. We too have been impressed with the reproducibility of the technique. As yet, however, we have been unable to show that these changes in $TcPo_2$ correlate with the severity of symptoms in people with claudication. It also remains to be shown that an improvement in the condition is reflected by a corresponding change in the $TcPo_2$ responses. Unless this proves to be the case then the technique is unlikely to be helpful.

1 Byrne P, Provan JL, Ameli FM, Jones DP. The use of transcutaneous oxygen tension measurements in the diagnosis of peripheral vascular insufficiency. *Ann Surg* 1984;**200**: 159-65.
2 Hauser CJ, Shoemaker WC. Use of transcutaneous PO_2 regional perfusion index to quantify tissue perfusion in peripheral vascular disease. *Ann Surg* 1983;**197**:337-43.

Sweet tooth maketh a sour disposition

Dr ALEX COMFORT (Cranbrook, Kent) writes: Dr George Dunea's amusing account of American dietary habits (12 July, p 120) is accurate, but it misses an important point of which, on coming home after 11 years in California, I am painfully aware. Whether or not Americans "eat prudently," the materials of prudent eating are available there. I particularly miss the excellent egg substitutes (made from egg white and corn lecithin), the non-cholesterol cream and cheese substitutes, and the quality of the polyunsaturated margarines, which taste like butter rather than soft soap. Even willing grocers cannot get hold of these products in Britain. One might hope that somebody would provide employment by making some of them under licence. If the American diet is to attract mockery the British diet should attract medical indignation, not least at the lethargy of food manufacturers.

Professor JOHN YUDKIN (London NW8) writes: Dr George Dunea's fascinating Letter from Chicago has one tiny error. It was not I who had the happy idea of coining the phrase "saccharine disease"; it was Surgeon Commander Cleave and Dr Campbell, who used this as the title of a book published in 1966.[1] They wrote about what is rather confusingly called "refined carbohydrate," which included sugar and highly milled cereals. A few years later I wrote a book dealing—like Dr Dunea—only with sugar, which was published with the title *Pure, White and Deadly*.[2]

1 Cleave TL, Campbell GD. *Diabetes, coronary thrombosis and the saccharine disease*. Bristol: Wright, 1969.
2 Yudkin J. *Pure, white, and deadly*. London: MacGibbon and Kee, 1972.

The doctor, the patient, and their contract

Dr STEPHEN J WATKINS (Department of Community Medicine, Oldham Health Authority, Oldham OL1 1JT) writes: In reviewing alternative contracts for general practitioners Professor Alan Maynard and colleagues (31 May, p 1438) assert that a salaried service offers the least degree of consumer control. This is certainly true if the contract of the salaried doctor is held by a remote bureaucracy. However, if the contract is held by a neighbourhood health committee elected by patients of the practice (as advocated by the Medical Practitioners' Union) the situation is different. Such an arrangement would offer maximum consumer control. It would also offer maximum professional freedom to the doctor provided the doctor retained the confidence of patients.

Correction

Asystole and electromechanical dissociation

We regret that two errors occurred in this letter by Dr C S Hopkins (14 June, p 1598). In the second paragraph there are two references to electromechanical dissociation, at the end of the second sentence and in the third sentence. In both cases this should have read external cardiac massage.

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