course in occupational health, then proceeding to obtain his Diploma of Industrial Health. He is now established as a full time occupational health physician, and has successfully moved from one industry to a totally different one.

Many general practitioners develop a special interest in one branch of medicine, which they successfully pursue not only in their own practice but often as a clinical assistant or hospital practitioner (one or more hospital sessions a week). Quite fortuitously I developed a special interest in the "third age" by being asked to give a lecture at the local college on "health in retirement"—a subject to which I had previously devoted no thought whatsoever. Several years later this led to my making a film on the subject (produced by a professional unit) using several good examples from patients of my own. By acquiring a knowledge of film making I have subsequently been invited to participate in assessment of medical films both for general distribution and for competition awards.

I hope that Dr O'Donnell's words of wisdom will not fall on deaf ears—both in encouraging not only potential career changers but also those who, at the final hurdle, make it possible—I refer to our senior colleagues who are on the appointment committees.

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Repeated renal failure with captopril

SIR,—We recently reported acute interstitial nephritis after treatment with the angiotensin converting enzyme inhibitor, captopril, in a cystinotic renal allograft recipient (27 November, p 1538). We have subsequently used the newly developed angiotensin converting enzyme inhibitor, enalapril (MK 421), to control this patient's hypertension without any adverse reaction.

After the withdrawal of captopril in this 8 year old girl blood pressure was poorly controlled on diazoxide 200 mg daily and metoprolol 200 mg twice daily, and she developed hypertensive encephalopathy (blood pressure 250/140 mm Hg) and severe hyperglycaemia (blood glucose 58 mmol/l (1045 mg/100 ml)). Blood pressure was controlled with intravenous hydralazine, but gradually rose to 180/110 mm Hg despite treatment with prazosin 7.5 mg eight hourly, hydralazine 25 mg six hourly, clonidine 100 µg six hourly, labetalol 400 mg eight hourly, frusemide 125 mg twice daily, and spironolactone 25 mg eight hourly.

Conventional hypotensive agents were then stopped, and the new oral angiotensin converting enzyme inhibitor, enalapril, which differs structurally from captopril and does not contain a sulphydryl group, was started in a dose of 1 mg increasing to 5 mg twice daily. Enalapril, together with frusemide 80 mg twice daily, controlled the blood pressure (see figure) with no consistent

change in renal function. The dose was subsequently adjusted to 7.5 mg daily, and the blood pressure has remained well controlled with stable renal function seven weeks after starting the drug.

We have thus successfully used enalapril in a patient with three previous episodes of captopril related deterioration in renal function. On the basis of the clinical course and the appearance of the renal biopsy sample we had considered the acute renal failure represented captopril induced acute interstitial nephritis rather than a renal functional deterioration secondary either to hypotension or angiotensin converting enzyme inhibition per se. The satisfactory response to enalapril in this patient confirms these conclusions and indicates that enalapril may be safely used in patients who have had adverse reactions to captopril.

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Confirming the diagnosis of mild hypertension

SIR,—Dr R M Hartley and others (22 January, p 287) say that the duration of the interval between visits to the doctor was not associated with a fall in blood pressure. This is true as far as their observations go, but I suggest that a longer trial would modify their view.

As long ago as 1948 Ayman¹ pointed out that patients attending a clinic weekly recorded lower pressures than when they were put on a monthly measurement, and my experience has confirmed this. Table I of the paper of Dr Hartley and others actually offers some support. Starting from comparable pressures, their group III cases, measured weekly, showed a mean fall of systolic pressure of 15.1 mm Hg in four weeks while group II, measured fortnightly, achieved only 10.5 mm Hg.

The only practical significance of this is that in treating patients one should not assume that control is deteriorating just because less frequent visits show higher pressures.

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¹ Ayman D. *Arterial hypertension*. New York: Oxford University Press, 1948.

Low phosphate arachidonic acid values in diabetic platelets

SIR,—Dr D B Jones and others (15 January, p 173) suggest that diabetics may have an inability to convert linoleic acid to arachidonic acid.

This has been firmly established in lower animals during the last 17 years by various workers, particularly by Brenner,¹ who has shown that the $\Delta 6$ -desaturase (which catalyses the first desaturation in the conversion of linoleic acid to arachidonic acid) in the microsomes of intact animals or cells in culture is increased by insulin and decreased in diabetic animals and by glucagon, adrenaline, thyroxine, dibutyl-adenosine 3':5'-cyclic phosphate, hydrocortisone, and ethanol. Hülsmann² showed decreased desaturation of fatty acids in diabetics. Earlier I had suggested³ that the

triopathy of diabetes might be caused by long-standing deficiency of essential fatty acids: the requirement of essential fatty acids is greatly increased in diabetic animals⁴; deficiency of essential fatty acids causes increased capillary permeability and fragility, which are found in diabetes, and there may be haematuria⁵; long-standing deficiency of essential fatty acids causes peripheral neuropathy, and perhaps in this the linolenate class of essential fatty acids is particularly important since the nervous system and retina are particularly rich in docosahexaenoic acid, which is formed from linolenic acid, and deficiency of this in man can cause neuropathy.⁶ Kinsell *et al*⁷ were the first to claim improvement of the retinopathy of diabetics with diets rich in linoleic acid, and this was subsequently confirmed by others.⁸⁻¹⁰ More recently linoleic acid has been shown to decrease platelet aggregation, as the authors state, and improve the increased aggregation in diabetes.¹¹

The values for the diabetics given in the two figures by Dr Jones and others must contain errors. The vertical coordinates in the two figures are the same (percentage of arachidonic acid), but only about six of the 20 values for diabetics appear to be identical in the two figures. For instance, figure 1 has two values above 22 and figure 2 none; figure 1 has four values below 12 and figure 2 six; the mean of the diabetic values in figure 1 appears to be 16.09 ± 4.94 and in figure 2 15.20 ± 4.23 . In view of the discrepancies perhaps the figures for "eicosapentaenoic" acid should have been given since the authors state that unlike the figures for arachidonic acid there was no significant difference between the means of diabetics and controls and no significant difference in the ratios of the two acids.

I have earlier¹² corrected the nomenclature of this acid as used by Dr Jones. Saturated fatty acids are -anoic, acids with one or more double bonds -enoic, and acids with one or more triple bonds -ynoic. The acid they refer to as eicosapentaenoic is not saturated. Their last reference contains three errors, one important (wrong volume) and two trivial.

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¹ Brenner RR. The desaturation step in the animal biosynthesis of polyunsaturated fatty acids. *Lipids* 1971;6:567-75.

² Hülsmann WC. Dieet en insulineresistentie. *Ned Tijds Geneesk* 1978;122:1133-6.

³ Sinclair HM. Essential fatty acids. In: Jolliffe N, ed. *Clinical Nutrition*, 2nd ed. New York: Harper, 1962:206-26.

⁴ Peiffer JJ, Holman RT. Essential fatty acids, diabetes and cholesterol. *Arch Biochem Biophys* 1955;3:520-1.

⁵ Burr GO, Burr MM. A new deficiency disease produced by the rigid exclusion of fat from the diet. *J Biol Chem* 1929;82:345-67.

⁶ Holman RT. Essential fatty acids in nutrition and disease. *Chemistry and Industry* 1981;704-9.

⁷ Kinsell LW. Prevention of vascular disease in the diabetic. *Diabetes* 1955;4:298-304.

⁸ Van Eck WF. The effect of a low fat diet on the serum lipids in diabetes and its significance in diabetic retinopathy. *Am J Med* 1959;27:196-211.

⁹ King RC, Dobree JH, Kok DA, Foulds WS, Dangerfield WG. Exudative diabetic retinopathy: spontaneous changes and the effects of a corn oil diet. *Br J Ophthalmol* 1963;47:666-72.

¹⁰ Houtsmuller AJ, van Hal-Ferwerda J, Zahn KJ, Henkes HE. Favourable influences of linoleic acid on the progression of diabetic micro- and macroangiopathy in adult onset diabetes mellitus. *Progr Lipid Res* 1981;20:377-86.

¹¹ Fleischman AL, Bierenbaum ML, Stier A, Somol H, Watson PB. In vivo platelet function in diabetes mellitus. *Thrombosis Res* 1976;9:467-71.

¹² Sinclair HM. Icosapentaenoic acid and ischaemic heart disease. *Lancet* 1982;ii:393.

Nifedipine in hypertensive emergencies

SIR,—The results of the study by Dr O Bertel and others (1 January, p 19) on the use of oral nifedipine for the rapid reduction of substantially raised blood pressure suggest that this is a reliable treatment with an attractively simple

Changes in plasma creatinine concentration and mean arterial pressure during treatment.

Conversion: SI to traditional units—Plasma creatinine concentration: 1 µmol/l = 0.01 mg/100 ml.