every time and write out a new prescription. The second, whereby carbon copies of prescriptions are made and postdated has always been cited by regional medical officers as the only way of repeat prescribing without seeing the patient. It is in the third method that he has failed in his duty to give legal guidance. He describes arrangements made "in most practices" whereby patients can collect repeat prescriptions. The fact that this is a common practice does not necessarily make it a legal practice. I note that he has carefully avoided stating that it is a legal practice, and it is this avoidance of a most important issue which requires clarification.

The only confirmation that I have had that this form of repeat prescription is officially recognised as correct is the recent issue of prescription forms suitable for use in a computer printer, such computer having been programmed to produce individual repeat prescriptions.

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* *We sent this letter to Dr Carne, who replies below.—ED, BMJ.

SIR,—Unlike the regulations controlling National Insurance Certification, I know of no regulation or law which obliges a doctor to see a patient face to face before writing a prescription. It has long been the practice, both in the UK and in most overseas countries, for a doctor to write a prescription which is repeatable without the patient seeing the doctor again: this was referred to in the original paper.

It is a common practice for doctors to advise and prescribe over the telephone. The regulations permit a doctor to phone the chemist and ask him to dispense a medication, though in such cases he has to follow this up with a written prescription—but without necessarily seeing the patient. In other words, by long custom and usage, it must be accepted that it is fully within the law and the NHS terms of service for a doctor to issue repeat prescriptions—or, indeed, to issue a prescription for a new episode without actually seeing the patient.

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Heat stroke and community runs

SIR,—Heat stroke during long-distance races is a serious problem, as evidenced by a race last summer near Washington DC in which two competitors died.

Dr M R Nicholson's recent letter (9 May, p 1544) mentions tight clothing, disease states resulting in reduced skin blood flow, pre-existing fever, and the use of anticholinergics and amphetamines as risk factors for heat stroke. Our research has suggested that there are a number of other factors the summer runner should be aware of so as to minimise the risks of running in warm weather. We recommend the following:

(1) Fluids: dehydration with the resulting reduction in sweating ability can be a major factor in the onset of heat stroke. The American College of Sports Medicine has recommended that runners drink one pint of liquid within 15 minutes of competing.¹ We recommend one

quart within three hours. Tea, coffee, and alcohol, all of which have diuretic properties, should be avoided for at least 12 hours prior to running. In order to minimise dehydration during a race, runners should drink one-and-a-half times what their thirst dictates. Particularly at early water stations they will have to force themselves to drink. Studies have shown that thirst is not an adequate measure of the need for liquids and will result in the consumption of only a half to two-thirds of what is needed totally to hydrate the body.^{2,3} Drinking an adequate amount of water has been called the most important supplement for the prevention of heat illness.¹

(2) Direct sunlight: because of the great amount of heat the body can absorb from direct sunlight, runners should run in the shade when possible and always wear light-coloured clothing, including a hat.

(3) Body cooling: a runner's primary means of dissipating heat is by the evaporation of sweat from the skin. Water evaporating off the body will work equally well for this purpose. Hoses and sprinklers provided by spectators as well as drinking water can all be used to keep runners wet.

(4) Drugs: phenothiazine tranquillisers are thought to act on the thermoregulatory centre of the brain and reduce the body's ability to regulate internal temperature.⁴ Anticholinergics reduce the body's ability to sweat.⁵ Diuretics promote water loss and can thereby help lead to dehydration.⁷ Runners should avoid hot weather racing if they are using any of these drugs.

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¹ American College of Sports Medicine. *Med Sci Sports* 1975;7:vii-viii.

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⁷ Craig FN. *J Appl Phys* 1970;28:779-83.

Beta₂-microglobulinaemia as index of diminishing renal function in diabetics

SIR,—I have read with interest the correspondence (6 June, p 1876) about renal function in diabetic nephropathy and β_2 -microglobulin. It seems possible that β_2 -microglobulin is a more sensitive index of reduced glomerular filtration rate (GFR) than serum creatinine or functions of serum creatinine, although why this should be so is not known.

Unfortunately, by the time either β_2 -microglobulin or creatinine are significantly elevated the diabetic patient is set on an inexorable course toward renal failure, which ensues a matter of months in some, several years in others, after the creatinine reaches 200 $\mu\text{mol/l}$ (2.3 mg/100 ml).¹ Nothing is known to prevent this happening and, although Mogensen² has demonstrated reduction in the rate of decline of GFR by aggressive treatment of hypertension, no relationship exists between the rate of change of GFR and either mean blood pressure or mean diastolic.¹ It is probable that by the time a diabetic has developed proteinuria on Albustix testing the situation is irreversible, for there is no convincing evidence that any therapeutic intervention modifies the course of diabetic nephropathy. For example, although abnormal platelet adhesion is corrected by the administration of an "antiplatelet" sulphonylurea (gliclazide), proteinuria is not affected and the

GFR continues to fall.³ The numbers of diabetics with chronic renal failure in need of dialysis and transplantation are rising, and treatment of these patients poses formidable problems. The important question to ask is whether much earlier phases of diabetic nephropathy, before clinical proteinuria develops, can be detected.

At present the outlook is not encouraging. Microalbuminuria, the passage of very small amounts of urinary protein, has been shown to be affected by the quality of diabetic control,⁴ but it is not known whether patients with microalbuminuria would go on to develop clinical renal disease. Jerums's work⁵ on the predictive value of measuring differential urinary protein clearances applies to patients who already have manifest renal disease. Urinary β_2 -microglobulin has been investigated in diabetic patients with proteinuria due to diabetic nephropathy⁶; when renal excretory function (GFR) is normal, urinary β_2 -microglobulin excretion and, by implication, renal tubular function are normal. There is a report, however, of a transient "tubular" pattern of proteinuria on urinary protein electrophoresis⁷ in diabetics, which the authors claim may be an early marker of renal malfunction. The patients were not followed up for long enough to see whether clinical proteinuria ensued. This does, however, raise the possibility of using other markers of renal tubular function such as N-acetyl glucosaminidase (NAG) to seek early renal damage. In this context, it is interesting that Yudkin *et al*⁸ recently reported a rise in urinary NAG excretion in young non-diabetic adults given a high-sucrose diet.

The earliest permanent structural change in diabetic nephropathy is probably basement membrane thickening in glomerular capillaries, as revealed by electron microscopy; this may be detected as early as one to two years after the onset of type-II diabetes. It is accompanied by increased basement membrane turnover.⁹ Whatever the cause of the basement membrane thickening it is possible that abnormal (perhaps glycosylated) basement membrane components remain to be detected in the blood or urine. Another approach may be to regard the thickened and glycosylated basement membrane as immunologically altered and capable of modifying certain immunological responses. There is evidence that this is the case in certain other renal lesions,¹⁰ raising the possibility of employing immunological methods to search for patients at risk of developing diabetic renal disease.

Once very early diabetic renal disease can be detected, the way will be clear for meaningful trials of, for example, agents which modify coagulation and to define the effect of control of the diabetes itself.

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² Mogensen CE. *Scand J Clin Lab Invest* 1976;36:383-8.

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⁵ Jerums G, Post RS, Miller M, Barzellato E. *Diabetes* 1973;22(2):104-10.

⁶ Jones RH, Marshall WJ, Myers RC, Watkins PJ, Parsons V. *Clin Chim Acta* 1980;108:375-83.

⁷ Lopes-Virella MF, Virella G, Rosebrock G, Sagel J, Gonzalez J, Colwell J. *Diabetologia* 1979;16:165-71.

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⁹ Brownlee M, Spiro RG. *Diabetes* 1979;28:121-5.

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Clinical and senior clinical medical officers' training

SIR,—All who have worked for a relevant training and an acceptable career structure for these clinicians will have been encouraged by the statement from the crafts evidence to the Review Body (23 May, p 1729).

"Broad agreement has been reached that the training of clinical medical officers should be defined and made compatible with that for