

Irrespective of any effects of antihypertensive drugs, systolic blood pressure itself correlates with risk of cancer.^{3,9} Though an increased incidence of cancer could be due to ascertainment bias because hypertensive patients are under closer medical surveillance than normotensive patients, this would not account for the association between mortality from cancer and blood pressure.^{3,10} Instead, it is typically hypothesised that this association is attributable to other confounding factors. For example, salt intake contributes to hypertension and is implicated as a causal factor in gastric cancer. Here, the concomitant decreases in salt intake, gastric cancer, and hypertensive cardiovascular disease during the twentieth century in most industrialised countries are noteworthy but fall short of establishing causality.¹¹ Analogously, heavy alcohol consumption contributes to both increased blood pressure and many malignancies.¹²

A powerful risk factor for hypertension is obesity. Because increased consumption of fat results in an increase in both weight and blood pressure and has also been implicated as a risk factor for some forms of cancer, dietary fat may explain the correlation between blood pressure and cancer.^{6,13} Similarly, low socioeconomic status simultaneously confers an increased risk of high blood pressure, certain cancers, and exposure to known carcinogens (for example, alcohol and tobacco). Nonetheless, no epidemiological study has yet shown (by multivariate analysis) that one or more of these hypothesised mechanisms truly accounts for the association between high blood pressure and cancer.

Lingering concerns therefore remain about the potential contribution of hypertension and its treatment to the incidence of cancer. Any increase in cancer with a particular antihypertensive drug will be difficult to detect because of both the dearth of large scale clinical trials which directly compare different treatments and the relative infrequency of cancer in prospective studies. Still, such evaluation is important because, for example, if the incidence of cardiovascular complications of hypertension is 5/1000/year and if a drug reduces risk by 30% (that is, by 1.5/1000/year) then with an increase in cases of cancer of just 2/1000/year a particular drug could do more harm than good. It becomes important, therefore, that any new antihypertensive drug should be

compared with other agents in clinical trials that are large enough to detect infrequent adverse morbid events. With respect to first line treatment with diuretics and β blockers, consolation is available in the fact that meta-analysis of large, randomised clinical trials have not identified any increase in deaths from cancer and the use of these drugs seems to reduce total, as well as cardiovascular, mortality.¹⁴

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Diabetic care in general practice

Adding quality to quantity

Treating eye, kidney, and foot lesions early in diabetic patients can prevent blindness, renal failure, and amputation,¹ and the sheer number of patients who would benefit from early diagnosis means that general practitioners have to play a part. Early papers on the topic showed that effective surveillance depends on an up to date register, a recall system, and a simple checklist,^{2,3} and many primary care teams are now grappling with Tudor Hart's challenge of "doing simple things well, for large numbers of people, few of whom feel sick."⁴

Some teams have made great progress: if well organised they can screen diabetic patients at least as well as outpatient clinics.⁵ Moreover, several different approaches have been found to be effective, including all day clinics,⁶ nurse coordinated care,⁷ and miniclinics.³ But what of practices that still do not have the infrastructure or confidence to offer diabetic surveillance?

On page 624 Hurwitz and colleagues report on a prompting

system for diabetes surveillance independent of practices themselves.⁸ This development meets the needs of local practitioners for support in personal education, patient recall, and retinal screening; flexibly integrates generalist with specialist care; and, perhaps best of all, puts patients first by sending the prompt directly to them. The evaluation shows that two thirds of surveillance activity shifted from hospital clinics to primary care, with more effective coverage in the prompted group than in the control group and acceptance levels well above 80%.

Although these findings are encouraging, only people regularly attending diabetic outpatient clinics were studied and only half of those eligible took part. Nor is it clear how much health gain resulted from this screening programme. Surveillance may be carried out adequately by a skilled practitioner in 10 to 14 minutes once or twice a year, but, to have any impact, discussions about management and lifestyle to reduce the risk of complications will take longer.

Health gain has been rather narrowly defined in primary care studies of diabetic care to date. When audits have gone beyond counting activity alone they have focused mainly on blood glucose concentration as a proxy measure of outcome.⁶ Pringle and coworkers do this in their large descriptive study of possible associations between a range of variables related to the patient, doctor, practice, and process of care and the concentrations of glycated haemoglobin in diabetic patients in Nottingham (p 630).⁹

Attempts to capture the contribution of more than 25 factors possibly associated with glycaemic control left 85% of the variance in glycated haemoglobin concentrations unexplained. Among modifiable factors only access to a dietitian and an interested general practitioner featured significantly in the final multiple regression analysis.

Such a study raises many questions. The findings support other evidence of the importance of doctors' attitudes towards helping patients to achieve good glycaemic control¹⁰ and that skilled strategies to support dietary change can make an appreciable contribution to glycaemic control¹¹ and may even affect survival.¹² The study also raises the question of which are the right outcomes to measure and how psychosocial variables should be taken into account.¹³ Future studies might pay more attention to the beliefs and behaviour of patients, doctors, and nurses as these probably strongly affect morbidity and mortality in diabetes.¹²⁻¹⁴ We might also base management more firmly still on a multifactorial model of the risks of microvascular and macrovascular disease, paying particular attention to smoking, blood pressure, and lipids as well as blood glucose.¹⁵ At the same time we need to remember that interventions, particularly those aimed at minimising hyperglycaemia, can themselves impair the quality of life.^{13, 16}

The increasing recognition that diabetes frequently coexists with other chronic diseases¹⁵ and may originate in early life¹⁷ suggests an increasing role for primary care in the manage-

ment and prevention of the condition in future. Attention to surveillance and the rational use of primary and secondary resources are still needed, but as we try to add "quality to quantity" we may need to look to social science as well as to epidemiology. Its methods may improve our understanding of our patients and ourselves and form the basis for behavioural changes, on which reduction of the risks of the disease still largely depend.¹¹

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Treating hypoglycaemia in general practice

Glucagon is the drug of choice for unconscious, agitated, or uncooperative patients

Severe hypoglycaemia is probably the commonest metabolic emergency seen in general practice. People with diabetes are mainly affected, but other causes (except in neonates) include excess alcohol consumption after inadequate food intake¹ and insulinomas and non-islet cell tumours, especially mesotheliomas. These and other uncommon causes should be considered when hypoglycaemia is suspected in non-diabetic patients.

Diabetic hypoglycaemia occurs mainly in insulin treated patients. It has been estimated that mild, self managed episodes may occur about twice weekly; severe episodes without unconsciousness but requiring help from another person twice yearly; and episodes of unconsciousness three times in 40 years.¹

Proper education of patients should reduce the incidence of severe hypoglycaemia. All patients with diabetes and their relatives or carers should understand the range of hypoglycaemic symptoms, appropriate self treatment, and when to call for help. In patients taking insulin this may mean deliberately inducing hypoglycaemia under controlled circumstances. Patients in whom there is a loss of warning of hypoglycaemia, possibly linked with the use of human insulin,³ should be identified, counselled, and given the

choice of switching to animal insulins (specific advice is available from the British Diabetic Association).

Medical help is likely to be sought only when hypoglycaemia is severe and the patient is unconscious, agitated, or uncooperative. These are genuine emergencies as prolonged hypoglycaemia can be damaging. Ideally, the diagnosis should be confirmed before treatment, and this can be done with capillary blood glucose test sticks. Although an unconscious patient is likely to have biochemical hypoglycaemia (blood glucose concentration <3.0 mmol/l), the relation between symptomatic and biochemical hypoglycaemia may be poor, with one study showing biochemical confirmation in less than a third of symptomatic patients.⁴ Ultimately, the diagnosis must therefore be clinical.

These patients should be treated parenterally because attempted oral treatment may be hazardous.⁵ The choice of treatment is between intravenous 50% dextrose (usually 50 ml) and subcutaneous, intramuscular, or intravenous glucagon (0.5 mg in children and 1 mg in adults); intranasal glucagon is also effective.⁶ Both treatments produce a good response, though dextrose acts almost immediately and glucagon takes one to two minutes.⁷ This time difference may be outweighed by the need to secure intravenous access for