age confirmed the view of Poskanzer that differences in age structure have relatively little effect on observed differences between surveys.⁶

The overall prevalence of 115/100 000 is the third highest recorded for a first survey of an area in the United Kingdom, exceeded only by 127 in north east Scotland⁷ and 134 in Shetland.³ The Shetland prevalence, however, is subject to the imprecision caused by a large sampling error mentioned earlier. The method of ascertainment in the present survey, which used Hospital Activity Analysis data and social services information, differed widely from methods used in the earlier surveys of Northern Ireland, north Scotland, and Durham and Northumberland so that comparison was not worth while. The most valid comparison was with the first survey of north east Scotland, which also used detailed methods of ascertainment.

Table IV compares the main results of the two surveys. Overall prevalence was slightly but not significantly higher in north east Scotland; age standardisation, however, accentuated the difference. Mean age, mean age at onset of multiple sclerosis, mean duration, and estimated mean incidence were remarkably similar in the two surveys. Sutton had a higher percentage of women and more people with probable multiple sclerosis and fewer with early probable or latent multiple sclerosis. Classification bias may have accounted for some of the difference between diagnostic groups as different methods of classification were used; in north east Scotland 77% of patients were classified after a physical examination, whereas in Sutton patients were classified using information abstracted from medical records. The second and third surveys of north east Scotland produced prevalences of 144 and 178/100 000 respectively, which were significantly higher than the prevalence in Sutton. How much of these differences were due to the effect of repeated studies, however, is unclear.^{2 5 6 8 9}

Using a nine year average mean is a crude method for estimating incidence. It assumes no change in the disease over time and is biased by people lost to follow up through death and migration. Using data for the latest nine years probably provided sufficient cases for a reasonable estimate of incidence. Interestingly, the mean annual incidence in north east Scotland in 1973 (5.3) was similar to that of Sutton in 1984 (5.0).

In support of Limburg's hypothesis that the frequency of multiple sclerosis increases with the distance of the surveyed area from the equator,¹⁵ Acheson proposed the existence of a simple linear relation between prevalence and geographical latitude in Europe, prevalence being higher in the north and lower in the south.¹¹ A comparison of the prevalence in north Scotland and Cornwall produced in the 1950s, however, failed to show any significant north-south gradient. Moreover, the prevalence found in our survey suggests that the latitudinal effect in the United Kingdom may be less than is generally accepted. Moreover, in a household survey of disabled people in the London Borough of Kensington and Chelsea carried out under the Chronically Sick and

Disabled Persons Act during the early 1970s the number of people who reported their disability as caused by multiple sclerosis gave a prevalence of 130/100 000,¹⁶ which was similar to the prevalence recorded in Scotland.

We conclude that to date the evidence provided by United Kingdom surveys of a north-south gradient in prevalence is less than convincing. Although it is generally accepted that the United Kingdom has a relatively high prevalence, usually taken as over 30-40/100 000, on the basis of this survey the prevalence for south east England is around 100/100 000. This prevalence may well apply more widely in the United Kingdom, but to establish this supposition will require further investigation.

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SHORT REPORTS

Captopril in elderly patients with heart failure

There are many reports on captopril but few data on its use in the elderly. We report our experience with captopril in an open study of an elderly population with heart failure.

Patients, methods, and results

Thirty elderly patients (mean age 81.6 (range 71-92) years) with congestive cardiac failure that had not responded to frusemide 40-120 mg or its equivalent were recruited into an open 12 week study of captopril. Cardiac failure had been present for an average of 11.9 (0.5-48) months. Seventeen patients were New

York Heart Association class II, eight class III, and five class IV. Sixteen were in-patients at the start of the study, and 14 were outpatients.

Eighteen patients completed the study; the remaining 12 were withdrawn because of non-compliance (failure to take more than 80% of the tablets) (five patients), intractable heart failure that did not improve when they took captopril (two), death from cardiac infarction (one), death from respiratory failure (one), vertebrobasilar insufficiency (one), death from gastrointestinal haemorrhage (one), and disseminated carcinoma (one). Multiple disease is a feature of this age group, and the deaths were probably unrelated to the drug.

The cardiac output was measured by a computerised densitometer with a diachromatic earpiece¹ and was taken to be the mean of three values. The dye was injected rapidly into the antecubital vein. The first dose of captopril (6.25 mg) was administered orally and the patient monitored in the supine position for three hours. Captopril 6.25 mg twice daily was then taken until the next assessment, when the dose was increased to 25 mg twice daily for the next four weeks. Thereafter the dose was increased if the cardiac failure was not controlled. If there was evidence of fluid retention the diuretic dose was increased but reduced if

failure was controlled. Variables were reassessed at two, four, eight, and 12 weeks.

The patients tolerated the first dose of captopril well, showing a mild transient hypotensive response. One patient had a notable hypotensive response to the first dose but was asymptomatic and improved when her legs were raised. Twelve patients who were taking captopril three times daily at eight weeks had the regimen changed to twice daily. At 12 weeks the median dose of captopril was 75 mg daily in two divided doses.

The table shows the haemodynamic measurements at the start and end of the study. The cardiac variables showed a significant improvement, but there was no significant difference between the variables measured when patients were taking captopril three times daily and those when they were taking it twice daily. Four

Means (SD) of important variables measured at beginning and end of 12 week study

	Beginning of study	End of study	Significance
Cardiac index (l/min/m ²)	2.21 (0.35)	3.09 (1.00)	p<0.005
Stroke volume index (ml/min/m ²)	28.04 (5.70)	39.32 (16.20)	p<0.005
New York Heart Association class	2.61 (0.78)	1.67 (0.49)	p<0.005

patients showed little or no improvement in haemodynamic variables and some required a higher dosage of diuretic. There were no significant changes in plasma electrolyte, urea, or creatinine concentrations. Five patients developed minimal and fluctuant proteinuria. The mean weight fell from 60.5 kg at the start of the study to 58.3 kg at 12 weeks, but this change was not significant. There were no side effects. Many subjects reported a sense of improved wellbeing, and this observation concurs with those of other workers.² Some subjects initially gained weight after starting to take captopril; this has been noted by other workers³ and did not require alteration of the diuretic dose.

Comment

This study shows that captopril is well tolerated by the elderly and results in an apparent improvement in the symptoms and variables of cardiac function when taken twice daily. Further studies in this age group are needed to clarify the role of this promising drug.

E R Squibb and Sons Limited supplied the drugs for this study.

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Department of Geriatric Medicine, University College Hospital Medical School, St Pancras Hospital, London NW1 0PE

PATRICK J MURPHY, MB, MRCP, senior registrar
TISCHA VAN DER CAMMEN, MD, research fellow
JAMES MALONE-LEE, MB, MRCP, senior lecturer

Correspondence to: Dr Malone-Lee.

Cannulation of difficult oesophageal strictures with angiographic catheters

Many non-operative techniques to relieve dysphagia due to benign or malignant oesophageal strictures have been described,¹ but the most widely used is peroral dilatation with or without intubation of the stricture once a guide wire has been passed under direct vision using a fiberoptic endoscope. Roughly 3-10% of strictures, however, remain impassable,² requiring laparotomy and gastrotomy for a traction technique with its attendant morbidity and mortality of 18-33%.^{3,4} We describe a method of cannulation of these difficult strictures with radiographic control.

Patients, methods, and results

Over the last 12 months endoscopic dilatation of 158 benign and malignant oesophageal strictures was attempted. In seven cases a guide wire could not be

passed through the stricture despite radiographic screening, and these patients proceeded to the angiographic catheter technique described below. Three strictures were due to oesophageal carcinoma, two were benign, one occurred after insertion of an Angelchik prosthesis, and one was due to extrinsic mediastinal node compression.

A 3 mm J guide wire 145 cm long with a steerable core (William Cook, Europe) is introduced into the upper oesophagus (figure, A). Either the wire is passed through the channel of an endoscope or the control catheter can be used to give it sufficient stiffness. A multipurpose 6.5 French torque control catheter (B) is then introduced over the guide wire, and with the wire just protruding from the catheter tip the stricture is gently probed. Often the wire slips easily through into the stomach, but if it does not it should be withdrawn and water soluble contrast (Conray 280, 10-20 ml) injected down the catheter to delineate the stricture. Tilting the table head up helps the contrast to run through the stricture, and the guide wire can then be passed into the stomach. The multipurpose catheter is then removed and an Amplatz dilator, 80 cm long (C), is passed over the wire into the stomach. This dilator is not tapered but, with the J wire removed, allows a heavy duty endoscopy guide wire (D) to be passed. The Amplatz dilator is removed, and dilatation can proceed in the usual manner.

J guide wire (A) for cannulating stricture used with multipurpose torque control catheter (B) if required; larger bore Amplatz catheter (C) introduced over (A) then admits endoscopy guide wire (D).

Cannulation of all seven strictures was achieved using this technique and was followed by successful dilatation, and by immediate intubation in one case. No patient sustained a perforation of the oesophagus during cannulation or subsequent dilatation and intubation. Five patients were discharged within three days; all had symptomatic relief of their dysphagia and were able to take a soft diet. Two patients died from carcinomatosis shortly after their strictures were relieved.

Comment

This new method of cannulating oesophageal strictures with a steerable angiographic guide wire passed under radiographic control was successful in seven consecutive strictures that were impassable at endoscopy; laparotomy, with its attendant high morbidity and mortality, was thus avoided. The procedure is simple and can be performed in any endoscopy suite with radiographic screening facilities or in radiology departments.

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Departments of Gastroenterology and Diagnostic Radiology, Royal United Hospital, Bath BA1 3NG

MANDY S SHARPE, MRCP, medical registrar
A H CHALMERS, FRCP, consultant radiologist
K R GOUGH, FRCP, consultant physician

Correspondence to: Dr Gough.