STROKE

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Office of Health Economics

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Introduction

In England and Wales, in 1986, stroke accounted for just over 12 per cent of mortality from all causes, most of it occurring in the elderly. Undoubtedly the death toll is considerable, however, it is the burden of morbidity and disability that stroke places on the community that is the real issue for concern. Each year approximately two people out of every 1,000 will experience a first stroke of whom about two-thirds will survive requiring some form of medical intervention. Indeed it is estimated in this paper that stroke patients consume just under one pound in every twenty-five of all National Health Service expenditure.

Analysis, by age, of the number of cases of stroke reveal that it is predominately a disease of the elderly. Present estimates indicate that 50 per cent of the total number of first strokes occur in the 75 and over age-group. If current demographic trends which point to future populations containing increased numbers of elderly people continue, the implications for stroke management is that the disease will remain a significant source of morbidity.

Despite available evidence highlighting the magnitude of the problem posed by the disease in this country, stroke remains a much misunderstood and mismanaged health issue. Concern has been growing regarding the quality of care offered to stroke patients, which, it is suggested does not meet the problems of long-term disability faced by many patients. The latter point raises an interesting debate regarding not only the efficacy of present rehabilitative methods but questions whether stroke patients are being allowed to recover in the most favourable environment. Indeed, there has been much criticism recently concerning the numbers of stroke patients hospitalised for long periods of time, who, it is suggested might fare better at home or in a specialised stroke unit.

Many aspects of the aetiology of the disease are still a mystery. The pathology of stroke has close links with ischaemic heart disease, in so much that both conditions develop from a background of vascular disease, and are associated with some of the same risk factors, albeit to varying degrees. However, unlike heart disease, epidemiology has revealed one risk factor, hypertension, to be more significant in the case of stroke than any others. Medicines aimed at lowering blood pressure have proved successful, in clinical trials, in reducing the incidence of stroke. In order to extend these beneficial effects to the population as a whole, requires an extensive screening effort at primary care level, and raises the debate surrounding the use of non-pharmacological means as an alternative to lowering blood pressure.

On a more optimistic note, however, the number of therapeutic options for stroke available to medical professionals has increased in recent years with, for example, the advent of clot lysing agents that increase cerebral blood flow immediately after a stroke, and new preparations that salvage tissue around the core of the lesion under threat from metabolic abnormalities, that occur during the vulnerable period.

This paper begins with a description of the natural history of stroke, and goes on to analyse the incidence and mortality patterns associated with the disease. The wide spectrum of contemporary issues central to the field of stroke management are evaluated and discussed – from advances in medical procedures, through to the rehabilitation of stroke survivors, and the extent of the financial burden the disease places upon the country's National Health Service.

The nature of stroke

Brain tissue has no oxygen reserve and, therefore, depends on a continuous flow of blood, providing oxygen and other essential nutrients, in order to maintain its survival. The blood supply is obtained from four main vessels: the right and left carotid arteries at the side of the throat and the two vertebral arteries which run up through the bones in the neck. These two pairs of major blood vessels, linked by the anterior and posterior communicating arteries, converge on the lower surface of the brain to form the 'circle of willis'. From this point, blood is transported through the brain via a network of branch arteries. Should a blockage in one of these arteries occur without being made good by a compensatory flow of blood from other vessels the area of the brain served by the occluded artery will be deprived of its oxygen supply.

Stroke is the term employed to describe the severe acute manifestations of cerebrovascular disease which result from interruptions to blood flow in the brain. In 'official' terminology, the World Health Organisation defines stroke as involving 'rapidly developed clinical signs of focal (or global) disturbance of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than a vascular origin' (WHO 1973). Acute events originating from head injury or neoplasm as well as those in which the signs and symptoms of the episode disappear within a short period of time (transient ischaemic attacks – see Box 1) are therefore excluded from the formal definition of stroke.

The clinical sequelae of stroke are wide ranging and include impairments to speech, balance, vision, touch sensation and movement. However, the nature of functional loss as well as the extent of disability, the speed of progression from the onset of symptoms and whether or not consciousness is lost, show a considerable degree of variation between patients. A key determinant in at least the first of these areas is the location within the brain at which the blood supply becomes impeded or ceases to flow. The brain is subdivided into a number of different regions each of which is more or less associated with a particular function or sensation. For example, diminished blood flow to the cerebellum may result in impaired muscle regulation and balance, whereas a corresponding loss to the occipital lobes may interfere with the sense of sight.

Box 1 **Transient ischaemic attack**

Thrombi and emboli are the intrinsic aetiological features of cerebrovascular disease instrumental in the development of temporary regions of ischaemic brain tissue which when prolonged are classified as infarctions. The former is in clinical terms referred to as a 'transient ischaemic attack' (TIA). The symptoms resemble very closely those manifested by infarction, the only difference being the time factor involved. The requirement for establishing a clinical diagnosis of TIA, as opposed to stroke, is that signs of neurological impairment have cleared within an arbitory limit of 24 hours. In reality, although symptoms are observed to last up to 24 hours, the period of ischaemia usually takes place after the acute event is over, therefore no abnormal neurological signs are present. An attack where mild symptoms remain is sometimes referred to as a reversible ischaemic neurological deficit (RIND) or a mild stroke. One patient in five presenting with the clinical picture of TIA will develop a complete stroke within a month, and in the light of this fact, should be regarded as important precursors of cerebrovascular accidents. Emboli from the heart, is a common source of TIA, accounting for approximately 30 per cent of cases, however 40 per cent of cases examined by angiography reveal the source to be disease of the carotid artery. Less common sources of TIAs are haematological disorders, vasospasm (e.g. due to trauma during neurosurgery) and transient reduction in blood pressure due to cardiac arrhythmia. It remains to be said that a considerable proportion of TIA patients on examination do not appear to have any serious or identifiable underlying disease.

Stroke pathology

Strokes may be divided into two broad categories according to the type of pathological process involved – infarction related and haemorragic. The former which accounts for around three-quarters of strokes, involves processes similar to those underlying many heart attacks. Arteries supplying particular parts of the brain may become blocked by the localised development of thrombi or through the deposition of emboli which had originally formed elsewhere in the circulatory system. The thrombotic process may originate with the development of atheromatous plaques at a vulnerable site in the arterial wall. These lesions may then grow in size and eventually lead to occlusion of the artery as material is shed from the plaque. Alternatively, vessels which have already narrowed from the accumulation of atheroma may finally become completely blocked by the arrival of emboli from erupting plaques located in other parts of the brain or at other sites in the circu-

latory system, such as the heart. In both of these instances, however, the outcome is a severe reduction in, or complete cessation of blood flow which deprives the affected area of the brain of oxygen and leads to the death of tissue.

The other major cause of stroke is haemorrage which accounts for about 20 per cent of acute cerebrovascular events. Within this group, most (75 per cent) strokes are the result of primary intracerebral haemorrhage. Typically, an artery within the brain bursts predisposing to the formation of a blood clot which may cause pressure in the part of the brain concerned and possibly cut off its blood supply. In contrast, subarachnoid haemorrhage involves the rupture of one of the arteries on the surface of the brain. Blood spills into the fluid-filled space between the brain and the skull. Events of this nature, which account for 25 per cent of haemorrhagic strokes, are most frequently the result of congenital berry aneurysms or vascular abnormalities.

The cause of stroke

In common with coronary heart disease, there is no single specific cause of stroke, but several risk factors have been identified which are associated with a raised probability of suffering an event of this nature.

Epidemiological studies are consistent in their findings that hypertension is far and above the most potent independent risk factor for stroke. Hypertension is associated with dietary factors and such activities as alcohol consumption, however, there is increasing evidence from on-going prospective epidemiologic studies such as the Heart Disease Study at Framingham, Massachusetts, that these and other adverse contributing factors exert an independent influence on the risk of stroke.

Hypertension

Increased risk of stroke is observed, for both sexes and all ages, with each increment of blood pressure throughout its range, i.e. from normotension (<140/90 mmHg) to borderline high blood pressure, to hypertension (>160/95 mmHg). Hypertension – mild, moderate or severe – exerts the greatest adverse effect on stroke incidence. As well as being a potent contributor to stroke morbidity and mortality, hypertension is an extremely prevalent condition – the World Health Organisation estimates that as many as 10 to 20 per cent of the population may be hypertensives.

Increased levels of systolic pressure with no corresponding increase in diastolic pressure, is prevalent in the elderly and is referred to as isolated systolic hypertension. It has been commonly assumed that the latter is a relatively harmless feature of the ageing process. However, data from the Framingham study has estimated at least a two-fold increase in the risk of cerebral infarction among individuals aged 65 to 84 years, with systolic pressures greater than 160 mmHg and amongst whom the diastolic pressure is consistently below 95 mmHg (Wolf *et al*, 1983).

Cigarette smoking

Cigarette smoking has been associated with an increase in the risk of stroke, although evidence for such a link, accumulated from prospective and retrospective studies of various populations, has not been as substantial and consistent as the evidence regarding the impact of cigarette smoking on coronary heart disease.

Nevertheless, results of recent investigations have confirmed that there is an association between the two variables that is statistically significant. For example, a case controlled study undertaken in Auckland, New Zealand, has suggested that cigarette smokers experience a three-fold increase in the risk of stroke compared with non-smokers (Bonita *et al*, 1986). Similarly the Honolulu Heart Programme revealed a two to three-fold increase in the risk of stroke after other risk factors were taken into account (Abbott *et al*, 1986). The most recent data to emerge from the Framingham Study, confirm this adverse effect of smoking on stroke incidence whilst also demonstrating a dose-response relationship – in other words – the risk of stroke increased as the number of cigarettes smoked increased.

Of more significance, as regards to preventive medicine and increasing our knowledge of the processes behind the effect of smoking on cerebrovascular disease, is the outcome on stroke incidence among subjects of the Framingham Study who quit the smoking habit. Seemingly, stroke risk falls to the level of non-smokers by five years from cessation of smoking, the effect decreasing significantly after only two years of abstinence, suggesting that it is never too late to give up smoking. On the strength of such evidence the pathological mechanisms underlying the impact of cigarette smoking on stroke is now decreasingly believed to be the result of the promotion of atherosclerosis, rather, it is thought that smoking precipitates the actual clinical event. A reason behind this is thought to be the increase in fibrinogen levels (the soluble protein in the blood that triggers the formation of fibrin – a blood-clotting agent) associated with smoking.

Heart Disease

Hypertension is the most important risk factor for stroke, but throughout the range of blood pressure, the threat of stroke is heightened by the presence in some subjects of coronary heart disease. The latter increases the risk of stroke by two-fold and congestive heart failure has an even more significant impact on stroke occurrence (Wolf *et al*, 1983).

Other cardiac impairments associated with an increase in stroke risk include evidence of left ventricular hypertrophy, which indicates the presence of sustained hypertension, and has been demonstrated to increase the risk of stroke by four-fold, independent of blood pressure and age. In addition, rhythm abnormalities such as atrial fibrillation (AF) and non-rheumatic AF have been shown to contribute to the threat of stroke. Indeed the results from the Framingham Study have revealed that non-rheumatic AF increases the likelihood of stroke by five times the rate for those subjects free from the condition.

Diabetes

Diabetic patients are prone to developing atherosclerosis, a precursor for stroke. This occurs more often in the leg arteries, but according to data from the Framingham Study, coronary and cerebral vessels are also affected (Wolf *et al*, 1983). The same study concluded that diabetes as a risk factor for stroke is more significant in women than men, and accounts for a greater proportion of brain than myocardial infarctions. It has been estimated that blood sugar levels for 160 mgm/dl, lead to an approximate doubling in the likelihood of an acute cerebral event. The impact of this condition does not wane with advancing age in either sex, however, as the prevalence of diabetes in the population is only about one or two per cent, the population attributable risk is relatively small.

Pill-associated stroke

The use of oral contraceptives also appears to be a risk factor for stroke, the first suggestion of such an association appearing about 20 years ago. Several stroke-promoting pathological features have been associated with the adverse effects of oral contraceptives, including, thrombosis, ebolism, arterial abnormalities and dysplasia (abnormal tissue development that can lead to occlusion of the arteries). Pill-associated stroke is more prevalent within certain high-risk category users, for example, patients with a history of migraine; persons aged over 35 years who have been taking the pill for a long period of time; diabetics; hypertensives; and most importantly cigarette smokers (Wolf *et al*, 1983). In addition contraceptive pills containing high levels of estrogren have been associated with a greater risk of stroke.

However, many studies have concluded that pill-associated stroke risk is minimal. To clarify the situation, the Oxford Family Planning Association Study has investigated contraceptive use by over 17,000 women, who when recruited between 1968 and 1974 were all married and aged 25–39 (Vessey *et al*, 1984). At the end of January 1984, data accumulating to 192,000 women, years of observations were analysed and suggested that any increase in the risk of subarachnoid haemorrhage is modest, probably not more than about 1.5 fold to 2 fold in comparison to non-pill users. The data were also suggestive of a somewhat greater increase in the risk of non-haemorrhagic stroke that appears to be confined to current users.

Alcohol

Excessive consumption of alcohol may be another – and to date underestimated – risk factor for stroke, especially among males. In a recent case-controlled study in Birmingham 230 patients with stroke between 20 and 70 years were compared for alcohol intake with controls matched for age, sex and race. Among the male cohort the relative risk of stroke was four times higher in heavy drinkers (heavy consumption = 300g or more per week) than in non-drinkers*. The effects of heavy

*Footnote: 8g of alcohol is equivalent to a half pint of beer, a glass of wine or a single measure of spirits.

drinking on the female population in terms of stroke risk could not be analysed as there were too few women in this consumption category. Adjustments for hypertension, cigarette smoking and medication led the investigators to suggest that high alcohol intake is an independent risk factor for stroke that can and should be prevented.

The association between excessive alcohol consumption and increase in stroke occurrence is usually attributed to raised blood pressure levels. However, other adverse effects of alcohol consumption that may contribute to stroke have been observed. Alterations to cardiac rhythm have been noted, prolonged alcohol consumption can cause cardiomyopathy (disease of the heart muscle) and alcohol has been demonstrated to encourage platelet aggregation (blood clotting). In addition it has been suggested that long-term alcohol abuse may reduce cerebral blood flow by causing vessels in the brain to contract or by interfering with cerebral metabolism (Wolf, 1986). Finally, smoking is more prevalent amongst heavy drinkers, who are therefore exposed to the additional risk factors associated with the former.

Hematocrit

Reduction in cerebral blood flow is experienced when blood viscosity increases. A determinant of the latter is the level of concentration of red corpuscles in the blood (haemoglobin). Hematocrit (a graduated capillary tube) is the means by which haemoglobin is measured. Pathologically raised hematocrit is a known indicator of increased stroke risk, however, recent investigation has centred around the possibility that even within the upper range, of what is recognised to be a normal hematocrit level, there is an increased risk of stroke, and cerebral infarction in particular. Evidence accumulated from the Framingham study, indicates that small penetrating arteries of the brain and also diseased major cerebral vessels that have become narrowed, are at risk, at such levels of hematocrit (Wolf *et al*, 1983).

Seemingly, venesection (blood-letting from a vein) relieves the condition by reducing blood viscosity. Investigations from the Framingham study conclude that although cigarette smoking and hypertension are associated with high normal concentrations of blood haemoglobin, it can be demonstrated that such levels of hematocrit exert an independent influence on cerebral infarction.

The occurrence of stroke

In Britain more than a dozen studies of the occurrence of stroke have been carried out over the past three decades. In theory, the findings of these separate investigations might be brought together to construct a time series of incidence rates over the last 30 years or so. In turn, these data might then be employed to shed some light on the uncertainties that surround the causes of the recent reductions in stroke mortality. It is not clear, for example, to what extent these trends reflect declining

case fatality rates rather than fundamental changes in the occurrence of cerebrovascular disease.

However, the results of the various surveys are wide ranging and it is difficult to discern any clear patterns with regard to incidence trends over time. The explanation for these inconsistencies lies in the differences that exist between the studies in a number of key areas of investigation. Complete case ascertainment, for example, is obviously important, yet several studies have established insufficient contact with general practice and consequently generated findings which underrepresent the true incidence of stroke. Further interstudy differences exist with regard to the age groups selected for analysis.

The results of epidemiological study are also critically dependent on the choice of diagnostic criteria and the precision with which they are applied. Since some of the clinical features underlying a diagnosis of stroke may result from other pathological events scope has certainly existed for both over and under-reporting of incidence in this respect. In addition, accurate pathological diagnosis is particularly necessary in order to distinguish haemorrhagic from occlusive stroke. The extent to which different studies have achieved this will vary according to the setting in which incidence has been measured. The point in time at which investigation has been carried out is another relevant factor. Studies undertaken before the era of the CT scan were able to achieve an accurate pathological diagnosis in only about 30 per cent of cases of stroke (Marquardsen 1976).

The potential for variation in incidence findings is, therefore, considerable and in an attempt to establish accurate contemporary data on the occurrence of stroke a major epidemiological investigation, known as the Oxfordshire Community Stroke Project, was started in the early 1980s. The research involved a prospective study of all new cases of stroke and transient ischaemic attack in a defined population of about 103,000 patients registered with 49 general practitioners. Every case was assessed by a neurologist and most patients underwent a CT scan or were examined post mortem, or both, to ensure accurate diagnosis.

The results from the first year of the study are shown in Table 1. For both males and females, first stroke incidence rates increase with age and peak in the 75 years and over grouping. With the sole exception of the 65–74 age group, the rates for males exceed those for females. However, in terms of caseload numbers it emerges that females account for the larger part of the annual burden of stroke morbidity. Coupling the age specific incidence rates shown in Table 1 with mid-1986 population estimates for England and Wales suggests a current toll of 102,000 first strokes per annum (equivalent to 2.04 per 1,000 population) and of these almost three out of five involve females.

The incidence profiles described above are broadly corroborated by the findings of the Third National Study of Morbidity in General Practice (Table 2). This research was carried out over a 12-month period during 1981/82 and the observed age specific incidence rates suggest a total of 128,000 new cases of cerebrovascular morbidity seen by general practitioners each year. This sum – which excludes transient

cerebral ischaemia – is approximately 25 per cent greater than that estimated on the basis of the Oxfordshire study findings. It is likely, however, that the discrepancy reflects the broader diagnostic basis of the general practice study – with the exception of transient ischaemia, the latter included all cerebrovascular disease – rather than genuine differences in incidence.

Incidence patterns may also be usefully examined from the perspective of the type of stroke involved. For example, available evidence indicates that haemorrhagic stroke is associated with a much worse early prognosis than cerebral infarction so that a clear picture of

Table 1 **Yearly age and sex specific incidence rates per 1,000 population for first stroke**

Age (years)	*Rate*
	Males
0–54	0.17
55–64	3.18
65–74	5.95
75 and over	17.82
	Females
0–54	0.16
55–64	2.92
65–74	6.89
75 and over	14.21
	Males and Females
0–54	0.16
55–64	3.05
65–74	6.45
75 and over	15.46

Source Sandercock *et al* 1983.

Table 2 **Incidence of cerebrovascular disease, rates per 1,000, 1981/82**

Age	*Males*	*Females*
0–4	—	—
5–14	—	—
15–14	0.1	0.1
25–44	0.2	0.0
45–64	2.0	1.5
65–74	9.1	7.5
75 and over	23.0	20.8

Source RCGP 1986.

Table 3 **Cerebrovascular disease: percentage frequencies of types of incident cases in community surveys**

Source	Location	Period	Subarachnoid	Cerebral			(n)
			Haemorrhage	Haemorrhage	Thromboembolism	Other CVD	Total cases
A. Earlier studies				(percentages)			
Whisnant 1971	Rochester MN	1945–54	5	10	78	7	(548)
Matsumoto 1973	Rochester MN	1955–69	6	10	79	5	(993)
Eisenberg 1964	Middlesex CT	1957–58	36		50	14	(191)
Wallace 1967	Goulburn, Australia	1962–64	6	17	72	5	(155)
Alter 1970	Fargo ND	1965–66	7	16	41	35	(408)
Kannel 1965	Framingham MA	1949–62	18	4	78	0	(90)
	Weighted mean		8[a]	12[a]	69	11	(2,385)
B. Recent studies							
Hansen 1977	Frederiksberg DK	1971–73	2	20	87[b]		(556)
Kagan 1978	Hawaiian Japanese	1972–77	11	14	71	4	(133)
Garraway 1979a	Rochester MN	1970–74	9	8	76	6	(323)
Aho 1975	Espoo, Finland	1972–73	17	17	61	5	(244)
Zupping 1976	Tartu, Estonia	1970–73	7	14	80	0	(667)
	Weighted mean		7	12	75[c]	6[c]	(1,923)
C. Latest studies							
Garraway 1983b	Rochester MN	1975–79	10	13	73	4	(295)
Herman 1982	Tilburg, Netherlands	1978–80	5	10	81	3	(526)
Sivenius 1982	Kuopio, Finland	1978–80	8	9	80	3	(373)
Sandercock 1985	Oxfordshire, England	1981–83	6	14	76	10	(320)
	Weighted mean		7	10	78	5	(1,514)

D. Oriental studies							
Tanaka 1981	Shibata, Japan	1976–78	7	23	58	5	(415)
Lin 1984	Hiroshima-Nagasaki	1950–74	5	17	76	2	(621)
Wang 1983	Beijing PRC	1980–81	3	22	72	3	(36)
Li 1985	6 cities PRC	1982	2	44	51	3	(115)
	Weighted mean		6	22	67	5	(1,187)

[a] Adjusted for Middlesex.
[b] 'Unknown' type (58 per cent) defined as no anatomic verification.
[c] Adjusted for Frederiksberg.

Source Kurtzke 1986.

specific occurrence trends may provide the basis for more reliable forecasts of future morbidity burdens. Such data may also play an important role in promoting a better understanding of the epidemiology of cerebrovascular disease. It is therefore unfortunate that accurate incidence data for the different types of stroke over time are not available.

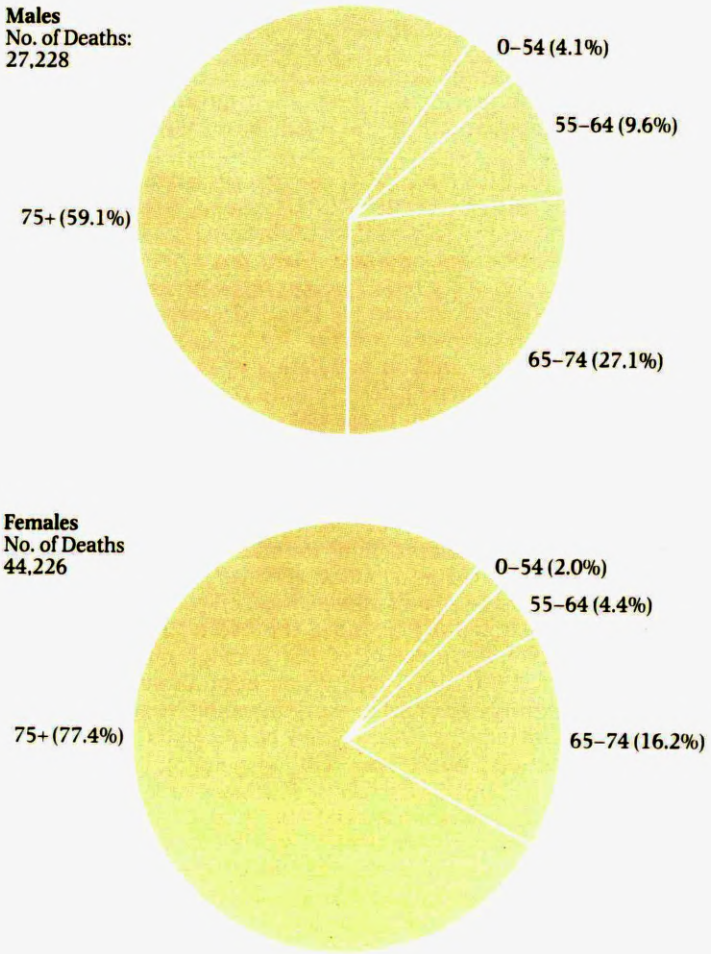
The Oxfordshire research project has nevertheless indicated that cerebral infarction is the most frequently seen type of stroke. Of the 168 first strokes indentified in the study, 127 or 76 per cent were the result of ischaemia caused by thrombosis or embolism. A further 22 or 13 per cent were classified as haemorrhagic strokes and within this group 13 were diagnosed as primary intracerebral haemorrhage and 9 as spontaneous subarachnoid bleeding. Nineteen cases (11 per cent) were designated strokes of unknown type. However, six of these patients had died within 24 hours of the onset of the stroke and had therefore probably suffered cerebral haemorrhages. If these cases are incorporated into the group of definite haemorrhagic strokes, the Oxford results suggest that the latter account for about 16 per cent of all first events and only 8 per cent remain unclassified.

The patterns described above have also emerged from other studies carried out in different parts of the world. Table 3 indicates that cerebral infarction is the predominant form of stroke, accounting for between three-quarters and four-fifths of all cases. The data also suggest that this pattern has probably changed little over time.

Mortality from stroke

In 1986, 71,454 deaths in England and Wales were recorded in the International Classification of Diseases category known as cerebrovascular disease. This total was equivalent to 26 per cent of all mortality from diseases of the circulatory system and slightly more than 12 per cent of deaths from all causes in that year. Detailed analysis of the fatalities attributed to cerebrovascular disease (Table 4) indicates that arterial occlusion is a more frequently observed cause of death than cerebral haemorrhage. The former is specified on 18.4 per cent of death certificates recording cerebrovascular disease as the cause of death compared with 13.1 per cent for the various forms of cerebral haemorrhage. However, these data require careful interpretation since information about the type of cerebral event involved is absent in a high proportion of deaths attributed to cerebrovascular disease. Table 4 shows that two-thirds of the latter total resulted from either 'acute but ill-defined' or 'other and ill defined' cerebrovascular disease. It is also relevant to note in this general context that attention has been drawn in recent years to inaccuracies in the diagnosis and recording of stroke mortality (Hughes 1985). Indeed from a study comparing death certificates and necropsy findings, Cameron and McGoogan (1981) concluded 'in cerebrovascular disease the diagnosis may be as often wrong as right'.

Figure 1 **Distribution by age of cerebrovascular disease mortality in England and Wales in 1986**



Source OPCS.

The age distribution of cerebrovascular disease mortality is markedly skewed to the right, that is, most deaths involve elderly persons. In 1986, 70 per cent of fatalities attributed to cerebrovascular events occurred among individuals aged 75 years and over (Figure 1). Distributed in this way, it might be anticipated that the disease is an important cause of death for the elderly population. This is indeed shown to be the case by the data contained in Table 5 which indicates that cerebrovascular disease accounts for at least one death in every ten in each of the five year age groupings after 75 years. For females, the disease is an especially significant cause of death – the corresponding proportion rises to nearly one in five for women in their ninth decade. The latter point is further underscored by the death probability data shown in Table 6.

In total female deaths from cerebrovascular disease outnumber male fatalities from this cause by a ratio of 1.65:1 Among those aged 75 years and over this ratio rises to 2.17:1. Most substantially, it reaches 5.63:1 for persons aged 95 years or more. However, these disparities are principally a reflection of the much greater representation of females in the elderly population rather than the result of a raised vulnerability to death from the disease among women. Table 7 contains cerebrovascular mortality rates per million population in England and Wales in 1985 and shows that female rates are only conspicuously higher (by about 18 per cent) than males in the oldest age group of 90 years and over.

Trends in cerebrovascular disease mortality over the past decade are shown in Table 8. Focusing on the age groups from 55 years onwards (which contained 97 per cent of the death toll from the disease in 1986) the data indicate mortality reductions for both sexes of between a fifth and a quarter over the 10-year period. The very much smaller figure shown for the decline in the all ages rate is therefore misleading. The apparent inconsistency arises because the overall rate, presented in this way, does not take account of the demographic changes that occurred between 1976 and 1986. When appropriate adjustment is made – by combining age specific mortality rates prevailing in 1976 with the population data for 1986 – it may be calculated that the overall rates fell by 20 per cent and 19 per cent for males and females respectively. At the same time, this exercise also suggests that in the absence of the mortality improvements experienced over the decade there might have been almost 17,500 more deaths from cerebrovascular disease in 1986 than were actually recorded. Expressed another way, the 1986 toll of cerebrovascular mortality might have been one-quarter as high again had the death rate reductions of the preceding decade not been achieved.

Despite these encouraging trends, it is clear that scope for further progress continues to exist. The international comparisons of cerebrovascular disease mortality shown in Table 9 indicate that although England and Wales are not ranked as disappointingly as occurs in comparisons of coronary heart disease mortality, many advanced nations have lower death rates for stroke. Focusing on females aged 75

years and over this observation is clearly valid for Sweden, Denmark, the Netherlands, France, Switzerland, Canada and the United States. Indeed, the rates shown for the latter two countries suggest that a reduction of a third or more may still be possible in the cerebrovascular disease death rate currently experienced by elderly women in England and Wales.

Table 4 **Analysis of cerebrovascular disease mortality by type of fatal event (based on 1985 data for England and Wales)**

	Percentage of annual CVD fatalities
Subarachnoid haemorrhage	4.2
Intracerebral haemorrhage	8.6
Other and unspecified cerebral haemorrhage	0.3
Occlusion and stenosis of precerebral arteries	0.4
Occlusion of cerebral arteries	18.0
Transient cerebral ischaemia	0.1
Acute but ill defined CVD	53.9
Other and ill defined CVD	12.4
Late effects of CVD	2.0
	100.0

Source OPCS.

Table 5 **Percentage of deaths at different ages attributed to cerebrovascular disease, England and Wales, 1985**

	Percentages	
	Males	*Females*
Under 15	0.36	0.46
5–24	1.16	2.04
25–34	3.18	6.76
35–44	4.76	6.78
45–54	5.21	6.71
55–64	5.70	7.95
65–69	7.54	10.19
70–74	9.64	13.34
75–79	11.82	17.01
80–84	12.91	19.03
85–89	13.52	19.37
90–94	13.25	18.34
95 and over	13.23	15.69

Source OPCS.

Table 6 **Chances per 1,000 of eventually dying from cerebrovascular disease at selected ages, England and Wales, 1985**

Age	*Males*	*Females*
0	97.4	155.1
1	98.4	156.4
15	98.8	156.9
45	100.9	158.7
65	112.4	170.2

Source World Health Statistics Annual 1987, WHO.

Table 7 **Cerebrovascular disease: mortality rates per million population in 1985 in England and Wales.**

	Males	*Females*
Under 15	4	4
15–24	9	6
25–34	27	31
35–44	81	77
45–54	281	223
55–64	976	771
65–74	3,888	2,925
75–79	10,391	8,451
80–84	17,521	16,280
85–89	27,312	28,322
90 and over	38,056	44,738

Source OPCS.

Table 8 **Cerebrovascular disease: mortality rates per 100,000 population in England and Wales in 1976 and 1986**

	Male			*Female*		
	1976	*1986*	*% change*	*1976*	*1986*	*% change*
Under 15	0.42	0.27	−36	0.22	0.35	+59
15–24	1.11	0.99	−11	1.05	0.65	−38
25–34	2.56	3.12	+22	3.19	2.34	−27
35–44	9.82	7.32	−25	10.49	6.96	−34
45–54	36.93	26.06	−29	34.04	19.78	−42
55–64	129.59	99.61	−23	97.71	69.74	−29
65–74	494.26	374.97	−24	372.39	287.78	−23
75 and over	1,778.78	1,461.38	−18	1,879.46	1,561.33	−17
All ages	119.96	111.57	−7	185.13	172.27	−7

Source OPCS.

Table 9 **International comparison of cerebrovascular disease mortality, rates per 100,000 population**

		Females		
Country	*Year*	*All ages*	*65–74*	*75 and over*
Portugal	1986	245.2	601.6	3,025.2
Greece	1985	212.0	447.2	2,575.0
Austria	1986	227.7	326.3	2,129.3
Scotland	1986	202.1	377.6	1,878.9
Italy	1983	148.9	314.3	1,777.3
Ireland	1984	108.5	316.7	1,616.4
Australia	1985	103.0	229.7	1,603.8
West Germany	1986	179.2	253.4	1,572.4
England and Wales	1986	172.3	287.8	1,561.3
Japan	1986	109.7	269.0	1,533.9
Norway	1985	148.6	222.4	1,468.0
Finland	1986	137.5	283.7	1,423.5
France	1985	129.9	176.4	1,277.3
Sweden	1985	132.3	188.0	1,178.7
Switzerland	1986	107.6	135.0	1,123.5
Netherlands	1985	93.7	179.2	1,122.3
Denmark	1985	113.4	182.6	1,087.0
USA	1984	76.3	154.9	934.9
Canada	1985	62.0	130.5	930.3

		Males		
Country	*Year*	*All ages*	*65–74*	*75 and over*
Portugal	1986	212.1	912.8	3,323.8
Greece	1985	156.7	475.7	2,207.0
Austria	1986	147.1	501.1	2,185.8
Italy	1983	125.0	485.0	1,944.7
Scotland	1986	131.0	497.3	1,857.7
Japan	1986	104.0	417.9	1,776.8
West Germany	1986	115.1	380.2	1,699.4
Ireland	1984	86.9	413.0	1,568.0
Norway	1985	109.2	311.5	1,463.4
England and Wales	1986	111.6	375.0	1,461.4
Finland	1986	94.8	451.6	1,417.3
France	1985	97.1	297.1	1,379.2
Australia	1985	67.1	312.4	1,340.2
Netherlands	1985	68.3	261.4	1,183.5
Denmark	1985	89.4	281.1	1,156.8
Switzerland	1986	74.9	231.6	1,119.7
Sweden	1985	99.1	261.7	1,112.3
Canada	1985	47.3	199.6	902.9
USA	1984	53.6	205.4	892.6

Source World Health Statistics Annual 1987, WHO.

Medical and surgical interventions for stroke

Treatment options to reduce the risk of stroke in patients with evidence of cerebrovascular disease

Patients with a known history of cerebrovascular disease who have experienced transient cerebral ischaemia or a minor stroke from which they made a full recovery, are at a particular risk of developing a further non-fatal or fatal cerebral event. Attempts to estimate the chance of a subsequent episode of this nature occurring have been reported many times in the literature, and although the results vary, it is suggested most authors would agree that approximately 40 per cent of patients with cerebrovascular disease who have not yet experienced a permanent stroke, will do so within the following year (Herskovits *et al*, 1981). Against this background, it becomes clear that a diagnosis of transient cerebral ischaemia in any form must be seen as an important precursor of stroke and underlines the need for effective pharmacological or surgical prophylatic intervention.

Anti-platelet therapy

Anti-platelet therapy, by ameliorating the risk of infarction, offers protection to patients with heart disease or who recently suffered a TIA/ minor stroke, from further vascular events, and as such is considered to be a secondary preventive measure.

Anti-platelet therapy such as aspirin and sulphinpyrazone inhibit the effects of the enzyme cyclo-oxygenase, and in doing so prevent the formation of endoperoxides and thromboxane A2. These latter agents induce platelets to aggregate thus increasing the risk of thromboembolic activity (platelet micro-aggregates are released into the cerebral circulation from thrombi formed on the surface of atheromatous plaques in the large vessels of the neck). The beneficial thrombosis – reducing properties of anti-platelet agents, are marred by the conversely dangerous tendency for these drugs, in large doses, to inhibit prostacyclin synthesis (active in reducing platelet adhesiveness), by the vessel wall, thus promoting the formation of thrombus. It is possible that these diverging characteristics vary in potency between different anti-platelet agents, illustrating the importance of indentifying the optimal dosage. Another drawback to this form of treatment is that the thrombosis-reducing properties of anti-platelet therapy might induce further bleeding in the case of a haemorrhagic stroke, therefore accurate diagnosis of the type of cerebral lesion is essential.

There have been many randomized clinical trials to assess the secondary prophylatic efficacy of various types of anti-platelet treatment, some of which are still in progress. In an attempt to reduce the margin of error encountered when analysing individual trials, and to ensure that all results are brought to light, the Antiplatelet Trialists' Collabora-

tion was established at the University of Oxford with the purpose of conducting an overview of the 25 completed studies. The published results revealed a reduction in vascular morbidity by 15 per cent and non-fatal vascular events, i.e. stroke or myocardial infarction, by 30 per cent (ATC, 1988). Patients included in the trials had histories of TIA, occlusive stroke, unstable angina and myocardial infarction. Interestingly enough, no differences in efficacy were recorded between patients with cerebral or cardiac disease. Similarly there were no differences in benefits accrued from the various types of treatment administered (low and high doses of aspirin, sulphinpyrazone and high dose of aspirin with dipyridamole). This last revelation underpins the present enthusiasm amongst neurologists for furthering the proplylatic use of aspirin in view of the fact that it is cheaper and less toxic than other anti-platelet medicines whilst matching their efficacy.

This view is shared by the investigators involved in the UK–TIA/aspirin trial (UK-TIA Study Group, 1988) one of the studies included in the APT collaboration overview. They also note that in the case of stroke patients, the reason for non-fatal events to be reduced more than fatal ones is in part due to the bleeding effect of aspirin possibly causing cerebral haemorrhage, which is more likely to prove fatal than the re-infarction hopefully being prevented. Reflecting the close aetiological links between cerebrovascular disease and coronary heart disease, it appears that aspirin therapy administered to all stroke patients would also reduce their risk of heart attack. In the final analysis it appears that if 100 patients with TIA, minor stroke or non-fatal heart attack were treated over 2 years with prophylatic aspirin, 2 non-fatal and one fatal stroke or heart attack would be prevented (MRC, funders of the UK-TIA/aspirin trial).

Surgery

Surgery for the prevention of stroke in patients with cerebrovascular disease was first introduced in the 1950s – yet it still remains a much disputed form of phrophylactic treatment (Horrocks, 1986). The most commonly performed operation of this nature is the **carotid endarterectomy**. In the case of TIA patients there is much debate as to whether this form of surgical intervention or medicinal therapy offers the best protection against a further cerebral episode. In the USA, enthusiasm for the former approach amongst surgeons results in approximately 85,000 of such operations being carried out each year – whereas their British counterparts, unconvinced that a proper randomised trial of the operation has been carried out, perform considerably less (*Doctor*, 1987).

Carotid endarterectomy is essentially a process of surgical cleansing, involving the removal of a stenosing plaque from the origin of the internal carotid artery. The effects of this procedure are two fold – firstly the restoration of high volume blood flow and secondly the removal of an important source of thrombo-embolic activity. The only controlled investigation of the value of the carotid endarterectomy was

completed over 20 years ago, the results of which were largely unfavourable. However an on-going study by the European Endarterectomy Trial should shed some light on this neurosurgical dilemma. In 1981 a team of investigators from the University of California, studied the appropriateness of the use of carotid endarterectomy, in the elderly, in three large geographical areas (Winslow *et al*, 1988). The team compiled 864 possible reasons for performing the operation and on the basis of this list, concluded that carotid endarterectomy was overused in the areas they studied.

In some TIA patients the lesion within the carotid artery to be operated on is located in such a way, that an endarterectomy would be impossible to carry out. In these cases an **extracranial-intracranial** (EC-IC) bypass can be performed instead. This operation involves the surgical joining of two blood vessels, usually the superficial temporal artery, and an intracranial artery, frequently the middle cranial artery. There has been much enthusiasm for this surgical procedure in the USA particularly where TIA patients are concerned. Recently, however, this enthusiasm has been dampened by the enlightening results of a collaborative investigation by neurosurgeons from the USA, UK and Japan, (EC-IC Bypass Study Group, 1985). Their findings have shown that although mortality and morbidity associated with the operation is low, patients who undergo this form of surgery are at no greater advantage than those on medical treatment alone. They go on to suggest that risks associated with this operation outweigh any benefits (EC-IC Bypass Study Group, 1985).

Treatment options after the acute event

Recent advances in therapy for the management of stroke victims immediately after the acute event, have increased the likelihood that in the near future, physicians will be in a position to improve the prognosis for stroke victims at this crucial stage in their illness. The two principal courses of action that medical management takes are, to increase cerebral blood flow (reperfusion) in order to save the area of brain tissue surrounding the site of the lesion, and secondly to mitigate the damage borne by brain cells (neurons) during the ischaemic period.

Vasodilators, in theory by causing blood vessels to dilate should ameliorate cerebrovascular resistance (prominent in this treatment area is **prostacyclin**). However, because blood vessels in the ischaemic regions are damaged they tend to be unresponsive to surgical or medical stimuli, so vasodilation therapy can encourage healthy vessels to dilate 'stealing' blood away from the deprived area (Thomas, 1984). Clinical studies of vasodilation therapy after stroke have not demonstrated favourable results for this form of treatment. Nevertheless, prostacyclin remains a popular therapeutic option, due in part to its adverse effects on platelet aggregation (blood-clotting) in stroke patients.

In occlusive stroke caused by thrombosis or embolism within a vessel, cerebral blood flow can be increased by the use of **thrombolytic**

agents such as **streptokinase**. They work by promoting the lyfis (disintegration) of the clot. Unfortunately, the fibrinolysis activity of this medicine (fibrin is the substance within the blood that causes the clotting) is not confined to the site of the blockage, and as such, can induce haemorrhage elsewhere. In response to this problem, **tissue plasminogen activator** (TPA) has been developed, which does not effect the circulating fibrinogen. Encouraging observations of the effects of TPA on non-haemorrhagic stroke patients have emerged from a USA open trial, which is still in progress. Seemingly, TPA promotes rapid reperfusion – one patient with signs of hemianopia (loss of half the usual area of vision) and hemiplegia (paralysis of one side of the body) regained vision and movement after 10 minutes, and a subject who presented with global aphasia (loss of speech) and hemianopia could speak and see again after 15 minutes of infusion with the treatment (SCRIP, 1988).

Anti-coagulant therapy, in particular intravenous **herapin**, nowadays is administered principally to patients who present with a 'stroke-in-evolution' (heparin is active in reducing the thrombo-embolic activity arising from the carotid and basilar arteries that cause infarction), or stroke due to emboli of cardiac origin (Grotta, 1987). Heparin is usually administered before any other anti-coagulant therapy, because its action is rapid at onset. This type of therapy is not recommended for TIA or haemorrhagic patients because of its tendency to induce bleeding in prolonged periods of treatment.

In practice, the inability to identify evolving strokes quickly and accurately poses some clinical problems. The timing of the onset of treatment is crucial if the natural course of the disease is to be halted effectively. Uncertainty in these respects may for example be associated with blanket administering of heparin to stroke patients who do not show signs of haemorrhage on a CT scan. Taking into account that only 15–25 per cent of cerebral infarctions are of an evolving nature, this over-compensatory approach leads to much unnecessary treatment with the medicine. As regards embolization to the brain due to the presence of heart disease (myocardial infarction or atrial fibrillation), studies have shown that herapin followed by **warfarin** (another anti-coagulant) cuts this risk by about two-thirds (Grotta, 1987).

A complication associated with ischaemic stroke, is the presence of increased cellular calcium concentrations, which have an adverse effect on neurons. **Calcium antagonists** such as **nifedipine** and **nimodipine** interrupt this process by inhibiting the entry of calcium into the brain cells and in addition provide vasodilatory action. Results from a recent trial, from the Netherlands, testing the effects of administering the calcium antangonist, nimodipine, suggests that male patients with acute stroke benefited from early treatment with the medicine but no advantage was found by treating women (Gelmers *et al*, 1988).

The mechanism behind calcium entry into neurons after ischaemia is unknown, but it is believed that some calcium channels are opened up by the action of excitatory amino acid neurotransmitters such as glutomate. In response to this discovery investigations are being carried out,

into the development of **glutomate antagonists**, a new medicine that promises to play an important role in the neuroprotection of stroke victims in the future. At the present time the suggested treatment to alleviate neuronal damage, is to administer barbiturates, in order to suppress the metabolism of brain cells during the ischaemic period, and also to block calcium channels. However, the dose of sedative medicines needs to be high, so treated patients could also need ventilatory and circulatory support (Thomas, 1984). One other important drawback of this treatment is the discovery by laboratory trials on animals, further backed-up by clinical trials on ischaemic patients, that treatment needs to be administered before the actual ischaemic period begins.

Prevention of stroke – the control of hypertension

Although hypertension is recognised to be extremely prevalent in Western society, and is closely associated with an increase in the risk of morbidity and mortality from cardiovascular events, the detection and management of the condition is a much disputed area. Problems arise in attempting to pin-point a level for blood pressure, which when reached, can no longer be classified within the boundaries of 'normal tension', and is indicative of unacceptably high risk from associated diseases. Seemingly, the upward trend in risk attributable to each increment in blood pressure is uniform, in other words, there is no obvious stage at which rising risk level accelerates. In clinical practice, using insurance levels, the upper limit for normal blood pressure is around 90 diastolic and 150 systolic, however, due to the unstable nature of blood pressure (blood pressure is subject to natural diurnal changes and short-term fluctuations due to exercise and stress), accurate readings are difficult to obtain.

Detection of hypertension is confounded further by the fact that the vast majority of subjects with this condition are asymptomatic, and no underlying disease can be diagnosed – this is termed 'primary' or 'essential' hypertension. Physical symptoms are only manifested when hypertension is severe and prolonged resulting in 'target organ' damage. The two organs most commonly affected are the heart and the kidneys, with patients frequently presenting with left ventricular hypertrophy and renal damage – clearly hypotensive treatment at this acute stage would be too late. The remainder of known hypertensives are classified as having 'secondary' hypertension, in these cases, an underlying causative disease has been established which when treated lowers the blood pressure.

The prevention of cardiovascular events attributable to elevated blood pressure levels, is concerned, therefore, with the detection and management of essential hypertension. Medical research has been unsuccessful so far in identifying the origins of the condition, so clinicians are dependent on epidemiology for clues to the possible causes.

Evidence concerning the influence of growing older on blood pressure confirms that average blood pressure increases with age in most populations (*Lancet*, 1981). However, when the blood pressure of individuals is considered over time, there appears to be little conformity. For many members of the population, elevated blood pressure levels are not an inevitable consequence of the ageing process, although subjects seen initially with abnormally high rates are in the most part prone to increasing levels with age. The latter sub-group probably account for the apparent rise in a population's mean blood pressure with age.

Non-medicinal intervention

The results of epidemiological investigation have led to public awareness campaigns that stress the contribution of non-medicinal, environmental influences on raised blood pressure levels throughout the population – the principal risks being obesity, salt intake and alcohol. Heredity is another important risk factor, which can be responsible for abnormally high pressures among children, and in recent years much emphasis has been placed on the role of pychosocial factors such as personality type, stress and socioeconomic status.

Salt consumption

It is a widely held belief that salt intake is causally related to essential hypertension, and on the basis of this assumption restriction of dietary salt is advocated frequently as a primary preventive measure for individuals at risk and for the population as a whole. On closer inspection of the available data, though, it appears that epidemiological studies have so far failed to corroborate this view convincingly (Boon and Aronson, 1985).

Advocates of a low sodium diet for all members of the population argue that although the evidence is not conclusive, there is nothing to be lost by implementing such a regime. However, like medicinal therapy, a salt restrictive diet might induce adverse reactions in some subjects, or fail because of lack of compliance in others. Further evidence is needed to determine the individuals who will respond positively to such a public health measure.

Obesity

Observation by way of cross-sectional studies has revealed that higher blood pressures are associated more with obese people than those of normal weight. How this correlation occurs is not fully understood yet, but some possible explanations are that over-eating might result in increased consumption of salt; raised insulin; a rise in estrogen levels; renal size remaining unchanged while body mass increases; an increase in vascular capacity; and a rise in sympathetic nerve activity as energy consumption increases (WHO, 1983). On a more fundamental basis, fat around the upper arms can increase the pressure recorded by sphygmomanometers, although, whether this leads to much inaccuracy of blood pressure readings is doubtful, as this effect has been recognised for some time and compensated for within epidemiological

studies. Although it has been shown that weight loss can reduce blood pressure, in terms of preventive action further investigation is required into the extent to which individuals need to reduce their body weight at various ages, to initiate this downturn in blood pressure. Certainly in individuals known to be hypertensive, and among those who are at high risk of developing the condition, the monitoring of body weight is advisable, furthermore, based on current understanding it would seem that this health measure could be applied to non-hypertensive adults and the young as an effective means of avoiding the risks associated with rising blood pressure and ageing.

Alcohol

It has been established by clinical and epidemiological studies that there is a link between heavy alcohol consumption and raised blood pressure. Saunders and his co-workers (1981) found a significant correlation between blood pressure and mean daily alcohol intake in subjects consuming in excess of 80g per day. The mechanisms involved are not fully understood, but some of the processes implicated are increased blood cortisol levels, increased catecholamine levels and effects on the renin-angiotensin system or on antidiuretic hormones. In the same investigation they discovered a significant correlation between hypertension and the severity of alcohol withdrawal syndrome. It seems that hypertension is present during the recovery period, after acute alcohol ingestion, particularly among heavy drinkers and alcoholics, and in a milder form in most regular consumers of alcohol whatever the quantity. Alcohol withdrawal is associated with a temporary hyperadrenergic state which can be controlled by the use of beta-adrenergic blocking drugs. After detoxification, however, blood pressures fall to normal levels in the majority of subjects and remains so unless consumption of alcohol is resumed, implying that alcohol-induced hypertension is labile in nature and therefore long-term raised blood pressure is not always a characteristic.

In the light of current evidence an important aspect of the management of hypertensive patients would seem to be the ability to recognise and modify an excessive alcohol consumption habit, and preferably before hypertension becomes apparent. Unfortunately, difficulties arise in attempting to assess the extent of the problem in the community, as studies tend to be hindered by poor compliance by alcoholics and unreliable responses to questioning on alcohol consumption levels.

On a more positive note, it seems that there is, as yet, no evidence to suggest that subjects who consume moderate to low levels of alcohol sustain higher blood pressure than persons who are teetotal, implying that for the majority of the population alcohol consumption is not making an important contribution to the prevalence of hypertension, and as such, does not warrant extensive interventive action. Having said that, there is recent evidence to suggest that alcohol consumption is a significant and under-recognised risk factor for stroke in men, independent of hypertension (Gill *et al*, 1986).

Psychosocial factors

There has been much conjecture of late regarding the effects of environmental and psychological factors on mental processes and whether or not they can contribute to sustained blood pressure elevation. The principal factors concerned are 'stress', personality traits and socioeconomic status. Although there is little doubt that such features can lead to short-term raising of blood pressure levels, available data on the subject, falls short of identifying an independent influence for psychosocial factors on long-term hypertension. Scientific investigations in this area are complicated by the fact that social groups are influenced to a varied degree by the aforementioned risk factors of alcohol, obesity and salt intake, as well as other variables (Greenberg, 1988). As regards preventing hypertension in the community, although there is insufficient empirical evidence to support mandatory screening and management of these possible psychological and social influences, by virtue of the fact that they are, in the main, remediable, interactive measures should not be ruled out entirely.

In the final analysis it might seem that non-pharmacological approaches to preventing hypertension in the community are more desirable than pharmacological means, due to the costs and harmful side-effects of the latter, however, scientific and epidemiological evidence for non-medicinal intervention is at present inadequate to influence clinical practice. Further investigation is required to determine what proportion of the population will accept dietary change and of that number, the proportion who will show a blood pressure response.

Medicinal intervention

For many hypertensives interventive action in the form of medicinal treatment is both necessary and effective, where non-pharmacological measures such as weight reduction, salt restriction and alcohol reduction are inappropriate or have failed to produce positive results. The objective is to reduce the diastolic pressure to a level considered to be 'normal' for the individual concerned. Systolic pressure tends to fall along with reduction in diastolic pressure. Typically, management using anti-hypertensive therapy follows a 'stepped-care' regime, whereby a series of different medicines are combined and introduced at controlled stages of treatment.

In the past, thiazide diuretics have been used as first choice in the initial stages of therapy. They work by altering the salt and fluid levels in the body. In more recent years, beta-blockers, that control the sympathetic nervous system, have tended to replace the diuretic as a first-line therapy due to their more potent effects that hopefully reduce the requirement for additional medicines. Interestingly, recent *post hoc* analyses of sub-group results from the Medical Research Council Trial (1985) of the treatment of mild hypertension, compared the efficacy of the two active treatments administered (bendrofluazide and propranolol) and discovered that the reduction in stroke rate on the diuretic was greater than that on the beta-blocker. However, these

particular findings alone are not a sufficient basis from which to formulate decisions about these forms of primary therapy. Commonly if one or other of these two hypotensive medicines does not succeed in reducing blood pressure, the next stage is to combine treatment.

In some cases further treatment is required, in the form of a calcium channel-blocker. By interfering with the entrance of calcium into the cells of the vessel wall, vasodilation takes place, causing peripheral blood vessels to relax, thus reducing blood pressure. When taken in conjunction with a beta-blocker and a diuretic, calcium channel-blockers have a 50 per cent success rate (Ramsay, 1985).

The newest area of anti-hypertensive therapy involves the renin angiotensin system. Renin produced by the kidneys, is responsible for the production of angiotensin I which is then converted by enzymes to angiotensin II. Angiotensin II is a potent constrictor of blood vessels and as such contributes to hypertension. There are three possible stages at which this process could be reversed. Firstly, the renin itself could be inhibited, drugs designed for this purpose are currently being developed. Secondly direct interference with the effects of angiotensin II. The third method, that of inhibiting the conversion of angiotensin II is already a reality, in the form of angiotensin converting enzyme (ACE) inhibitors.

The effect of anti-hypertensive therapy on stroke incidence and mortality.

In the majority of patients hypertension can be reversed by the use of medicinal treatment. Early controlled clinical trials of anti-hypertensive therapy have demonstrated that treatment of severe to moderate levels of hypertension will reduce the incidence of cardiovascular events and strokes. However, the value of treating mild blood pressure levels, as found in the majority of the known hypertensive population is less clear. In response to this shortcoming in medical practice, a number of major clinical trials have been carried out on populations with mild hypertension. The results have proved useful not only in assessing the impact of anti-hypertensive regimes on morbidity and mortality from all cardiovascular events but in demonstrating the efficacy or otherwise of different forms of treatment and in identifying sub-groups of patients who respond particularly well to treatment.

Five trials of this nature have been completed so far and although their design characteristics vary, they have all shown that incidence of stroke is reduced on active treatment. The most recent and largest trial to be completed was by the Medical Research Council (MRC Working Party, 1985). Their results revealed that if 850 mild hypertensive patients are given anti-hypertensive medicines for one year about one stroke will be prevented, which, they suggest is an important but infrequent benefit. Many patients participating in the trial suffered chronic, though minor side-effects. *Post hoc* analysis of sub-group data revealed an interesting though inconclusive result – for all categories of events, and in both treated and placebo groups, stroke rates were lower in non-

smokers than in smokers, thus reflecting epidemiological evidence that cigarette smoking increases the risk of cerebral events. A reduction in stroke rate was shown in both men and women with no statistically significant differences in response to treatment.

Therapeutic trials of anti-hypertensive treatment have proved that such treatment can reduce stroke occurrence in individuals. Furthermore it is often speculated that improved management of hypertension is a major contributory factor to the observed decline in mortality from stroke seen in recent years and in several countries. Although it is a well established fact that hyptertension is the most important independent risk factor for stroke, it has proved difficult to establish a definite association between the increased detection and treatment of hypertension and the apparent decline in stroke deaths. In New Zealand a team from the University of Auckland used data from studies of the prevalence of blood pressure in the community, plus data from therapeutic trials of hypertensive treatment to attempt to show the proportion of the decline in stroke mortality that is attributable to increased use of pharmacological means for reducing hypertension (Bonita and Beaglehole, 1986). The latter it was estimated, accounted for 10 per cent of the observed decline. A larger estimate might have been reached if the population studied had been extended to include the 70 and above age group – evidently, it seems that three-quarters of the decline in stroke deaths occurred in this age category, but concern regarding the administering of anti-hypertensive drugs to the elderly prevented them from being included in the study. It was also suggested that recent decreases in stroke deaths could be attributable to a small decrease in the average blood pressure of the population due to dietary and lifestyle changes rather than the widespread use of anti-hypertensives, although as yet there is no evidence to support this view.

A study of the population of Rochester, Minnesota, was carried out between 1950 and 1979 with the similar purpose of examining the changing pattern of hypertension control and determining whether there is a direct cause and effect association with the observed declining incidence of stroke during that period (Garraway and Whisnant, 1987). The results showed a definite association between the two variables for women during the period under observation, however, for men although control of hypertension increased at the same period in time the corresponding decline in stroke was delayed for ten years. Whether this result reflects actual physiological differences in the way in which women respond to hypotensive treatment could not be proved.

The effects of anti-hypertensive therapy in the elderly

High blood pressure is commonly observed in the aged, and in particular raised systolic pressure. Analysis of age-specific stroke rates reveals that morbidity and mortality from stroke is concentrated in the elderly population. It is universally accepted that hypertension is a major risk factor for stroke, therefore, the control of hypertension in

older patients would seem to be a logical means of reducing the burden of stroke in the community.

Unfortunately treating hypertension in the elderly population is fraught with complications. Not only is it difficult to establish the definition of hypertension in this age category (underlying vascular disease causing rigidity of the arterial walls can give a high blood pressure reading when hypertension and its clinical sequelae are not present), the situation is further confounded by unwanted side-effects as the elderly are often taking additional drugs for separate complaints and harmful interactions can occur. Furthermore among the elderly population, there is sometimes poor compliance as regards the regular taking of the drugs prescribed. More importantly though, the elderly are prone to unwanted side-effects from the anti-hypertensive agents themselves, for example, some older diuretics can increase the risk of glucose intolerance and gout and certain beta-blockers aggravate bronchial disease in the elderly.

Paradoxically, anti-hypertensive treatment in the elderly can actually induce stroke (Jansen *et al*, 1987). This is caused when the hypotensive effect of the drugs is too rapid. Under such circumstances in younger patients, the process of cerebral autoregulation (when blood pressure rises cerebral vessels dilate, and when blood pressure falls they constrict, thus keeping blood flow at a constant pressure) protects the brain but in older patients this natural process can fail.

Most of the therapeutic trials to assess the risks and benefits of anti-hypertensive treatment have not included an elderly cohort, and taking into consideration the complications associated with treating the elderly hypertensive patient, it is not possible to extrapolate the results out to include this older age group. In response to this situation, a trial by the European Working Party on High Blood Pressure in the Elderly (EWPHE 1985) was carried out. The mean age of entry was 72 years, and 70 per cent of the patients were women. The results revealed that active treatment with anti-hypertensive drug therapy reduced the incidence of non-fatal strokes by 50 per cent although the occurrence of fatal strokes remained the same. These findings were further backed up by a more recent general practice study (Coope and Warrander, 1986) of patients aged 60–79 years, again active treatment was successful in reducing the incidence of stroke.

In the light of this evidence it would seem that the screening and treatment of hypertension in the elderly, provided that side-effects are kept to a minimum, is a feasible and beneficial means of primary prevention of stroke in this age category. However, contradictory evidence has been revealed by Grimley Evans, (1987) in a four-year follow-up study of the relation between blood pressure and stroke in an elderly population in South Tyneside. It appeared that the treatment of hypertension in elderly women is not advantageous due to the fact that no relation could be found between stroke incidence and raised blood pressure in females. However, in men raised blood pressure was significantly associated with increased risk of stroke.

The cost of stroke

The costs associated with stroke may be analysed from a number of different perspectives. From the point of view of the individual who experiences a non-fatal stroke, the event may generate additional monetary costs as well as cause personal suffering and hardship. The former may include, for example, the travel and time costs involved in obtaining medical care and those arising from the purchase of aids to daily living. In some cases, much greater expenditures may be necessary if significant adaptations have to be made to the patient's home. The costs of pain and handicap cannot be reduced to monetary expression and therefore tend to be neglected in assessments of the financial burden of disease. Understandable as this may be, it is clearly especially unfortunate in connection with stroke since survey data have shown the condition to be the most important single cause of disability among individuals classified as being very severely handicapped (Harris 1971).

From the perspective of society as a whole, ill health may give rise to indirect costs if it causes the individuals concerned to lose time from work. These sickness absences lead to production losses which are conventionally valued on the basis of gross earning levels. Stroke, however, is a relatively insignificant source of this type of economic burden because the vast majority of cases involve individuals who have reached retirement age. In 1982/83, before the current sickness benefit regulations came into force, cerebrovascular disease gave rise to 7,900 spells and 6.8 million days of certified incapacity for work in Britain. These totals were equivalent to just 0.1 per cent and 1.8 per cent respectively of all spells and days of absence recorded in that year.

At the same time, the small number of fatalities from the disease that occur below 65 years of age each year implies that it is only a very limited source of permanent loss to the nation's potential work force. It may be estimated that the toll of deaths below 65 years of age from cerebrovascular disease in 1985 gave rise to about 60,000 lost years of possible working life. This sum was equivalent to less than four per cent of the total loss from all causes in that year.

The most readily quantifiable component of the financial burden of disease is the direct treatment expenditure borne by the health service. In this respect, stroke gives rise to significant costs both in the primary care and hospital sectors of the NHS. (The following analysis is based on cerebrovascular disease as a whole rather than just stroke because of the difficulties of extracting the latter from the available statistical information.)

Data on the extent of treatment provided in the primary care setting may be obtained from the Third National Survey of Morbidity in General Practice. The study covered a 12-month period during 1981/82 and found that 6.4 persons per 1,000 consulted their general practitioner because of cerebrovascular disease. Application of this rate to 1986 population estimates suggests a total of 320,000 patients

consulting family doctors for the disease each year.

The consultation rate for these patients observed by the study was 19.9 per 1,000. On average, therefore, each patient consulted his or her doctor approximately three times during the year. In conjunction with the other findings of the survey, these data indicate that cerebrovascular disease accounts for 0.59 per cent of general practitioner consultations each year. When applied to the cost of the general medical services in England and Wales, this proportion yields an estimated cost for cerebrovascular disease consultations of £6.4 million in 1985.

It is possible, however, that this approach to costing understates the true level of expenditure on the general practitioner element within the primary sector. This would be the case if patients suffering cerebrovascular disease required longer and more resource intensive consultations than the average. Information to clarify this specific point is not available from the Third Morbidity Survey although it was found that a much higher proportion of cerebrovascular disease consultations required a home visit – 59 per cent compared to the figure for all causes together of 12 per cent. As a means of taking account of the additional costs involved in a home visit, Carstairs (1976) has suggested that such consultations might be weighted, albeit arbitrarily, by a factor of four. If this approach is followed in the costing of cerebrovascular disease, it can be re-estimated that the latter accounts for 1.2 per cent of general practice 'consultation units', thereby costing £13 million in 1985.

The Morbidity Survey also provides information on the extent to which general practitioners refer stroke patients to hospital outpatient departments. From the 1981/2 survey it may be estimated that 3.4 per cent of the 320,000 cerebrovascular disease patients who consult their family doctor each year are further investigated and treated as hospital outpatients. The cost of the average (all causes) case seen in this setting was estimated at £94 in acute non-teaching hospitals in 1985/86, thereby yielding a total expenditure for cerebrovascular disease of £1.02 million. This estimate has, however, to be treated with caution because there is some degree of uncertainty about its accuracy. For example, average outpatient cost figures may inadequately reflect the true resource burden of managing the disease in this setting. In addition, the calculation excludes those cases entering the outpatient system from sources other than general practice, such as accident and emergency departments or following inpatient discharge. Considerations such as these suggest that the outpatient cost shown above may, therefore, be an underestimate. However, the fact remains that cerebrovascular disease accounts for only 0.3 per cent of all outpatient referrals by general practitioners and that adjustments to the estimated cost figure as might be appropriate are unlikely substantially to alter its absolute magnitude.

The largest part of the direct health care costs associated with cerebrovascular disease results from the treatment that is provided on a hospital inpatient basis. The Hospital Inpatient Enquiry estimates that

there were 118,500 admissions to hospitals in England for cerebrovascular disease in 1985. The mean duration of inpatient stay for each case was 48.8 days. Consequently the disease currently generates a total of 5.78 million hospital bed days per annum. Adjusting this figure pro rata to include Wales and combining the result with the 1985/86 average cost per inpatient day in acute hospitals with a minimum of 51 beds (£87), it may be estimated that hospital inpatient treatment for cerebrovascular disease currently gives rise to an annual NHS expenditure of £532 million.

Again, this estimate contains an element of ambiguity because of the use of a single average per diem cost figure in the calculations. The potential for inaccuracy in this respect is implicit in the Hospital Inpatient Enquiry data which show that patients with cerebrovascular disease are cared for in a number of different medical and surgical specialties. The 1985 report indicated, for example, that 42 per cent of these patients were managed on general medical wards and 41 per cent by the specialty of geriatric medicine. Another 7 per cent were accommodated on neurological or neurosurgical wards whilst 6 per cent were admitted to beds under the care of general practitioners. At the same time, potential for costing imprecision exists even within given specialties if the balance between the treatment and hotel components in the average per capita cost for all diseases combined differs markedly from that in cerebrovascular disease alone – a possibility raised by the prolonged mean inpatient spell observed for elderly cerebrovascular patients.

In total, cerebrovascular disease is estimated to have cost the NHS nearly £550 million in 1985 in England and Wales. This sum was equivalent to 3.9 per cent of total NHS expenditure. A similar proportion has been reported by other studies of the direct treatment costs of cerebrovascular disease (Drummond and Ward, 1986). It should be noted, however, that the figure calculated in this paper does not include two important elements of expenditure: the ingredient and dispensing costs of medicines prescribed by general practitioners and NHS spending on community support services such as home nursing. In both cases it is not possible to identify the costs that are specifically attributable to cerebrovascular disease from the available data. Focusing on medicines by way of illustration, official data recorded NHS spending of more than £150 million on 35 million prescriptions for antihypertensives and diuretics in Britain in 1985. However, raised blood pressure is a risk factor for a number of other conditions in addition to cerebrovascular disease and there is insufficient data to provide a basis from which to allocate the sum spent between different diseases which may not yet have become clinically evident.

It therefore follows that it is not possible to derive a comprehensive figure for the cost of cerebrovascular disease to the NHS and that the estimate of £550 million calculated above contains an element of understatement. Against this background, perhaps the key point to emerge from the foregoing analysis is that a very high proportion of total costs stems from the provision of hospital inpatient care. In the

foreseeable future, cost pressures in this sector may be expected to increase as the numbers of elderly people in the population continue to grow – in 1991 there will be 350,000 more people aged 75 and over than in 1985 – and with the advent of new hospital-administered pharmaceutical approaches to promoting survival after an acute cerebral event. It can, therefore, be confidently predicted that as the general search for economic efficiency in the use of scarce health care resources intensifies, the costs and benefits of admitting patients suffering acute cerebrovascular disease to hospital will be a subject that attracts increasing attention (Bamford *et al*, 1986).

The place of care for stroke patients

From the estimate that stroke patients consume approximately 3.9 per cent of all National Health Service resources, and although mortality from stroke is on the decline the disease still remains a major source of morbidity and disability in the UK. As such it requires very careful management by health care professionals if the optimal balance is to be struck between the allocation of resources for hospital and community services, whilst continuing to maximise the stroke patient's chances of survival and recovery. Indeed, in recent years there has been much debate surrounding the value of referring stroke victims to hospital. In view of the fact that admission of stroke patients to general wards is more about nursing care and rehabilitative therapy than life-saving medical treatment, it has been suggested that patients would fare as well if not better at home, in the hands of family carers and the community.

If a general practitioner has reason to believe that a patient's illness, which is manifested by the clinical symptoms of a stroke, has a different underlying cause, then this is usually reason enough to seek confirmation of diagnosis and further investigation by a hospital specialist.

In reality, though, it would seem that most patients are dealt with by a general physician or geriatrician, uncommonly by a neurologist, and there is no evidence to suggest that general practitioners are less skilled at providing accurate diagnoses (Wade *et al*, 1985). Nevertheless, differentiation between types of stroke based on clinical assessment alone can lead to misdiagnosis, thus hospitals provide, by way of computerised tomographic scanning, an opportunity to distinguish between pathological entities such as infarction and haemorrhage, and in doing so could reveal a chance for early neurosurgical intervention. However, this is a rare occurrence, and it is generally believed that treatment regimes remain unaffected by the results of such investigations.

The primary reason for admission, seems to be for long-term remedial therapy. The beneficial effects of rehabilitative therapy for

stroke victims, by maximising the patient's chances of recovery from disablement, are well documented. Admission to hospital is at present the means favoured by general practitioners, of gaining access to this form of treatment. Despite this fact, studies have indicated that the organization of acute medical wards is such that long-term admission for stroke can be detrimental to the patient's recovery. This is due to the fact that the role of the nurse is to provide support by carrying out the tasks that the disabled person is no longer able to perform, and the danger being that the longer a patient remains in hospital the more dependent he will become on this aid. It is also true to say that although most patients will see a physiotherapist while in hospital other important specialist staff such as speech therapists, occupational therapists and social workers are not introduced into the patient's recovery programme until much later, if at all. General nursing is probably the most consistent form of care that a stroke patient will receive while in hospital and as Wade points out 'nursing involvement is unresearched, and probably underestimated'.

Many patients are admitted for largely social reasons. General practitioners will frequently deem it necessary to refer a stroke patient for admission to a general ward, simply because the nursing care at home is either inadequate or non-existent. The majority of stroke victims in hospital are elderly patients, who prior to their illness lived alone or with a partner who would not be able to cope with the demanding role of carer. In fact research has shown that nursing-care, as a reason for referring patients to hospital is given in 87 per cent of cases (Bamford *et al*, 1986).

It has been suggested that some of the above shortcomings of hospital provision that are proving detrimental to stroke patient welfare, could be alleviated by the introduction of specialist stroke wards. In brief, these would be general medical wards for use only for stroke patients. Their objective would be to provide a therapeutic environment that encourages the stroke patient to strive for independence, and return home as soon as possible. An obvious benefit to be derived from assembling all stroke patients in one unit, would be the incentive to establish common medical, nursing and rehabilitative practices. However, contradictory evidence has been provided by a controlled trial of the management of acute stroke in the elderly (Garraway *et al*, 1980). The study compared the outcome of two groups of patients, who were admitted to either general wards of a district hospital or a stroke unit. Observation of the stroke unit patients on discharge from hospital, revealed a higher level of independence, as would be expected, but after re-establishment at home for about a year, this initial advantage over the general-ward patients was lost. According to Garraway there are two main contributory factors to the outcome of the trial. Firstly, he speculates that stroke patients from the general wards might have been released before they had achieved maximum recovery, so it could be that the home environment provided the stimulus needed to establish independence. As for the stroke unit patients, Garraway suggests that

the diminishing independence level on return from hospital, was caused by overprotective families and carers, who carried out tasks that the patients were capable of performing themselves thus undermining the skills learnt at the unit.

In view of the fact that 80 per cent of strokes occur at home, and that 80 per cent of all surviving stroke victims return to the community eventually (Wade, 1987), the provision of an adequate domicillary – care service would seem to be an essential part of the overall management of stroke patients. It is assumed that the decrease in hospital expenditure, as a result of fewer admissions, would more than cover the additional spending that would then be required on community services such as physiotherapists and occupational therapists. However, although it is postulated that stroke victims who remain at home for the entirety of their illness, make a greater functional recovery than do patients discharged from general medical or stroke wards, there is little conclusive evidence to support this idea.

In order to assess the value of home-care management for stroke victims, an investigation by way of controlled trial was carried out in Bristol (Wade *et al*, 1985). A home-care service was already available for stroke victims as an alternative to hospital care, but in practice this was underused by general practitioners. The trial assessed the outcome of increasing this domicillary care in the hope that it would reduce the number of patients admitted to hospital, encourage hospitals to discharge patients earlier, improve functional recovery, and lessen the anxiety and stress of carers. The trial group had access to the improved home-care service for the first six months following acute stroke, and after the trial period a number of assessments were made, the results of which were disappointing. It appears that the trial group used more hospital bed-days and had a higher admission rate for stroke. The social activities of both groups after six months were also studied – the tests performed in this category examined physical function (Barthel ADL scale), social functioning (Frenchay Activities Index) and depression (Wakefield self-assessment inventory) but no significant differences were revealed. Even amongst those patients who were not admitted to hospital at any stage during the trial, the results were surprisingly disappointing overall for the control patients and trial patients who remained at home, but functional and social recovery levels at six months were very similar.

Further analysis of the trial results brought to light attitudes amongst hospital staff and general practitioners, towards the experiment, that were counterproductive. For example, despite the better home-care service hospital staff were 'suspicious of moves encouraging early discharge to save money'. General practitioners ignored the incentives of domiciliary care and continued to refer patients to hospital, under the influence of a relatively easy admission procedure in the locality.

Rehabilitation of stroke patients

Although many would argue that the current provision of rehabilitative services is not tailored to the needs of stroke patients, it is generally accepted that remedial therapy should remain an integral part of the overall management of stroke patients who are left disabled after the acute episode, and for whom it is believed therapy will be beneficial. For permanently disabled patients the priority in terms of rehabilitation is to regain independence within the home environment as quickly as possible. Physiotherapy is the principal form of remedial treatment required – occupational therapy is less relevant to the rehabilitation of stroke patients, than it is to other victims of brain damage, because the majority of stroke patients are beyond retirement age.

Rehabilitation services are very labour intensive, and as such require careful evaluation before recommendations for improving the treatment strategies employed or for increasing the scale of provision are put into operation. Many studies from centres with an interest in rehabilitation after stroke have been carried out to assess the effectiveness of different remedial regimens, however, reviews of their findings have failed to provide conclusive evidence for any beneficial effects. Indeed direct comparison of the results of treatment between different rehabilitation units is not possible. Even if the pattern of remedial treatment is very similar between centres, patient characteristics are going to vary, thus influencing the outcome of therapy.

With these concerns in mind a group of investigators that included geriatricians, neurologists and epidemiologists from all over Britain was funded by the Medical Research Council to assess the current status of research into the rehabilitation of acute brain damage in adults (they included in their study patients with persisting disability after stroke or head injury) (*Lancet*, 1982). The purpose of the investigation was threefold. Firstly, the team looked at the various methods that are currently favoured for assessing the extent of post-stroke disability before rehabilitation commences. They stressed the importance of a uniform method of categorising patients according to their deficit, if the effects of alternative forms of intervention are to be properly examined by comparing rates of recovery or degree of recovery at any given time. Methods currently employed include, neurological examinations for hemiplegia, dysphagia, etc, cognitive tests to assess memory loss and problem solving ability and Activities of Daily Living (ADL) scales that measure the extent to which stroke patients are dependent on others for everyday physical tasks. In the case of the latter it was discovered that twenty-seven different scales were in use throughout the country. The team went on to investigate and compare the actual remedial programmes currently in service. They proposed that some questions central to the issue of rehabilitation are yet to be resolved, for example, the optimum

time to commence various remedial techniques, the length of time that therapy should last and how intensively should techniques be applied. Furthermore the group stressed the importance of following-up patients after leaving hospital, to evaluate whether progress is maintained. Lastly, the team recommended further research into the formulation of strategies that reduce dependency and improve social functioning. Although treatments aimed at improving one particular function (eg speech) are effectively contributing to such an outcome, it is suggested that the effect of being cared for by an enthusiastic, dedicated rehabilitation team could have an important beneficial impact also.

The group made a final recommendation. To aid the patient selection procedure for rehabilitation services, account should be taken of 'premorbid' variables – in other words – the characteristics of patients prior to their stroke that could have an adverse effect on prognosis. Relevant to this issue is a review by Jongbloed (1986) of 33 studies that aimed to identify these factors. Her findings revealed that previous stroke, older age, urinary and bowel incontinence, and visuo-spatial deficits all adversly affect functional outcome. No relationship was found between sex and hemisphere of stroke, and she concludes that severity of paralysis and delays in admitting patients for treatment require further research as results were inconclusive.

Depression after stroke

Depression is a general term used to describe many different mood states, and is commonly encountered after any major illness. However, it has been suggested that 'depression' is a specific pathological feature of stroke illness and as such is underdiagnosed. Problems reported to be encountered by patients with post-stroke depression include: chronic behavioural problems; sexual difficulties and dysphasia (House, 1987). House, an MRC Research Officer from the Oxfordshire Community Stroke Project has reviewed the literature on rates of depression after stroke. Although figures of 22 per cent and 27 per cent have been reported in the first weeks after stroke and subsequently maintained over a period of six months to two years, House proposes that due to many faults in the designs of the studies he reviewed, it can not be proved, at the present time, that depression is more common among stroke patients than it is among the elderly with different physical illnesses. Due to the paucity of evidence regarding the aetiology of post-stroke depression and because clinical diagnosis is unreliable, House suggests that is it not possible at the present time to support claims that the condition is underdiagnosed and undertreated. He concludes that depression after stroke should be managed in the same way as depression after any major illness, but stressed the importance of discussing associated problems with the patient as part of the rehabilitation programme.

Conclusion

Despite the magnitude of the burden of the disease in the community, many aspects of stroke management have until recent years been subject to disappointing attitudes within the medical world. The reasons behind this apparent disinterest in stroke patients is in part due to the lack, until recently of effective, interventive medical therapy. The majority of stroke patients admitted to hospital are taken onto general wards of district hospitals, specialising in diagnosis, treatment and nursing care. In the absence of a therapeutic strategy for reversing the effects of an acute stroke, admission for diagnostic reasons have been unnecessary. Furthermore, it is suggested that the traditional role of nursing care may in fact be detrimental to the long-term prognosis of stroke patients. It has been said of hospital care for stroke, that it is 'haphazard and suboptimal' (Wade, 1987) with no one group of medical professionals taking responsibility.

This paper has demonstrated that deaths from stroke have been on the decline. Unfortunately due to the paucity of incidence data over time it cannot be confirmed as yet, if this is a direct result of a reduction in new cases of stroke or whether case-fatality has improved. Whatever the reason, there is a general consensus of opinion that the observed decline in mortality is linked to an increase in the control of hypertension. Moreover the recent flurry of controlled trials to assess the effectiveness of anti-hypertensives in preventing cardiovascular disease have confirmed this view.

New data on risk factors from epidemiological studies have provided greater insight into the aetiology of stroke. Variables such as excessive alcohol consumption and cigarette smoking are now identified as contributing independently to the risk of stroke.

The aim of this paper has been to demonstrate that although the field of stroke management has been neglected in the past, the future appears in a more optimistic light. Not only are the chances of surviving an acute cerebral event increasing, due to advances in medical and surgical techniques, but a better understanding of the needs of stroke survivors and how to organise effective rehabilitation programmes is also on the horizon.

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