Prospects for Patients with Strokes, with Special Reference to the Hypertensive Hemiplegic

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"A disease of old age, which might surprise a man, and yet not immediately kill him, and of which there might possibly be a removal, at least for a season, that there might some space be given him to recover a little strength, before he go home and be no more seen."—King Solomon's Portraiture of Old Age.

Contradictory views on the prognosis of strokes have been the rule in medical writings, and for centuries hemiplegics seeking reassurance on recovery have had a wide range of opinion to choose from.

Hippocrates wrote briefly that it was impossible to cure a severe apoplexy and not easy to cure a mild one, but Soranus of Ephesus gave more positive encouragement. He distinguished apoplexy from residual paralysis, and practised a system of treatment, interpreted by Caelius Aurelianus in the fifth century (Drabkin, 1950), which included much that might be considered "modern" to-day. He recommended passive and active movement; heat, as warm wax or bathing in hot springs; appliances such as weights, pulleys, and a special walking-chair; swimming with inflated bladders to support paralysed limbs; and he particularly required the patient to join in the effort himself and not to rely on others for his exercises.

Conflicting Opinions on Prognosis

It was probably this hopeful tradition that appealed to the author of the quotation given above, which reflects the quiet moderation of John Smith's outlook in 1666. He explained that "an apoplexy" was not necessarily a "Harbinger of immediate dissolution," and he, too, distinguished between apoplexies degenerating into "Palsie" and those progressing to coma and death. This difference was not always acknowledged, and the failure to recognize it may account for some of the conflicting opinions on prognosis. However, John Smith's shrewd observations evidently made little impression, because 125 years later Thomas Kirkland (1792), dissatisfied with the prevailing opinions concerning nervous apoplexies and palsies, was "led from the confusion in which I found the subject involved, to make a new arrangement and to distinguish the different species of these maladies." He used the word "stoke," and described the treatment of a pressure sore and severe spastic paralysis in a neglected hemiplegic invalid. Commenting with satisfaction on the healing of the bedsore, he wrote: "Nor did we with less pleasure behold an amendment of the paralytic side, for her arm and leg, though they did not get perfectly well, recovered very much. The spasms had entirely left her and her health and faculties were greatly restored, so that she enjoyed several years the remainder of her life comfortably."

Another half-century found Copland (1830) still trying to combat discouragement in the prognosis of strokes. A colleague had said: "The mortality of apoplexy is fearfully great, and proves either that the proximate causes of the disease are beyond the reach of art, or that the measures usually adopted as remedies are inapplicable, inefficient, or prejudicial." Copland preferred to believe that, "Although perfect recovery so rarely occurs, the state of the patient may be ameliorated, and he may live many years without the occurrence of any of the unfavourable consequences of the malady." It is surprising, therefore, to read a comment made a generation later by Charcot (1881) that, if an apoplexy was not immediately fatal, in most cases the patient "only retains life at the expense of deplorable infirmities. He is left with more or less complete hemiplegia, most frequently rendering him incapable and even condemning him to perpetual confinement to bed. The intelligence itself rarely escapes completely, and remains more or less profoundly clouded, a condition which may go on to complete dementia." This is a most depressing outlook compared with the more moderate opinion of his contemporary Gowers (1888), who knew that "most cases improve and many recover," provided they were given the kind of care, and the time, necessary to make a recovery that is characteristically slow. Gowers believed that "the tendency to improvement by cerebral compensation, and by the spontaneous disappearance of indirect symptoms, is very marked and makes it difficult to estimate the actual influence of treatment that is employed, especially of methods that extend over a long period and thus have time on their side; moreover, it renders these cases a tempting field for the assumptions of the quasi-therapist."

These differences may have been an expression of different kinds of clinical practice. One was founded on institutional care and biased by an excess of patients who had failed to recover; of 2,000 inmates in the Salpêtrière in 1800, 1,000 were chronic invalids, and 200 of these were hemiplegic. The other, with few supporting hospital beds, was mainly consulting practice outside hospital, more in touch with recovery and optimism than with chronicity and lost hopes. In the first half of this century in Great Britain, the care of most recovering hemiplegics devolved on the family doctor or on the inadequate resources of "chronic" hospitals. Neither offered much opportunity for controlled study. Medical textbooks, with a few distinguished exceptions, gave a gloomy prognosis for residual disability from stroke, and as a houseman in a teaching hospital before the war one gained the impression that patients with cerebrovascular accidents were admitted to die, or, at the end of a few weeks, to choose between a struggle for independence at home or a bed for life in the workhouse infirmary.

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Research into Apoplexy

By 1950 a disease which topped the Registrar-General's mortality returns was only beginning to attract research interest, and more so in America than here. Disparity in views on prognosis was as wide as ever, varying from dismal forecasts to some most extravagant claims. These extremes were derived from selective experience, but support for a more moderate view came from Droller (1955), from Robinson et al. (1957), and from our earlier inquiries (Adams and McComb, 1953; Adams and Merrett, 1961). Our patients were severe or moderately severe hemiplegics who had survived the onset and the period of immediate mortality in the first fortnight after a stroke, and were transferred from home or other hospitals in the third or fourth week. Intellectual and physical deterioration, or cardiac and respiratory complications, accounted for 20% of them within two months of the onset, and the remaining 80% were about equally divided between recovery, defined as independence in walking and self-care (42%), and chronic incapacity (38%). The main factors in prognosis of residual disability appeared to be intellectual impairment (which is often proportional to the severity of the episode); the time elapsing after onset before efforts to restore activity began; persistence with treatment while improvement, however slight, continued; the patient's willingness or ability to co-operate; and the support he could count on at home. These could influence recovery much more than the extent of paralysis, sensory deficit, impaired postural control, poor physical condition, hypertension, or limited exercise tolerance. This half-chance of recovery in our first series was discouraging, but it provoked an inquiry into the causes of failure which has changed attitudes towards "difficult" patients, and their treatment (Adams and Hurwitz, 1963). However, although staff was increased, and the management of hemiplegics seemed to improve, we had no evidence that patients were doing any better, or, if you prefer it, that we were doing any more for them. Therefore we have examined the results of treatment since a new wing specially designed for rehabilitation was opened in 1958, comparing them with those of the earlier inquiry, and the findings, as you will see presently, are rather surprising.

Stages of Recovery

Consideration of the results of treatment of cerebrovascular accidents should take account of the stages of recovery and the settings in which treatment is given. The stages we defined in our previous inquiry were the first two weeks, when immediate deaths directly attributable to the vascular accident occur; the first two months, when there is a second wave of deaths owing to the indirect causes mentioned earlier; and the first two years, the period of prolonged supervision often needed to decide whether residual disability can be overcome. The settings are the home, the general hospital ward, and the geriatric hospital wards, active or long-stay. My only source of information about the prognosis of patients treated at home after a stroke is Dr. John Rankin's review of patients referred to him by his colleagues (Rankin, 1957). The outlook for admissions to general wards has been assessed from the records of the Royal Victoria Hospital, Belfast, in 1963 and 1964, and has been considered in relation to clinical state and electroencephalogram by Melville and Renfrew (1961). Our recent review shows the prospects for those transferred to geriatric wards. At home the fate of many patients with cerebral vascular accidents is decided by rapid progression of signs towards death, or by equally rapid recovery, and admission to hospital may never be considered. Of 252 patients referred to Dr. Rankin immediately after the onset of strokes more than half (58%) died within a few days. Of the remainder 76% regained ability to walk unaided (and 60% were independent also in self-care), 17% had moderately severe or severe residual disability which, without help, confined them to a chair or bed, and 7% died within five months of the onset. This was a difficult and a demanding kind of inquiry, but one that should be pursued and extended, preferably as a part of a comprehensive prospective study of cerebrovascular disease. There were 343 patients with cerebral vascular accidents admitted to the Royal Victoria Hospital in 1963 and 1964 in the following proportions (Table I).

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of Cases</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral haemorrhage</td>
<td>97</td>
<td>28</td>
</tr>
<tr>
<td>16 thrombosis</td>
<td>214</td>
<td>62</td>
</tr>
<tr>
<td>16 embolism</td>
<td>32</td>
<td>9.4</td>
</tr>
<tr>
<td>Total</td>
<td>343</td>
<td>100</td>
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</table>

Records of patients filed under "cerebral tumour" or other space-occupying lesions were not investigated.

(a) Cerebral Haemorrhage (28%).—Subdural in a few; subarachnoid in many as emergencies or transferred for angiography for overlap in other hospitals. Intracerebral haemorrhage established at post-mortem examination or assumed from rapidly deepening coma and death within a day or two.

(b) Cerebral Thrombosis (62%).—I use this term to cover all forms of "non-embolic cerebral infarction" because it rolls more easily off the tongue. New interest in cerebrovascular disease is breeding a new terminology, and while recognizing the need for more precise definitions as a guide to more accurate diagnosis one must protest against terms that are too cumbersome or too short. Why introduce a meaningless abbreviation like T.I.A. to describe a transitory cerebral episode that any self-respecting doctor or patient knows as a wee turn or little stroke?

(c) Cerebral Embolism (10%).—Almost all from rheumatic heart disease, a few after valvotomy, and some established clinically, or more often at post-mortem examination, as the sequelae of cardiac infarction.

Cerebral Episodes

After cerebral haemorrhage (Table II) mortality, although high, was lower than would be expected, because the Royal Victoria Hospital is the neurological centre for Northern Ireland, and these figures are biased by the number of patients transferred for investigation of suspicion of subarachnoid haemorrhage. Raised cerebrospinal pressure and xanthochromia were recorded in most of these subjects, but the diagnosis could not always be established beyond doubt, and many were discharged within a few days without residual disability for further follow-up in their own area hospitals; most victims of intracerebral haemorrhage died, and only 5% of this group were transferred to geriatric wards for rehabilitation. Although badly handicapped, these patients responded well to prolonged care because on average they were younger than those with cerebral infarction.

Of the cerebral thrombosis patients (Table II), whether male or female, one-quarter returned home from the general hospital ward, half were transferred to the geriatric department for further treatment, and one-fifth died within two weeks. The
only patients transferred to a mental hospital came from this group (3%).

The findings for embolic infarction were not consistent for males and females (Table II). Half of the females died, and half of these deaths (and the associated emboli) were attributed to coronary occlusion. There was only one male death, that of a 49-year-old patient who had had a mitral valvotomy seven years before. He died three days after a massive cerebral embolism, which was confirmed at post-mortem examination. Only two of the 12 men had cardiac infarcts, and both made good recovery; the others had cerebral embolism associated with rheumatic heart disease. Survivors of this, whether male or female, are almost always disabled to an extent which seems disproportionate to their ages. In this they resemble many patients who survive strokes after subarachnoid or intracerebral haemorrhage from an aneurysm, or the neurological surgery that often follows. In these the same combination of relative youth and disastrous initial intellectual and physical impairment often applies. Being relatively young as age goes in relation to cerebral vascular disease, it is reasonable to suppose that the victims of haemorrhage from intracerebral aneurysm, or of embolism from a fibrillating auricle, would have healthier cerebral vessels and better circulatory reserves than older patients with arteries affected by atheroma. Perhaps in old age the effect of sudden occlusion in some section of a tottering cerebral circulation, which has become adjusted to its insufficiency, is less disastrous than the devastating ischaemic reaction which, for example, an embolus arriving out of the blue may be able to provoke in healthy vessels.

It seems, then, that the proportion of patients from the general hospital wards needing geriatric rehabilitation after cerebral infarction (50%) is ten times the proportion of those after cerebral haemorrhage (5%). Policy relating to the admission to hospital of patients with strokes varies in different areas, so the ratio of geriatric beds to population where the ratio is high, more strokes may be admitted to geriatric wards at onset as emergencies. Our figures reflect the practice in Belfast whereby most patients with cerebrovascular accidents needing hospital care spend the critical fortnight after onset in an acute admission ward, and survivors are transferred for rehabilitation about the end of the second week. These transfers account for about three-quarters of our hemiplegic admissions to the geriatric wards, and we may look at the results of their treatment next.

In the five years 1959-63 there were 777 hemiplegics admitted to Wakehurst House. Thirty-four with rheumatic heart disease and cerebral embolism are considered separately and 14 were untraced (compared with 26 in the former series), leaving 729 patients in the new series to compare with 710 in the old, both composed predominantly of patients with cerebral thromboses.

Recent Improvement in Results

The demand for hospital care for cerebrovascular disorders seems to be increasing, but this is less unexpected than the changes found in the results of treatment. There were no differences in age distribution between men and women in the two series,

\[ \chi^2 = 3.66, \quad \text{D.F.} = 8, \quad 0.9>P>0.8 \]
\[ \chi^2 = 4.36, \quad \text{D.F.} = 8, \quad 0.9>P>0.8 \]

but fewer patients died within two months, and what was formerly only a 3% difference between recovery and failure in the survivors became 33% in favour of recovery (Fig. 1). This was a gradual change (Fig. 2) brought about perhaps by the earlier transfer of hemiplegics to us (average duration of stay in the wards of the Royal Victoria Hospital of 124 hemiplegics transferred in 1963 and 1964 was 18 days). Alternatively, we may have become more selective in perhaps we chose better, and picked more winners. Despite these possibilities one must conclude that the trend towards a better rate of recovery is a measure of improved understanding and treatment, because we accepted any patient who had a history of independent activity prior to the stroke, who was not moribund on recovery from it, and who had not been neglected to the extent of irreversible physical or mental contractures. Almost two-thirds of our hemiplegics regained independence (although less than half of these had useful hemiplegic hands), only one-quarter became chronic invalids, and the rest died within two months owing to complications of one kind or another. We have shown before (Adams and Merrett, 1961) that patients of either sex who died were on average older than those who became chronic invalids, and these, in turn, were older than those who recovered, but there was no significant age difference between grades of recovery; and, contrary to expectation, there were more patients with dominant hemisphere lesions in the recovered group, a point noted earlier by Hobhouse (1936).

This applies to the new series too (54.9% of the recovered patients had dominant hemisphere lesions). The explanation was thought possibly to be that the patient with a non-dominant hemisphere lesion can afford to ignore the paralysed side and concentrate on the practised use of his “master” limbs, whereas the patient with the dominant hemisphere lesion, finding these limbs relatively useless, is driven to redoubled effort to regain the use of the paralysed side.

These results are encouraging, but they probably mean only that methods of treatment known for centuries are being applied with greater precision and intensity to many more patients. It should reassure hemiplegics everywhere in these islands to know that owing to the development of geriatric departments...
their chances of this kind of care are greater now than ever before, but it would probably interest them more to know their prospects of survival. Unfortunately, as our former inquiry showed, survival is greatly foreshortened after a stroke compared with normal average life expectation (Adam and Merrett, 1961). The recovered hemiplegic patient of either sex appeared to have an even chance of relatively active life for about six years if under 65 years of age at onset, or for three and a half years as a man, or four years as a woman, over that age. Comparable normal average expectations estimated for like age groups were more than 15 years for the under-sixties, nine years for those aged 65–75, and five years if over 75.

This reduced life expectancy may bring welcome release to many tragically handicapped invalids, especially those who are relatively young, but it is the most sinister effect of their underlying arterial disease, and this, of course, is the real arbiter of their fate.

I propose to consider now two controversial topics related to the prognosis of strokes—the treatment of hypertension and the use of anticoagulants.

Hypertension

Raised blood-pressure is said to be an adverse factor in the prognosis of survival after strokes, and advice to lower it, with antihypertensive drugs if necessary, has come from several sources (Marshall and Shaw, 1959; Marshall and Kaeser, 1961; Carter, 1963, 1964). Our experience leaves us less assured of the adverse effect of hypertension after strokes, and less confident about the wisdom of blood-pressure-lowering in apparently hypertensive hemiplegics. Consider, for example, the following case:

A minister was admitted to hospital 12 years ago, following a stroke when aged 59. She was moderately obese, and her blood-pressure was 215/135. Left ventricular enlargement was confirmed by chest x-ray examination and electrocardiography, which also showed evidence of coronary ischaemia. There were grade II hypertensive retinal changes. Two weeks later, on transfer to our wards, her pressure was 220/120; she had residual dysphasia and hemiparesis with a relatively useless hand. She recovered quickly and went home towards the end of the fourth week. Now, aged 71, she still has an untreated pressure of 185/115, but has never had a day’s illness since her recovery, and residual evidence of her hemiparesis is minimal. She works as a housekeeper at the opposite side of the city to her own home, where in her spare time, aided only by a district nurse, she looks after two invalids, a 95-year-old mother, and a sister, who, she explains, is old and has had a stroke.

This patient’s survival might well have been attributed to some antihypertensive drug had she been given one. Her history is not exceptional. Excluding two young people (one with post-puerperal thrombosis and the other with tertiary syphilis), 18 (72%) of the female survivors of the original series had systolic pressures greater than 185 mm. Hg, and 13 (52%) had diastolic pressures of 110 mm. Hg or more. The shortest period of follow-up of these patients was nine years and the longest 15 years. All six male survivors, however, had characteristically low pressures in comparison, with systolic levels below 140 mm. Hg and diastolic pressures of 90 mm. Hg or less.

Case histories such as these are impressive, but inferences drawn from such small numbers could be misleading. Dr. J. D. Merrett was therefore invited to make a statistical study of possible relations between blood-pressure and survival in the whole series, and I am indebted to him for a most thorough analysis. This was omitted from the former inquiry in 1961 because the life tables were so incomplete, and because the blood-pressure was recorded in the early years by different doctors and not by the strict criteria used to set standards of normal pressure in old age by Hamilton et al. (1954), Miall and Oldham (1955), or Anderson and Cowan (1959a, 1959b).

However, the records made each week in our rehabilitation wards and subsequently at out-patient review seemed likely to be at least as reliable as some of those used to brand hypertension as an adverse factor in strokes. The levels used for analysis, taken from the hemiplegia card-index, were the highest resting pressures noted in convalescence—that is, while in the rehabilitation ward.

Women had higher pressures on average than men, and women less than 70 and those 70 and over had to be considered separately owing to certain dissimilar age distributions. For each sex, and at each of these age levels for women, Dr. Merrett calculated life tables to compare survival in the blood-pressure groups defined in Table III. These are described in detail elsewhere (Merrett and Adams, 1965), and I need only refer to the broad conclusions. It appeared that, although patients with low blood-pressures after strokes could expect to survive somewhat longer than those whose pressures were high, the difference was marginal. Out of 100 possible differences, only 10 were statistically significant, and three of these favoured the patients with high blood-pressures; indeed, far from lending convincing support to the belief that hypertension seriously limits survival after a stroke, our results might as easily have been said to show that it is best to be hypertensive as a hemiplegic patient if only to improve the chances of surviving the first two months. The higher mortality in the period among patients with low pressures was one of the more consistent findings in the analysis.

We have no evidence of a critical need to lower the blood-pressures of all patients who are hypertensive after strokes, nor can we reconcile our findings with the belief that the risks of reactions to hypotensive therapy “are not to be compared with the risk of leaving hypertension untreated in these patients” (Marshall, 1964). Much depends on what is meant by “treatment.” Reassurance, with appropriate sedation, is one thing, but treatment with antihypertensive drugs is quite another, although this much-advertised approach is becoming harder to resist. Hemiplegic invalids have enough to worry them without being made pressure-conscious, or having further restrictions imposed on their limited activities. Anti-hypertensive therapy adds the risk of drug-induced dizziness or falls, troublesome complaints well known to Anderson (1965), who has dealt with them successfully in old people referred to the Rutherglen Consultative Health Centre by arranging with the family doctor to have the offending treatment withdrawn.

The discovery of a raised blood-pressure is first a lead to a search for a primary cause, such as renal, endocrine, or collagen disease. If it is determined that it is “essential,” there are clear indications for treatment in the malignant phase, in rapidly progressive hypertension with high fixed diastolic levels, or in heart failure. If, however, the patient has symptomless hypertension there are various factors to be considered before attempting treatment, and in older people blood-pressure level is usually the least of these.

Age is significant, because pressure levels that cause anxiety in middle life can be accepted with less concern a decade or two later. Sex has some influence, because women withstand
raised pressure better than men. How and when pressure was recorded must be considered: "how" because inconsistencies arise from different methods, and "when" because pressures vary so much under different circumstances, and particularly after a cerebrovascular accident. We referred briefly in our previous investigation to the distinctive pressure changes that may follow a stroke, thinking that they were common knowledge. The changes we described (Adams and Merrett, 1961) have since been challenged, but the following evidence can now be added.

Among the patients transferred from other hospitals recently there were 35 (10 males, 25 females) for whom reliable records of blood-pressure within 24 hours of the onset, and again six to eight weeks later, were available, and there were 19 of these (8 males, 11 females) for whom four comparable readings were obtained on admission and at intervals of four weeks. Average readings of systolic and diastolic pressures of either sex on admission were higher than those six or eight weeks later (Table IV and Fig. 3), and analysis of the sets of four readings showed that the third reading on average tended to be slightly below the first and second, but the fourth at follow-up after discharge returned to the level of the second, although still less than the average on admission (Table V and Fig. 4).

### Table IV. Mean Blood-pressure Readings on Admission and Six to Eight Weeks After a Stroke

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<tr>
<th></th>
<th>Males</th>
<th>Females</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Admission</td>
<td>6-8 Weeks</td>
</tr>
<tr>
<td>Systolic B.P.</td>
<td>179-0</td>
<td>127-5</td>
</tr>
<tr>
<td>Diastolic B.P.</td>
<td>106-5</td>
<td>83-0</td>
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</tbody>
</table>

### Table V. Mean Blood-pressure Readings on Admission, on Transfer, and at Four-week Intervals After a Stroke

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
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<tbody>
<tr>
<td></td>
<td>Admission</td>
<td>Transfer</td>
</tr>
<tr>
<td>Systolic B.P.</td>
<td>176-3</td>
<td>152-5</td>
</tr>
<tr>
<td>Diastolic B.P.</td>
<td>105-6</td>
<td>88-8</td>
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We conclude that blood-pressure tends to be high on admission with cerebral infarction, falling gradually throughout the next two or three months in hospital, despite considerable physical activity towards the end of this time. At review after four weeks at home the pressure has usually returned to the resting level at four to six weeks after the stroke.

The stroke, therefore, may decide whether treatment should be given to lower blood-pressure. The hypertensive patient who has not had a stroke faces the risks of cerebral haemorrhage, ischaemic heart disease, cardiac failure, or uraemia. If hypertension is severe the indication for treatment is unquestioned, because each of these can be attributed to a direct relation between hypertension and arterial disease on clinical, if not always on well-established pathological, grounds. The association between raised systemic pressure and cerebral haemorrhage is notorious (although no one yet knows whether the vascular weakness exploited by the raised pressure is a micro-aneurysm, necrotizing arteritis, or ischaemic capillary damage); the Framingham survey (Dawber et al., 1962) has established hypertension as a prime mover in the causation of coronary heart disease; raised pressure overloading an ischaemic myocardium is the direct cause of hypertensive heart failure; and renal failure results from the arteriolonecrosis associated with malignant hypertension. These conditions occur mainly in patients in middle life, accompanying grades of raised systemic pressure that are severe in relation to age, and mortality is high in them all.

![Fig. 3.—Comparison of blood-pressures at onset of stroke and six to eight weeks later.](image)

![Fig. 4.—Blood-pressures at onset of stroke and at approximate monthly intervals after.](image)

### The Clinical Picture

The clinical picture in the hypertensive hemiplegic is different. Owing to heavy mortality, few survivors of intracerebral haemorrhage are represented, and younger patients with congenital aneurysms or cerebral embolism are not usually hypertensive. Most hemiplegic invalids with raised pressures therefore have had cerebral thrombosis. They differ from the pre-hemiplegics at risk of haemorrhage in being older, in having blood-pressure levels which are not disproportionately high in relation to age, and in being exposed to different mortality risks—respiratory complications (34%), another cerebral vascular accident (32%), coronary infarction (18%), and other causes (16%) (Adams and Merrett, 1961).

Therefore we are reluctant to accept the idea that hypertension in these patients should invariably be treated, not only because of the results of our investigations, but because of the theoretical possibility that their cerebral circulations are less vulnerable to the direct hazards of raised systemic pressure (cerebral haemorrhage) than to the indirect effects of arterial disease (vascular insufficiency or occlusion, subintimal haemorrhage, or micro-embolism). This is in keeping with suggestions made by Leishman (1963) to explain his findings that, although reduced mortality in successfully treated patients with severe hypertension was due to the prevention of cerebral haemorrhage and uraemia, the mortality in later years of the follow-up included a high proportion of fatal strokes even in patients with well-controlled pressures. He thought that with severe untreated hypertension cerebral haemorrhage arose, like renal failure, from necrotizing arteritis, and could be prevented.

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1 Statistical analysis suggested that these differences were significant, but this particular analysis requires that blood-pressure in all patients should be recorded at exactly the same intervals. Although variations in these intervals in this group of patients were slight, they were not precise enough to satisfy Dr. Merrett’s requirements.
by early treatment, whereas the later strokes were caused by atherosclerosis, and reduced pressure could not protect the victims from the consequences of this.

If most elderly patients who have survived strokes have run their risk of cerebral haemorrhage, and if they are not threatened with cardiac or renal failure, why lower their blood-pressure? Presumably the intention is to limit the spread of atherosclerosis, which hypertension is said to promote in the larger arteries and to extend into the arterioles (Carter, 1964). The similarity and uniformity of the vascular complications in all the conditions of diverse aetiology characterized by raised arterial pressure, and the reversal of these complications when the blood-pressure is lowered, support its primacy as the cause of the complications, whether atherosclerotic or arteriosclerotic, according to Sokolow and Harris (1962). However, there has been no counterpart of the Framingham study for cerebral vascular disease, and, although its development may be brought forward in time by hypertension, evidence of this is not solid enough yet to justify indiscriminate blood-pressure lowering after strokes.

We should treat patients, not arbitrary blood-pressure levels.

Anticoagulants

There are two clear indications for the use of anticoagulants in cerebrovascular disease: first, in patients with mitral stenosis, auricular fibrillation, and cerebral embolism, and, secondly, in some patients with vertebral-basilar insufficiency, because the prognosis for either is sometimes so poor without treatment that anticoagulants are at least worth a trial.

It has been estimated that the chances of developing a left auricular thrombus are as high as 84% in patients with "pure" mitral stenosis and auricular fibrillation (Wells, 1961), and that half of these patients will develop peripheral embolism. Moreover, a patient who has had an embolism is almost certain to have others within a few days or weeks unless treated with anticoagulants. Carter (1960) believes this treatment should be given early in cerebral embolism, and comments that it may have to be continued indefinitely, but the source of embolism in one-third of his patients was coronary infarction.

Since 1948 we have admitted 64 patients with cerebral embolism attributable to mitral stenosis with auricular fibrillation.

Fifty-three had neither valvotomy nor anticoagulants, and almost a third of these died within six months of the onset of embolism, and more than half were dead within two years (Table VI). The average age of these untreated patients was 54.5, and there was a preponderance of older patients among the 25 who died in the first two years (their average age was 57.4 years). Nine of these untreated patients still survive. Their average age is 56.0 years, and survival varies from just over two years in those most recently added to 10 years for the oldest survivor.

Eleven patients were treated with anticoagulants; nine of them are still alive. Two died in the fourth year after the stroke when 45 years old. One died in congestive heart failure attributed to subacute bacterial endocarditis, the other was a sudden "cardiac" death, but as both deaths occurred at home neither cause could be confirmed by necropsy. The remaining patients (average age 53.2) have survived from 4 to 11 years since their cerebrovascular accidents. The 11-year survivor, originally untreated and unfit for valvotomy, was given anticoagulants when, after five years, the recovery she had painstakingly worked for was suddenly lost owing to another cerebral embolism.

The benign course of transient cerebral ischaemia is fully appreciated (Acheson and Hutchinson, 1964), but some patients become bedridden because of constant unsteadiness, recurrent drop attacks, or loss of confidence owing to repeated falls, although still mentally alert and showing little or no evidence of focal pyramidal damage (Adams and Hurwitz, 1963). Treatment with anticoagulants appears to abolish the symptoms and restore confidence in some of these patients, and anyone who is deprived of independence or made bedridden in this way deserves at least a therapeutic trial for 10 to 12 weeks. The response to anticoagulants is less likely to be explained by cerebrovascular insufficiency (Denny-Brown, 1951; Corday et al., 1953), than by thromboembolism (Milliken and Siekert, 1955; Fisher, 1959; Russell, 1961; Ashby et al., 1963). Millar (1965) has suggested that platelet stickiness may play some part in this mechanism, and stickiness, therefore, was assessed in a number of healthy octogenarians and nonagenarians living in a residential home, and in some patients with cerebral infarction immediately after the onset of strokes and again in the second week. I am indebted to Miss Anne Dalby for the results of these tests, which were made by a modification of the technique of Wright (1942) described by McDonald and Edgill (1958) that was adopted by Bridges et al. (1965), to whom I am also indebted for the results of platelet stickiness in the controls under 60 years of age and for comparison in Fig. 5. The numbers were small for accurate statistical assessment, but it seems that while platelet stickiness is low in the younger controls it is scattered over a wide range in old people, and although increased in patients with strokes there is no consistency in the stickiness levels found in them either.

Other Factors of Interest

Time does not permit full consideration of some other interesting factors related to recovery or to survival after strokes. The future is brighter for patients with impaired postural control. This is a common feature of cerebrovascular disorders, but the extent of the handicap it imposes on recovery is not always appreciated. Studies such as those of Kremer (1958), Sheldon (1960, 1964), and Martin and Hurwitz (1962) have defined the physiological mechanisms required for static or moving postural control, and their work has made it possible to recognize specific patterns of disturbance and so devise techniques of treatment. We are interested, too, in establishing

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**Table VI.—Cerebral Embolism in Rheumatic Heart Disease**

<table>
<thead>
<tr>
<th>Time since Onset (Years)</th>
<th>Untreated</th>
<th>Treated (Warfarin)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Died (44)</td>
<td>A &amp; W (9)</td>
</tr>
<tr>
<td>0-1</td>
<td>18</td>
<td>—</td>
</tr>
<tr>
<td>1-3</td>
<td>25</td>
<td>—</td>
</tr>
<tr>
<td>3-5</td>
<td>33</td>
<td>1</td>
</tr>
<tr>
<td>5-7</td>
<td>39</td>
<td>1</td>
</tr>
<tr>
<td>7-9</td>
<td>44</td>
<td>4</td>
</tr>
<tr>
<td>9-11</td>
<td>10</td>
<td>5</td>
</tr>
</tbody>
</table>

Mean age = 54.5, Standard deviation = 10.7

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**Fig. 5.—Platelet stickiness in old age and strokes.**
the distinguishing features of those whom Sheldon (1964) described in characteristically well-chosen words as "the physical aristocracy of the advanced years." These are the people we have to accept, for want of true random samples, as average "normals" in old age, and they usually show a combination of exceptionally well preserved ectoderm and intellect. Even when their mesoderm lets them down through arterial disease their recovery can be built on solid foundations.

The prospects of recovery have been advanced immeasurably by improved postgraduate education, by better dissemination of knowledge, and, as a result of this, by more accurate diagnosis and better medical care of impending stroke, established stroke, or residual hemiplegia. The early promise of vascular surgery (Eastcott et al., 1954) was offset by discouraging results and prognostic uncertainties (Fazekas et al., 1963), but as gaps in knowledge of the natural history of the disease are filled in, and with better selection, the outcome of cerebrovascular surgery and its prognosis must improve (Irvine et al., 1965; Lancet, 1965).

Underlying all this, and of paramount importance, is arterial disease, the source of almost all the trouble, and the dominant factor determining the prospects of survival after a stroke. The importance attached to it is emphasized by the columns of new research references it acquires in each volume of the Cumulated Index Medicus. But I am ill-equipped to enter Sir George Pickering's Augean stable of atherosclerosis and contend with the cosmic dust he raised there recently (Pickering, 1964). Let me instead commend to you the beautifully presented, comprehensive, yet concise review of arterial disease recently written by Mitchell and Schwartz (1964). To anyone with an inquiring mind there is an open invitation in their description of associations between arterial disease, hypertension, and cerebrovascular disorders as "a field where conjecture and wishful thinking have predominated far too long."

Summary

Prospects for recovery have been distinguished from prospects for survival in the prognosis of strokes. Comparison of the results of treatment of 736 hemiplegics during 1948-56 with 777 patients treated during 1959-63, suggests that prospects for recovery have improved. Survival, however, depends almost entirely on the extent of the underlying arterial disease, and there is no means of changing the course of this. Hypertension has been said to have an adverse effect on the prognosis of strokes. This is questioned. Anticoagulant treatment may improve the prognosis of cerebral embolism attributable to rheumatic heart disease and of vertebral-basilar insufficiency.

I wish to thank my patients for their courtesy and co-operation, and the physicians and family doctors who referred them for treatment. I am indebted to Mr. L. M. Payne, Librarian of the Royal College of Physicians of London, for historical researches; to Miss J. B. Webster and her staff in the medical library of the Queen's University of Belfast; to Brigadier T. W. Davidson, Group Medical Superintendent, and Mrs. J. Easterby, Medical Records Officer, for access to the records of the Royal Victoria Hospital; to Dr. J. D. Merrett for statistical help; to Mr. G. Smith for the illustrations; and to Mr. J. A. Robin and his assistant Miss A. Fitzpatrick for photographic reproduction of them. I am most grateful to the staff of the geriatric department for their direct and indirect help, to Dr. R. S. Allison for his continued interest and encouragement, to Dr. Mary Adams and to Dr. Louis Hurwitz for helpful comment on the manuscript, and especially to Mrs. Vera Stewart for the time and care given to preparing it and the tables.

REFERENCES

(Ibid., 18, 123.