

Cerebrovascular incidents after myocardial infarction

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SUMMARY Pathological studies show a high correlation between the degree of atheroma in coronary, cerebral, and carotid arteries. Necropsy evidence of myocardial infarction also shows a high prevalence of severe atheroma in the carotid arteries. A further pathological finding is that obstruction in cerebral and carotid circulations is commonly due to embolism from the heart. In contrast, long-term follow-up of survivors of myocardial infarction indicates a low prevalence of cerebrovascular disease. To test if this low prevalence is due to lack of clinical ascertainment, a study was made of 260 survivors of myocardial infarction followed for five years. Specific attention was given to eliciting any clinical manifestations of cerebrovascular disease. In this study it was confirmed that in survivors of myocardial infarction the prevalence of cerebrovascular disease is surprisingly low: completed strokes 4%, transient cerebral ischaemic attacks 2%. A possible explanation of the low prevalence is that after the acute episode of myocardial infarction attacks of cardiac dysrhythmia predisposing to systemic embolism become infrequent.

The generalised nature of atheroma leads one to expect a high correlation between the prevalence of lesions at various sites. Pathological studies have shown this to be so. Correlation between the degree of atheroma of the coronary and cerebral arteries measured at necropsy is high, though not so high as between cerebral arteries themselves (Young *et al.*, 1960). Correlation between the degree of atheroma of coronary and carotid arteries is likewise high (Young *et al.*, 1960; Mitchell and Schwartz, 1965). When necropsy evidence of myocardial infarction is made the starting point, the prevalence of carotid stenosis is again high, the lumen of the sinus being reduced by half in 59% of men with myocardial infarction against 35% in unselected necropsies (Mitchell and Schwartz, 1965). Conversely, when atheroma of the carotid sinus is the starting point the prevalence of myocardial infarction rises from 8% in cases with mild atheroma to 67% in severe cases (Martin *et al.*, 1960).

Embolisation from the heart to the carotid and cerebral circulation is also a common pathological finding. In a series of cases of occlusion of the internal carotid artery 20% were caused by emboli

from the heart (Berry and Alpers, 1957); in another series 45% of cerebral infarctions were similarly caused (Blackwood *et al.*, 1969). In a monumental study of 994 consecutive necropsies comprising 40% of deaths in Oslo over a six month period, 47% of occlusions of cerebral vessels and 46% of cerebral infarcts without verified occlusions were judged to be embolic, the majority coming from the heart (Torvik and Jørgensen, 1964, 1966; Jørgensen and Torvik, 1966, 1969). The pathological evidence suggests, therefore, that whether one starts with patients with ischaemic heart disease or with cerebral ischaemia, a high prevalence of the other condition will be found.

The evidence provided by clinical studies on this point is conflicting. Large series of unselected strokes presenting acutely attribute less than 10% to emboli (Glynn, 1956; Groch *et al.*, 1961; Carter, 1964). Conversely, although cerebral embolism from endocardial thrombi is well recognised in the acute stage of myocardial infarction, follow-up studies of survivors provide less evidence of a relationship. Five major studies of survivors of myocardial infarction followed from two to 10 years, comprising 2662 patients, make no mention of strokes (Honey and Truelove, 1957; Pell and D'Alonzo, 1964; Woodhouse, 1969; Mulcahy *et al.*, 1975; Vedin *et al.*, 1975). Five other studies

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involving 1094 patients record a cerebrovascular accident as the cause of 1–8% of deaths (Cole *et al.*, 1954; Helander and Levander, 1959; Juergens *et al.*, 1960; Dimond, 1961; Gertler *et al.*, 1964). In only one of these studies (Gertler *et al.*, 1964) were transient cerebral ischaemic attacks mentioned specifically and this was to say that none had been recorded.

While pathological studies, therefore, show a high correlation between cerebrovascular and cardiovascular disease, clinical studies show a less close relationship. It is important to try to resolve this discrepancy because if a large proportion of strokes have a cardiac cause, preventive measures should be directed to the heart. The present study was undertaken with the specific objective of seeking clinical manifestations of cerebrovascular disease in survivors of myocardial infarction on the hypothesis that the low reported incidence is due to lack of ascertainment.

Patients and methods

Between 1 October 1970 and 30 September 1971, 334 patients (265 males and 69 females) were admitted to the coronary care unit of the West Middlesex Hospital with acute myocardial infarction or acute coronary insufficiency. Of these 260 (212 males and 48 females) survived to be discharged from hospital after four weeks or more. Patients with recent myocardial infarction (214; 82%) had typical cardiac pain with either characteristic serial electrocardiographic abnormalities of infarction or non-specific serial electrocardiographic abnormalities accompanied by increase of serum enzymes (McAllen, 1971). Patients with acute coronary insufficiency (46; 18%) had typical cardiac pain and non-specific serial electrocardiographic abnormalities without elevation of serum enzymes. For 58 of the males and 13 of the females it was not their first infarct. In addition to the routine follow-up which had taken place, as many as possible were re-examined clinically and electrocardiographically by one of us (PM McA) during 1975–76, giving a follow-up period of five years, specific attention being paid to eliciting any history of stroke or transient ischaemic attacks (TIAs) or signs of cerebrovascular disease. Evidence of past or present systemic embolism or cardiac dysrhythmia was also carefully sought. Those in whom there was any suggestion of cerebrovascular disease were also seen independently by one of us (JM). In the case of patients who could not attend hospital information was obtained from the family doctor or relatives. Death certificates were sought for all who had died.

Results

The age and sex distribution of the patients is given in Table 1. The results of follow-up are given in Table 2, which shows that 158 of the 188 patients not known to have died were seen personally by one of us. At the time of follow-up 72 patients were found to have died and Table 3 lists the cause of death given on the death certificate.

Table 1 Age and sex distribution of patients

Age group (yr)	Males	Females	Total
25–34	2	0	2
35–44	18	4	22
45–54	63	7	70
55–64	94	19	113
65–74	34	17	51
75–84	1	1	2
	212	48	260

Table 2 Result of follow-up

Result	Number of patients
Seen personally	158
Information from family doctor or relative	11
Died	72
Lost to follow-up	19
	260

Table 3 Cause of death

Cause of death	Number of patients
Myocardial infarction	44
Cardiac failure	2
Ruptured aorta	1
Cerebrovascular accident	6
Carcinoma	7
Other causes	7
Unknown	5
	72

In five instances, though we had reliable information that the patient had died, the death certificate could not be traced. It can be seen that the majority were certified as having died from a further myocardial infarction. Six of the patients were certified as having died of cerebrovascular accident; three of these were hypertensive when last seen (BP 210/130, 180/130, 210/110 mmHg).

Abnormal features relevant to the cardiac and cerebrovascular status of the 158 survivors who were examined personally are listed in Table 4.

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Table 4 *Condition at follow-up of 158 survivors seen personally*

Condition	Number of patients
Atrial fibrillation	6
Congestive failure	1
Bundle branch block	4
left	3
right	3
both	1
Pacemaker inserted	69
Angina pectoris	4
Completed stroke	5
Transient ischaemic attacks	8
Carotid bruit	

Four patients experienced non-fatal completed strokes, all diagnosed as cerebral infarction. None was hypertensive and none had carotid bruits. One, a man in sinus rhythm, had his stroke three months after his myocardial infarction, and a second man with atrial fibrillation after one year. A third man in sinus rhythm had a mitral valve replacement after his myocardial infarction; two years later he experienced two strokes within an interval of a few weeks. The one woman was in sinus rhythm and had her stroke four years after her myocardial infarction.

Five patients experienced TIAs, two in the carotid territory, two in the vertebrobasilar, and one in both. All were in sinus rhythm, one was hypertensive (BP 210/130 mmHg) and only one had carotid bruits. The hypertensive patient was numbered among the six who had a fatal stroke. The TIAs started between seven months and four years after the myocardial infarction.

Table 5 *Influence of factors on survival*

	Months					
	0	6	12	24	36	48 60
	% surviving					
Males	100	98	94	86	78	71 65
Females	100	91	87	80	80	73 65
Males 55 years or more under 55	100	98	92	82	71	65 56
	100	98	97	90	86	77 77
Males no previous infarct previous infarct	100	98	95	86	77	71 68
	100	96	89	84	75	68 58
Males no previous angina previous angina	100	98	93	84	76	69 65
	100	97	97	94	78	73 57
Males diastolic BP < 100 mmHg at discharge	100	98	96	88	81	71 64
diastolic BP 100 mmHg or more at discharge	100	96	85	79	66	66 66
Males systolic BP < 160 mmHg at discharge	100	98	96	88	82	74 67
systolic BP 160 mmHg or more at discharge	100	95	85	77	62	62 62

The influence of a number of factors on survival calculated by life table technique (Merrell and Shulman, 1955) is shown in Table 5. Because of the small number of females the factors other than sex were examined only in relation to the males. In our series age and previous myocardial infarction had an adverse influence on the survival of males up to 60 months. Raised blood pressure, both diastolic and systolic, also had an adverse effect. The occurrence of angina before the myocardial infarction did not have an adverse influence except in those who survived for 60 months.

Discussion

The hospital from which these patients were discharged serves a well-defined catchment area with a population of approximately 300 000. Patients admitted with myocardial infarction are cared for initially in the coronary care unit. Selection factors, are, therefore, unlikely to have affected greatly the composition of the group which may be taken as typical of patients who have experienced a myocardial infarction.

The five year survival rate of 65% calculated by the life table method falls within the range found in other series, which extends from a low of 39% (Honey and Truelove, 1957) to a high of 74% (Pell and D'Alonzo, 1964). Precise comparison is not possible because of the differing degree to which factors known to influence long-term survival after myocardial infarction present in various series (Cole *et al.*, 1954; Honey and Truelove, 1957; Helander and Levander, 1959; Juergens *et al.*, 1960; Dimond, 1961; Gertler *et al.*, 1964; Pell and D'Alonzo, 1964; Woodhouse, 1969; Mulcahy *et al.*, 1975; Vedin *et al.*, 1975). The importance of these factors is well demonstrated in the present study, age, previous myocardial infarction, and systolic and diastolic blood pressure having an adverse effect.

The effect of blood pressure is of particular interest in that its adverse influence is seen mainly during the first two to three years after myocardial infarction. Better control of blood pressure might eliminate this early wastage leaving the currently intractable problem of loss due to complications of atheroma to be faced (Breckenbridge *et al.*, 1970).

The incidence of cerebrovascular episodes after myocardial infarction in this series is extremely low. Only 4% of the patients suffered a completed stroke and 2% TIAs. This was not due to lack of ascertainment as might occur in a routine follow-up when TIAs may not be mentioned by patients

or, if mentioned, are misdiagnosed. Most of the survivors were seen personally and specific enquiry was made into any of the many manifestations which might represent an episode of cerebral ischaemia. Despite this careful enquiry the number of cases found was small.

The discrepancy between the reported frequency of cerebrovascular episodes due to emboli from the heart in pathological and clinical studies remains unexplained. Certainly in the present study, heart disease as manifested by myocardial infarction did not carry a high risk of cerebrovascular episodes, either major or minor, during the period of the patient's survival. It is noteworthy that in pathological series of cerebral infarction caused by embolism from the heart, myocardial infarction is not the commonest form of heart disease, being present in 23% in one series (Blackwood *et al.*, 1969) and 42% in another (Jørgensen and Torvik, 1966).

It may be that it is not the coexistence of atheroma as such which is responsible for the occurrence of cerebrovascular incidents in patients with cardiac disease but the presence of cardiac dysrhythmia. It has long been recognised that atrial fibrillation carries a high risk of embolism, particularly cerebral. Recently, systemic embolism as a hazard of chronic sinoatrial disorder has been suggested (Rubenstein *et al.*, 1972), denied (Eraut and Shaw, 1973), but increasingly documented (Samarasinghe and Senanayake, 1973; Radford and Julian, 1974; Krishnaswami and Geraci, 1975; Fairfax *et al.*, 1976). In one study it occurred in 16% of patients with chronic sinoatrial disorder against 1.3% of matched patients with complete heart block (Fairfax *et al.*, 1976). Likewise transient cerebral ischaemia in other dysrhythmias has also been documented (Walter *et al.*, 1970) though again its frequency has been denied (Reed *et al.*, 1973).

In the present study the prevalence of cardiac dysrhythmia was low. On discharge from hospital one patient was fibrillating and one had complete heart block; at the time of follow-up only six had atrial fibrillation. Of course there is no means of knowing of patients who had episodes of dysrhythmia which passed unrecognised but it does seem that, in the absence of recognisable cardiac dysrhythmia, cerebrovascular incidents are uncommon in survivors of myocardial infarction.

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