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Cerebral Embolism in the Michael Reese Stroke Registry*

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SUMMARY Infarction secondary to cerebral embolism was diagnosed in 127 (23.5%) of 540 patients in the Michael Reese Stroke Registry. Coronary artery disease, atrial fibrillation, valvular heart disease, mitral annulus calcification, and cardiomyopathy were the commonest etiologies. Echocardiography documented a potential embolic source in 7 patients without previously known heart disease, and clarified the cardiac pathology in many of the patients with known heart disease. The left anterior circulation was affected in 48%, right anterior in 37%, and posterior circulation in 15% of patients. CT was abnormal in 71% of the patients, and was approximately equally helpful in all locations. Nineteen percent of emboli presented with a deficit that was other than maximal at onset. Concurrent systemic embolism was unusual (2.3%). Prognosis was somewhat worse than in thrombotic stroke. Grouping of patients according to embolic source (intra-arterial, cardiac, and uncertain source) showed no differences in activity at onset, early course, or in subsequent course of the illness.

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CLINICAL CONCEPTS OF CEREBRAL EMBOLISM have changed dramatically during the past decade. Before 1970, embolism was seldom diagnosed; criteria for diagnosis usually included at least two of the triad of (1) known cardiac source (recent myocardial infarct or rheumatic mitral stenosis with atrial fibrillation), (2) sudden, maximal at onset neurological deficit, and (3) associated systemic embolism. When cerebral angiography became commonplace, atherosclerotic plaques in the extracranial arteries were identified as a source for intra-arterial emboli. Matsumoto et al. recognized that the 8% of 993 strokes attributed to embolism at the Mayo Clinic between 1955 and

1969 underestimated embolism because diagnosis depended upon awareness of a known source.¹ Strokes are readily separated into a hemorrhagic group (subarachnoid and intracerebral hemorrhage) and ischemic group but further division of the ischemic group into thrombotic and embolic mechanisms is often difficult and controversial. Dalsgaard-Nielsen considered thrombosis and embolism together^{2,3} and the National Cooperative Study Group even coined the term "thorem" to indicate those instances when the differentiation between thrombosis and embolism could not be made with certainty.⁴

The Harvard Cooperative Stroke Registry estimated that fully 31% of their 694 patients had cerebral embolism,⁵ a figure far higher than any prior study.^{1,6-8} The Harvard registry noted (1) atrial fibrillation was an important cause of embolism even without fresh cardiac infarction or valvular disease, (2) embolism did not always cause a deficit that was maximal at onset, (3) systemic embolism was rarely recognized in patients with cerebral emboli, (4) cerebral angiography often documented distal embolism if performed within 48

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hours of stroke onset and could define an intra-arterial source.⁵ Subsequently, further evidence has established atrial fibrillation as an important cause of embolism and has identified other cardiac disorders especially mitral valve prolapse, cardiomyopathy, intra-atrial defect with paradoxical embolization, and marantic endocarditis as important sources of emboli.

Newer cardiac imaging techniques (such as echocardiography, gated cardiac scans, and labelled platelet cardiac scintillation scans) increase the potential for recognition of cardiac pathology. The advent of digital subtraction angiography promises less invasive visualization of intra-arterial embolic sources. These newer technologies should improve the separation of thrombotic from embolic disease. The Harvard registry antedated the widespread application of CT and echocardiography.⁵ We reviewed and now report our experience in the Michael Reese Stroke Registry with patients with the sudden onset of neurological deficits that we diagnosed as cerebral embolism in order to further clarify clinical and laboratory aspects of this disorder. These patients were seen between August 1979 and June 1982.

Methods

The Michael Reese Stroke Registry is a prospective computerized data bank written in the PASCAL language utilizing an Apple microcomputer. Patients were initially entered by Stroke Service neurologists at the time of their stroke; laboratory evaluation, subsequent clinical course of illness, and final diagnosis were later added at discharge. All patients were examined and entries checked by two of the authors (D.B.H. or L.R.C.). Five hundred and forty patients had been entered by June 1982, when the registry was searched for patients with a discharge diagnosis of cerebral embolism. The Harvard Stroke registry criteria for embolism were used.⁵ Patient summaries were checked and cases in whom the diagnosis of embolism was insecure were eliminated leaving 127 patients for analysis. This group was divided into 3 smaller groups according to the source of embolism: (1) intra-arterial embolic source (10), (2) cardiac source (72), and (3) uncertain source (45). The source was considered uncertain if investigation was incomplete, or if the source

was equivocal. Echocardiography (M mode and two-dimensional) was performed using either a Picker system 80c, with an oscillating mechanical sector scanner (sector angles up to 60 degrees) and ultrasound crystals of 2.25 MHz frequency, or an Advanced Technical Laboratories Mark III system, with a rotating mechanical sector scanner (sector angle 90 degrees) and ultrasound crystals of 3 MHz frequency. Two dimensional echographic recordings on videotape in real-time were made in standard long-axis, short-axis, apical and (when possible) subcostal views. Group comparisons between atherosclerosis and embolism were made by chi-square tests on 2 × 2 contingency tables.

Results

Age, sex and racial characteristics did not differ significantly among the patients in the different embolism groups (table 1) nor did the characteristics of the embolism patients differ from those with large vessel thrombosis (called herein atherosclerosis) or from those of patients in the registry as a whole.

Prior strokes occurred in 27% of the embolism patients. Four old infarcts were found on CT in clinically uninvolved vascular territories without a corresponding history of prior stroke. The number of old strokes in the embolism patients was not significantly different from either patients in the atherosclerosis group or patients in the entire registry. TIAs were far commoner in the atherosclerosis group but of course the occurrence of TIA in the same vascular territory as the stroke was a major criterion for placement in the atherosclerosis group. Seizures were uncommon in the entire registry (7%) and did not distinguish thrombosis from embolism. Systemic embolism was infrequently recognized (2.3%, of the cerebral embolism patients). Ten patients (8%) had a second embolic stroke after entry into the registry.

Seventy-six patients with a diagnosis of cerebral embolism had echocardiograms. Among the 45 patients in group 3 (embolic source uncertain), 23 had no previously known heart disease. Ten of these patients had no echocardiography and 5 had a normal echo. Four patients had nonspecific minor echocardiographic findings, (e.g. ventricular or atrial enlargement). Four patients had a potential embolic source identified

TABLE 1 Characteristics of Embolic Stroke Groups as Contrasted with the Atherosclerosis Group and All Registry Patients

	All registry cases (N = 540)	Atherosclerosis (N = 180)	Embolic groups by source			All emboli (N = 127)
			Group 1 (arterial) (N = 10)	Group 2 (cardiac) (N = 72)	Group 3 (uncertain) (N = 45)	
Age (years)	63.0	66.1	65.3	68.4	67.5	67.7
Sex (% male)	52	56	50	49	62	54
Race (% black)	71	70	70	63	69	66
Old strokes (%)	27.5	28	30	22.5	33	27
TIA (%)	21	41.5*	40	11	6.6	11*
Seizures (%)	7	5.7	10	4	6.6	5.5
Systemic embolism (%)	1.7	0*	0	2.8	2	2.3*

*Atherosclerosis group differs from embolism group, chi-square test, $dF = 1, p < .05$.

by echo; 3 had akinetic regions, and one a cardiomyopathy. In 20 patients, angina or a prior myocardial infarction was present. Of these, 11 had no echo and 4 had a normal echo. Three patients had ventricular enlargement, 1 had mitral annulus calcification, and 1 had an akinetic region. Arrhythmias were present in 2 patients (sick sinus syndrome and atrial fibrillation) but neither of these patients had an echocardiogram. In group 3 (embolic source uncertain), 17 patients had normal noninvasive vascular studies in the form of Doppler flow studies, OPG, and carotid phonoangiography; and 8 patients had cerebral angiography. Angiograms documented distal emboli in 4 patients and 2 had proximal minor plaque disease. Four arteriograms were normal.

Fifty-four of the 72 patients in group 2 (presumed cardiac source) had echocardiography (table 2). In most cases the patients had known arrhythmias or known ischemic, valvular, or cardiomyopathic disease. Six patients without prior known cardiac disease had a mural thrombus (3), atrial myxoma (1), cardiomyopathy (1), or mitral annulus calcification (MAC) (2) found by echo. In 4 patients, post-mortem examination revealed a cardiac source not documented by echo; these lesions included marantic endocarditis (2), cardiomyopathy with a mural thrombus, ventricular aneurysm with recent MI, and myocardial infarction with mural thrombosis.

Fifty-one of the 127 cerebral emboli patients (40%) had either intensive care unit or Holter cardiac monitoring for arrhythmia as compared to 30% of the entire registry and 23% of the atherosclerosis patients. Angiography was performed in 21 patients, 7 patients in each of the diagnostic groups.

CT was performed in 124 patients (98% of the em-

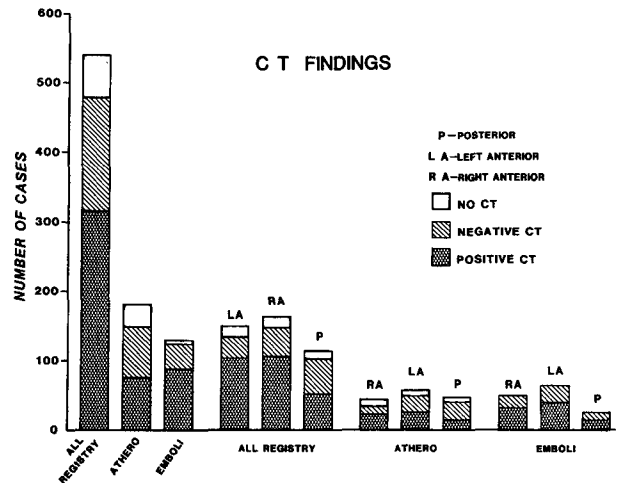


FIGURE 1. CT scan findings in the 540 registry cases, 180 atherosclerotic infarction cases, and 127 embolic infarction cases.

bolism patients compared to 77% of atherosclerosis patients and 89% of the entire registry). Seventy-one percent of patients with embolism had an abnormal CT; there was no significant difference in CT positivity in the 3 embolic groups. Clinically 48% of patients had a left anterior circulation lesion, most often in the middle cerebral artery distribution (62.8% of these had a positive CT); 37% had a right anterior circulation lesion (64% positive CT) and 15% had a posterior circulation lesion (65% positive CT). In 4 patients CT defined an unsuspected old stroke in a clinically unaffected vascular territory. Figure 1 depicts the CT data. Patients with embolic strokes had a higher percentage of CT's and more positive CT's than the patients with

TABLE 2 Group 2 (Cardiac Source) Echo Data

	Cardiac diagnosis prior to echo	Number having an echo study	Echocardiographic findings suggestive of an embolic source			
(1)	Coronary artery disease (MI or angina)	40	27	mitral annulus calcification (MAC)	2	
	recent MI			11	akinetic region	3
	atrial fibrillation			15	mural thrombus	2
	heart block			2	ventricular aneurysm	3
					calcified aortic valve	2
(2)	Atrial fibrillation	33	20	diffuse cardiac dysfunction	3	
	angina			7	mitral regurgitation	2
	MI			13	mitral annulus calcification (MAC)	2
	valve disease			6	aortic valve calcification	1
					mural thrombus	1
					diffuse cardiomyopathy	2
					ventricular aneurysm	1
(3)	Valve disease	13	11	mitral regurgitation	1	
	atrial fibrillation			6	akinetic region	1
					IHSS	1
					aortic stenosis	1
					rheumatic mitral stenosis	3
(4)	Cardiomyopathy	8	6	bacterial endocarditis	1	
					prosthetic valve	3
(5)	No known clinical heart disease	9	6	cardiomyopathy	6	
					mural thrombus	3
					cardiomyopathy	1
					mitral annulus calcification (MAC)	2
			atrial myxoma	1		

TABLE 3 Activity at Onset and Course

	Total registry (N = 540)	Atherosclerosis (N = 180)	Group 1 (Arterial) (N = 10)	Group 2 (Cardiac) (N = 45)	Group 3 (Uncertain) (N = 72)	All emboli (N = 127)
Activity at onset (%)						
activity of daily living	58	50	10	66	59	60
on awakening	35	44*	70	26	28	31*
exertion	5	3		5	7.5	6
medical procedure	2	3.5	10	3	5	4
Early course (%)						
maximal at onset	59	55*	80	80	82	81*
stepwise	10	15*		7	8	7*
fluctuating	12	18*	10	8	4	7*
progressive	19	12*	10	5	4	5*
Subsequent course (%)						
stable	35	34	20	38	35	35
improved	51.5	49	70	53	54	55
abrupt deterioration	5	8*		3	2	2*
gradual deterioration	9	9	10	7	8	7.5

*Atherosclerosis group differs from embolic group, chi-square test, $dF = 1, p < .05$.

atherosclerosis or patients in the entire registry. The higher percent of positives was true for both anterior and posterior circulations. There were more left than right anterior circulation strokes in all categories but the percentage having CT's and the percentage with positive CT did not differ. There were fewer recognized posterior circulation emboli than atherosclerotic lesions. Shape, size, or density of the CT lesion was not helpful in predicting the source of embolism. Frank hemorrhage, or increased density on an unenhanced CT scan was not noted in any of the patients with cardiac source embolism.

Table 3 considers activity at onset, early course, and subsequent course. The cardiac, arterial and uncertain source groups are quite similar with respect to each of these parameters. Table 4 compares condition on discharge in each subgroup. Embolic patients had a higher incidence of severe deficits or death than either the atherosclerosis patients or patients in the entire registry.

Discussion

Defining and diagnosing cerebral embolism has proven as difficult as capturing an attractive but elusive

butterfly. Purely clinical criteria have emphasized suddenness of onset, previously recognized source, and co-existent systemic embolism⁹ but some emboli do not produce a deficit that is maximal at onset, sources of embolism in the heart and great vessels are frequently not identified prior to the stroke, and co-existent systemic embolism has been rarely recognized in other series of patients with cerebral emboli. Laboratory criteria are also not always definitive since the presence of a possible embolic source (e.g. myocardial infarction with a mural thrombus or atrial fibrillation) does not exclude a coexistent carotid occlusion that could have been the real culprit. On occasion angiography defines the distal embolus, but it is frequently normal. Even pathological criteria are not absolute, for the embolus may have left its source and may have been lysed after embolization to a distal site. At present, the diagnosis is best made by using a combination of clinical, laboratory, and pathologic criteria while excluding other known causes of ischemic stroke (e.g. large vessel occlusive disease, lacunes, primary hemological disorders with clotting diathesis, and venous occlusive disease).

Many questions remain. What is the usual activity at

TABLE 4 Condition on Discharge

	Total registry (N = 540)	Atherosclerosis (N = 180)	Group 1 (Arterial) (N = 10)	Group 2 (Cardiac) (N = 72)	Group 3 (Uncertain) (N = 45)	All emboli (N = 127)
No deficit (%)	17	23*	20	15	13	15*
Slight deficit (%)	36	33	40	28	38	33
Moderate deficit (%)	20	22	20	20	15	18
Severe deficit (%)	12	14	20	16	21	18
Death due to stroke	10	6	0	12	8.5	10
Death other causes (%)	4	2*	0	9	4	7*

*Atherosclerosis group differs from embolic group, chi-square test, $dF = 1, p < .05$.

onset and the clinical course of patients with cerebral emboli? Are there uncommon cardiac diseases which might on occasion cause cerebral embolism? How useful is echocardiography in detecting an unsuspected cardiac source, in verifying a potential source in patients with known heart disease, and as a routine screening test in all patients with stroke, or as a screening test only in patients with relatively sudden onset deficits and no known occlusive extracranial disease? What is the role of CT in the patient with a cerebral embolus? Is systemic embolism a potentially useful diagnostic criterion? What is the prognosis of embolic strokes? Is prognosis different from large vessel occlusive disease? We have reviewed our data to try to answer some of these conundrums.

It is often taught that thrombotic strokes due to in situ occlusion are generally noted on awakening, the thrombus being formed when the circulation is sluggish. Embolism might, in contrast, be precipitated by sudden activity dislodging a fragment from its resting place. In our patient material, most strokes occurred during ordinary daily activities; 44% of thrombotic strokes were noted on awakening as compared to 35% of all registry cases and 31% of embolic cases. This difference between emboli and thrombotic strokes is statistically significant ($p < 0.02$). In the Harvard registry 14% of emboli were discovered on awakening as compared to 20% of thrombotic strokes.⁵ In the series of Wells, which consisted mostly of patients with rheumatic heart disease, 67% of embolic strokes occurred while the patients were sitting quietly or lying in bed and 7 patients awoke with their deficit.¹⁰ Thus, noting the deficit when awakening from a nap or nocturnal sleep only slightly favors a non-embolic mechanism.

A deficit that was maximal at onset occurred in 81% of the embolic strokes as compared to 55% of atherosclerotic strokes and 57% of the total registry strokes. Subsequent fluctuations or changes in deficit occurred with fully 19% of emboli, a figure that is comparable to the 21% figure in the Harvard registry.⁵ Dalal et al.¹¹ have reported 9 examples of emboli seen on initial angiography which later disappeared. Fisher and Perlman later called attention to the "non-sudden embolism" which they explained by the distal passage of embolic material.¹² Others have documented disappearance or movement of emboli.¹³ Furthermore, angiography after 48 hours has a low yield due to disappearance of emboli.⁵ Our study verifies that emboli do not always cause a deficit maximal at onset; 1 in 5 patients with embolism have a fluctuating deficit, probably due to clot movement and initial instability of collateral circulation. The later course of illness did not differ substantially between the thrombotic and embolic groups except that more embolic strokes improved quickly and fewer embolic patients subsequently sustained abrupt deteriorations. The embolism groups did not differ significantly among themselves regarding early or late clinical course.

The most common cardiac disorders associated with embolism were coronary artery disease, (56%), atrial fibrillation (47%), valvular disease, (18%) and cardio-

myopathy (11%), all recognized causes of cerebral embolization. Combinations of these disorders were common. Unexpectedly 7 patients had mitral annulus calcification (MAC) 2 of whom also had coronary artery disease and 2 had co-existent atrial fibrillation. DeBono and Warlow studied 151 consecutive patients with retinal or cerebral ischemia and found MAC in 8 as compared to no instances of MAC in age and sex matched controls without cerebrovascular ischemia.¹⁴ They argued that this epidemiological data supported MAC as an embolic source. In Korn et al.'s original description of MAC, 4 patients had cerebral infarcts, 3 multiple, a fact noted in a table but not commented upon in the body of the paper.¹⁵ The pathology of MAC is consistent with a possible site for embolism. Ulcerated calcific material can occasionally be extruded through a mitral cusp into the left atrial cavity, occasionally a myxoid thrombus can be attached to the calcified ulcerated annulus, and bacterial endocarditis may complicate this condition.¹⁶ Our study also incriminates MAC as a possible source of cerebral embolization. Because MAC correlates with advancing age, hypertension, and atherosclerosis, it is possible that annulus calcification is merely a marker for these other associated disease processes and may not be the true cause of stroke in an individual patient. We also had one patient with idiopathic hypertrophic subaortic stenosis (IHSS); this obstructive cardiomyopathy has been associated with cerebral embolism especially when complicated by atrial fibrillation.¹⁷ On the negative side, there were no patients with mitral valve prolapse (MVP) complicated by cerebral embolism among the 540 registry patients, though we have seen occasional patients with embolism and MVP and do not doubt its embolic potential.

The utility of echocardiography in ischemic stroke is controversial. Larson et al., in an editorial, argued that echocardiography had some false positives and too many false negatives to be a useful screening procedure in unselected stroke populations.¹⁸ Bergerson and Shah found only 5 potential cardiac sources by echo (2 MVP and 3 cardiac thrombi or vegetations) among 184 consecutive patients with acute cerebral events and felt that echocardiography had a low yield in stroke patients.¹⁹ Greenland et al. studied an unselected population of 100 consecutive ischemic strokes;²⁰ although 59 potentially important cardiac embolic sources were detected (11 with MAC), these authors discouraged routine use of echo but urged its use in patients with evidence of heart disease or those under 45 years of age. Lovett, Sandok et al. used echo in a somewhat different group of patients, those in whom extracranial occlusive disease and primary hematological disease had been excluded, or patients suspected of harboring a cardiac source;²¹ forty-one of their patients had an embolic source documented, and 9 had mural thrombi. Donaldson et al. found echo useful in patients with heart disease as 7 of 32 such patients had a cardiac "mass" demonstrated echocardiographically which might serve as an embolic source. Only 1 of 30 patients without known heart disease had a potential source

(MVP) for embolism.²² In our study, the echocardiogram complemented other clinical techniques in clarifying the nature of the heart disease. Eight of our patients with unsuspected cardiac disease had a potential embolic source detected only by echo — 3 MAC, 3 mural thrombi (1 with MAC) 1 cardiomyopathy, 1 myxoma, and 1 IHSS. Our cases were a select group of stroke patients; patients with lacunes or known extracranial vascular disease were not included. When proper technique and procedures are used,²³ echocardiography can be a useful adjunct to the clinical exam.

The incidence of thrombus within the heart in coronary disease and atrial fibrillation, the two commonest disorders associated with embolism, is high. Garvin²⁴ found 81 examples of left mural thrombi among 133 autopsied patients dying of myocardial infarction. In a larger series of 327 patients with acute or healed myocardial infarction, 108 had left ventricular thrombi, 9 right ventricular thrombi, and 31 thrombi within the atrial appendages.²⁵ In patients with atrial fibrillation, more thrombi were found in the atria than the ventricles even in the presence of coronary disease.²⁶ It should be emphasized that tiny emboli, the size of a shotgun pellet, have the potential to block the middle cerebral artery producing a catastrophic deficit.²⁷ Unfortunately echocardiography has limitations in ability to detect smaller lesions but future technological advances may diminish this limitation. We agree that the routine use of echo in all ischemic strokes is not presently warranted. We would favor echocardiography in patients with cardiac symptoms or known heart disease especially atrial fibrillation and coronary artery disease and in patients in whom lacunar disease and occlusive vascular disease are clinically unlikely. In patients with multiple TIAs in the same vascular territory, and in those with documented appropriately situated extracranial vascular disease and no known cardiac disease, echocardiography is likely to have a very low yield.

In our series, the most frequent site of embolic occlusion was the left anterior circulation especially the left middle cerebral artery. In McDowell's series right hemiparesis was also slightly more common.²⁸ Gacs and colleagues have shown by embolizing small balloons into the cerebral circulation, that the destination of the balloons does depend on rheological factors.²⁹ The middle cerebral artery stem, the major upper division branches, and the angular artery predictably and repeatedly received the majority of balloons.²⁹ Anterior cerebral artery embolization was rare. Posterior circulation emboli represented only 15% of our series, at least partially reflecting the percentage of brain supplied by the vertebrobasilar system. As in prior series, the posterior cerebral artery was the commonest posterior circulation vessel affected.

Though CT is now widely used, little data is available concerning its utility in ischemic stroke. In our series, 124 of 127 embolism patients (98%) had CT of which 87 (71%) were positive; 75 patients had 1 lesion, 11 had 2 lesions, and 2 patients had 3 lesions. Four patients had a clinically unsuspected lesion detected by CT in an uninvolved vascular territory. CT

was equally useful in the 3 anatomical regions (right and left anterior circulation and posterior circulation) and in the three subgroups of embolic stroke, (arterial, cardiac, or uncertain source).

Systemic embolism, an important finding in older criteria for the diagnosis of cerebral embolism, was rarely recognized (2.3%) in our series. This low figure is similar to the 2% figure in the Harvard registry.⁵ Systemic embolism is more commonly documented at necropsy. In one necropsy series of patients with acute MI, 27 had renal, 19 brain, 17 splenic, and 13 extremity emboli.³⁰ In all, only 23 of 87 organs involved by embolism caused recognized clinical symptoms. Fairfax *et al.* studied 100 patients with chronic sino-atrial disorders and found 16 examples of embolism which included 19 cerebral events and 7 systemic events.³¹ Because the brain is divided into a myriad of uniquely functioning areas, sudden interruption of its blood supply is likely to disrupt function at least temporarily. In systemic vessels, limb emboli, if small, might cause a cramp or temporary coldness of the limb; renal, splenic, or other visceral emboli might cause transient abdominal or flank pain or diarrhea, symptoms usually not considered as important clues to serious disease. Although systemic embolism occurs often and is frequently documented at necropsy, it is difficult to recognize clinically. If one waits for an obvious systemic embolism to confirm a cerebral event as embolic, the diagnosis will be missed in most patients.

In our series, the prognosis for recovery from embolism was not as good as for stroke due to atherosclerosis or for strokes in the registry as a whole. Eighteen percent of embolism patients had a severe deficit at discharge compared to 14% of thrombotic mechanism patients and 12% of patients in the entire registry. Death occurred in 17% of the embolism patients (10% thought directly due to their stroke and 7% of other causes, most often cardiac). Fewer embolism patients left the hospital free of any deficit. Wells¹⁰ and Carter³² commented on the unfavorable prognosis of cerebral embolic disease, and McDowell emphasized the importance of the underlying disease in determining prognosis.²⁸ Cerebral embolism is a very serious disorder with frequent residual disability, common recurrence, and a high risk of death from stroke or heart disease.

Necropsy studies have documented a high incidence of cerebral infarcts in patients with heart disease^{24, 26, 30, 33, 34} even when rigid criteria for the morphological diagnosis of embolism, such as identification of the embolic material grossly or microscopically,³⁴ are used. The clinical diagnosis of embolization has lagged behind post-mortem diagnosis and will remain elusive because of the small size of an embolic fragment necessary to produce a devastating clinical deficit.^{27, 28} This study has corroborated the high incidence of emboli found in the Harvard registry, and has emphasized the utility of CT and echo in the diagnosis of this disorder. Patients with emboli due to cardiac, intra-arterial, and uncertain sources showed little clinical differences in demographic features,

course, or CT findings. Systemic embolism was unusual and prognosis was often poor. Hopefully newer diagnostic tools such as digital subtraction angiography and cardiac scintillation scanning³⁵ might improve our diagnostic capabilities and lead to more accurate recognition of this serious disease.

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