Brain (1991), **114**, 51–70

THE STROKE SYNDROME OF STRIATOCAPSULAR INFARCTION

by GEOFFREY A. DONNAN,[1] PETER F. BLADIN,[1] SAMUEL F. BERKOVIC,[1] WENDY A. LONGLEY[2] *and* MICHAEL M. SALING[2]

(From the [1]Department of Neurology, Austin Hospital, the [2]Department of Psychology, University of Melbourne, Parkville, and the [2]Department of Neuropsychology, University of Melbourne, Austin Hospital, Melbourne, Australia)

SUMMARY

Striatocapsular infarction has recently been described as a distinct stroke entity and forms an important subgroup of subcortical infarctions. In a prospective study of 50 consecutive patients over a 10 yr period with this syndrome, clinical and neuropsychological features, pathogenesis and outcome were studied to provide information concerning management and prognosis. The most common clinical presentation was that of a stroke affecting mainly the upper limb with cortical signs such as dysphasia, neglect or dyspraxia. Evidence from EEG, angiographic and neuropsychological data supported a vascular/haemodynamic basis for the presence of the acute neuropsychological changes, while the chronic changes were more likely to be due to diaschisis. A study of risk factors and cerebral angiography enabled 4 pathophysiological subgroups to be identified: (1) cardiac emboli to the origin of the middle cerebral artery; (2) severe extracranial carotid artery occlusive disease with presumed embolism to the same site and/or involvement of haemodynamic factors; (3) proximal middle cerebral artery abnormalities causing occlusion of multiple lateral striate arteries at their origins; (4) normal angiography where pathogenesis was uncertain. The risk factors of cardiac disease and smoking were significantly increased as compared with age and sex-matched controls with other forms of ischaemic stroke. Stroke or vascular death rate was 2.7% per yr during a mean follow-up period of 2.25 yrs. Predictors of an excellent recovery with return to normal lifestyle were younger age, only brachial or brachiofacial weakness with absence of cortical signs at presentation and minimal change on angiography. This stroke entity deserves particular recognition in the spectrum of subcortical infarctions because of its specific pathogenesis, distinct neuropsychological features and reasonable prognosis.

INTRODUCTION

Striatocapsular infarction involves the territory of the lateral striate branches of the middle cerebral artery with sparing of the overlying cortex (Bladin and Berkovic, 1984). The clinical syndrome is usually one of a hemiparesis, affecting mainly the upper limb, in association with cortical abnormalities such as aphasia, neglect and/or apraxia. The area of infarction seen on axial CT slices is typically comma shaped, with the rostral aspect in the head of the caudate and anterior limb of the internal capsule and the tail in the putamen (fig. 1). The dimension at the point of maximal diameter is usually at least 3 cm. Lacunes may also occur in the territory of the lateral striate arteries but they are restricted to the territory of a single penetrating vessel, are therefore of smaller size (usually less than 1.5 cm diameter) and in this region usually do not produce symptoms.

Correspondence to: Dr G. A. Donnan, Department of Neurology, Austin Hospital, Melbourne, Australia 3084.

Recognition of striatocapsular infarction is important, since it differs from lacunar infarction (Donnan *et al.*, 1982; Fisher, 1982; Mohr, 1982) by virtue of involvement of large extracranial and intracranial vessels in the mechanisms of infarction. The development of infarction in the cases demonstrated angiographically depends either on complete proximal occlusion of the middle cerebral artery with adequate colateral flow to the overlying cortex via transcortical and transdural anastomoses (Vander Eecken and Adams, 1953), thus restricting the area of infarction to the deep subcortex or, alternatively, during partial embolic occlusion of the middle cerebral artery origin, the lateral striate artery origins may be blocked but allow adequate flow past the occlusion (Bladin and Berkovic, 1984).

We have now studied prospectively 50 consecutive patients with striatocapsular infarction over a 10 yr period and present here a complete account of the clinical and radiological features, neuropsychological characteristics, associated risk factors and long-term prognosis of this important stroke syndrome. By studying angiographic findings and risk factors, 4 pathophysiological subtypes have been identified.

PATIENTS AND METHODS

Patients were identified prospectively as a part of an on-going stroke subtype categorization in the Austin Hospital Stroke Unit (Chambers *et al.*, 1983). During the period of study, from July 1977 to November 1988, 3630 patients entered the Stroke Unit and were evaluated accordingly. From these, 50 patients were selected on the basis of the recent onset of an acute focal neurological deficit which related to the finding of an area of hypodensity in the striatocapsular region consistent with recent infarction. The area of infarction had to include at least two elements of the striatocapsular area: head of caudate plus internal capsule or putamen plus internal capsule. Patients with small infarcts of 1.5 cm diameter or less consistent with lacunar infarction were excluded (Bladin and Berkovic, 1984).

All patients were examined neurologically by one of us (G.A.D., P.F.B., S.F.B.) where particular care was paid to the bedside examination of cortical function. A minimum assessment included tests for neglect (visual, sensory and motor) dyspraxia (motor, ideational, dressing), speech (spontaneous, reception, expression, repetition, reading, writing), frontal perseverative tasks and graphic tests of construction. Approximately 6 months after admission a neuropsychological examination was performed on 12 consecutive patients who had exhibited cortical signs in the acute phase. The purpose of the chronic phase study was to attempt to document any residual subtle neuropsychological deficits (Skyhøj Olsen *et al.*, 1986; Vallar *et al.*, 1988) and to characterize these further. This aspect of the study may shed further light on the underlying mechanism of genesis of the acute neuropsychological changes observed (*see* Discussion for fuller review).

CT scans were performed on admission (noncontrast) and 7–10 days later (contrast enhanced). EEG was performed in the majority of patients, usually shortly after admission. In cases of identified striatocapsular infarction this was repeated 2 wks later if the first EEG was abnormal. Cerebral angiography was only performed on those patients in whom a reasonable clinical recovery had occurred in the period shortly after the stroke (from 1 to 4 wks) and in whom the pathophysiological mechanism of infarction was considered likely to relate to the extacranial vasculature by virtue of the presence of cervical bruits or other clinical indicators such as preceding transient ischaemic attacks or amaurosis fugax.

After discharge all patients were followed up in the Stroke Unit Outpatient Clinic at regular intervals. This was continued during the study period, except for 3 patients in whom telephone contact was necessary because of geographic isolation. The following categories of final outcome were used: (1) resumption of normal lifestyle and/or employment; (2) unable to resume normal activities, but managing at home (functional recovery); (3) total dependence with nursing care required (no functional recovery).

In order to determine whether patients with striatocapsular infarction had a markedly different risk factor profile than other forms of ischaemic stroke, 2 control patients for each case of striatocapsular infarction were selected who were the next sequential admissions to the Stroke Unit of the same sex and age (±5 yrs).

These had a clear diagnosis of ischaemic stroke. For smoking as a risk factor, a similar comparison was made with the number of current smokers in patients with ischaemic stroke entering the Stroke Unit from 1985 to 1986. This was done because patients before 1985 were not questioned closely concerning their smoking habits, unlike the patients with striatocapsular infarction who were questioned specifically as a part of the current study.

Statistical analysis were performed using the χ^2 test and, where appropriate, applying Yates' correction for continuity (Yates, 1934). For the case control section of the study, odds ratio with 95% confidence intervals were calculated. For other nonparametric distributions, the Mann-Whitney test was used.

RESULTS

Of the 50 patients with an acute stroke due to striatocapsular infarction, there were 27 males and 23 females. This represented 1.4% of all Stroke Unit admissions during the period 1977–1988. Mean age was 63 (range 24–88) yrs.

Clinical features

The most common presentation, observed in 31 patients (62%), was that of a single event producing a hemiparesis and hemisensory loss with accompanying cortical features (Table 1). Ten patients had preceding transient ischaemic attacks in the ipsilateral carotid

TABLE 1. PRESENTING CLINICAL FEATURES IN 50 PATIENTS WITH STRIATOCAPSULAR INFARCTION

Presenting clinical features	*No. of patients*
Single event	31
Preceding TIAs (ipsilateral)	
Hemisphere	5
Capsular warning syndrome	2
Amaurosis fugax	3
Stuttering progression	6
Gradual progression	1
Postcardiac surgery	1
Collapse and loss of consciousness	1
Total	50

artery territory. Two of these had repetitive bursts of hemiplegia before the stroke within a brief period (1 had 5 events within 3 h and the other 10 events over 2 days) similar to the 'capsular warning syndrome' seen before lacunar stroke (Donnan *et al.*, 1982; Donnan and Bladin, 1987). One patient was standing at the race-course when he suddenly fell to the ground unconscious and on recovery several minutes later had right arm weakness and slurred speech. Another patient was observed to have a gradual and smooth progression of right face, arm and leg weakness to complete hemiplegia over 24 h, while 6 others had a stuttering progression over approximately the same period. The patient who developed striatocapsular infarction associated with cardiac surgery awoke from the anaesthetic with a dense right hemiplegia and aphasia.

The most common pattern of weakness was mainly upper limb (21 patients, 42%), then face, arm and leg equally (13 patients, 26%) followed by a variety of other patterns, including mainly facial weakness (14 patients, 28%), dysarthria only (1 patient), and

arm clumsiness only (1 patient); 30 patients (60%) had sensory, as well as motor involvement, while the remaining 19 (38%) had only motor signs and 1 dysarthria only. Cortical signs, such as dysphasia, dyspraxia, motor or sensory neglect were present in 35 patients (70%). Acute ipsilateral eye deviation was seen in 4 patients.

CT characteristics

In general, the topography of the striatocapsular infarcts were similar to those that we described previously (Bladin and Berkovic, 1984) (fig. 1), although several were

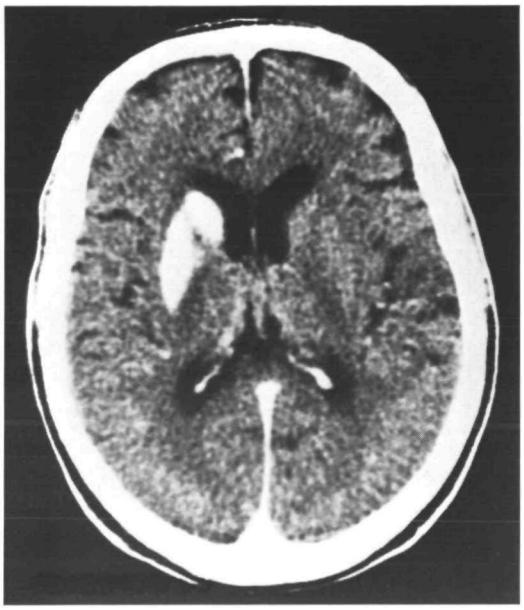

FIG. 1. Typical striatocapsular infarction showing involvement of head of caudate, anterior limb of internal capsule and putamen (contrast enhanced scan with right striatocapsular infarction in 63-yr-old male).

larger, particularly in the paraventricular region, and reached dimensions of width 3 cm, depth 5 cm and length 4.5 cm. Several scans showed incomplete involvement of the striatocapsular region, e.g. involvement predominantly of the head of caudate (figs 2A, 3A) or putamen and capsule (fig. 4A). Surrounding oedema may have contributed greatly to the hypodensity in the case shown in fig. 2A, since a later scan was quite normal despite the persistence of subtle motor signs in the right upper limb. Scans repeated in other cases showed collapse of the infarct to a slit-like structure indistinguishable from old subcortical haemorrhage (fig. 5A, B).

While the very large infarcts always produced a dense hemiplegia at onset and the few examples of incomplete striatocapsular involvement produced an incomplete syndrome (e.g., arm clumsiness only, in the case shown in fig. 2A), the topography of infarction was a poor predictor of the pattern of weakness at presentation.

Risk factors

A higher proportion of patients had cardiac disease as compared with the control patients with other forms of cerebral ischaemia (Table 2): 25 of the 50 patients with striatocapsular

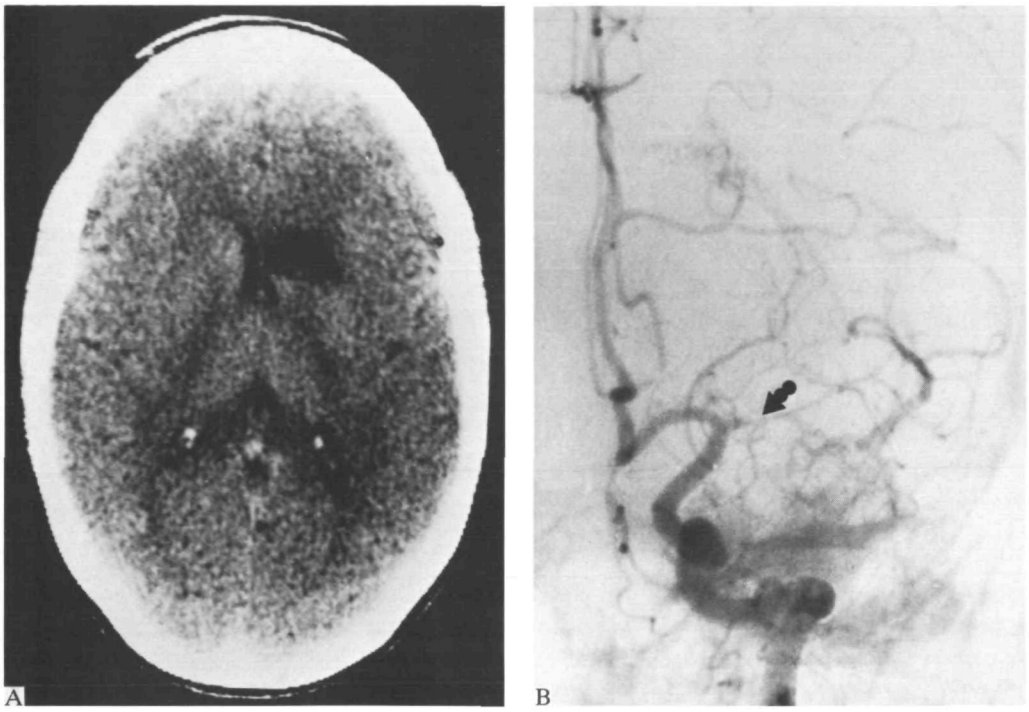

FIG. 2. A, partial left striatocapsular infarction in a 24-yr-old woman. B, angiogram showing middle cerebral arterial changes consistent with dissection or arteritis (arrow).

infarction (50%) had either atrial fibrillation (15 patients), cardiomyopathy (1 patient), ischaemic heart disease (14 patients) or a combination of these compared with 16 of 100 control patients (16%) (Odds Ratio (OR) 5.25, 95% Confidence Intervals (CI) 2.43−11.3, χ^2 19.4 for 1 df, $P < 0.001$). Five of the patients with striatocapsular infarction had had recent coronary artery bypass grafts and 46% were current smokers compared with 27% of controls (OR 2.30, CI 1.13−4.69, $\chi^2 = 5.4$ for 1 df, $P < 0.05$). The risk factors of hypertension (OR 0.70, CI 0.35−1.38), peripheral vascular disease (OR 0.85, CI 0.21−3.43), diabetes (OR 0.73, CI 0.19−2.90) and ipsilateral cervical bruit (OR 0.78, CI 0.23−2.63) did not differ between the two groups.

Cerebral angiography and relationship to cardiac risk factors

Twenty-seven patients (54%) underwent cerebral angiography (Table 3). Twelve patients (24%) had either complete internal carotid occlusion (fig. 4B) or greater than 95% stenosis ipsilaterally (fig. 6B). All 6 of the latter went on to have a carotid endarterectomy. Three patients had significant disease of the ipsilateral middle cerebral arteries in the absence of arterial change elsewhere. One had middle cerebral artery stenosis at the level of the lateral striate arteries and another had an aneurysm just beyond these arteries. The third patient was a 24-yr-old woman with arterial change in the proximal middle cerebral artery consistent with either arteritis and/or arterial dissection

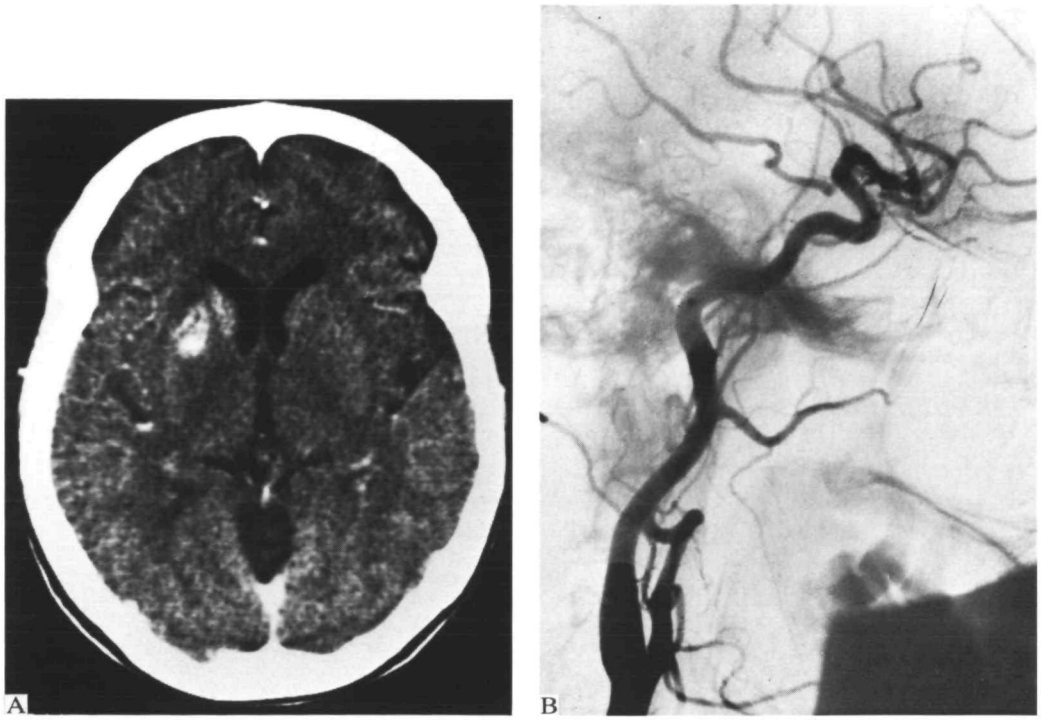

FIG. 3. Right striatocapsular infarction in a 67-yr-old male. A, contrast enhanced scan showing that the infarct mainly involved the caudate and anterior limb of the internal capsule. B, angiography was normal.

(fig. 2B). Twelve patients had normal angiograms or minimal change (fig. 3B). The timing of angiography in this group was not different from the remainder, i.e., ranging from 10 to 25 days poststroke. In the 23 patients in whom angiography was not performed, 20 had significant cardiac risk factors: 11 had atrial fibrillation, 4 had atrial fibrillation associated with ischaemic heart disease, 4 had ischaemic heart disease alone and 1 had cardiomyopathy. Of the remainder, only 6 of 27 patients had cardiac disease. In the 12 patients in whom angiography showed no change or minimal change, 4 (33%) had cardiac disease compared with only 2 of 15 in the group with severe carotid occlusive disease.

Cortical signs and EEG: relationship to angiography

Of the 50 patients 35 (70%) had cortical signs at presentation (*see* Methods). None of these had CT evidence of cortical infarction. An EEG was performed on 41 patients and focal ipsilateral slow waves were seen in 27 (66%). In general, EEG changes tended to parallel focal cortical signs in the ipsilateral hemisphere ($\chi^2 = 12.7$; $P < 0.001$) (Table 4), although it was of interest that 5 of 15 patients with no cortical signs at bedside examination had abnormal EEGs over the infarcted hemisphere, suggesting that the EEG

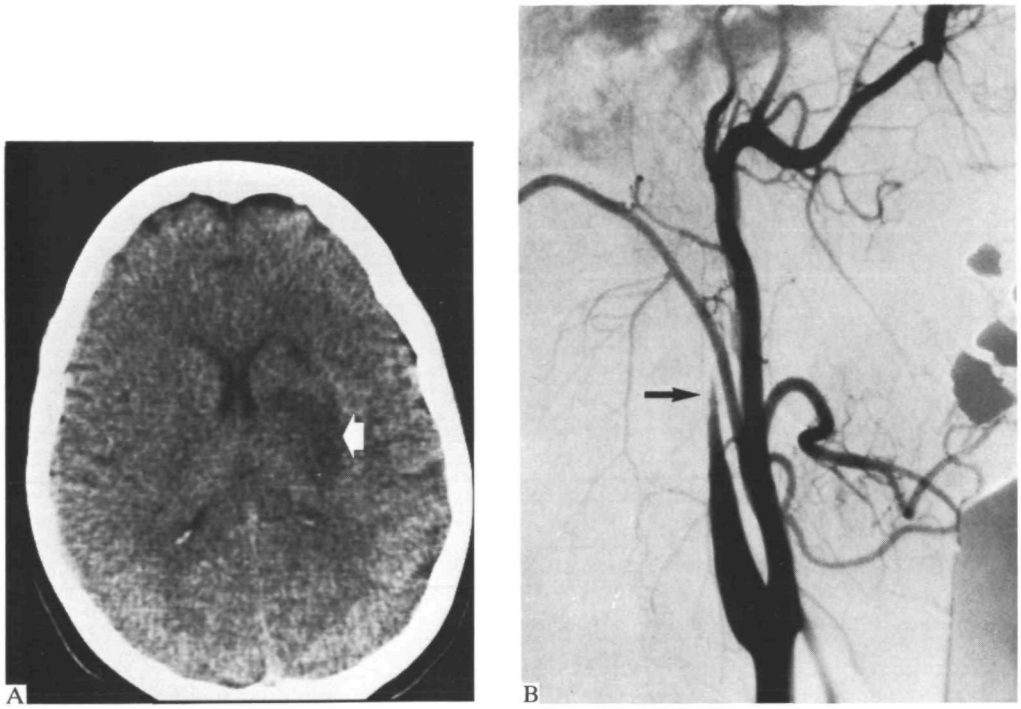

FIG. 4. A recent left striatocapsular infarction in a 35-yr-old woman. A, the infarct involved mainly the putamen (arrow) and anterior limb of the internal capsule. B, angiogram showing complete occlusion of the left internal carotid artery (arrow).

may be sometimes more sensitive than the bedside examination in detecting generalized hemisphere cortical dysfunction.

Of the 27 patients who had angiography, patients with complete internal carotid occlusion or greater than 95% stenosis were more likely to have cortical signs that those with no major extracranial carotid occlusive disease ($\chi^2 = 13.7$, $P < 0.001$) (Table 5). This was not the case with EEG changes, where focal slowing was seen as often ipsilateral to severe as to minimal carotid occlusive disease.

Neuropsychological changes

Acute. The distribution of cortical signs seen during the acute phase is shown in Table 6. Cortical signs were generally consistent with the laterality of the infarction. Examples of bedside testing included dysgraphia in a patient with left hemisphere infarction (fig. 7) and left neglect on clock construction in a patient with right hemisphere infarction (fig. 8). Frontal perseverative signs were sometimes present (fig. 9), presumably due to interruption of frontal connections in the anterior limb of the internal capsule. Of interest is the presence of right neglect without language signs in 2 of the left hemisphere patients.

Chronic. The gross language and visuospatial features seen in the acute phase had resolved. However, some degree of neuropyschological dysfunction was evident in the

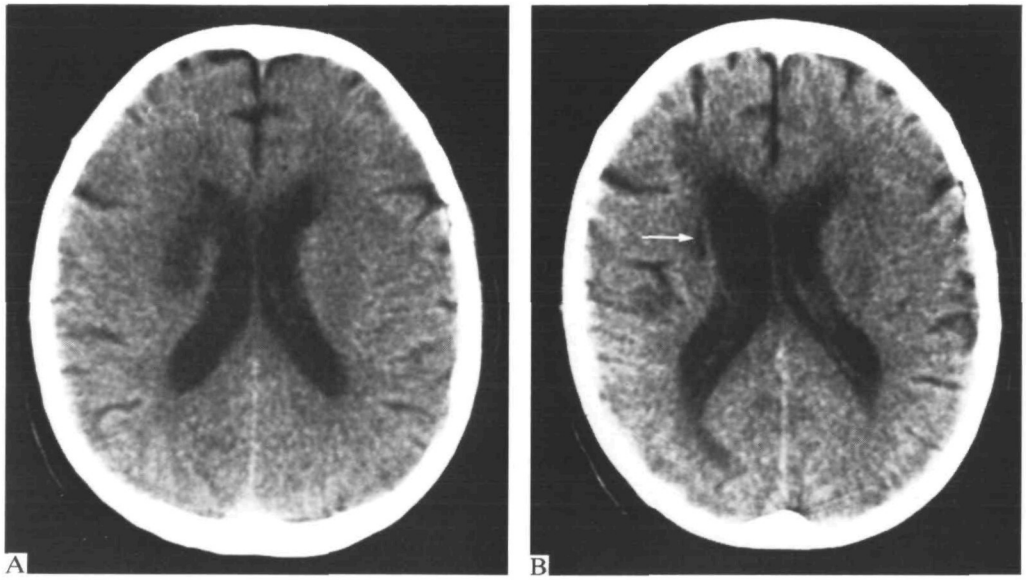

FIG. 5. A, unenhanced CT scan showing upper portion of a right striatocapsular infarct in the paraventricular region several days after the onset of left hemiparesis. B, repeat scan 3 months later showing collapse of the infarct to a slit-like structure (arrow).

TABLE 2. RISK FACTOR COMPARISON BETWEEN STRIATOCAPSULAR PATIENTS AND AGE AND SEX-MATCHED CONTROLS WITH OTHER FORMS OF CEREBRAL ISCHAEMIA

Risk factor	*Patients with striatocapsular infarction** (*n* = 50)	*Control patients with other forms of cerebral infarction*** (*n* = 100)
Cardiac disease		
Atrial fibrillation	15	11
Ischaemic heart disease	9	5
Cardiomyopathy	1	0
Total	25 (50%)	16 (16%)[a]
Hypertension	25 (50%)	59 (59%)
Current smokers	23 (46%)	27 (27%)[b]
Diabetes	3 (6%)	8 (8%)
Peripheral vascular disease	3 (6%)	7 (7%)
Cervical bruits (ipsilateral)	4 (8%)	10 (10%)

* Mean age 64 ± 13 (SD) yrs, range 24–88 yrs. ** Mean age 64 ± 13 yrs, range 20–84 yrs. [a] $P < 0.001$; [b] $P < 0.05$. Types of cerebral infarction in the control group were as follows: cortical or cortical plus subcortical 60, lacunar 25, hindbrain 11, watershed 4.

chronic phase (Table 7, Appendix). First, both patient groups tended to achieve low scores on a coding task (Digit Symbol Substitution), which in the case of the right hemisphere group differed significantly from the control group. This task is particularly sensitive to cerebral impairment, being dependent on response speed, sustained attention

TABLE 3. RELATIONSHIP BETWEEN CEREBRAL ANGIOGRAPHY AND THE PRESENCE OF CARDIAC DISEASE IN THE 50 PATIENTS WITH STRIATOCAPSULAR INFARCTION

Angiography	*n*	*No. of patients with cardiac disease*
Ipsilateral internal carotid artery		
Normal or Minimal change	12	3
Occlusion	6	1
90% + stenosis	6	1
Middle cerebral artery		
Aneurysm	1	0
Stenosis (50%)	1	0
Dissection/arteritis	1	0
Total	27	5
Not done	23	20
Total	50	25

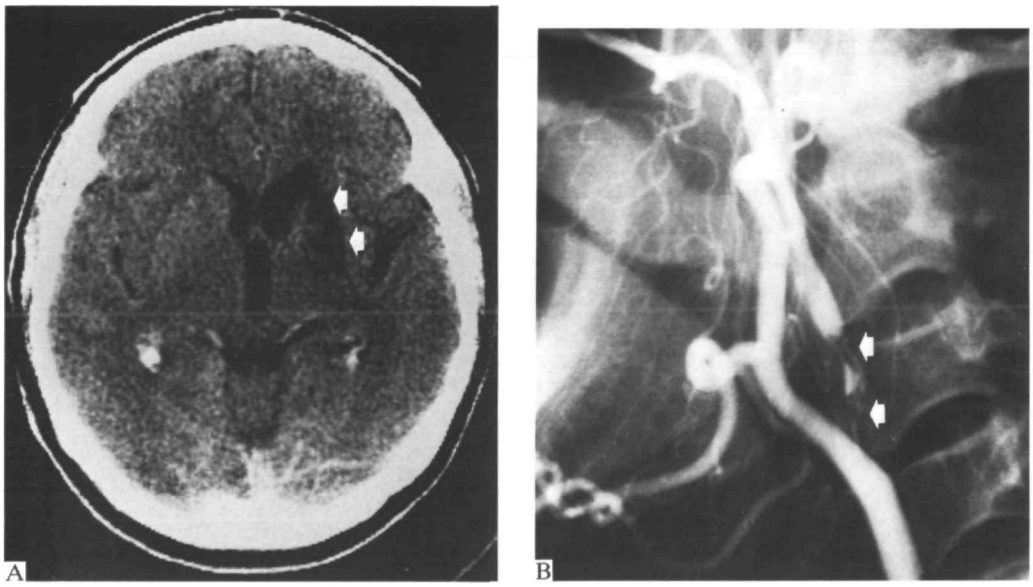

FIG. 6. A, unenhanced CT scan showing left striatocapsular infarction (arrows). B, angiogram showing ipsilateral high grade internal carotid artery stenosis (arrows).

and persistence; it is of no lateralizing significance, and is *relatively* independent of intellectual factors or learning (Lezak, 1983).

Secondly, the patients with left hemisphere infarctions had difficulty with the oral production of words starting with a designated letter, as measured by the Controlled Oral Word Association Test. This word-finding task is regarded as a sensitive indicator of 'higher level' language dysfunction in clinically nonaphasic patients, particularly if there is left or bilateral prefrontal involvement (*see* Walsh, 1987, for review). By

FIG. 7. Dysgraphia shown by a 57-yr-old woman with left striatocapsular infarction. She was right-handed and used her nondominant hand (left). The sentence was to read 'Pack my box with twelve dozen jugs of liquid veneer'.

TABLE 4. RELATIONSHIP BETWEEN CORTICAL SIGNS AND EEG IN 50 PATIENTS WITH STRIATOCAPSULAR INFARCTION

	Cortical signs	*No cortical signs*
Focal EEG changes ipsilaterally	23	4
EEG normal	2	9
Bilateral EEG changes	2	1
EEG not done	8	1
Total	35	15

TABLE 5. RELATIONSHIP BETWEEN ANGIOGRAPHY PERFORMED IN 27 PATIENTS AND EEG/CORTICAL SIGNS

	Angiography	
	Carotid occlusion or high grade stenosis ipsilaterally (no. of patients)	*No haemodynamically significant change (no. of patients)*
Cortical signs clinically		
Focal hemisphere abnormalities	15	3
Normal	0	9
Total	15	12
EEG findings		
Focal ipsilateral showing	9	8
Normal	2	6
Generalized slowing	1	1
Total	12	15

TABLE 6. DISTRIBUTION OF CORTICAL SIGNS IN THE ACUTE PHASE*

Left-sided lesion	*n*	*Right-sided lesion*	*n*
Aphasia only	13	Neglect only	8
Aphasia + ideomotor apraxia	1	Neglect + anosognosia	2
Aphasia + Gerstmann's syndrome	1	Neglect + constructional apraxia	2
Aphasia + right neglect	3	Neglect + anosognosia + Constructional apraxia	2
Right neglect only	2		

* n = 34; details of the cortical findings in 1 patient were not available.

constrast, the right hemisphere patients were impaired on reasonably complex block construction task (Block Design), suggesting the presence of residual visuospatial difficulties in this group.

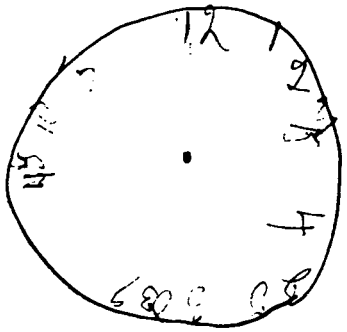

FIG. 8. Left neglect shown in clock drawing by a 65-yr-old man with right striatocapsular infarction.

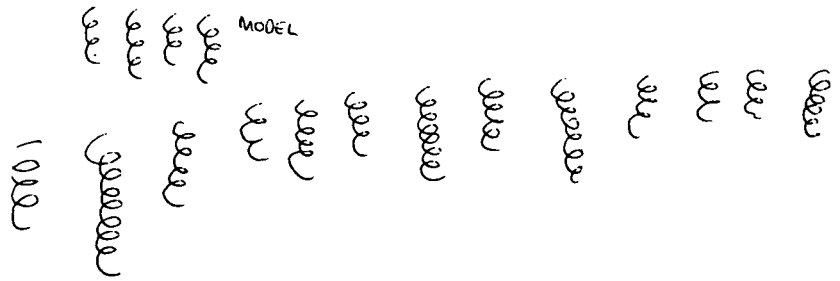

FIG. 9. Perseveration shown in the number of springs and number of coils within each spring copied from the model (*top row*). This 35-yr-old woman had a left striatocapsular infarction. (CT scan and angiography shown in fig. 4.)

Thirdly, the chronic phase was characterized by material-specific impairment in new learning in both patient groups. Patients with left hemisphere infarctions were able to learn a list of 15 words (Rey Auditory Verbal Learning Test (RAVLT)) almost as well as the control subjects or right hemisphere patients; however, their spontaneous recall of the list was markedly reduced after an interpolated interference condition (trial B of the RAVLT; *see* Table 7). They also exhibited subtle problems in recalling prose passages (Logical Memory) when compared statistically with the other two groups. Patients with right hemisphere infarctions showed impaired learning of visuospatial information, exemplified by their failure to learn the Milner Pathway of the Austin Maze; an examination of the error date in Table 7 reveals that, unlike the controls and left hemisphere patients, the patients with right hemisphere lesions maintained high error rates over 10 successive learning trials. Right hemisphere patients also differed from controls in terms of their ability to reproduce complex visuospatial information from memory (Rey's Complex Figure). This latter finding should be regarded as suggestive only, since the performance of the left hemisphere groups was approximately intermediate between that of the right hemisphere and control subjects.

Outcome

During a follow-up period of 1 month to 8 yrs (mean 2.25 yrs), 11 patients had further progression of their deficit in hospital. Two patients were lost to follow-up after being

TABLE 7. NEUROPSYCHOLOGICAL TEST PERFORMANCES OF CONTROL SUBJECTS AND PATIENTS WITH LEFT OR RIGHT-SIDED STRIATOCAPSULAR INFARCTIONS

	Controls (*n* = 5) (*mean ± SD*)	*Left infarcts* (*n* = 6) (*mean ± SD*)	*Right infarcts* (*n* = 6) (*mean ± SD*)
WAIS-R (scaled scores)			
Picture completion	9.4 (2.4)	9.3 (3.2)	9.4 (2.4)
Digit symbol substitution	9.4 (2.2)	6.8 (2.9)	7.0 (1.1)[a]
Block Design	9.4 (3.2)	10.6 (1.9)	7.3 (3.5)[c]
WMS			
Logical memory	14.6 (3.4)	11.4 (3.2)[b]	14.8 (2.5)
Rey Auditory Verbal Learning Test (Correct responses out of a total of 15)			
Trial 1	4.6 (1.7)	5.4 (2.7)	4.6 (1.5)
2	6.6 (1.7)	6.0 (3.0)	7.8 (2.2)
3	9.0 (1.0)	8.4 (3.5)	8.6 (3.1)
4	10.4 (1.1)	8.2 (3.5)	10.2 (1.8)
5	11.0 (2.6)	8.4 (3.1)	10.8 (1.8)
B	5.0 (1.4)	4.6 (1.5)	6.2 (4.6)
6	8.8 (3.5)	4.0 (2.9)[b]	10.0 (1.2)
Recognition	13.6 (1.1)	11.8 (2.6)	13.8 (1.5)
Rey's Complex Figure (Units correct out of a total of 36)			
Copy	32.2 (4.7)	33.7 (3.8)	29.8 (4.2)
Recall at 5 min	18.6 (6.7)	13.3 (2.9)	9.5 (6.5)[a]
Benton Controlled Oral Word Association Test (percentiles)	66.8 (29.8)	14.8 (26.1)[a]	43.6 (19.1)
Austin Maze (Milner Pathway) (errors)			
Trial 1	17.6 (5.0)	16.6 (5.0)	20.6 (6.2)
2	11.8 (2.2)	14.8 (11.1)	19.2 (6.2)
3	13.4 (9.5)	10.6 (4.6)	21.6 (7.8)
4	9.8 (5.3)	10.6 (9.2)	19.2 (6.6)[a]
5	9.0 (6.9)	12.2 (11.9)	17.4 (7.5)
6	7.8 (8.9)	8.4 (6.6)	16.6 (7.1)
7	8.8 (6.2)	7.0 (4.3)	16.8 (7.3)[b]
8	5.4 (4.2)	6.0 (4.3)	18.0 (7.1)[b]
9	6.8 (8.0)	6.2 (5.8)	19.0 (6.7)[b]
10	3.6 (4.0)	6.6 (6.8)	15.0 (6.9)[a]

[a] Differs from control group only (5% level, Mann-Whitney test). [b] Differs from remaining two groups (5% level, Mann-Whitney test). [c] Differs from remaining patient group (5% level Mann-Whitney test).

transferred back to their countries of origin. One patient had a brief TIA affecting the same side of the body as the previous stroke 2 yrs earlier. Another patient, who was in chronic atrial fibrillation, developed a striatocapsular infarct on the opposite side 5 yrs later. Eight patients died, 5 from cardiac conditions, 1 from renal failure and 2 from uncertain causes. Stroke or vascular death rate for the follow-up period was therefore approximately 2.7% per yr.

Overall, the majority of patients either had functional recovery (23 patients) or a more complete recovery with return to work or normal lifestyle (15 patients). Twelve patients had no functional recovery. Although numbers were low for an angiographic correlation with outcome, a suggestion that minimal change or normal angiography was associated

with a better outcome was seen, since 13 of 15 such patients had a functional recovery or return to work. Four of the 6 patients with carotid occlusion made a functional recovery, but none was able to return to work or its domestic equivalent.

Patients who were able to return to work to resume normal lifestyle were significantly younger (56.9 ± yrs) compared with those with no functional recovery (69.9 ± yrs) (two-tailed Mann-Whitney test, $U = 37$, $P < 0.02$) and fewer had cortical signs at presentation (8/15 vs 2/12, $\chi^2 = 3.8$, $P < 0.05$). Topography of infarction at the onset of stroke was not a good predictor of outcome excepting for the few cases with only partial striatocapsular infarction (all returned to work) or massive infarction (3 cases, all with no functional recovery). The pattern of motor weakness at onset did influence outcome, since 10 of the 12 with no functional recovery had hemiplegia equally affecting face, arm and leg while this pattern was present in only 3 of the 15 who returned to normal lifestyle ($\chi^2 = 8.3$, $P < 0.01$). Brachiofacial weakness or mainly arm weakness was the commonest pattern in the latter group. The presence of cardiac or other risk factors had no impact on outcome.

DISCUSSION

The current study expands on our previous findings concerning the clinical features and presentation of patients with striatocapsular infarction (Bladin and Berkovic, 1984). The most common presentation is that of a single episode of the sudden onset of a hemiparesis affecting the upper limb more than lower limb or face, with accompanying sensory and cortical signs. The neuropsychological features frequently include language disturbances or neglect attributable to the affected hemisphere, together with difficulties with new learning tasks, which may persist for a greater period than has previously been appreciated. Less frequently a pure motor hemiparesis with minimal cortical signs may be seen and rarely, subtle change such as dysarthria alone or merely upper limb clumsiness. An examination of risk factors associated with striatocapsular infarction in this study shows that this form of stroke is associated with cardiac disease more than all other forms of ischaemic stroke combined. Fifty per cent of patients had either atrial fibrillation, cardiomyopathy, ischaemic heart disease or a combination of these, compared with 24% of controls. Five patients had had recent coronary artery bypass grafts. Smoking was the only other significant risk factor greater than other forms of ischaemic stroke, perhaps because of its strong association with cardiac disease (Doll and Peto, 1976).

By taking into account the cardiac risk factors described and the angiographic findings, 43 cases of striatocapsular infarction studied could be divided into four pathophysiological subgroups. Seven patients had less adequate investigation and could therefore not be classified.

Groups 1. Presumed cardiac source of embolus (17 patients)

All patients in this group had relatively severe cardiac disease which may have acted as a potent source of embolism. This was recognized by Santamaria *et al.* (1983) who reported 8 patients with deep subcortical infarction secondary to cardiac embolism and in our population was certainly the most common mechanism of development of striatocapsular infarction. Fifteen of our patients had atrial fibrillation with or without ischaemic heart disease and 1 had cardiomyopathy. All patients had a single abrupt clinical

event without premonitory symptoms. The embolus most likely lodged, either transiently or permanently, at the origin of the middle cerebral artery, as demonstrated in one of these cases reported previously (Bladin and Berkovic, 1984). On these occasions, collateral flow from the anterior cerebral artery may take over much of the function of the superior division of the ipsilateral middle cerebral artery while collateral flow from the posterior circulation may supply the territory of the inferior division of the middle cerebral artery and thus preserve cortical tissue (Vander Eeken and Adams, 1953; Bladin and Berkovic, 1984). If the arterial occlusion is only temporary, cortical flow may also be restored via anterograde flow through the middle cerebral artery stem.

Group 2. Severe carotid occlusive disease (12 patients)

A haemodynamically significant ipsilateral high grade internal carotid artery stenosis was seen in 6 patients and complete occlusion in a further 6 patients. One of the former had a long intraluminal clot, the details of which have been described previously (Donnan and Bladin, 1979). Five patients had premonitory events (3 with amaurosis fugax) and 2 had a stuttering onset, confirming previous observations that severe carotid occlusive disease may be associated with premonitory and intermittently progressive symptoms (Fisher, 1951), unlike cerebral infarction due to cardiac emboli where this pattern is unusual (Dyken *et al.*, 1986). Cardiac disease was present in only 2 of these patients, confirming the paramount role of the extracranial vascular disease in the pathogenesis of striatocapsular infarction in this group. The mechanism of infarction may involve emboli from the stenotic, occluded or occluding internal carotid artery which may lodge at the middle cerebral artery origin as in the first group. However, in the angiograms studied, crossflow from the opposite hemisphere via the anterior communicating artery to the ipsilateral middle cerebral artery was commonly the means by which hemispheric flow was restored as well as via anastomotic flow from anterior and posterior cerebral arteries. Whether deep striatocapsular infarction can occur in these cases based entirely on reduced middle cerebral artery flow and its effect on the lenticulostriate end arteries without involving embolic occlusion as a mechanism is uncertain.

Group 3. Normal angiography (or minimal change), mechanism uncertain (11 patients)

This group comprises those patients in whom angiography was normal or only minimal change was present. Three patients had ischaemic cardiac disease and it is conceivable that cardiac embolism was responsible for the striatocapsular infarction in these cases. Indeed this mechanism must remain a possibility in other cases with relatively normal angiograms, since the cardiac disease or dysrhythmia may remain occult. However, 5 patients had premonitory symptoms and 2 had a stuttering progression of deficit, perhaps making a cardiac source of embolism less likely. Two patients presented with the bursts of hemiplegia characteristic of the 'capsular warning syndrome' previously described as a prelude to lacunar infarction (Donnan *et al.*, 1982; Donnan and Bladin, 1987), but on these occasions evolved into striatocapsular infarction involving multiple rather than single lenticulostriate vessels. In 2 patients there were features in the clinical presentation which suggested that vasospasm may have played a role; emotional stress was present before the event, mild throbbing headache was noted during and after the event and a 'washed out' sensation persisted for many hours later. Another patient lost

consciousness for a brief period at the onset, suggesting a more global circulatory disturbance during the infarct genesis.

Groups 4. Middle cerebral artery abnormalities (3 patients)

Here disease of the middle cerebral artery was seen in the absence of cardiac or extracranial occlusive disease. In one 24-yr-old woman, middle cerebral artery dissection or arteritis was suspected with resultant occlusion of the origins of several penetrators to the striatocapsular region (fig. 2). Similar occlusion of the origins of penetrators was likely in the patient where atheromatous middle cerebral artery stenosis of approximately 50% was seen. The single patient in whom an aneurysm was demonstrated distal to the lateral striate arteries did not have evidence of subarachnoid haemorrhage, so the relationship of the aneurysm to striatocapsular infarction must remain speculative. While middle cerebral artery disease was a less common mechanism of striatocapsular infarction in our series, this may not be the case in Japanese and American black populations where middle cerebral artery occlusive disease is said to be more common (Kieffer *et al.*, 1967; Gorelick *et al.*, 1984).

There is obvious confusion in the literature concerning the nature and terminology of subcortical infarctions. We suggest that the term striatocapsular infarction be used for the clearly recognizable infarctions restricted to the territory of the lenticulostriate vessels of the middle cerebral artery with its typical clinical correlates as described here. A clear distinction may then be made from other 'deep subcortical' infarction such as in the anterior choroidal artery territory (Decroix *et al.*, 1986), lacunar (Fisher, 1982; Mohr, 1982) and internal watershed infarctions (Wodarz, 1980; Bogousslavsky and Regli, 1986), all of which have differing clinical presentations, CT topography and pathogenesis.

Early studies of the pathological correlates of carotid and middle cerebral artery occlusions contained descriptions of what could now be classified as striatocapsular infarctions (Foix and Lévy, 1927; Torvik and Jörgensen, 1966; Blackwood *et al.*, 1969). The proposed mechanisms of infarction were similar to our Group 2 patients, particularly in the study of Trovik and Jörgensen (1966). Similarly, recent descriptions of patients with aphasia attributed to deep subcortical infarction contained some cases of striatocapsular infarction (Damasio *et al.*, 1982; Naeser *et al.*, 1982), although the pathogenesis of infarction was not discussed, thereby perhaps minimizing the cortical contribution to the aphasia due to depressed cortical blood flow (*see below*). Rascol *et al.* (1982) described a variety of deep subcortical infarctions of which only type I was clearly striatocapsular. In broader clinical studies of occlusion of internal carotid and middle cerebral arteries both Ringelstein *et al.* (1983) and Caplan *et al.* (1985) had some examples of striatocapsular infarction but did not focus on the clinical features of this group particularly. However, the patients described by Adams *et al.* (1983); Santamaria *et al.* (1983) and Levine *et al.* (1988) more clearly had striatocapsular infarction and had clinical features similar to our own cases.

It was of interest that ipsilateral EEG slow waves tended to parallel the presence of focal cortical signs (Table 4). Cortical signs are extremely useful in the initial bedside distinction of the two commonest forms of ischaemic hemisphere stroke, subcortical lacunes and cortical infarcts. The former rarely have cortical signs and typically have a normal EEG, whereas the latter usually have both cortical signs and focal slow waves

(Chambers *et al.*, 1983; Macdonell *et al.*, 1988). Striatocapsular infarction presents an interesting paradox; cortical signs and focal EEG slow waves are usual, and yet the anatomical lesion is exclusively subcortical. There is debate in the literature as to whether 'cortical' features in such subcortical lesion are due to dysfunction of the overlying cortex or represent true subcortical signs (Damasio *et al.*, 1982; Naeser *et al.*, 1982; Perani *et al.*, 1987; Vallar *et al.*, 1988).

Our clinical, EEG and angiographic data, together with blood flow studies of other workers, strongly suggest that the cortical signs in the acute phase are due to disruption of cortical function, rather than a subcortical effect per se. It is well known that focal slow waves in acute cortical lesions are due to involvement of the immediate subcortical white matter, resulting in cortical deafferentation; such slow waves are absent or inconstant features of lesions limited to the internal capsule or deep grey matter (Ball *et al.*, 1977; Gloor *et al.*, 1977; Macdonell *et al.*, 1988). The association of focal slow waves with cortical signs in our patients thus strongly suggests that there is functional disruption lateral to the anatomical margins of the striatocapsular lesion, involving the immediate subcortical white matter and the cortex itself. This electroclinical deduction is supported by cerebral blood flow studies showing acute focal depression of cortical blood flow in patients with striatocapsular infarction (Perani *et al.*, 1987; Vallar *et al.*, 1988).

This depression of blood flow and presumably metabolism represents, in part, the 'ischaemic penumbra' of the infarction (Astrup *et al.*, 1981). Our finding that cortical signs are more likely to be present in patients with severe extracranial carotid occlusive disease, where the ability to limit the size of the ischaemic penumbra may be reduced compared with those patients with uninterrupted anterograde flow through the internal carotid and middle cerebral systems, further supports this contention. Any role played by diaschisis due to involvement of fibres of passage to the cortex in the genesis of cortical signs (Perani *et al.*, 1987) is therefore likely to be overwhelmed by local haemodynamic factors, at least in the acute phase.

However, in the chronic phase, our neuropsychological findings would suggest that the remote effect of depressed cerebral blood flow and metabolism that follows focal ischaemia, that is, diaschisis (Høedt-Rasmussen and Skinhøj, 1964) may be responsible for the subtle, significant and persisting deficits. Amongst these deficits were material specific learning impairments which are classically due to unilateral diencephalic or medial temporal lobe lesions; both areas are supplied by the anterior choroidal and posterior cerebral arteries, well outside the territory of the vessel supplying the striatocapsular regions, the middle cerebral artery. A possible explanation for persisting deficits of this type is diaschisis involving fibre connections crossing the vascular territories of middle cerebral and posterior cerebral arteries.

Our finding that the neuropyschological impairment in the acute phase was always attributable to the affected hemisphere has been shown by others (Damasio *et al.*, 1982; Skyhøj Olsen *et al.*, 1986; Perani *et al.*, 1987; Levine *et al.*, 1988; Vallar *et al.*, 1988; Højer-Pedersen and Petersen, 1989), although Cambier *et al.* (1984) described a case of a left subcortical lesion in which autotopagnosia, amongst other neuropsychological changes, was present. However, the pathology was haemorrhage, rather than infarction, and pressure effects may have come into play.

Up until this time there has been disagreement in the literature as to whether the

neuropsychological deficits associated with striatocapsular infarction may persist over a prolonged period. Levine *et al.* (1988) found that more than 75% of surviving aphasics in their series had obvious speech deficits some months after the stroke. On the other hand, Skyhøj Olsen *et al.* (1986) demonstrate that in 6 out of 8 aphasic patients clinical remission was complete at 3 months after the stroke. At 6 months, 3 of their patients showed subtle language signs on detailed neuropsychological examination. It is possible that the learning functions assessed in our study represent a more sensitive index of neuropsychological impairment, and our findings suggest that the chronic phase is not devoid of such impairment in a significant proportion of patients with purely subcortical lesions. This may be of particular importance when assessing such patients for re-entry into the workforce.

We found the outcome of patients with striatocapsular infarction appears to be intermediate between the excellent prognosis of lacunar infarction (Gandolfo *et al.*, 1986; Bamford *et al.*, 1987) and the poor prognosis of patients with cortical/subcortical infarction. The latter are faced not only with motor impediments, but also gross neuropsychological dysfunction. Two-thirds of our patients had at least a functional recovery and half of these were able to return to work. Predictors of a better outcome were of younger age, absence of cortical signs at presentation and no haemodynamically significant disease seen on cerebral angiography. A poor prognosis was likely in older patients with equal motor involvement of face, arm and leg at onset, the presence of cortical signs and, if performed, angiographic findings of severe carotid occlusive disease. Based on these findings a relatively early prediction of likely outcome should be possible in many cases.

REFERENCES

ADAMS HP, DAMASIO HC, PUTMAN SF, DAMASIO AR (1983) Middle cerebral artery occlusion as a cause of isolated subcortical infarction. *Stroke,* **14,** 948–952.

ASTRUP J, SIESJÖ BK, SYMON L (1981) Thresholds in cerebral ischemia—the ischemic penumbra. *Stroke,* **12,** 723–725,

BALL GJ, GLOOR P, SCHAUL N (1977) The cortical electromicrophysiology of pathological delta waves in the electroencephalogram of cats. *Electroencephalography and Clinical Neurophysiology,* **43,** 346–361.

BAMFORD J, SANDERCOCK P, JONES L, WARLOW C (1987) The natural history of lacunar infarction: the Oxfordshire Community Stroke Project. *Stroke,* **18,** 545–551.

BENTON AL, HAMSHER, K DE S, VARNEY NR, SPREEN O (1983) *Contributions to Neuropsychological Assessment: A Clinical Manual.* New York and Oxford: Oxford University Press.

BLACKWOOD W, HALLPIKE JF, KOCEN RS, MAIR WGP (1969) Atheromatous disease of the carotid arterial system and embolism from the heart in cerebral infarction: a morbid anatomical study. *Brain,* **92,** 897–910.

BLADIN PF, BERKOVIC SF (1984) Striatocapsular infarction: large infarcts in the lenticulostriate arterial territory. *Neurology, Cleveland* **34,** 1423–1430.

BOGOUSSLAVSKY J, REGLI F (1986) Unilateral watershed cerebral infarcts. *Neurology, Cleveland,* **36,** 373–377.

CAMBIER J, ELGHOZI D, GRAVELEAU P, LUBETZKI C (1984) Hémiasomatognosie droite et sentiment d'amputation par lésion gauche sous-corticale. Rôle de la disconnexion calleuse. *Revue Neurologique,* **140,** 256–262.

CAPLAN L, BABIKIAN V, HELGASON C, HIER DB, DEWITT D, PATEL D, STEIN R (1985) Occlusive disease of the middle cerebral artery. *Neurology, Cleveland,* **35,** 975–982.

CHAMBERS BR, DONNAN GA, BLADIN PF (1983) Patterns of stroke: an analysis of the first 700 consecutive admissions to the Austin Hospital Stroke Unit. *Australian and New Zealand Journal of Medicine*, **13**, 57–64.

DAMASIO AR, DAMASIO H, RIZZO M, VARNEY N, GERSH F (1982) Aphasia with nonhemorrhagic lesions in the basal ganglia and internal capsule. *Archives of Neurology, Chicago*, **39**, 15–20.

DECROIX JP, GRAVELEAU P, MASSON M, CAMBIER J (1986) Infarction in the territory of the anterior choroidal artery: a clinical and computerized tomographic study of 16 cases. *Brain*, **109**, 1071–1085.

DOLL R, PETO R (1976) Mortality in relation to smoking: 20 years' observations on male British doctors. *British Medical Journal*, **2**, 1525–1536.

DONNAN GA, BLADIN PF (1979) The stroke syndrome of long intraluminal clot with incomplete vessel obstruction. *Clinical and Experimental Neurology*, **16**, 41–47.

DONNAN GA, TRESS BM, BLADIN PF (1982) A prospective study of lacunar infarction using computerized tomography. *Neurology, New York*, **32**, 49–56.

DONNAN GA, BLADIN PF (1987) Capsular warning syndrome: repetitive hemiplegia preceding capsular stroke. *Stroke*, **18**, 296.

DYKEN ML, FISHER M, HARRISON MJG, HART RG, SHERMAN DG (1986) Cardiogenic brain embolism. *Archives of Neurology, Chicago*, **43**, 71–84.

FISHER CM (1982) Lacunar strokes and infarcts: a review. *Neurology, New York*, **32**, 871–876.

FISHER M (1951) Occlusion of the internal carotid artery. *Archives of Neurology and Psychiatry, Chicago*, **65**, 346–377.

FOIX C, LÉVY M (1927) Les ramollissements sylviens: syndromes des lésions en foyer du territoire de l'artère sylvienne et de ses branches. *Revue Neurologique*, **11**, 1–51.

GANDOLFO C, MORETTI C, DALL'AGATA D, PRIMAVERA A, BRUSA G, LOEB C (1986) Long-term prognosis of patients with lacunar syndromes. *Acta Neurologica Scandinavica*, **74**, 224–229.

GLOOR P, BALL G, SCHAUL N (1977) Brain lesions that produce delta waves in the EEG. *Neurology, New York*, **27**, 326–333.

GORELICK PB, CAPLAN LR, HIER DB, PARKER SL, PATEL D (1984) Racial differences in the distribution of anterior circulation occlusive disease. *Neurology, Cleveland*, **34**, 54–59.

HØEDT-RASMUSSEN K, SKINHØJ E (1964) Transneural depression of the cerebral hemispheric metabolism in man. *Acta Neurologica Scandinavica*, **40**, 41–46.

HØJER-PEDERSEN E, PETERSEN OF (1989) Changes of blood flow in the cerebral cortex after subcortical ischemic infarction. *Stroke*, **20**, 211–216.

KIEFFER SA, TAKEYA Y, RESCH JA, AMPLATZ K (1967) Racial differences in cerebrovascular disease: angiographic evaluation of Japanese and American populations. *American Journal of Roentgenology*, **101**, 94–99.

LEZAK MD (1983) *Neuropsychological Assessment*. Second edition. New York and Oxford: Oxford University Press.

LEVINE RL, LAGREZE HL, DOBKIN JA, TURSKI PA (1988) Large subcortical hemispheric infarctions: presentation and prognosis. *Archives of Neurology, Chicago*, **45**, 1074–1077.

MACDONELL RAL, DONNAN GA, BLADIN PF, BERKOVIC SF, WRIEDT CHR (1988) The electroencephalogram and acute ischemic stroke: distinguishing cortical from lacunar infarction. *Archives of Neurology, Chicago*, **45**, 520–524.

MILNER B (1965) Visually-guided maze learning in man: effects of bilateral hippocampal, bilateral frontal, and unilateral cerebral lesions. *Neuropsychologia*, **3**, 317–338.

MOHR JP (1982) Lacunes. *Stroke*, **13**, 3–11.

NAESER MA, ALEXANDER MP, HELM-ESTABROOKS N, LEVINE HL, LAUGHLIN SA, GESCHWIND N (1982) Aphasia with predominantly subcortical lesion sites: description of three capsular/putaminal aphasia syndromes. *Archives of Neurology, Chicago*, **39**, 2–14.

OSTERRIETH PA (1945) Le test de copie d'une figure complexe; contribution à l'étude de la perception et de la mémoire. *Archives de Psychologie*, **30**, 205–353.

PERANI D, VALLAR G, CAPPA S, MESSA C, FAZIO F (1987) Aphasia and neglect after subcortical stroke: a clincial/cerebral perfusion correlation study. *Brain*, **110**, 1211–1229.

RASCOL A, CLANET M, MANELFE C, GUIRAUD B, BONAFE A (1982) Pure motor hemiplegia: CT study of 30 cases. *Stroke*, **13**, 11–17.

RINGELSTEIN EB, ZEUMER H, ANGELOU D (1983) The pathogenesis of strokes from internal carotid artery occlusion. Diagnostic and therapeutical implications. *Stroke*, **14**, 867–875.

SANTAMARIA J, GRAUS F, RUBIO F, ARBIZU T, PERES J (1983) Cerebral infarction of the basal ganglia due to embolism from the heart. *Stroke*, **14**, 911–914.

SKYHØJ OLSEN T, BRUHN P, ÖBERG RGE (1986) Cortical hypoperfusion as a possible cause of 'subcortical aphasia'. *Brain*, **109**, 393–410.

TORVIK A, JÖRGENSEN L (1966) Thrombotic and embolic occlusions of the carotid arteries in an autopsy series. Part 2. Cerebral lesions and clinical course. *Journal of the Neurological Sciences*, **3**, 410–432.

VALLAR G, PERANI D, CAPPA SF, MESSA C, LENZI GL, FAZIO F (1988) Recovery from aphasia and neglect after subcortical stroke: neuropsychological and cerebral perfusion study. *Journal of Neurology, Neurosurgery and Psychiatry*, **51**, 1269–1276.

VANDER EECKEN HM, ADAMS RD (1953) The anatomy and functional significance of the meningeal arterial anastomoses of the human brain. *Journal of Neuropathology and Experimental Neurology*, **12**, 132–157.

WALSH KW (1985) *Understanding Brain Damage: A Primer of Neuropsychological Evaluation*. Edinburgh: Churchill Livingstone, pp. 161–162.

WALSH KW (1987) *Neuropsychology. A Clinical Approach*. Second edition. Edinburgh: Churchill Livingstone.

WECHSLER D (1945) A standardized memory scale for clinical use. *Journal of Psychology*, **19**, 87–95.

WODARZ R (1980) Watershed infarctions and computed tomography. A topographical study in cases with stenosis or occlusion of the carotid artery. *Neuroradiology*, **19**, 245–248.

YATES F (1934) Contingency tables involving small numbers and the χ^2 test. *Journal of the Royal Statistical Society*, **1**, Supplement, 217–235.

(Received May 17, 1989. Revised December 28, 1989. Accepted January 9, 1990)

APPENDIX

Explanatory notes for neuropsychological tests used

Picture Completion

The subject is presented with a series of incomplete pictures from which an important part is missing; the task required the missing part to be identified. As a subtest of the Wechsler Adult Intelligence Scale-Revised (WAIS-R) this test is scored in terms of the number of correct missing parts identified, and this can be converted into an age-scaled score as above.

Digit Symbol Substitution

This coding task is a subtest of the WAIS-R. The subject is presented with a coding table, the top row of which displays the numerals 1 to 9, while bottom row displays 9 different symbols, each of which is arbitrarily but uniquely associated with 1 of the numerals. The subject is also presented with a series of numerals ranging between 1 and 9 in a recurring random sequence. He/she is required to write the associated symbol below each numeral, and is allowed to refer to the coding table throughout the task. The score is the number of symbols correctly coded in 90 s; this score can be converted to an age-scaled score with a mean of 10 and an SD of 3.

Block Design

This test is a block construction task. Each block has 2 red sides, 2 white sides and 2 red and white sides. The subject is required to replicate a series of designs. The test is timed and the score can be converted to an age-scaled score as above since it is also a subtest of WAIS-R.

Logical Memory (Wechsler, 1945)

A passage of prose is read to the subject, who is then required to recall as much of the passage as possible. The response is scored in terms of the number of ideas correctly recalled. This score can then be age-scaled as for WAIS-R subtests. The test is then repeated using a second passage.

Rey Auditory Verbal Learning Test (Lezak, 1983)

A list consisting of 15 common nouns is presented 5 times to the subject, and spontaneous recall is examined after each presentation. Following the fifth trial, the subject listens to a second list of 15 common nouns, none of which was contained in the previous list (trial B). The subject is then examined for spontaneous recall of the original word list (trial 6). A comparsion between the fifth trials and trial 6 reflects the extent to which the interpolated list interferes with retention of the original list. The test is scored in terms of the number of words correctly recalled on each trial.

Rey Complex Figure (Osterreith, 1945; Lezak, 1983)

The subject is required to copy a complex but meaningless design, and is then asked to reproduce it from memory 5 min later. In this study, the Controlled Oral Word Association Test was administered during the 5 min gap. The test is scored in terms of the number of elements correctly copied or reproduced.

Controlled Oral Word Association Test (Benton et al., 1983)

This test elicits the oral production of words beginning with a designated letter. The letters used in this study were 'F', 'A' and 'S', and the subject is allowed 1 min for each letter. Certain restrictions are placed on the subject, i.e., proper nouns are disallowed, as are words with a common stem but different suffixes and repetitions of a word already given (*see* Walsh, 1985). The score is the total number of words allowed; the norms are standardized in terms of age and years of education, yielding percentiles.

Austin Maze (Walsh, 1985)

The subject is seated in front of 10×10 matrix of electrically activated 'stepping stone' switches, and is required to learn a predetermined rectangular pathway through the matrix. Switches which are on the pathway momentarily activate a green light and those not on the pathway activate a red light and buzzer. Milner's (1965) pathway was used in the present study and the task was scored in terms of the number of errors on each of 10 trials.