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Subcortical lobar intracerebral haemorrhage: clinical-computed tomographic correlations

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SUMMARY Fifty patients with spontaneous lobar haematomas were reviewed. Thirty-two were normotensive and eighteen were hypertensive. Vomiting, seizures, and altered consciousness were more common in hypertensive than in normotensive patients. Haematoma size (greater than 4·0 cm) and intraventricular extension occurred most commonly in hypertensive and rarely in normotensive patients. Twenty-eight per cent of hypertensive patients died, whereas none of the normotensive patients died.

The majority of intracerebral haematomas are hypertensive in aetiology. These are usually located in the thalamic or basal ganglionic region; less commonly, they are in the cerebellum, brain stem, or subcortical white matter of the cerebral hemispheres (lobar haematomas). The finding of a lobar haematoma should initiate a thorough investigation for a specific neuropathological aetiology for the haemorrhage such as angioma, aneurysm, neoplasm, or an underlying medical condition (for example bleeding disorder, collagen vascular disease).

The present report is an analysis of the clinical-computed tomographic correlations in 50 patients with non-traumatic acute lobar haematoma in whom a thorough clinical and laboratory investigation did not demonstrate a specific aetiology for the intracranial haematoma.

Methods and patients

Of 489 consecutive patients who were studied prospectively with CT evidence of intracerebral haematoma, there were 68 patients with lobar haematomas. There were 18 patients in whom neurodiagnostic findings, laboratory studies and/or pathological findings showed a specific aetiology for the lobar haematomas (table 1).

Four patients with anterior cerebral or anterior communicating artery aneurysms had frontal haematomas, and two patients with middle cerebral artery aneurysms had temporal lobe haematomas. Of the patients with arteriovenous malformations, the haematoma was frontal (1 case), temporal (2 cases), and parietal (1 case). The intracranial neoplasms were metastatic in three cases and were located in the frontal lobes. One case of glioblastoma multiforme had a temporal haematoma.

There were 50 other patients with CT evidence of lobar haematoma in whom no aetiology was determined by CT findings, angiography, laboratory studies, and in some cases by surgical or necropsy studies. Patients with a history of trauma were excluded from this analysis. In an effort to ensure that patients with traumatic haemorrhagic contusions were not included, patients in whom trauma was even a remote consideration were excluded. The clinical and CT findings were reviewed by the author. The diagnostic criteria used by Kase *et al*[1] to support the diagnosis of lobar haematoma were used. These included: (1) absence of clinically detectable cardiac source of cerebral embolism; (2) CT evidence of a hyperdense lesion located in the subcortical white matter which extended beyond a specific arterial territory; (3) the haematoma was sometimes surrounded by an irregularly marginated hypodense region (representing oedema), but the irregular margination of the hypodense component excluded the diagnosis of infarction; (4) there was sometimes ventricular extension; (5) ring enhancement was sometimes seen if scan was performed 7 to 28 days after the haemorrhage; (6) when performed, angiogram showed evidence of an avascular mass and not a cortical branch occlusion (which would be consistent with cerebral embolism). Haematomas which appeared to originate in the ganglionic-thalamic region and then secondarily extended into the white matter of the cerebral hemispheres were excluded from this analysis, as were those patients with multiple lobar haematomas.

CT scans were performed on either a Picker Synerview high resolution body scanner (matrix size of 256 × 256, scan time of one second, slice thickness of 10 mm) or GE CT/T 8800 body scanner (matrix size of 256 × 256, scan time of 48 or 9·6 seconds, slice thickness of 10 mm). All

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Table 1 *Aetiology of lobar haematomas in 18 patients*

Condition	*Number of cases*
Saccular aneurysm	6
Arteriovenous malformation	4
Intracranial neoplasm	4
Anticoagulant induced	1
Blood dyscrasia	1
Collagen vascular disease	1
Septic embolism-bacterial endocarditis	1

Table 2 *Location of 50 spontaneous lobar haematomas*

Location	*Number of cases*
Frontal	11
Frontal-temporal	4
Temporal	8
Temporal-parietal	5
Parietal	15
Occipital	7

scans were performed before and immediately following the intravenous infusions of 300 ml of iodinated contrast material. The maximal diameter of the haematoma was measured on the axial CT sections; however, no attempt to determine haematoma volume was made. All scans were performed within 48 hours of the onset of clinical deficit. Follow-up scan was performed in 28 patients. Twenty-nine patients had an angiogram within 10 days of the ictal event. Eighteen patients who had sustained elevated blood pressure did not have angiography. Six of 50 patients had surgical intervention for clot evacuation. Three patients had surgical intervention to evacuate the lobar haematoma without prior angiography.

Findings

The age of the patients ranged from 32 to 68 years old (2 were in their thirties, 13 were in their forties, 25 were in their fifties, 10 were in their sixties). Fifteen patients were known to be hypertensive prior to the development of the lobar haematoma. These patients had at least two of the following: (1) systolic pressure exceeding 150 mm Hg and diastolic pressure exceeding 100 mm Hg, (2) treatment with antihypertensive medication, (3) funduscopic evidence of hypertensive retinal disease, (4) radiographic evidence of left ventricular enlargement, (5) electrocardiographic evidence of left ventricular strain pattern. Thirteen patients had no prior history of hypertension but were hypertensive on admission to the hospital. Ten of these patients were normotensive within 48 hours and required no further antihypertensive treatment, whereas three other patients required treatment with antihypertensive medication for sustained elevated blood pressure. Twenty-two patients had no prior history of hypertension and were normotensive on admission.

The onset of symptoms was sudden in all cases. Following the onset of clinical deficit, there was progression to maximal deficit in 11 patients; whereas the deficit was maximal at onset in 39 other cases. This neurological worsening consisted in progressive deterioration in the level of consciousness (eight cases) and evidence of transtentorial herniation (three cases). There was headache at onset in 36 patients. Vomiting accompanied the headache in 16 patients. Seizures were the presenting symptom in 14 patients, with nine patients having focal seizures and five having generalised seizures. Only six patients had signs of meningeal irritation. The neurological findings observed on examination were correlated with the location of the haematoma.

The CT findings consisted of a hyperdense lesion located in the cerebral hemispheres (table 2). This lesion was not confined to a specific arterial distribution so that it is unlikely that any of these lesions represented haemorrhagic infarcts. The size, shape, and location of the haematoma were not consistent with a contusion. The haematoma was usually round or oval in shape. There was irregularly marginated hypodense oedema surrounding the haematoma in 78% of cases. The oedema was most extensive with the larger lesions and was least extensive with smaller lesions. Mass effect was seen in 62% of cases. Intraventricular extension of the haemorrhage was seen in 38% of cases. Follow-up CT was performed in 28 cases 7 to 21 days after the initial study and ring enhancement was seen in six cases. This ring enhancement was seen in haematomas located primarily in the parietal region.

The mortality of patients with lobar haematomas was 10%. Follow-up of patients who survived the intracranial haemorrhage was obtained for 6 to 12 months. Three patients subsequently had a seizure. This developed within 6 weeks of the intracranial haemorrhage. Of the 14 patients who initially presented with seizures, five had recurrent seizures. The occurrence of seizures did not correlate with the size of the haematoma. Seizures occurred most frequently in patients who had surgery and those patient who showed ring enhancement on the scan after contrast injection. No patient had a second episode of intracranial haemorrhage.

LOCATIONS OF HAEMORRHAGE

Frontal haemorrhage The 11 frontal haematomas were unilateral. There were two locations of these haematomas: (1) superior to the lateral ventricle, (2) inferior or contiguous with the frontal horn of the lateral ventricle. Haematomas located superior to the lateral ventricle were round or oval in shape. Four haematomas were 1·7 to 2·1 cm in size. These patients presented with frontal headache and contralateral leg weakness. (fig 1) Four haematomas

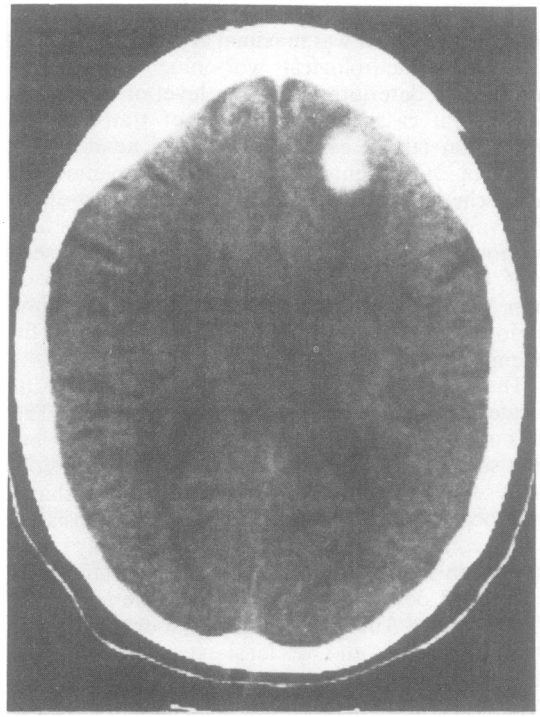

Fig 1 *48-year-old normotensive woman suddenly developed right leg weakness. Neurological examination showed pure motor hemiaparesis. CT findings: left (reader's right) oval hyperdense superior frontal haematoma.*

were 2·2 to 3·3 cm in size. These patients initially had contralateral leg weakness (monoparesis) and this rapidly progressed to hemiparesis. These eight patients had normal consciousness and their neurological deficit rapidly stablised. They were treated nonsurgically. Repeat CT showed resolution of the haematoma and none showed ring enhancement.

The three patients with inferior and midfrontal haematomas initially presented with headache and vomiting. There was rapid progression to impaired consciousness., lateral gaze paresis and hemiparesis. These haematomas were triangular in shape with the apex pointing toward the frontal horn of the lateral ventricle. There was surrounding oedema, significant mass effect and intraventricular extension of the haematomas which were 4·0 to 5·4 cm in size. Rapid neurological deterioration occurred in two patients and these patients died within 96 hours despite corticosteroids but without surgical intervention. One patient was treated nonsurgically with good clinical outcome.

Frontal-temporal haemorrhage

Four normotensive patients developed headache followed by difficulty expressing themselves and right-sided weakness. Findings were nonfluent expressive aphasia and right hemiparesis. Consciousness was not impaired in these patients. The haematomata were ovoid in shape and extended across the sylvian cistern. They were 3·8 to 4·9 cm in size. No blood was seen in the basal cisterns or ventricles. The haematoma extended into the basal ganglia but not across the internal capsule. Angiography showed an avascular mass without evidence of neovascularity, angioma, or aneurysm. In these patients, the hemiparesis resolved rapidly; however, the language disturbance showed only mild improvement six months later. Because of the extension into the basal ganglia and the initial clinical improvement, surgical intervention was not performed in these patients. Follow-up CT showed resolution of the haematoma size with no evidence of ring enhancement.

Temporal haemorrhage

All eight patients presented with the sudden onset of headache and vomiting. Six normotensive patients had left temporal haematomas. These patients initially developed language dysfunction. Findings were aphasia characterised by impaired comprehension, fluent speech and marked anomia. All patients had right homonymous hemianopsia. Only three patients had evidence of hemiparesis. CT showed round or oval hyperdense haematoma. There was surrounding oedema with mass effect. The haematomas were 3·3 to 4·7 cm in size and extended superficially to the cortex. There was no cisternal or ventricular blood. Angiogram showed an avascular mass. Three patients showed progressive impairment of the level of consciousness, and therefore these patients underwent surgical evacuation of the haematoma. These patients survived. Two patients had significant residual neurological dysfunction and one patient had mild deficit. Three other patients were treated nonsurgically. They survived and had mild residual language deficit.

Two hypertensive patients developed right temporal haematomas. They presented with headache and then had a seizure. Following the seizure they were confused. Neurological examination showed left pronator drift and unilateral Babinski sign. CT showed haematoma which was 3·5 and 4·3 cm in size without intraventricular extension. The patients improved with nonsurgical management.

Parietal haemorrhage

Fifteen patients had parietal haematomas, of whom six initially developed a seizure. This was observed to be focal in onset in three and generalised in three others. Four patients presented with a headache immediately followed by hemiparesis and five

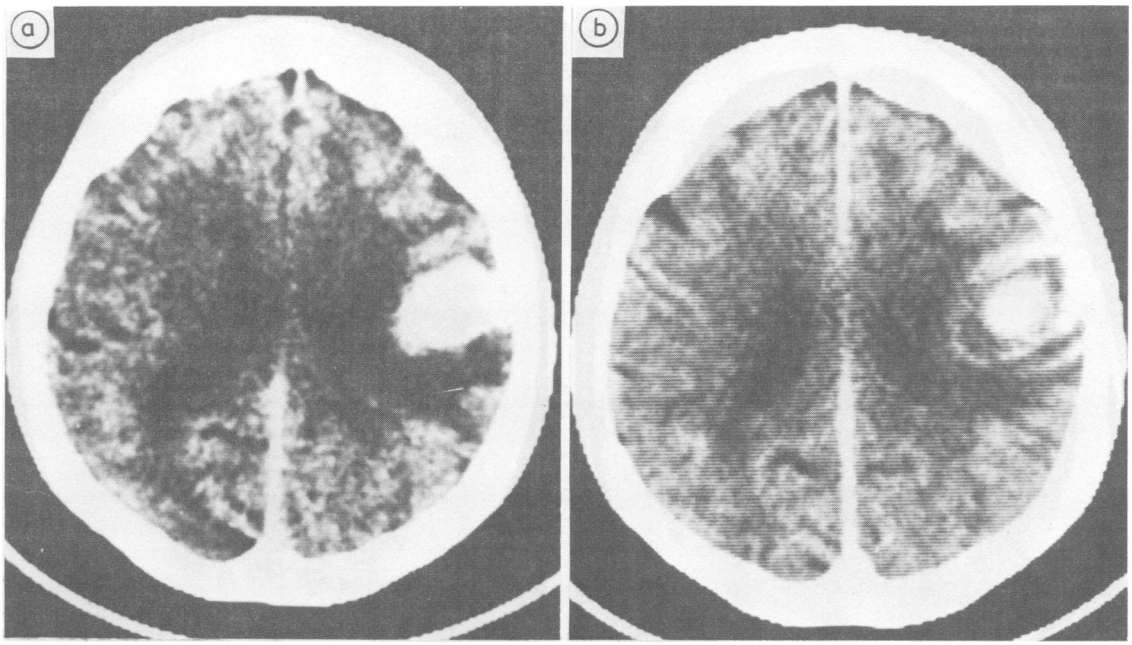

Fig 2 *52-year-old hypertensive woman suddenly developed right-sided weakness. Findings were right hemiparesis-hemianaesthesia. CT findings: left parietal haematoma with surrounding oedema (A). She showed clinical improvement. Repeat CT (10 days later) showed some resolution of haematoma size with peripheral ring enhancement (B).*

developed sudden onset of motor weakness without accompanying headache. Eight haemorrhages involved the left and seven were in the right hemisphere. All patients had evidence of hemiparesis and hemisensory deficit. Three patients had a homonymous hemianopsia. Of the eight patients with right hemispheric haematomas, six had abnormalities involving body schema and/or elements of anosagnosia. Of the seven patients with left hemisphere haematoma, one showed evidence of fluent word-finding aphasic disturbance. Five patients had mild impairment of consciousness which resolved within 48 hours (two of these patients received corticosteroids and three did not). All patients had hemiparesis and hemisensory deficit. All patients recovered without surgical intervention.

CT scan showed a round or overall hyperdense parietal lesion. The size ranged from 1·5 to 4·4 cm. They had surrounding hypodense rim which represented vasogenic oedema. There was evidence of ventricular compression in four cases. None of these haematomas extended into the ventricles. Repeat study was performed one to three weeks after the initial CT in 10 cases; six showed evidence of peripheral rim enhancement (fig 2). A third scan was performed in six patients who showed peripheral ring enhancement and there was resolution of the enhancement within three to four weeks.

Temporal-parietal haemorrhage

Five patients presented with the sudden onset of headache and vomiting. This was followed by rapid development of hemiparesis. Three patients had a seizure. In three patients there was marked deterioration of the level of consciousness and these patients showed pupillary and oculomotor findings consistent with descending transtentorial herniation. This progression developed over a 4 to 8 hour interval. Surgical evacuation of the haematoma was performed but these three patients became comatose and died. Necropsy examination was performed and showed the parenchymal haemorrhage with no evidence of infarction and no evidence of abnormal vessels on gross or microscopic examination of the brain. Two other patients developed hemiparesis and had normal consciousness and the deficit did not progress. These patients were treated nonsurgically and they showed clinical improvement.

In the three patients who died, CT showed a hyperdense round haematoma which was 4·5 to 6·6 cm in size. There was surrounding vasogenic oedema with significant mass effect. There was ventricular extension of the haemorrhage in these three cases (fig 3). In the two other patients, the temporal-parietal haemorrhage was 3·5 and 3·8 cm in size. There was minimal mass effect and no intraventricular extension. None of these five

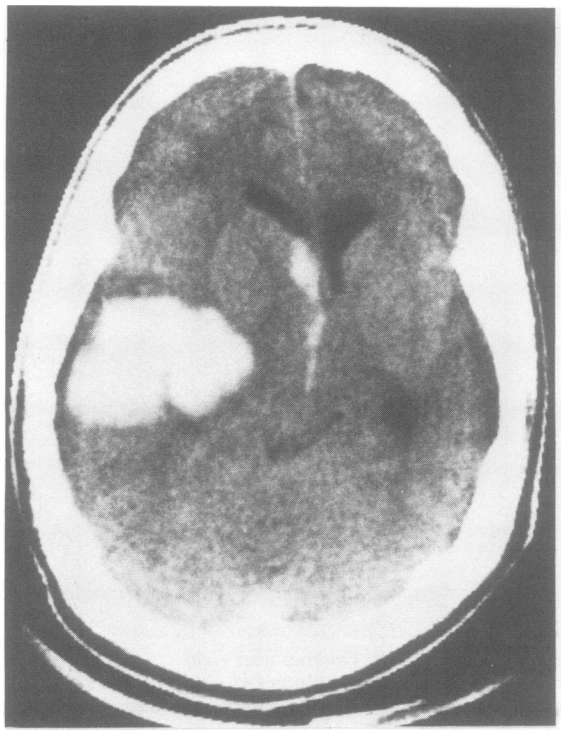

Fig 3 *46-year-old hypertensive man developed headache and vomiting. He became obtunded with left hemiplegia and dilated right pupil. CT findings: right temporal-parietal haematoma with marked mass effect and intraventricular extension.*

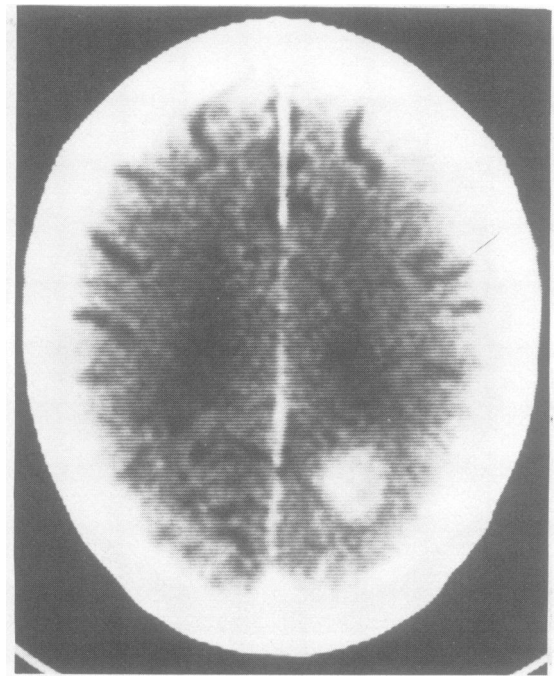

Fig 4 *53-year-old normotensive man developed headache and visual blurring. Findings were right homonymous hemianopsia. CT findings: left hyperdense round occipital haematoma.*

haematomas showed contrast enhancement.

Occipital haemorrhage

These seven patients initially developed headache. All patients described visual symptoms including blurring, haziness, or difficulty due to bumping into objects. All patients had a contralateral homonymous hemianopic defect on confrontation visual field examination. No motor, sensory, or reflex abnormalities were detected. The occipital haematomas were round in shape and 1·8 to 3·3 cm in size (fig 4). There was no surrounding oedema, mass effect or intraventricular extension. None showed evidence of contrast enhancement. The headache resolved within 5 days and confrontation visual field examination showed resolution of the visual defect within 28 days. Surgery was not performed in patients with occipital haematomas.

Discussion

Prior studies of non-traumatic spontaneous subcortical lobar haematoma have emphasised two features: (1) the need to investigate the patient for an identifiable and potentially remediable cause of the intracranial haemorrhage[2,3]; (2) the lower incidence (10 to 32%) of systemic arterial hypertension in patients with lobar haematomas compared with patients basal ganglia and thalamic intracranial haemorrhage.[4] For example, it has been reported that 10% of lobar haematomas are caused by primary or metastatic intracranial tumour.[5] It is usually possible to predict the pathological finding of an intracranial neoplasm based upon the clinical presentation, CT and angiographic findings. Congenital berry aneurysms may rupture into the subarachnoid spaces and dissect into the brain parenchyma to cause lobar haematoma. In these cases, there are usually characteristic clinical (prominent symptoms and signs of meningeal irritation), CT (blood in the basal cisternal spaces) and angiographic (demonstration of the aneurysm, vasospasm) findings. Large arteriovenous malformations may rupture to cause lobar haematomas. It has been reported that if the angiogram is performed too early after the haemorrhage, the clot may compress the arteriovenous malformation so that the abnormal vessels are not visualised. In certain patients with lobar haematomas, angiography may be performed several weeks later

Table 3 *Comparison of features of hypertensive and normotensive lobar haematomas*

Characteristics	*Incidence in normotensive patients*	*Incidence in hypertensive patients*
Vomiting at onset	6%	78%
Seizures	16%	33%
Altered consciousness	9%	66%
Progression of deficit	6%	50%
CT size of haematoma 4·0 cm	6%	50%
Intraventricular extension of blood	0%	33%
Mortality	0%	28%
Outcome		
Significant neurological disability	34%	77%
Mild or minimal neurological disability	66%	23%

when haematoma resolution has occurred. Based upon the initial complete CT findings (including a noncontrast and post-contrast scan), it is frequently possible to predict the presence of an underlying large arteriovenous malformation even prior to angiography.[8] Subcortical lobar haematomas may result from small or cryptic vascular malformations. These may be compressed or even destroyed by the bleeding episode such that these vascular malformations are angiographically and sometimes even pathologically occult.[6 7] In this series of intracranial haemorrhages, there were 14 cases in which an aneurysm, neoplasm, or angioma was identified by neurodiagnostic studies and confirmed by pathological findings. There were certain important diagnostic features which differentiated these 14 patients from the 50 patients with spontaneous lobar haematomas. Clinically, evidence of neurological deterioration more than 24 hours after the initial haemorrhage was *not* consistent with spontaneous lobar haematoma. Certain CT findings were never seen with spontaneous lobar haematoma: (1) blood in the cisternal spaces, (2) post-contrast enhancement on the scan performed within the initial week after the haemorrhage.

The age predilection of these 50 patients with lobar haematomas is older than would have been expected for bleeding from a cerebral vascular malformation. In this series only 4% of patients were in their thirties, whereas the majority of patients with angiomas have an initial bleeding episode prior to age 30. Thirty per cent of patients in this series had definite evidence of systemic arterial hypertension prior to the lobar haematoma. In two reported series of lobar haematomas, 31 and 36% of patients had prior history of elevated blood pressure.[1 4] An additional 13 patients had elevated blood pressure on admission; however in 10 cases these patients were normotensive within 48 hours and only three required antihypertensive medication. The five patients who died were hypertensive. All patients who had haematomas which were larger than 4·5 cm in size and had intraventricular extension were hypertensive. Arteriolar micro-aneurysms are most commonly seen in the lenticulostriate vessls but may also be seen in subcortical vessels.[9] Rupture of these micro-aneurysms due to hypertension appears to be the aetiology of the lobar haematomas in 30% of cases. These hypertensive haematomas are larger and are associated with 28% mortality (five died of 18 patients). In two other series of lobar haematoma, the mortality was 11 and 32%;[1 4] however, both these series include patients with recognisable aetiological factors for the haematoma in addition to hypertension.

The incidence of vomiting, seizures, and altered consciousness was higher in hypertensive than normotensive patients (table 3). There was clinical evidence of subsequent neurological progression in 50% of hypertensive and 6% of normotensive patients. Of the 13 survivors of hypertensive lobar haematomas, 10 showed significant residual neurological disability due to motor weakness and language disturbance (77%); whereas three patients (23%) showed good functional recovery. All patients with normotensive lobar haematomas survived. Twenty-one showed almost complete recovery or only mild recovery (66%) whereas 11 patients (34%) showed more significant neurological deficit. These findings suggest that spontaneous lobar haematoma which occur in normotensive patients has a better neurological outcome than those which occur in hypertensive patients.

The findings in the present series may provide useful data concerning the rationale for surgical intervention in patients with lobar haematomas. Conclusions based upon data derived from the pre-CT era should probably be discarded because certain of the smaller haematomas were probably incorrectly diagnosed as infarcts and these series were biased towards patients with large haematomas. In a prospective but pre-CT scan study, McKissock *et al* concluded that surgical intervention offered no advantage over conservative nonsurgical management.[10] Ropper and Davis reported that since the lobar haematomas were fre-

quently small and peripheral in location, surgical intervention was not beneficial.[4] Kase *et al*[1] indicated the potential benefit of clot evacuation in selected patients with lobar haematomas. This included those patients who had medium and large size clots (with volume greater than 20 cc) who had impaired level of consciousness and showed continued signs of deterioration. Patients who had clots of comparable size but remained stable or improved after admission showed good recovery without surgical intervention. Based upon the findings in this group of 50 patients, it appears that normotensive patients with lobar haematoma have a low mortality; however there may be a sub-group in whom surgical clot evacuation may improve functional recovery (34% had significant neurological disability). In hypertensive patients with lobar haematoma, the mortality was 26% and 77% of survivors had significant neurological disability.

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