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Computerised tomography after recent severe head injury in patients without acute intracranial haematoma

J. SNOEK, BRYAN JENNETT, J. HUME ADAMS, D. I. GRAHAM, AND D. DOYLE

From the University Departments of Neurosurgery and Neuropathology, Institute of Neurological Sciences, Southern General Hospital, Glasgow

SUMMARY Sixty patients with severe head injury who did not have an acute intracranial haematoma on CAT scanning are reviewed. The scans are correlated with the level of consciousness at the time of scanning and with the outcome six months after injury. The initial scan was interpreted as being normal in 38% of the cases. In the remainder the most common abnormalities were small ventricles and areas of mixed increased and decreased density interpreted as contusions. All the patients with small ventricles were under 20 years of age. Post-mortem examinations were undertaken on 15 of the 19 fatal cases. There was evidence of a high intracranial pressure in 12, cerebral contusions were absent or minimal in 10, there was diffuse immediate impact damage to white matter in six, and there was moderate or severe hypoxic damage in four.

The most obvious value of CAT scanning after recent head injury is to identify and localise acute intracranial haematomas, and this it does with a high degree of accuracy (Galbraith *et al.*, 1977). But even in neurosurgical units which operate a selective admission policy, only a proportion of the patients referred have a haematoma, and the management of other patients who are in coma remains a difficult problem.

We review here the CAT scans of 60 patients with a severe head injury who did not have an acute intracranial haematoma. Scans are correlated with the level of consciousness at the time of scanning, with the outcome six months after injury and, in those who died, with the neuropathological findings.

Patients and methods

The patients had all sustained non-missile injuries, over 80% being the result of road accidents. Males were five times more common than females, and 68% of the patients were younger than 20 years. A fractured skull was found in 42% and hemiparesis in 53%. All of the patients were admitted to the regional neurosurgical unit in Glasgow, to which patients are referred from the primary surgical wards of other hospitals, because they are considered to need special investigation or treatment. All patients were severely injured by the criterion of the international collaborative study (Jennett *et al.*, 1977b)—that is, they were in coma for at least six hours, coma being defined as the inability to obey commands, to utter words, or to open the eyes. Only two-thirds had their first scan within 24 hours of injury, and by the time the first scan was done, 42% of the patients were out of coma by this definition.

In 10 patients angiography was performed before a scan was done, usually because the scanner was not immediately available, and an urgent investigation was considered necessary; these angiograms were all normal. General anaesthesia was employed in 60% of the patients in order to ensure scans of good diagnostic quality; inthree patients, however, the quality of the scans was suboptimal but was regarded as sufficient to exclude a significant intracranial haematoma. Patients whose scan showed any evidence of an extracerebral collection of blood or the presence of a circumscribed

Address for correspondence and reprint requests: Professor Bryan Jennett, Department of Neurosurgery, Institute of Neurological Sciences, Southern General Hospital, Glasgow G51 4TF.

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intracerebral haematoma were excluded from the present series, even when surgery was not considered necessary. The remaining abnormalities were classified into five categories: (i) lateral shift of the ventricles; (ii) a compressed ventricular system, suggestive of diffuse swelling of the brain; (iii) localised areas of reduced density, interpreted as "oedema" or as infarction; (iv) areas of mixed increased and decreased density, interpreted as contusions; (v) other abnormalities, most often evidence of blood in the ventricles or in the Sylvian fissures.

The outcome six months after injury was classified according to a scale previously described (Jennett and Bond, 1975). There were 19 deaths, and in 15 of these necropsy and detailed neuropathological examination were carried out.

Results

The initial scan, although not disclosing a haematoma, was abnormal in 62% of the 60 patients. Some patients had more than one abnormality (Table 1). More than a third of the abnormal scans showed narrowing of the ventricles, sometimes to the point of being scarcely discernible; when the ventricular system could be visualised in these cases there was no shift. The brain parenchyma was either normal or showed homogeneously reduced density in such cases, and these patients were all young, seven being 5, seven aged 5–13, and one aged 17 years. Areas of mixed reduced and increased density were the most common other findings, and in some cases these were associated with lateral shift. Abnormal scans were found somewhat more often in patients who were still in coma or who had a skull fracture but were *less* common in patients with hemiparesis than in those without (Table 2).

Repeat scans could not be performed routinely because of limited availability of the scanner, but they were done in 15 patients. In four cases the scan was initially abnormal but was normal when repeated at 1, 2, 15, and 27 days. In seven cases the repeat scan showed continuing abnormality. In four patients whose initial scan was normal, it was still normal when done again. During the study there were two patients in whom a large intracerebral haematoma was found on repeat scan; in one of these the initial scan on the previous day had shown only a small area of contusion (Fig. 1), while in the other case the initial scan, also the day previously, had been considered normal. Both of these patients had deteriorated dramatically and it was this which had led to the scan being repeated. Both died in spite of surgical intervention. Because they had intracranial haematomas, albeit only on their second scan, they are not included in the present analysis.

Table 1 *Frequency distribution of 41 scan abnormalities in 37 patients*

Abnormality	*Patients*	
	Number	%
Small ventricles	15	37
*Mixed density change	8	20
Lateral shift only	6	14
*Local reduced density	5	12
Other findings	7	17

*In each of these categories two patients had lateral shift also.

Table 2 *Incidence of abnormal CAT scans soon after head injury*

Status of patient	*Number of patients*	*Abnormal*	
		Number	%
Out of coma	25	13	52
Still in coma	35	24	69
No skull fracture	34	18	53
Skull fracture	26	19	73
No hemiparesis	28	19	70
Hemiparesis	32	18	56
No hemiparesis + no skull fracture	18	11	61
Hemiparesis + skull fracture	16	10	63

None of the differences in incidence between pairs is significant at 0.05.

The outcome distribution six months after injury was similar in this series of patients to that found in the international collaborative head injury study for patients without intracranial haematoma (Table 3). The outcome was much better for patients who had emerged from coma by the time their scan was done, but it was only marginally better for those with a normal as distinct from an abnormal CAT scan (Table 4). An abnormal CAT scan did not appear to influence the outcome either in patients already out of or in those still in coma. Of 15 patients with compressed ventricles, eight survived and became independent. The necropsy findings in those who died are discussed below.

NEUROPATHOLOGY (Table 5)

Postmortem examinations were undertaken in 15 of the 19 fatal cases. The brains were fixed intact, and then subjected to a comprehensive neurohistological analysis (Adams *et al.*, 1977). These 15 cases did not differ significantly from the rest of the series in respect of age, frequency of skull fracture, or incidence of abnormal scans. The age

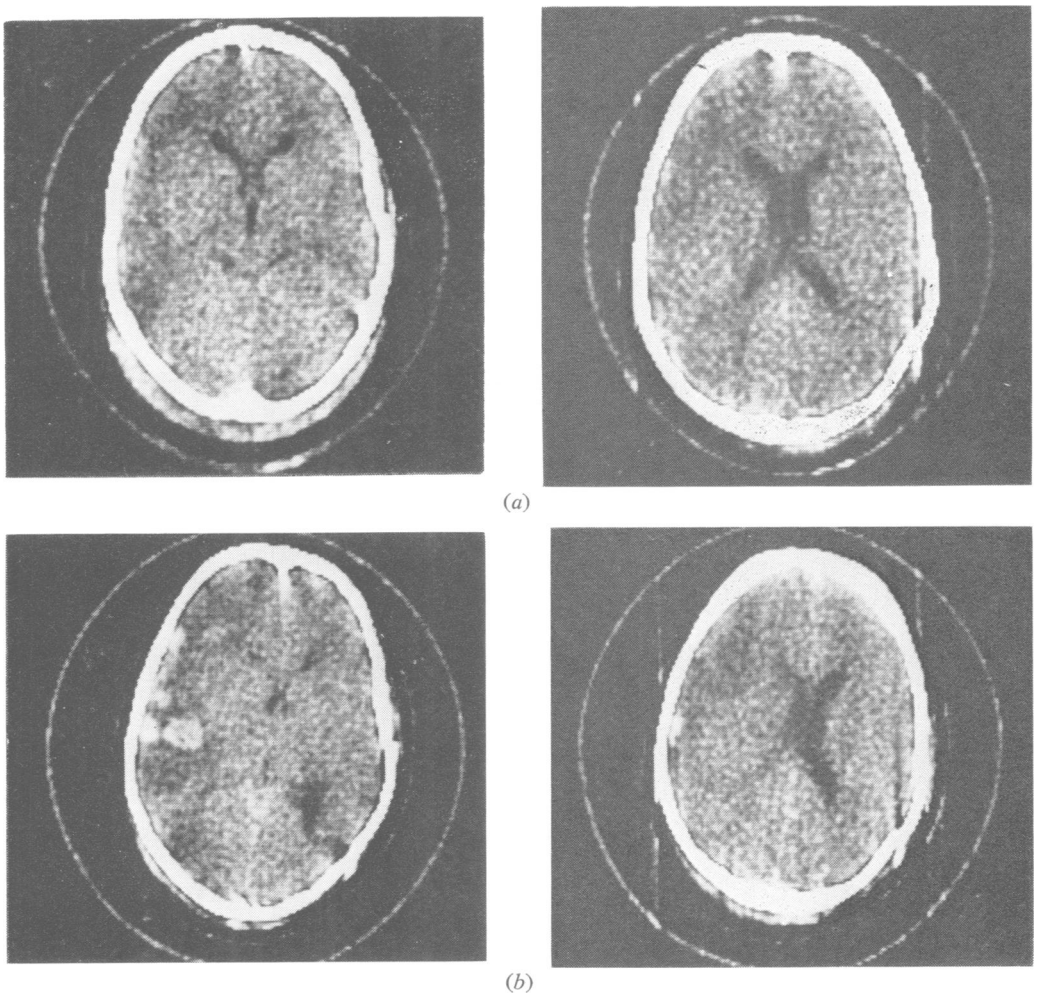


Fig. 1 *(a) CAT scan two and a half hours after injury. There is no shift of the ventricles. An area of mixed reduced and increased density in the left frontoparietal area is suggestive of contusion. (b) CAT scan 24 hours after injury. There is now marked displacement of the ventricles to the right, contusions are obvious and these amount to an intracerebral haematoma in the left posterior frontal region. JMcS, aged 5 years. Fell out of a fourth floor window. He was unconscious on admission, reacting to painful stimuli by abnormal flexion of limbs. Radiography revealed a right parieto-occipital fracture. The patient's condition improved to the state where he was localising to pain, but the next day he started having apnoeic attacks. After the repeat CAT scan, craniotomy revealed a left frontal intracerebral haematoma, and a left frontal lobectomy was performed. No material improvement after operation, died four days after the accident.*

Table 3 *Outcome of severe head injuries*

	Number of patients	*Dead* (%)	*Severe disability* (%)	*Moderate disability/ Good recovery* (%)
Scan series	60	32	8	60
Cases in data bank without haematoma	472	42	8	50

None of the differences between series is significant at 0.05.

Table 4 *Mortality of severe head injuries without haematoma*

	Number of patients	*Normal CAT scan*		*Abnormal CAT scan*	
All cases	60	5/23	22%	14/37	38%
Out of coma	25	0/12	0%	0/13	0%
Still in coma	35	5/11	45%	14/24	59%

Differences in mortality between normal and abnormal scan series are not significant at 0.05.

Table 5 *Summary of neuropathological findings in 15 fatal cases*

Case number	*Age (yr)*	*Survival*	*Time between latest scan and death*	*Scan finding*	*Fracture of skull*	*High ICP*	*Contusions†*	*Diffuse damage to white matter*	*Hypoxic§ brain damage*	*Ventricles*	*Other abnormalities in brain*
1	8	5d	3d	Normal	−	+	+++	−	+	Small	−
2	10	51h	24h	Normal	−	+	+	−	+++ (D)	Small	−
3	21	20d	2d	Normal*	−	+	−	+	+	Normal	−
4	58	5d	11h	Normal	−	−	+	−	−	Normal	Ventriculitis and meningitis
5	59	2m	2m	Normal	−	−	+	+	−	Normal	−
6	3	3d	6h	Small ventricles	+	+	−	−	++ (BZ)	Small	−
7	4	8d	8d	Small ventricles	−	+	−	−	+++ (D)	Small	−
8	5	30h	6h	Small ventricles	+	+	+	−	−	Small	−
9	8	17h	13h	Small ventricles	−	+	+	+	+	Small	−
10	11	5d	5d	Small ventricles	−	+	+++	+	−	Small	−
11	12	44h	36h	Small ventricles	+	+	++	+	++ (BZ)	Small	−
12	2	7d	7d	Reduced density in right cerebellar hemisphere	+	−	−	−	−	Small	Infarct in right cerebral hemisphere
13	9	20h	11h	Shift of ventricles to left	+	+	++	−	+	Shift to left	Swelling of right cerebral hemisphere
14	11	8d	2d	Blood in ventricles	−	+	+	+	+	Blood in ventricles	−
15	29	5d	4d	Right frontoparietal contusions. Shift of ventricles to left	+	+	+++	?‡	+	Shift to left	Swollen right cerebral hemisphere in relation to contusions. Severe secondary brainstem haemorrhage.

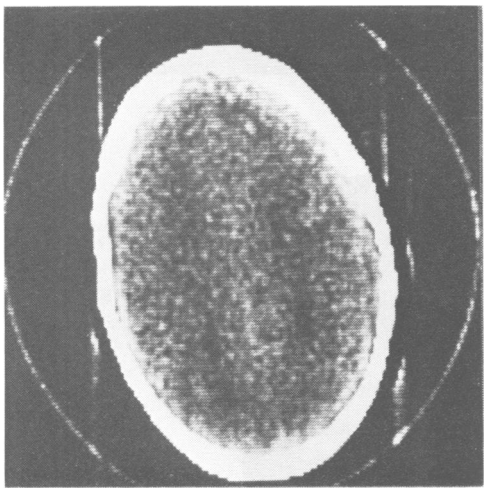
* Blood in ventricles in CAT scan 18 days earlier.
† Graded mainly in depth: + = superficial; ++ = full thickness of cortex; +++ = extend into white matter.
‡ See text for doubt about classification of this case.
§ Based primarily on severity of damage in neocortex: + = mild; ++ = moderate; +++ = severe. D = diffuse; BZ = boundary zone type.
ICP = intracranial pressure.

range was from 2 to 59 years, and 11 were children aged 12 years or less. Survival ranged from 17 hours to two months after injury. Thirteen of the patients had been unconscious from the moment of injury until death. The two who "talked" (Reilly *et al.*, 1975) were cases 2 and 4 (Table 5) where the principal neuropathological findings were intracranial infection and severe hypoxic brain damage respectively.

Cerebral contusions were absent in four cases, minimal (restricted to the outer layers of the cortex) in six, moderate (affecting the full thickness of the cortex) in two, and severe (extending into the white matter) in three (Fig. 2).

Diffuse damage to white matter of immediate impact type (Adams *et al.*, 1977) was identified in six cases (Figs. 3, 4). None of these patients had talked at any time after their injury, and not one had a fracture of the skull. Contusions were absent or minimal in four, and moderate or severe in two. There was probably a seventh patient (case 15, Table 5) with diffuse damage to white matter but this could not be confirmed beyond doubt because of the presence of severe secondary brainstem damage.

Hypoxic damage in the cerebral cortex of the type we have defined elsewhere (Graham *et al.*, 1978) was identified in 10 cases. This was severe



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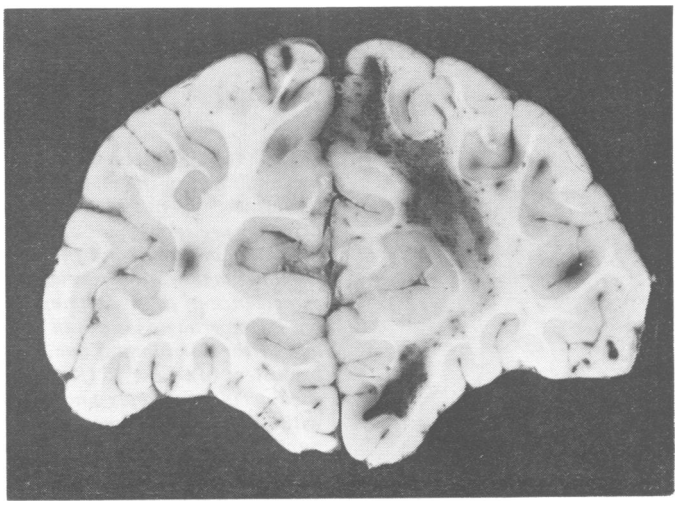

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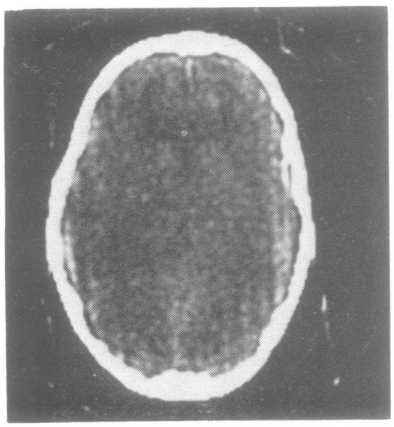
Fig. 2 *(a) CAT scan 48 hours after injury, interpreted as showing no definite abnormality. (b) Pathology: there are severe haemorrhagic contusions extending into the white matter at the right frontal pole. Case 1 (MMcN) aged 8 years. Pedestrian road accident (mentally defective). Deeply unconscious, pupils reacting, no focal signs, no skull fracture, no other injuries. Died on the fifth day.*



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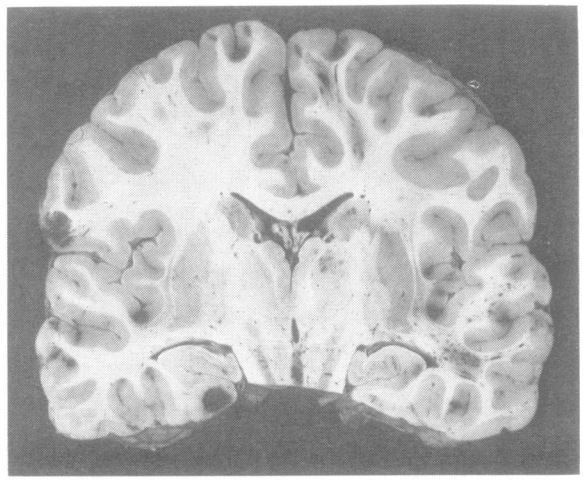

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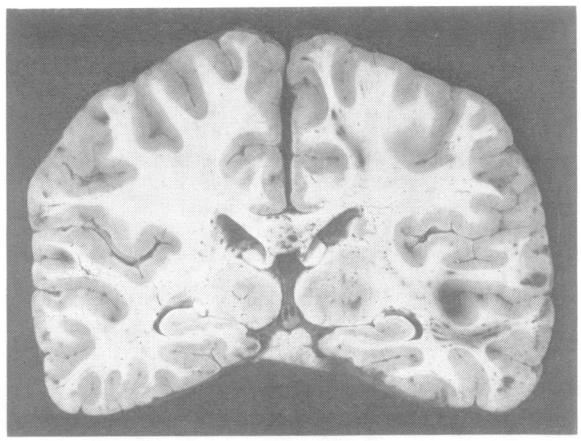

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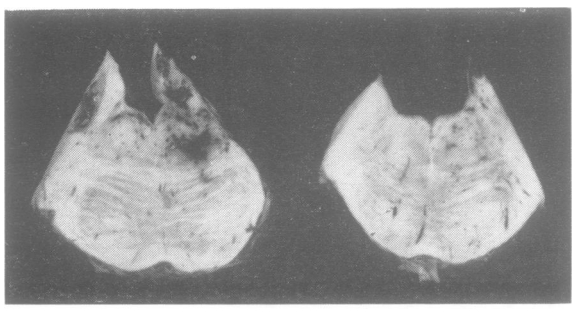
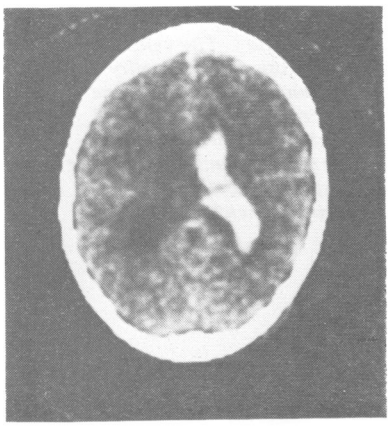


(d)

Fig. 3 (a) *CAT scan on day of injury. The ventricles are small and in the midline, and no parenchymal lesions were identified at any level. The laterally placed densities are movement artefacts. Pathology: the ventricles are small and there are numerous haemorrhagic contusions in the cortex (b) and (c); as evidence of diffuse damage to white matter, there are haemorrhagic lesions in the corpus callosum (c) and in the dorsolateral quadrant of the rostral brainstem (d). Case 10 (KA) aged 11 years. Pedestrian road accident. Deeply unconscious from beginning with extensor motor responses in the limbs, unequal pupils, no skull fracture. Fractured femur and pubic ramis; also silent, rigid abdomen which yielded blood on peritoneal lavage but no ruptured viscus on laparotomy. Died five days after injury.*



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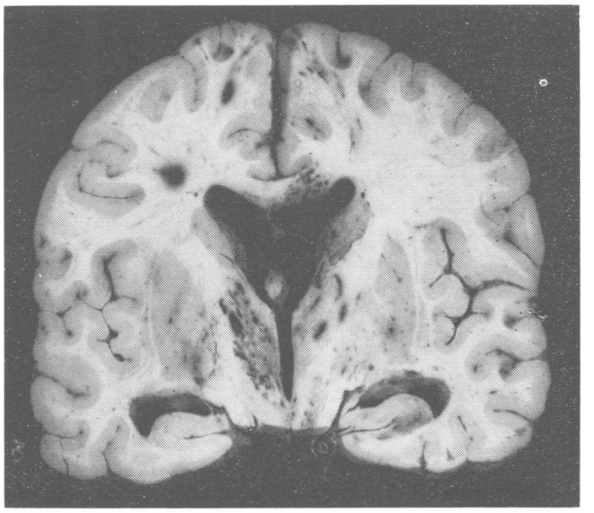

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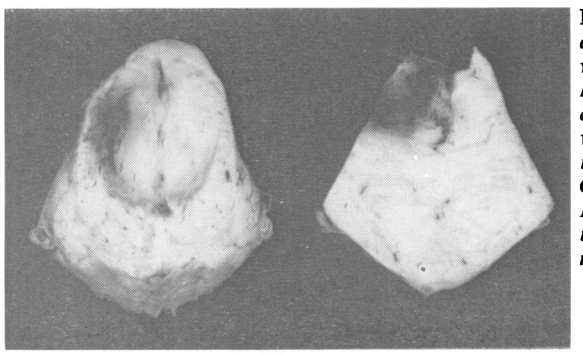


(*c*)

Fig. 4 (*a*) *CAT scan on day of injury. There is an almost complete blood cast of the right lateral ventricle. No parenchymal brain lesion detected. Pathology: there is a large haemorrhagic lesion in the corpus callosum* (*b*) *and there was blood in the ventricles; there is also a large haemorrhagic lesion in the dorsolateral quadrant of the rostral brainstem* (*c*). *Case 14* (*AM*) *aged 11 years. Pedestrian road accident. Deeply unconscious with extensor motor responses in the limbs, pupils reacting, no skull fracture; only other injury was a fractured clavicle. Died on eighth day.*

and diffuse in two, and moderately severe and of boundary zone type in two. One of the two latter cases had been severely hypotensive on admission to hospital.

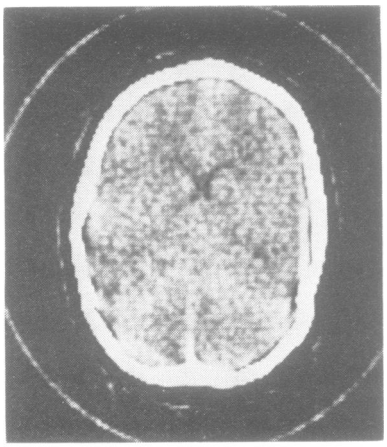
Neuropathological evidence of a high intracranial pressure, using the criteria we have already defined (Adams and Graham, 1976) was found in 12 of the 15 cases despite the absence of any case with an acute intracranial haematoma.

The ventricles were small in all of the cases in whom the CAT scan had shown compressed ventricles (Table 5, cases 6–11). Three of these cases had diffuse damage to white matter (Fig. 3), in two there was moderate or severe hypoxic brain damage, while the sixth (case 8) could only be classified as an example of "idiopathic brain swelling" (Fig. 5), a well-recognised entity in children (Lindenberg *et al.*, 1955; Adams, 1975).

CORRELATION BETWEEN CAT SCAN AND NEUROPATHOLOGICAL FINDINGS

Two factors limit the comparison of scans with pathological findings. One is the interval between scanning and necropsy, the other the difficulty of matching scan cuts with brain slices. Fixed brains can be sliced in approximately the same planes as the CAT scans but since so much vital information is impossible to identify in such slices, we have adhered to conventional coronal sections in order to maintain continuity in our pathological studies on head injuries over many years.

The most striking feature was the severity of the



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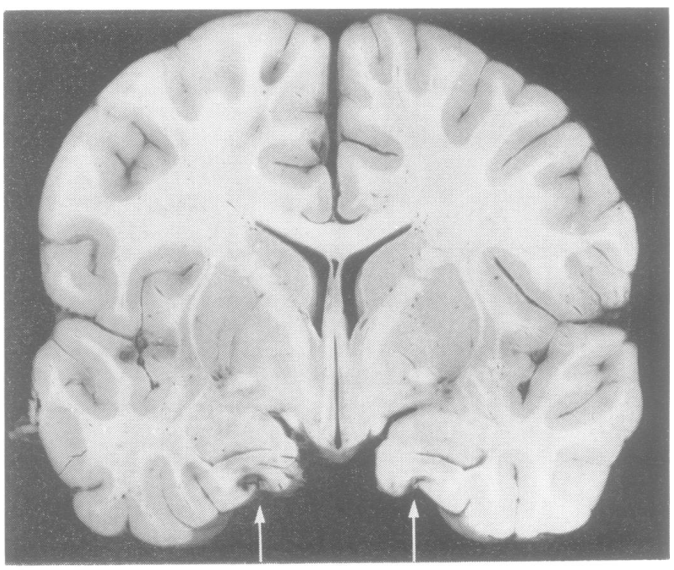

(b)

Fig. 5 *(a) CAT scan on day of injury. The ventricles are small and in the midline, no other abnormality identified. (b) Pathology: the ventricles are small and there are bilateral tentorial herniae (arrows). Case 8 (TMcP) aged 5 years. Pedestrian road accident. In coma from beginning, pupils reacting and limbs flexing at first; limbs became less responsive and pupils fixed after 20 hours. Fractured femur, clavicle, mandible, and skull. Died at 30 hours.*

brain damage found in the five cases with normal scans and in the six with small ventricles. No parenchymal lesion was detected in the scans of the six patients with severe impact damage to white matter, but two had blood in the ventricles soon after injury (Fig. 4). It is likely that the lesions in the corpus callosum and in the dorsolateral quadrant of the rostral brainstem, which are such a consistent pathological finding in such cases, might be shown up in scans if a tomographic cut happened to be at the right level. But we did not see such lesions on any of the six scans from the patients with this type of brain damage, nor in another who had a particularly large lesion in the corpus callosum. This case was not included in the present analysis because she was seen only in consultation a month after injury when she was in a vegetative state. A scan then showed only some ventricular dilatation, but at necropsy the classical lesions of diffuse white matter damage were seen (Fig. 6).

In two of the three cases which were found post-mortem to have severe contusions—that is, contusions extending through the full thickness of the cortex into the white matter (Fig. 2)—the contusions had not been identified in the CAT scans. None of the less severe contusions was seen in the CAT scans.

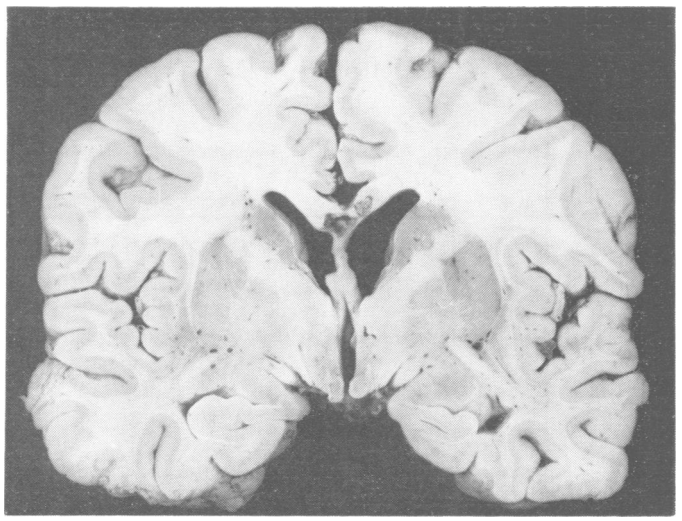

Fig. 6 *CAT scan four weeks after injury showed some ventricular dilatation but no parenchymal abnormality in cerebral or posterior fossa cuts. Pathology: there is a large organising and partly cystic lesion in the corpus callosum which was not identified in the CAT scan. MMcV, aged 16 years (not in consecutive series). Fell down lift shaft. Deeply unconscious with extensor limb responses, no skull fracture. Haemopneumothorax, ruptured right kidney, lacerated liver and ovary. Blood pressure 90/60 mmHg before resuscitation and laparotomy. Mechanical ventilation (tracheostomy) for four weeks, by which time she was in a vegetative state and had developed a series of epileptic fits. Died three months after injury.*

There was no case in the series in which an intracranial haematoma was disclosed after death. The two cases which developed delayed intracerebral haematomas, which were detected on repeat scanning (Fig. 1) and operated on, both died. At necropsy after such surgery it is not easy to determine what the previous state of the brain had been, but we concluded that each of these patients had had significant contusions.

The pathological lesions which are typical of raised intracranial pressure, of diffuse white matter damage, and of recent hypoxic brain damage are either small or only recognisable at microscopic level, and it is therefore, not surprising that they are not evident on CAT scanning. Their presence may be suspected from circumstantial evidence, both clinical and from the scan. But it is clear that caution is called for in drawing conclusions from the scan alone, particularly in the direction of regarding a normal or near normal scan as excluding severe brain damage.

Discussion

The value of CAT scanning in the recognition and localisation of intracranial haematoma is undeniable (Galbraith *et al.*, 1977). How much scanning can help in the diagnosis and prognosis of other traumatic lesions has still to be defined. However, it is already clear that it makes it possible to detect during life the presence of some pathological lesions which could previously only be suspected until necropsy. These include contusions, oedema, infarction, and intraventricular blood. Moreover, serial scanning enables the evolution and resolution of these lesions to be visualised, which was previously impossible.

One of the earliest reports of head injuries examined by CAT scanning was from Boston (Merino-de Villasante and Taveras, 1976). This stated that abnormalities on scanning increased proportionately with clinical severity. All patients with injuries "more severe than contusion" had abnormal scans but the authors admitted that their classification of clinical severity lacked "a very precise characterisation of each group." A larger and more representative series was reported the following year from Sacramento (French and Dublin, 1977). While the more severely injured more often had abnormal scans, no less than 44% of those with "deep coma and abnormal motor movements" had a normal scan, as did 15% of those in coma with lateralising signs.

Our experience is much closer to that of Sacramento than that of Boston. All the Glasgow patients were in coma by a rigorous definition, and yet 38% had normal scans; 26% of fatal cases had no definite CAT abnormality. In this series only four patients had contrast enhancement because this was not considered likely to contribute to management in the acute stage after injury. It may be argued that some scans which were regarded as normal might have shown abnormalities if more extensive and careful examination had been carried out, with more brain slices, including posterior fossa cuts, and contrast enhancement in every case. Certainly more subtle abnormalities may be revealed by careful retrospective review, including evidence available from serial scans over several weeks, as recently reported by Zimmerman *et al.* (1978a). But head injuries present urgent problems, often at night, and not only the scan-

ning but also the interpretation must often depend on junior radiographic and medical staff, without the benefit of an experienced radiologist. It is in these circumstances that clinicians need to know what conclusions to draw when a scan is normal, or when various abnormalities are seen which are less obvious and less easy to interpret than a haematoma.

The neuropathological findings in the 15 cases in this series exemplified the type of brain damage one would expect to find in fatal cases without an intracranial haematoma, on the basis of a study of a large number of consecutive head-injured brains already analysed and reported (Adams, 1975). Thus many did not have a fracture of the skull, and the postmortem examination frequently revealed neither severe contusions nor lacerations of the brain. There were six cases—possibly seven—with severe diffuse damage to the white matter of immediate impact type, and four with moderate or severe hypoxic brain damage. There were two cases with swelling of one cerebral hemisphere in relation to contusions, and one with idiopathic post-traumatic brain swelling.

Although this study confirms the reliability of CAT scanning in excluding an intracranial haematoma it cannot yet be claimed that there are characteristic scan appearances for various other types of traumatic brain damage. Indeed the CAT scan may be normal even when severe and irreversible brain damage has occurred, and a normal scan cannot, therefore, be equated with a good prognosis. If, however, the scan is interpreted in the context of the clinical picture it may be possible to derive useful data from a normal or near normal scan, other than the exclusion of gross lesions.

The group of patients with small, undisplaced ventricles is particularly interesting. It was the most frequent single abnormality, and on reviewing the scans of other patients which had been reported as normal there were some which had relatively small ventricles. There are as yet no clearly defined criteria for the range of size in normal individuals at various ages, and caution is needed in interpreting this finding until more data are available and methods of measuring ventricular size and establishing the normal range have been evolved. Six cases with small ventricles on scanning came to necropsy; only one had severe contusions while all had marked swelling of the white matter. Other findings in such cases were diffuse impact damage of white matter, and severe hypoxic brain damage. Bruce *et al.* (1978) reported small ventricles in one-third of their series of severe head injuries in children. In the present series 14 out of 15 patients with this finding were under 14 years of age, and the exception was only 17 years old. Bruce and his colleagues postulated that this appearance was often the result of cerebrovascular dilatation, on the grounds that the brain parenchyma showed increased density and that in cases where blood flow was measured it was increased. Our findings, however, indicate that this CAT scan appearance can be associated with a variety of pathological lesions.

Only two patients whose initial scans did not show an intracranial haematoma subsequently developed this complication; each showed marked clinical deterioration which led to the repeat scan (Fig. 1). When all patients in coma on admission were routinely scanned in one American series (Sweet *et al.*, 1979) almost all significant intracranial haematomas were found to be already visible on the scan done soon after admission. Nevertheless our two cases emphasise that the absence of a haematoma on an early scan does not exclude its later development. This could be a matter of some importance if large numbers of patients were to be scanned soon after injury as a means of deciding whether complications were likely to develop. How many later developing lesions would have been revealed if repeated scanning had been routine is at present not known. No doubt some lesions which were not of clinical significance might have been found, as we have previously reported (Jennett *et al.*, 1977a), but no significant expanding lesions were found at necropsy which had not been diagnosed on at least one scan. When pursuing a policy of repeated scanning French and Dublin (1977) encountered several focal collections, particularly subdural hygromas, some time after injury. When such lesions have not caused obvious clinical deterioration their significance is doubtful. Their removal did not produce dramatic improvement in any of the Sacramento cases.

Computerised tomographic scanning provides more data about recently head-injured patients than any other investigation, and the only hazard to the patient comes from the use of sedation or anaesthesia, which is sometimes necessary if good quality scans are to be obtained in unco-operative patients. Its most useful role still appears to be the detection of intracranial haematoma. This study shows that a normal scan, or one which shows small but undisplaced ventricles, does not exclude severe impact damage to white matter, extensive secondary hypoxic brain damage, or raised intracranial pressure. Moreover, patients with marked focal signs (hemiparesis) not infrequently have a normal scan. This has also been the experience of others. Lanksch *et al.* (1978), reviewing 910 recently

head-injured patients, emphasised that unconscious patients with marked morphological lesions could have normal scans. They concluded that "utilising only CAT findings it is neither possible to predict the patient's outcome nor to decide whether to intervene neurosurgically." Zimmerman *et al.* (1978b) have reported that the small haemorrhagic lesions, which are commonly associated with diffuse white matter damage of impact type, can often be seen on the scan if examinations are repeated with thin cuts. However, even with careful retrospective review, including serial scans, they found a normal scan in 30% of patients without haematoma who had impaired consciousness (Zimmerman *et al.*, 1978a).

Until more experience is available, with careful comparison between CAT scans and what is found in the brain at operation or at necropsy, it must remain uncertain what significance attaches to certain findings—and particularly to a normal or near normal scan. The contribution of the scan to prognosis as distinct from diagnosis, may be limited to patients with certain combinations of clinical features and scan appearances. As with other laboratory investigations CAT scans will be most useful only when they are intelligently interpreted in the light of the whole clinical picture.

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