Serial MRI and neurobehavioural findings after mild to moderate closed head injury

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Abstract

Fifty patients who sustained mild to moderate closed head injury (CHI) underwent a CT scan, MRI, and neurobehavioural testing. At baseline 40 patients had intracranial hyperintensities detected by MRI which predominated in the frontal and temporal regions, whereas 10 patients had lesions detected by CT. Neurobehavioural data obtained during the first admission to hospital disclosed no distinctive pattern in subgroups of patients characterised by lesions confined to the frontal, temporal, or frontotemporal regions, whereas all three groups exhibited pervasive deficits in relation to normal control subjects. The size of extraparenchymal lesion was significantly related to the initial Glasgow Coma Scale score, whereas this relation was not present in parenchymal lesions. One and three month follow up MRI findings showed substantial resolution of lesion while neuropsychological data reflected impressive recovery. The follow up data disclosed a trend from pervasive deficits to more specific impairments which were inconsistently related to the site of brain lesion. These results corroborate and extend previous findings, indicating that intracranial lesions detected by MRI are present in most patients hospitalised after mild to moderate CHI. Individual differences in the relation between site of lesion and the pattern of neuropsychological findings, which persist over one to three months after mild to moderate CHI, remain unexplained.

Since the application of MRI to neurosurgical patients several reports¹⁻⁷ and our quantitative study of 20 patients⁸ have shown that this technique is more sensitive than CT scanning in detecting intracranial abnormalities after closed head injury (CHI). In view of our preliminary description⁸ of individual patients with mild to moderate CHI exhibiting distinctive neurobehavioural sequelae associated with frontal *v* temporal lobe hyperintensities, we extended this study to 50 cases. We evaluated the neuroanatomical distribution of abnormalities visualised by MRI in patients sustaining mild to moderate CHI; resolution of these apparent lesions over one to three months; and the relation between cerebral site of lesion and neurobehavioural sequelae.

Patients

PATIENT POPULATION

Fifty patients who met criteria for mild (40) or moderate (10) injury according to their lowest Glasgow Coma Scale (GCS)⁹ score were selected from consecutive admissions to the neurosurgery service for CHI (table 1). The criteria for mild impairment of consciousness included an initial period of unconsciousness for 20 minutes or less and no deterioration from an admission GCS score of 13-15;^{10 11} whereas moderate impairment of consciousness was defined according to an admission (or lowest) GCS score 9 to 12 with no deterioration below this range.¹² Although the GCS score was 7 during helicopter evacuation for one of the patients whose injury was classified as moderate, his level of consciousness rapidly improved to a GCS score of 11 in the emergency room. Patients previously admitted to hospital for head trauma or with a history of another neuropsychiatric disorder, alcoholism, or drug abuse were excluded from the study. Patients and their families consented to participate in this investigation. To evaluate neurobehavioural outcome of these head injured patients, 21 control subjects were recruited from the community by newspaper advertisements. Respondents selected were comparable to the patients in their distribution of demographic features (table 1) and had no history of neurological disease or hospitalisation for head injury or major psychiatric disorder.

CT AND MRI

The protocol specified completion of MRI within 72 hours after the first (admission) CT scan and after any operations. In cases in which MRI was delayed (for example, due to agitation) a second CT scan was obtained to provide a concurrent comparison of these neuroimaging techniques. An MRI was scheduled for moderately injured patients when they no longer needed life support equipment for clinical management (typically, on the day of transfer from the neurological intensive care unit to a hospital room).

The CT scans were obtained on a General Electric 8800 (General Electric Company, Medical Systems Division, Milwaukee, WI) in the transaxial plane without contrast enhancement. MRI was performed on a Teslacon (Technicare, Cleveland, OH) system with a 0.6-Tesla magnetic field strength with a proton resonant frequency of 25.4 MHz. Images were acquired in contiguous 8 mm slices in transaxial and coronal planes with two spin-echo sequences: a repetition time (TR) of 500 msec

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Table 1 Demographic and clinical features of patients with closed head injury and normal control subjects*

	Glasgow Coma Scale score		Total (n = 50)	Controls (n = 21)
	13-15 (n = 40)	9-12 (n = 10)		
Mean (SD) age (years)	25.9 (8.0)	27.4 (9.3)	26.2 (8.2)	25.8 (6.4)
Mean (SD) education (years)	12.0 (2.0)	12.3 (2.7)	12.1 (2.1)	12.0 (1.3)
Male	27	7	34	13
Female	13	3	16	8
Cause of injury				
MVA	20	8	28	
Assault	6	1	7	
Bicycle	2	0	2	
Sports	1	0	1	
Fall	2	0	2	
Other	9	1	10	
Mean (SD) GCS score	14.4 (0.66)	9.7 (0.82)	13.4 (2.0)	
Mean (SD) time to obey commands (days)	0.009 (0.01)	1.33 (1.25)	0.27 (0.75)	
Mean (SD) duration of PTA (days)	1.85 (3.21)	9.44 (8.16)	3.24 (5.32)	
Mean (SD) injury test (days)	5.95 (4.20)	11.8 (8.0)	7.12 (5.59)	
Surgical treatment				
Evacuation of				
EDH	0	2	2	
SDH	0	2	2	
Repair depressed fracture	0	2	2	
Insert Richmond bolt	0	1	1	

MVA = motor vehicle accident; EDH = epidural haematoma; GCS = Glasgow Coma Scale; SDH = subdural haematoma.
 *Overall comparison of head injured patients v controls by one way analysis of variance revealed no differences in age or education.

and echo delay time (TE) of 32 msec; and a TR of 2000 msec and a TE of 60 and 120 msec. A board-certified radiologist interpreted the neuroimaging findings independently of the neurobehavioural data. The CT and MRI findings, which were reviewed on different days, were coded on research forms which were compatible with computer entry. Intracranial abnormalities on MRI were coded as lesions provided that they were present on both the transaxial and coronal T₂-weighted images (spin echo sequences: TR 2000 msec, TE 60 and 120 msec). The radiologist estimated the volume of each lesion in cubic centimetres by placing a grid on the area of interest. In cases with lesions consisting of more than one component (such as ring lesions) the constituent parts were summed to estimate a total volume.

Neurobehavioural examinations were performed at baseline during the initial hospitalisation (within 24 hours after the MRI scan) after resolution of post-traumatic amnesia¹³ and at one and three months after injury (table 1). The MRI (but not CT) was repeated on each follow up examination. Of the total series, 39 patients (78%) returned for the one month follow up examination, whereas 11 patients (22%) declined or were unable to return. Comparison of these subgroups disclosed no difference in demographic features. Patients who returned, however, had slightly less impaired consciousness as reflected by their lowest GCS score (mean 13.8, SD 1.7) than patients who did not return at one month (mean 12.2, SD 2.6, $F(1,48) = 5.95$, $p < 0.02$). Twenty six patients (52%) returned for both the one month and three month follow up examinations.

NEUROPSYCHOLOGICAL TESTS

In view of the vulnerability of the orbitofrontal and temporal lobes to CHI^{14,15} the neurobehavioural examination emphasised func-

tions dependent on the integrity of these areas. To evaluate frontal lobe functioning, the modified card sorting test¹⁶ of concept formation and flexibility in problem solving was given while measures of verbal fluency^{17,18} (generation of words beginning with a designated letter within a time limit of 60 seconds) and design fluency¹⁹ (drawing novel designs under a time limit of five minutes) were also administered. Temporal lobe functioning was evaluated by the verbal selective reminding procedure,²⁰ which tested recall of 12 words administered over 12 trials followed by a delayed recall trial 30 minutes later. Long term memory for relatively non verbal information was evaluated by recall of a spatial configuration of eight marbles on a board consisting of 16 locations.²¹ On each of 12 trials the examiner corrected false-positive errors (marbles placed in incorrect locations) and reminded the patient of locations that had been missed on the preceding trial. Similar to verbal selective reminding, retention of the spatial locations was tested 30 minutes after completion of the 12 recall trials. Recognition memory for geometric and random designs²² was also assessed. The token test¹⁸ was used to assess the potential contribution of receptive language deficit to cognitive and memory performance.

Results

NEUROANATOMIC DISTRIBUTION OF ABNORMALITIES ON CT AND MRI

Of the total series 40 patients (80%) had intracranial abnormalities (hyperintensities) visualised by the initial MRI, including 13 (26%) who had normal CT findings, 26 (52%) in whom MRI showed more lesions than were seen on CT, and a single patient who had identical findings on CT and MRI. In contrast, nine patients (18%) had lesions on CT that were not seen on MRI, including eight

Figure 1 Incidence and neuroanatomical distribution of parenchymal and extraparenchymal lesions seen on MRI and CT at baseline plotted separately for patients with GCS scores in 13–15 (a) v 9–12 (b) ranges.

parenchymal lesions, three extraparenchymal (that is, subdural and extradural) lesions, and two cases of subarachnoid haemorrhage. Ten patients (20%) had normal images on both CT and MRI.

Figure 1 depicts the number, type, and neuroanatomical distribution of lesions shown by CT and MRI at baseline for patients with GCS scores of 13–15 and 9–12. Of the 144 hyperintensities which were present on MRI, parenchymal abnormalities in the frontal and temporal region predominated with relatively few lesions in the parietal and occipital areas. Figure 1 also shows that lesions involving deep grey structures were rare in this series.

SIZE OF FOCAL LESION ON MRI IN RELATION TO IMPAIRED CONSCIOUSNESS AND TIME SINCE INJURY
Figures 2a and 2b plot the mean parenchymal and extraparenchymal lesion size separately for patients who had mild v moderate impairment

of consciousness and underwent MRI at all three examinations. A dissociation between lesion size and impairment of consciousness was seen depending on whether the parenchymal or extraparenchymal compartments were affected. As shown in figure 2a extraparenchymal lesions at baseline were larger in patients who had moderate impairment of consciousness compared with patients with GCS scores in the 13–15 range ($F(1, 38) = 4.11$, $p < 0.05$). This relation between impaired consciousness and extraparenchymal lesion size was confirmed at one month ($F(1, 30) = 8.99$, $p < 0.005$) and three months ($F(1, 32) = 4.38$, $p < 0.05$). In contrast the groups defined by GCS score did not differ in size of parenchymal lesion at baseline or follow up (figure 2B). The group with GCS scores of 9–12 included large extraparenchymal lesions (50 cc, 100 cc, 150 cc) that were not surgically evacuated in three patients.

Figure 2 Mean size of (a) extraparenchymal lesions and (b) parenchymal lesions at baseline, one month, and three months in head injured patients sustaining mild to moderate impairment of consciousness according to Glasgow Coma Scale (GCS). Patients had lesions on baseline MRI. For GCS scores 13–15, baseline $n = 9$, one month $n = 5$, three months $n = 4$.

Table 2 Relationship between site of cerebral abnormality on MRI and neurobehavioural findings at baseline

	Localisation of injury			Controls (n = 21)	F value
	Frontal (n = 8)	Temporal (n = 5)	Frontotemporal (n = 11)		
Severity of injury					
GCS score	13.62 (1.92)	13.20 (2.04)	13.80 (1.16)		0.24
Time to obey commands (days)	0.37 (1.05)	0.60 (1.34)	0.03 (0.08)		0.86
Duration of PTA (days)	2.00 (2.44)	6.80 (10.98)	3.54 (5.00)		0.89
Lesion size (cc)					
Parenchymal	16.37 (17.54)	8.80 (6.70)	18.50 (17.18)		0.65
Extraparenchymal	9.12 (13.53)	4.00 (8.94)	12.36 (10.74)		0.92
Frontal lobe functioning					
Card Sorting					
Total correct	33.38 (7.78)	25.80 (10.35) ^a	28.00 (10.81) ^b	36.43 (2.52) ^{ab}	4.94**
% Perseverative errors	5.11 (6.59)	14.61 (15.77)	13.39 (17.21) ^a	2.21 (3.30) ^a	3.80*
Word association score	27.13 (10.20) ^a	23.20 (14.72) ^b	23.36 (9.93) ^c	43.33 (11.28) ^{abc}	10.69***
Perseverative errors	1.00 (2.14)	1.20 (1.64)	1.91 (4.21)	0.33 (0.80)	1.08
Design fluency (fixed)					
% Perseverative errors	26.08 (26.24) ^a	18.22 (13.73)	13.74 (11.25)	3.12 (5.55) ^a	6.28**
Memory					
Recall					
Verbal CLTR (total words)	44.88 (45.80) ^a	39.00 (42.77) ^b	37.00 (34.74) ^c	119.81 (17.76) ^{abc}	24.22***
Verbal 30' recall	6.25 (3.37) ^a	4.60 (5.27) ^b	6.50 (5.19) ^c	11.76 (0.43) ^{abc}	11.51***
Spatial CLTR					
(total locations)	67.87 (31.97)	59.80 (27.68)	65.80 (33.37) ^a	89.95 (8.78) ^a	4.24**
Spatial 30' recall	6.75 (1.38)	6.80 (1.64)	6.77 (1.30)	7.80 (0.67)	3.19*
Recognition					
Geometric (net score)	24.57 (4.85)	15.00 (14.25) ^a	16.00 (8.50) ^b	25.71 (3.75) ^{ab}	6.51***
Random (net score)	11.28 (5.40)	11.75 (11.14)	3.55 (5.87) ^a	17.47 (5.36) ^a	11.00***
Language comprehension					
Token test	40.00 (4.44)	39.40 (5.46)	38.90 (4.50) ^a	43.33 (0.97) ^a	5.17**

*p < 0.05.

**p < 0.01.

***p < 0.001.

GCS = Glasgow Coma Scale; PTA = post-traumatic amnesia; CLTR = consistent long term retrieval.

F value pertains to overall effect of groups whereas common superscripts (^{abc}) denote significant contrasts between two groups.

Restricting the analysis of lesion to those patients who returned for follow up examination showed significant reductions in lesion size, particularly during the first month, whereas changes between one and three months were less impressive (see figs 2a and 2b). Overall analysis of parenchymal and extraparenchymal lesions showed that the change in lesion size from baseline to one month was highly significant ($F(1, 36) = 8.32$, $p < 0.006$). In contrast the reduction in lesion size between one and three months fell short of significance. Although the interaction between severity of impaired consciousness (GCS score 9–12 *v* 13–15) and occasion was significant ($F(2, 51) = 3.39$, $p < 0.04$), a “floor” effect was present as the lesions were essentially resolved by three months irrespective of the initial GCS score. The triple interaction of GCS score with occasion and type of lesion was not significant.

SITE OF PARENCHYMAL ABNORMALITY IN RELATION TO NEUROBEHAVIOURAL SEQUELAE AND RECOVERY

In view of the predominance of focal hyperintensities in the frontal and temporal regions, subgroups of CHI patients were formed according to sites of lesions on the baseline MRI (table 2). The frontal and temporal lobe groups in table 2 included patients with lesions confined to one of these regions, whereas patients with overlapping lesions were classified as frontotemporal. Table 2 also shows that there were no differences in the acute neurological indices of the frontal, temporal, and frontotemporal groups. Nine patients with lesions affecting the frontal region and seven patients with temporal lobe involvement were deleted from the analysis of site effects because

their lesions extended outside of the fronto-temporal region, including the parietal (5), occipital (5), parieto-occipital (4), internal capsule (1), and superior sagittal sinus (1). The 10 patients who had no evidence of brain lesion on the baseline MRI (10) were also excluded from this analysis.

NEUROBEHAVIOURAL FINDINGS AT BASELINE

Analysis of the baseline data disclosed neuropsychological impairment in all three of the lesion site groups relative to normal subjects (see table 2). Deficits on tests of frontal lobe dysfunction are reflected by a higher percentage of perseverative errors on word association or design fluency, or both, in all three groups. Although performance of the CHI patients on the modified card sorting test tended to fall below the controls, the difference reached significance only for the temporal and frontotemporal lesion groups. Impaired verbal recall (verbal CLTR in table 2) was confirmed for the total CHI series and in all three groups, whereas defective reproduction of a visuospatial array from memory (spatial CLTR) was confined to the frontotemporal group. Table 2 shows that recognition memory for designs was impaired in the temporal and frontotemporal lesion groups. In summary, the baseline findings showed pervasive impairment on tests of cognition and memory without consistent relations between specific neurobehavioural deficits and site of lesion. In view of the possibility that subacute diffuse brain swelling or oedema might have obscured the more restricted effects of focal lesions on the baseline examination, we performed a similar analysis on the MRI and neurobehavioural data collected at one month.

Table 3 Proportion of test scores for executive function and memory domains falling below 95% of controls in patients grouped according to MRI findings at one month

			Type of test at baseline		Type of test at one month		Type of test at three months	
Patient	One month	Baseline	Executive function	Memory	Executive function	Memory	Executive function	Memory
Frontal lesion								
1	F-R	FT-B	0.00	0.75	0.50	0.50	0.50	0.25
2	F-B	F-B	0.50	0.50	0.00	0.00	—	—
3	F-B	O-B	1.00	0.75	0.75	0.50	0.75	0.25
4	F-B	FT-B	0.25	0.50	0.25	0.25	—	—
5	F-L	FT-B	0.50	0.25	0.00	0.25	—	—
6	F-B	F-B	0.75	0.50	0.00	0.25	0.00	—
7	F-L	F-L	0.50	0.25	0.25	0.00	0.00	—
8	F-R	FT-B	0.25	0.50	0.25	0.75	0.00	0.25
9	F-B	O-B	0.25	0.25	0.00	0.50	—	—
Temporal lesion								
1	T-B	O-B	0.25	0.75	0.25	0.25	—	—
2	T-L	T-L	1.00	1.00	0.25	0.25	0.25	0.25
3	T-R	FT-R	0.75	1.00	0.50	0.50	0.50	0.50
4	T-R	FT-B	1.00	0.75	0.50	0.00	—	—
Frontotemporal lesion								
1	FT-B	O-B	1.00	1.00	0.25	0.50	0.25	0.25
2	FT-B	O-B	0.25	0.25	0.25	0.25	—	—
3	FT-B	O-B	0.50	0.50	0.50	0.75	0.25	0.75
4	FT-R	FT-R	1.00	0.50	0.25	0.00	0.50	0.00
Resolved lesion								
1	Resolved	F-L	0.25	0.00	0.00	0.00	0.00	0.00
2	Resolved	FT-B	0.75	1.00	0.50	0.50	0.25	0.50
3	Resolved	T-L	0.50	1.00	0.75	0.50	0.66	0.75
4	Resolved	T-L	0.25	0.25	0.00	0.00	0.00	0.00
5	Resolved	F-L	1.00	0.50	0.50	0.25	0.25	0.00
6	Resolved	O-B	0.00	0.50	0.33	0.25	—	—
7	Resolved	FT-B	0.50	0.75	0.25	0.25	0.50	0.50
8	Resolved	F-B	0.50	0.75	0.50	0.25	—	—
Other lesion								
1	O-L	O-L	0.25	0.25	0.25	0.00	0.25	0.00
2	O-R	O-B	0.50	0.50	0.25	0.25	—	—
3	O-L	O-B	0.00	0.00	0.50	0.00	0.00	0.00
4	O-L	O-B	0.00	0.00	0.50	0.25	0.00	0.00
5	O-B	O-B	0.50	0.25	0.25	0.25	0.50	0.25
6	O-R	T-R	0.50	1.00	0.25	0.25	0.00	0.25
No lesion								
1			0.00	0.50	0.00	0.25	0.00	0.00
2			0.00	0.50	0.50	0.00	0.25	0.00
3			0.25	0.25	0.00	0.00	0.00	0.00
4			0.00	0.25	0.00	0.00	—	—
5			0.50	0.33	0.00	0.00	0.50	0.25
6			0.50	0.25	0.25	0.00	—	—
7			0.25	0.25	0.50	0.00	0.25	0.00
8			0.25	0.25	0.00	0.25	—	—

B = bilateral; F = frontal; L = left; R = right; T = temporal.

NEUROBEHAVIOURAL FINDINGS AT ONE AND THREE MONTHS AFTER INJURY IN RELATION TO SERIAL MRI DATA

Comparison of the one month and baseline MRI findings showed a pattern of changes in the site of lesion (table 3). Frontotemporal hyperintensities tended to resolve to lesions confined to the frontal or temporal regions whereas multifocal lesions changed to frontotemporal lesions. Multifocal abnormalities at baseline (denoted by other lesion group) tended to partially resolve from bilateral to unilateral lesions. Most of the patients with complete resolution of lesions by one month had initial findings restricted to the frontal or temporal region. Analysis of the follow up neurobehavioural data confirmed the persistence of deficits relative to control subjects on several measures of frontal lobe functioning and memory. Site of lesion based on the one month MRI in the 39 patients who returned for follow up often differed from their baseline findings (table 3). Patients whose lesions had resolved were deleted from the comparison of the groups with the one month data. Repeating the group comparisons with the one month data still failed to demonstrate impressive

differences in neurobehavioural functioning (table 4). Similarly, comparison of the frontal, temporal, and frontotemporal lesion groups at three months (not shown) again showed no effect for site of lesion.

Table 3 summarises for individual patients (grouped according to site of lesion at one month) the proportion of test scores which fell below the 5th percentile of the control group on measures of frontal ("executive") function and memory. A trend of multiple deficits on the baseline examination changing to more focused impairments at one and three months after injury was evident. Patients with residual lesions which affected areas outside of the frontotemporal region, however, also exhibited persistent neurobehavioural deficits (table 4). Six of the nine patients whose lesions resolved by one month, nevertheless continued to exhibit deficits on tests of executive and memory functioning. In contrast, none of the seven patients without MRI evidence of an intracranial lesion at baseline or follow up had defective scores on both the executive function and memory domains one month after injury. Comparison of the one month neuropsychological performance of patients with resolved

Table 4 Relation between site of cerebral abnormality on one month MRI and concurrent neurobehavioural findings

	Localisation group				
	Frontal (n = 9)	Temporal (n = 4)	Frontotemporal (n = 4)	Controls (n = 21)	F value
Frontal lobe functioning					
Card sorting					
Total correct	37.35 (1.50)	35.00 (7.61)	35.75 (3.68)	36.42 (2.52)	0.67
% Perseverative errors	1.73 (2.99)	2.60 (3.12)	4.33 (4.77)	2.21 (3.30)	0.59
Word association score	33.22 (8.75)	26.25 (9.53) ^a	27.00 (4.96) ^b	43.33 (11.28) ^{ab}	5.90**
Perseverative errors	0.77 (0.83)	0.25 (0.50)	0.25 (0.50)	0.33 (0.79)	0.89
Design fluency (fixed)					
% Perseverative errors	8.86 (9.96)	5.95 (7.89)	0.00 (0.00)	3.12 (5.55)	1.98
Memory					
Verbal CLTR (total words)	85.77 (33.03) ^a	80.25 (31.47)	74.00 (60.74) ^b	119.80 (17.75) ^{ab}	5.47**
Verbal 30' recall	10.22 (2.58)	10.00 (0.81)	9.00 (3.55) ^a	11.76 (0.43) ^a	4.35**
Spatial CLTR (total locations)	81.55 (27.16)	94.00 (2.16)	95.50 (1.00)	89.95 (8.79)	1.19
Spatial 30' recall	7.77 (0.66)	7.75 (0.50)	8.00 (0.00)	7.80 (0.67)	0.14
Recognition					
Geometric (net score)	25.88 (3.44)	26.50 (2.38)	17.00 (16.52)	25.71 (3.75)	2.55
Random (net score)	14.00 (7.53)	19.50 (9.70) ^a	2.33 (10.01) ^{ab}	17.47 (3.56) ^b	4.99**
Language comprehension					
Token test	42.22 (2.04)	42.50 (2.38)	41.50 (3.00)	43.33 (0.96)	1.90

p* < 0.05.*p* < 0.01.****p* < 0.001.F value pertains to overall effect of groups whereas common superscripts (^{abc}) denote significant contrasts between two groups.

Figure 3a MRI of patient 7 of frontal group (see table 4), 27 year old man whose initial GCS score was 14 after sustaining CHI when struck by tow line under high tension. Baseline MRI performed three days after injury showed area of increased signal (33 cc) in left frontal cortex, interpreted as contusion. Follow up MRI showed clear resolution of lesion. Neurobehavioural findings at one month reflected specific problem in verbal fluency which resolved by three months. (b) MRI obtained from patient 4 of the frontotemporal group (see table 4), 41 year old woman who sustained CHI when struck by car. Despite GCS score of 13 indicating mild impairment of consciousness, post-traumatic amnesia persisted for 17 days. Large predominantly right temporal contusion/haematoma (60 cc) detected on baseline MRI was partially resolved on follow up scans. Patient's initial memory deficit resolved, but she still showed defects on tests of executive function at one and three months.

Table 5 Pearson correlation coefficients (*r*) between duration of post-traumatic amnesia and neurobehavioural performance*

	Baseline (<i>n</i> = 50)		One month (<i>n</i> = 39)		Three months (<i>n</i> = 26)	
	<i>r</i>	<i>p</i> value	<i>r</i>	<i>p</i> value	<i>r</i>	<i>p</i> value
<i>Frontal lobe functioning</i>						
Card sorting						
Total correct	-0.51	0.0002	-0.25	0.13	-0.24	0.23
% Perseverative errors	0.49	0.0004	0.04	0.82	0.23	0.25
Word association score	-0.32	0.03	-0.51	0.0008	-0.36	0.06
Perseverative errors	0.07	0.65	-0.01	0.98	-0.29	0.14
Design fluency (fixed)						
% Perseverative errors	0.17	0.25	0.02	0.91	0.02	0.92
<i>Memory</i>						
Recall						
Verbal CLTR (total words)	-0.38	0.008	-0.52	0.0007	-0.34	0.08
Verbal 30' recall	-0.47	0.0007	-0.40	0.01	-0.23	0.24
Spatial CLTR (total locations)	-0.44	0.002	-0.33	0.04	-0.67	0.0001
Spatial 30' recall	-0.38	0.01	-0.34	0.04	-0.12	0.53
Recognition						
Geometric (net score)	-0.57	0.0001	-0.24	0.15	-0.29	0.13
Random (net score)	-0.27	0.09	-0.24	0.15	-0.19	0.33
<i>Language comprehension</i>						
Token test	-0.42	0.003	-0.36	0.03	-0.11	0.58

CLTR = consistent long term retrieval.

Minor variation in sample size across different tests due to scheduling and equipment problems.

lesions *v* the group who had consistently normal MRI findings showed that verbal memory was impaired in the patients who had transient lesions ($F(1,14) = 7.12, p < 0.02$). There were no other differences in neuropsychological performance, however, between the resolved and no lesion groups.

The transition from pervasive to specific neurobehavioural sequelae by one month after injury is illustrated by the findings of two patients. Figure 3a shows the MRI scan obtained at baseline in patient 7 of the frontal group (table 3) who had a left frontal lesion which was still present at one month. As summarised in table 3 there was considerable resolution of his multiple deficits by one month, leaving him with a specific verbal dysfluency. Figure 3b depicts a predominantly right temporal lobe lesion in patient 4 of the frontotemporal group whose neurobehavioural findings evolved from pervasive deficits initially to a specific impairment on measures of executive function by one month.

RELATION BETWEEN DURATION OF POST-TRAUMATIC AMNESIA AND NEUROBEHAVIOURAL FINDINGS

The duration of post-traumatic amnesia corresponded to the interval beginning when the patient began to obey commands until attaining normal scores on a brief test¹³ of orientation and memory for ongoing events. Table 5 depicts the Pearson correlation coefficients between duration of amnesia and the neuropsychological test scores at each examination. The most consistently significant correlations across the three examinations were obtained for the verbal and visuospatial selective reminding tests of multitrial recall. In contrast the correlations for recognition memory and executive function were greatly diminished by the three month examination.

Discussion

Our findings confirm our preliminary data⁸ and studies at other centres,¹⁻⁷ indicating that

intracranial hyperintensities are more often present on MRI compared with focal CT abnormalities in patients sustaining mild to moderate CHI. Our initial report⁸ showed focal lesions in 17 of 20 patients (85%) studied during the first week after sustaining mild to moderate head injury, a figure which closely approximates the present MRI finding of focal abnormalities in 40 of 50 cases (80%). Parenchymal lesions on MRI were distributed primarily in the frontotemporal region, a pattern consistent with neuropathological findings in fatal head injuries¹⁴ and experimental models of CHI.¹⁵ Our study also shows that moderate impairment of consciousness rather than mild confusion or disorientation is associated with larger intracranial lesions on the one month MRI (but not at baseline). The size of parenchymal lesion, however, was not related to the severity of impaired consciousness.

Our preliminary observations⁸ had suggested that the pattern of initial neurobehavioural deficits was related to a frontal *v* temporal site of lesion detected by MRI. In this expanded study neurobehavioural deficits on the baseline examination were pervasive on tests of purported frontal lobe function and on measures of memory irrespective of whether the intracranial lesion was confined to the frontal or temporal lobes or overlapped these regions. The relation of focal brain lesions to specific neurobehavioural sequelae one month after injury also showed inconsistencies across patients. Individual patients with resolved lesions in the frontotemporal region, however, tended to have a higher proportion of tests with defective scores than patients who had no lesions on their initial MRI. The inconsistent relation between site of lesion and pattern of neurobehavioural deficit did not change appreciably over three months in our patients with mild to moderate CHI. The small number of patients in the lesion site groups, however, is a limitation of the present study because of the reduced power of the statistical tests.

In contrast to the present findings in mild to

moderate CHI previous MRI study of patients representing a wider range of injury severity has revealed that the relation between depth of lesion and neurobehavioural functioning tends to strengthen over time.⁵ Wilson *et al*⁵ found that the relation between MRI findings and neuropsychological functioning in 25 adults with CHI of varying severity changed over time. Depth of lesion had a stronger relation to cognitive deficit at follow up (five to 18 months) than on the initial examination.

Our study also shows the heterogeneity of mild to moderate CHI in regard to the persistence of cerebral abnormality detected by MRI; notably 19 of 27 patients (70%) studied at all three examinations still had evidence of an intracranial lesion one month after sustaining a mild to moderate head injury. At the same time, it should be acknowledged that four of the eight patients whose hyperintensities had resolved by one month still exhibited deficits in one or both domains of the neurobehavioural examination. Whether the pathophysiological basis of these sequelae of mild to moderate CHI could be shown by metabolic imaging awaits further research.

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