Recognition memory, and head injury

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SYNOPSIS Severely head injured adults were tested on a recognition memory procedure involving the identification of eight recurring shapes among a series of 160. Compared with a control group, the tested patients showed many fewer correct responses. Their type of error was commonly a failure to recognize rather than a false recognition. The severity of the memory deficit was related to the length of post-traumatic amnesia (PTA), but to neither the presence of neurological signs at the time of memory testing, nor to the time after injury at which the patients were tested. The older patients showed a more significant relationship between PTA and memory score than the younger patients.

Although head injuries are common and the disabilities, physical and mental, that result from them take so much time and effort in rehabilitation, there is still relatively little information concerning the prospects and time scale of eventual recovery. This is particularly true in the case of cognitive deficits, with the exception of relatively few papers (Conkey, 1938; Ruesch, 1944; Carlsson et al., 1968; Russell, 1971). The samples used by both Conkey and Ruesch appear to have been restricted to lower social class patients who suffered mild injuries. In the range of patients reported by Russell (1971), the assessment of cognitive recovery was clinical and by Carlsson et al. (1968) vague. Neither reported objective psychological testing. The persistence and severity of memory deficits in a sample of severe head injuries in adults has already been shown using a continuous recognition task (Brooks, 1972). Deficits in memory were detected often many months after the injury had taken place, and long after the patient had emerged from post-traumatic amnesia (PTA). In the previous study, the score was based only on the number of correct responses, with a correction for guessing. The present paper reports memory testing in a rather larger sample of head injured adults using a continuous recognition procedure in which the process of learning itself can be studied.

METHODS

PATIENTS The 34 patients (all but one, males) had all suffered severe diffuse head injury which had resulted in transfer to the Division of Neurosurgery at the Institute of Neurological Sciences, Glasgow. Patients were seen at varying times after injury (mean 12.0, range 1 to 32 months), but each patient was seen once only. In no case was the patient seen until completely orientated in time and place, and fully out of PTA. The length of PTA assessed clinically was defined as the time between the injury and the regaining of continuous day to day memory for

| TABLE 1 |
| DURATION OF PTA |

<table>
<thead>
<tr>
<th>PTA (days)</th>
<th>&lt;=20</th>
<th>21-40</th>
<th>41-60</th>
<th>&gt; 60</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases (no.)</td>
<td>15</td>
<td>10</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>

at least 24 hours. The mean PTA duration was 43.4 days (range 2 to 300 days), and the PTA distribution is shown in Table 1. The mean age of the patients was 31.2 years (range 16–60 years).

Patients were examined neurologically at the time of memory testing (Table 2). Dysphasia was shown by 14 patients, in two of whom severe difficulties in word-finding and propositional speech were evident.
TABLE 2
TESTING IN RELATION TO NEUROLOGICAL EXAMINATION

<table>
<thead>
<tr>
<th>Grade</th>
<th>Patients</th>
<th>Mean testing time</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>22</td>
<td>11.4</td>
<td>1-27</td>
</tr>
<tr>
<td>II</td>
<td>8</td>
<td>14.1</td>
<td>8-32</td>
</tr>
<tr>
<td>III</td>
<td>4</td>
<td>9.2</td>
<td>4-16</td>
</tr>
</tbody>
</table>

No signs, or focal neurological signs so minor as not to interfere with day-to-day living. These patients have made a good recovery. Marked focal neurological signs, but patient still capable of independent existence: a moderate disability. Disabling neurological signs: dependent existence: severe disability.

The other 12 showed mild difficulties such as slight hesitations, slight mispronunciations, or occasional difficulty in finding a word. Care was taken to ensure that all patients understood the requirements of the test.

A control group was also tested consisting of 34 orthopaedic outpatients undergoing rehabilitation after injuries of the lower limbs. The head injured and control group were compared using $t$ tests on age and educational level, and proved to be well matched ($t$ age = 0.91 NS; $t$ education = 0.29 NS).

CONTINUOUS RECOGNITION TEST In this test, developed by Kimura (1963), the patient is presented with a series of 160 2 in. square cards, on each of which there is either a simple 'geometrical' design or a 'nonsense' figure—that is, an asymmetrical pattern that is very difficult to encode verbally. Each card is displayed for 3 s and of the 160 designs eight (four geometric and four nonsense) reappear a total of eight times, each recurring design doing so once in every block of 20 cards. The test can therefore be considered to consist of seven 'trials'; each trial comprising 20 shapes, 12 completely new ones appearing once only, and eight that have appeared before. The first 20 shapes are, of course, all new to the patient who is not required to make any response to them. For the remaining 140 shapes, the patient must identify each card as 'new' (first appearance) or 'old' (a recurrence). The maximum possible score in each trial is 8. A correction for guessing is made by subtracting from the number correct, the number of false positive errors (incorrectly identifying a 'new' shape as a recurrence) to produce the final corrected score. Three measures are derived in all: (1) corrected score, (2) false positive errors, and (3) false negative errors (failure to identify a recurring item).

RESULTS

Patients and controls were compared using the trend analysis of variance procedure described by Edwards (1968), in order to investigate not

![Figure 1](http://jnnp.bmj.com/)

**FIG. 1.** Continuous recognition: corrected score.

<table>
<thead>
<tr>
<th>TABLE 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>RANK CORRELATION: MEMORY SCORES WITH PTA</td>
</tr>
<tr>
<td>Corrected score</td>
</tr>
<tr>
<td>False positives</td>
</tr>
<tr>
<td>False negatives</td>
</tr>
</tbody>
</table>

* $P < 0.01$. 

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**FIG. 2.** Continuous recognition: false positive errors.

**FIG. 3.** Continuous recognition: false negative errors.
only differences between the two groups, but also differences from trial to trial within the groups, due to learning. Any difference in the shape of the learning curves between the two groups is indicated by the Groups × Trials Interaction.

MEASURES OF COMPARISON Each measure is discussed as follows:

1. Corrected score (Fig. 1) The two groups are clearly significantly different (F = 10·05, P < 0·01). The head injured patients achieved a lower score overall. There is a significant trials effect (F = 41·39, P < 0·01) showing the improvements due to learning, and the interaction is significant (F = 2·78, P = 0·05) reflecting the more rapid rate of learning in the control group.

2. False positives (Fig. 2) There is no significant difference between the groups (F = 0·05 NS). Both showed a similar and significant (F = 32·1, P < 0·01) reduction in false positive errors over trials. The interaction term was not significant.

3. False negatives (Fig. 3) On this measure there was a significant difference between the groups, the head injured patients gained a consistently higher number of false negatives (F = 14·99, P < 0·01). The reduction in errors of this kind due to learning was less marked, although still significant (F = 4·75, P < 0·01) and the rate of error reduction did not differ significantly in the two groups.

There are, therefore, significant differences between head injured patients and controls on two of the three measures chosen, the number of false positive errors being the one not showing such a difference.

TIME OF TESTING The head injured patients were tested at varying times after injury, each patient being seen once only, making it possible to investigate the change in recognition memory, with time after injury. This was done by correlating the memory scores with the time after injury at which the patient was tested, a recovery effect being indicated by a positive correlation. The Spearman Rank Order correlation coefficient was used throughout the study, as further correlations were carried out on subgroups of the head injuries, where the smaller numbers necessitated the rank order procedure rather than the product moment correlation. The correlations between scores and time proved to be small and insignificant for each measure used, the highest being +0·13 for the corrected score. In the head injury group as a whole, therefore, there appears to be no relationship between time and memory, as all coefficients are insignificant. If, as clinical experience and experimental evidence suggests (Carlsson et al., 1968), intellectual recovery is an exponentially rising function of time, then the relationship between memory and time may be more obvious early after injury, where the function is still showing a steep rise. To investigate this, the head injured patients were subdivided into two groups: an ‘early’ group (N = 19) tested one year or less after injury (mean 6·6, range one to 12 months) and a ‘late’ group (N = 15) tested more than one year after injury (mean 18·0, range 13–32 months). Although there was some slight tendency for the late group to achieve higher scores, the difference between the two subgroups tested, using the Mann Whitney U test were not significant. Within each subgroup the correlations between memory score and time were computed, and these proved to be insignificant, the largest coefficient being −0·28 for errors in the early group. Clearly there is no obvious relationship in either group between time and memory.

ASSOCIATION BETWEEN MEMORY AND SEVERITY OF INJURY Two measures of severity were used—the duration of PTA, and the neurological grade at the time of testing. The two indices correlated +0·43 which, although significant at the P < 0·01 level, is low enough to suggest that the two indices are assessing different aspects of severity, the PTA being fixed, but the neurological grade diminishing with time.

The effects of severity were calculated in two ways: by correlating the memory scores summed across all seven trials, with the length of PTA, as shown in Table 3; and by comparing patients with moderate–severe neurological signs (grades II and III) with those with mild or no signs (grade I), using the Mann Whitney U test.

PTA correlated significantly with two of the three memory scores used. It is interesting that
there is no relationship between PTA and false negatives, despite the fact that of the two error scores—false positives and negatives—it was the latter alone that distinguished the patients from the controls. Although there was no patient-control difference in the number of false positives, there was a relationship within the head injury group, between the severity of the injury and the number of false positives, the more severe injuries scoring more false positives. The level of false negatives is significantly higher in the head injuries regardless of the severity of the injury, suggesting some kind of threshold, or all-or-none result of cerebral trauma which (in this group of severe injuries) is independent of the severity of the trauma.

The relationship between memory and neurological signs is very much less obvious: although there was a slight tendency for the patients with moderate or severe signs to achieve a lower score, and more errors, the differences between the two subgroups were not significant (Table 4). In the group as a whole, therefore, there is no relationship between the severity of damage judged by the presence of neurological signs, and the severity of the memory deficit.

EFFECTS OF AGE This was investigated by dividing the head injuries into two groups of ‘younger’ (age 15–30 years, N = 18), and ‘older’ (age over 30 years, N = 16), which were then shown to be similar in respect to PTA, education, and time of testing using U tests. There was a slight but insignificant tendency for the younger patients to achieve higher scores (Table 5). Having investigated differences in scores between the two age groups, the relationship between severity and memory was investigated within each age group by correlating PTA with each memory score, as shown in Table 6. There is rather a different relationship between score and severity in the two age groups for two of the three memory measures. For the corrected score, and false positives, the correlation is higher in the older than in the younger patients, each coefficient being significant in the older patients but smaller and not significant in the young patients. However, the correlations for these two measures are still far from zero in the young group. This age difference supports a previous finding (Brooks, 1972), although the differences in size of coefficient are not as great as found previously using a smaller group of patients. For the false negatives, the coefficient is insignificant in both groups, showing that in neither does the severity of damage determine the number of items incorrectly missed, despite the fact that the patient group as a whole differed significantly from the control group on this measure. The hypothesized threshold effect resulting in a higher level of false negatives regardless of severity, appears to hold, therefore, for both age groups.

LATERALITY OF DAMAGE TO CEREBRAL HEMISPHERES A further variable that may determine memory recovery, is the site of brain damage. In
acceleration-deceleration head injuries it is difficult to get any accurate statement about the localization of damage. The damage is often widespread or multifocal and it is unrealistic to attempt to make any precise localization. However, one can use the presence of dysphasia to indicate left hemisphere damage, although this does not exclude right hemisphere damage also. There is now a large body of neuropsychological work summarized by Milner (1971) showing that left hemisphere damage typically impairs memory for material that can be encoded verbally, whereas right hemisphere damage impairs memory for non-verbal material, assuming left hemisphere dominance. In the test used here, two kinds of stimuli were used—the ‘geometric’ (verbal) and the ‘nonsense’ (non-verbal), and it was expected that dysphasic patients would show more errors on the geometric than on the nonsense shapes. This was investigated by comparing the 14 dysphasic patients with the 20 patients who showed no signs of any language disturbance. These two groups were compared initially on severity variables as shown in Table 7. Although the dysphasics were not significantly worse on PTA, they were so on the neurological grading. This is to be expected, as the presence of dysphasia is one index used in determining the neurological grade.

The performance of the two groups on corrected score, and on the proportion of errors gained on the geometric items was compared using the Mann Whitney U test. The dysphasic patients tended to achieve slightly higher corrected scores than the other patients (dysphasics 23.5, ‘others’ 18.7) but the differences are not significant. On the proportion of geometric errors, although there is no significant difference between the two groups, the dysphasics achieve, if anything, a smaller proportion of geometric errors than the other patients (dysphasics 0.42, ‘others’ 0.39).

### DISCUSSION

It is interesting to speculate on why the two kinds of error should be differently affected by a head injury. A false negative error is a failure to identify a previously displayed item as familiar: this could result from a number of processes. These include poor initial learning, so that less material is committed to memory, or is committed in a distorted or degradable form; or, an alternative is a strategy whereby the patient adopting very strict decision criteria would identify an item only if he were quite certain. This explanation would require a low level of false positives, as the patient would be unwilling to make guesses, and this was indeed the case. A difficulty in retrieving the information from memory seems unlikely as this would result in an increase in both kinds of errors in the head injured patients. A further possibility is that head injured patients are suffering perceptual difficulties, but this is unlikely as perceptual deficits should cause at least as many false positives as negatives, and this was not the case.

At present the results suggest that the poorer performance of the head injured group is caused by either or both of two processes—a poor initial learning, and a strategy of increased caution. The data are not available to distinguish completely between these two alternatives.

The time of testing after injury appeared to bear little relationship to the memory performance. Even when the patients were subdivided into ‘early’ (tested 1 to 12 months after injury) and ‘late’ (tested more than 12 months after injury) there was still no relationship, although using an exponential model, referred to earlier, one might have been expected in the early group but not in the late. Despite the wide range in times, few patients were seen in the very early stages, within days or weeks of emerging from PTA, and it is in these stages that rapid recovery is most striking. Had more patients been included at this very early stage, it is likely that the time/score relationship would have become more obvious. However, the fact remains that in this sample of patients, all of whom are fully out of PTA, there is little correlation between their
memory and the time since their injury, suggesting that in the time range chosen here, recovery of memory is dependent upon factors other than the time elapsing since the injury, or, more likely, that recovery has finished by the time the patients were tested (six to seven months in the ‘early’ group). This finding has important implications for the rehabilitation of the brain injured. If, as appears to be the case from these data and those of other workers (Conkey, 1938; Ruesch, 1944), memory recovery ceases at a relatively early stage after the injury, then the efficacy of, for example, occupational therapy procedures designed to improve memory, is thrown into question. It may be that the value of such therapy lies not so much in assisting the re-learning of old skills, or in the formation of new ones, but in giving the patient strategies for coping with a deficit that may have to be considered to be permanent.

The influence of severity of injury on memory performance depended, not only on the severity index chosen, but also on the age of the patient. PTA correlated significantly with two of the memory scores in the whole group, whereas the presence of neurological signs did not appear to be associated with a more severe memory deficit. When patients were subdivided into age groups, PTA correlated more highly in old than in young patients.

The different relationship between memory and the two indices of severity calls for comment. PTA is perhaps best thought of as expressing the damage to the brain as a whole, sustained at the time of injury: the neurological grade, obtained at the time of memory testing, reflects the severity of localized damage. In the head injuries as a whole, the severity of diffuse damage shows a clear relationship with the severity of memory deficit. The focal damage, however, is much less important in determining memory performances. The memory deficits found, even months or years after injury, appear to be a result of diffuse brain damage. When age is taken into account, the picture becomes a little less clear, in that PTA bears a stronger relationship with memory in older (over 30 years of age) than in younger (age 15–30 years) patients, suggesting that in the older group diffuse brain damage may be more severe in terms of cognitive deficit than in the younger group. The recovery of memory in the younger patients appears to be determined not just by the extent of the injury (which it undoubtedly is to some extent), but probably by other factors such as the site of the injury, the pre-traumatic intellectual level, or perhaps by the degree of stimulation during recovery. In the older patients these factors are probably overridden to some extent by the reduced capacity of the older brain to tolerate trauma.

Of the two error scores only the number of false negatives correlated significantly with PTA—more so in older patients than in younger, although in this variable there is no difference between patients and controls. However, on false negatives, where patients did differ significantly from controls, there is no correlation with PTA. The poor performance in terms of false negatives is therefore appearing largely independently of the severity of diffuse damage, suggesting some kind of threshold effect, so that even a relatively mild but still diffuse injury leads to a deficit of the kind observed here, whereas severe focal damage need not do so unless crucially placed.

Comparing the performance of the dysphasic and non-dysphasic patients, there was no significant difference either on the corrected score, or on the proportion of geometric errors. In fact, the dysphasic patients as a group made fewer geometric errors than the other patients. In a patient population of this kind, it is perhaps not surprising that findings concerning hemispheric differences in verbal memory that were derived from clearly lateralized cases (Milner, 1971) should not hold. The presence of dysphasia in a diffuse injury does not preclude right hemisphere damage, just as absence of dysphasia does not rule out left-sided damage.

The work here highlights an area where further study is necessary. The underlying nature of the deficit is unclear, being caused either by a learning deficit, or by an approach typified by extreme caution, or both. Work on choice reaction time after head injury (Miller 1970) suggests that the head injury deficit occurs at the input side of cognitive information processing, but does not exclude a strategy of caution. Further analysis using more refined techniques would help to throw light on this problem.
This work was carried out jointly in the Departments of Psychological Medicine and of Neurosurgery, and the Institute of Neurological Sciences, Glasgow. It is part of a wider study of head injury supported by a grant from the National Fund for Research into Crippling Diseases, to Professor Bryan Jennett, Department of Neurosurgery. Thanks are due to the neurosurgeons of the Institute of Neurological Sciences, Glasgow, who allowed their patients to be studied.

REFERENCES


