DO AMNESICS FORGET COLOURS
PATHOLOGICALLY FAST?

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ABSTRACT

We tested amnesic and control subjects on a task which required the recognition of single, difficult to name colours, after delays ranging from 7 seconds to 120 seconds after performance of the two subject groups had been matched at the shortest delay by giving the amnesic patients longer study time. The amnesic patients showed abnormally fast forgetting over the two minute period. Furthermore, a subgroup of nine subjects with presumed damage to midline diencephalic structures (Korsakoff's syndrome) were found to forget as fast as a group of six subjects with presumed medial temporal lobe damage (herpes simplex encephalitis). These results contrast both with studies using the Huppert and Piercy procedure and those using the Brown-Peterson task, none of which have shown convincing evidence of accelerated forgetting in medial temporal lobe or diencephalic amnesia.

Key words: amnesia, Korsakoff' syndrome, herpes encephalitis, forgetting.

INTRODUCTION

Both the Brown-Peterson task (Brown, 1958; Peterson and Peterson, 1959) and the Huppert and Piercy task (e.g., Huppert and Piercy, 1978, 1979) have been used to assess whether amnesic patients forget information abnormally quickly. The Brown-Peterson task traditionally tested recall of verbal material. For example Peters and Peterson (1959) required subjects to recall a consonant trigram. Another characteristic of the task was that the retention interval was filled with verbal distraction. By contrast, the original Huppert and Piercy task tested memory for pictorial material and did not have an explicit distractor task in the retention interval. A further difference was that this task tested recognition of material whereas the Brown-Peterson task tested recall. Subsequently analogues of both these tasks have been developed which test memory for material in other modalities. Nonverbal versions of the Brown-Peterson task have been developed (e.g., Sullivan, Corkin and Growdon, 1986; Cermak, Reale and De Luca, 1977) and the Huppert and Piercy task has been used with verbal material (e.g., Hart and O'Shanick, 1993). Although different types of test material have been used, the Huppert and Piercy task has continued to be a test

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of recognition memory and very few studies have used a recognition version of the Brown-Peterson task (Butters and Cermak, 1974).

The Brown-Peterson and Huppert and Piercy tasks differ in two other important ways. One major difference is the time course over which they typically assess forgetting. The Brown-Peterson task has assessed memory over short delays of a matter of seconds usually starting with a delay of zero seconds. By contrast, the Huppert and Piercy task has assessed memory from a delay of 10 minutes onwards. Thus, the two tasks have been considered to be assessing forgetting rate from short-term memory (STM) and long-term memory (LTM) respectively.

The second difference is related to the initial delay at which memory is tested. When the initial delay is greater than a few seconds the baseline memory performance against which forgetting is measured differs between amnesic and control groups and this causes problems in interpreting rate of forgetting data (Mayes, 1986). To overcome this problem Huppert and Piercy (1978) ensured that amnesics' and controls' recognition performance was matched at the shortest delay by allowing amnesic patients longer exposures to target items during the study phase. As Huppert and Piercy were interested in the rate at which information was forgotten from LTM, the performance of the amnesic and control subjects was matched at ten minutes, a delay at which STM was thought to be no longer contributing to performance. Although this choice of initial delay is arbitrary, most subsequent studies have also used ten minutes as the shortest delay. In contrast, the tasks developed by Brown (1958) and Peterson and Peterson (1959) did not require a matching manipulation as both subject groups performed equally (at ceiling) on the initial delay of zero seconds.

The results regarding the forgetting rate of amnesic patients which have been obtained using these tasks are not clear cut. There have been conflicting findings concerning forgetting rate of subjects with medial temporal lobe and diencephalic damage on the Huppert and Piercy task. A number of research groups have reported abnormally fast forgetting by amnesic patients with damage to medial temporal lobe structures, but not by patients with damage to midline diencephalic structures (Huppert and Piercy, 1978; Huppert and Piercy, 1979; Squire, 1981). However, at least one study failed to show abnormally fast forgetting for either medial temporal lobe or diencephalic amnesics (e.g., McKee and Squire, 1992). Also, Freed, Corkin and Cohen (1987) reported normal rate of forgetting by patient HM and patients with Alzheimer's disease, the neuropathology of which includes damage to medial temporal lobe structures, have also been reported to exhibit normal rates of forgetting over periods of three days (Freed, Corkin, Growden et al., 1989) and of one week (Kopelman, 1985).

There is also controversy concerning rate of forgetting of amnesic patients on the Brown-Peterson task. The pattern that has emerged is that patients with amnesia resulting from medial temporal lobe damage perform at near normal levels (e.g., Butters, 1984), whereas Korsakoff patients, with presumed diencephalic damage, are impaired (e.g., Starr and Phillips, 1970). However. after reviewing this work, Kopelman (1992) concluded that no study had

provided unequivocal evidence of accelerated forgetting even in Korsakoff amnesics. Following Kinsbourne and Wood (1975), he argued that this would take the form of a significant group by delay interaction effect *beyond the zero delay interval* as at the zero delay ceiling effects, which obscure interpretation, have invariably been reported. In addition, Mair, Warrington and Weiskrantz (1979) reported very good performance in two Korsakoff patients on the Brown-Peterson task. In fact their performance was slightly better than controls out to a delay of 60 seconds on all but one variant of the task. It is clear that the issue of whether amnesic patients show accelerated forgetting over both short and long delays is far from resolved, but the weight of evidence at present suggests a normal rate of forgetting in amnesia.

In the present study we looked at forgetting rate in a group of amnesic patients who had either medial temporal lobe damage or diencephalic damage. We used a task which was similar to the Brown-Peterson task in that the delays at which memory was tested were relatively short (7 seconds to 120 seconds), but instead of recall we assessed recognition of very difficult stimuli. The task also involved a matching procedure at the shortest delay similar to that used by Huppert and Piercy.

As discussed above, previous rate of forgetting studies which have been concerned with LTM have tested memory at delays longer than 10 minutes However, if consolidation into long-term memory is at its peak during the first 10 minutes, subjects may well show pathologically fast forgetting only during the period of seconds or minutes immediately following exposure. That is, pathologically fast forgetting may occur in the period preceding the shortest delay at which performance has usually been matched. To date, relatively few studies have explored forgetting in this time window (Carlesimo, Sabbadini, Fadda et al., 1995; Carlesimo, Sabbadini, Loasses et al., 1997: Hart, Kwentus, Harkins,1988; Holdstock, Shaw and Aggleton, 1996; Issac and Mayes, submitted; Leng and Parkin, 1989; Mayes, Downes, Symons et al., 1994). In the present study, therefore, we decided to look at the rate of forgetting of amnesic patients over these shorter delays. Investigating the performance of human amnesic patients at these delays is also important bocause similar delays have been used to assess : recognition memory in primate models of medial temporal lobe and diencephalic amnesia (Aggleton and Mishkin, 1983; Alvarez-Royo, Zola-Morgan and Squire, 1992; Murray and Mishkin, 1986; Zola-Morgan, Squire, Amaral et al., 1989).

One of the problems of testing memory at short delays is that the performance of control subjects is often at ceiling. In order to eliminate this problem the Farnsworth-Munsell colour hue set was used. The target and distractors comprising a particular recognition set were selected so that only a single colour name could be applied to each set, thereby preventing the use of verbal labels as memory aids. Using this criterion, a test of a high difficulty level was produced, so that control subjects were not at ceiling even when recognition memory was tested for single targets.

The development of a recognition memory test for single targets which did not suffer from ceiling effects at the shortest delay made it possible to reduce a problem of the Huppert and Piercy procedure which may result in

underestimation of forgetting rate in amnesic patients. This problem is particularly evident when memory is being tested for a multi-item list and results from the manipulation used to match performance at the shortest delay. In the Huppert and Piercy task recognition performance of the patient and control groups is matched by giving the amnesic patients longer item exposures during the study phase. At the end of presentation of a multi-item list timing of the presentation to test delay begins. Although this ensures that the delay between offset of the last item in the list and the beginning of the test phase is equivalent in the two subject groups, the delay between presentation *onset or offset* of *any individual item* in the list (apart from the last) and its test is longer for the amnesic group than for the control group. The result is that initial memory for each item is tested at a later point on the forgetting curve for the amnesic patients compared with the controls. As forgetting has been found to be best described by a function that decelerates with time (Baddeley, 1976), the rate of forgetting of the amnesics may be underestimated (see Mayes, 1986). When one tests memory for single items, rather than for a multi-item list, this potential problem is reduced. In the present procedure, just as in the Huppert and Piercy procedure, the amnesic patients were given longer exposure to items than controls. However, only one item was presented before test. As a result the increased item exposure lengthened the delay between *onset* of stimulus presentation and initial memory test for the amnesics compared with controls, but the delay between offset of each individual item and test was equivalent in the amnesic and control groups. If we make the reasonable assumption that the point at which equivalent learning of the stimulus has been reached by the two groups is at the end of stimulus presentation and that subjects do not start to forget until stimulus offset, then, in the present study, the two groups will have been tested at equivalent points on the forgetting curve. Even if one assumes forgetting starts at the beginning of the study exposure (which is not very reasonable), with our procedure, it is possible to match amnesic patients and their controls at approximately the same study onset-test delay.

In summary, the present study investigated the rate of forgetting of single items by amnesic patients on a task which tested recognition memory for very hard to verbalise colours thereby avoiding ceiling effects in the control subjects. Amnesic and control subject performance was matched at a delay of 7 seconds and performance was measured at further delays of 15 seconds, 30 seconds, 60 seconds and 120 seconds.

MATERIALS AND METHODS

Subjects

The amnesic group consisted of 21 amnesic patients (15 males, 6 females) fron the North West region of England. The amnesia of these patients was due to a variety of aetiologies, but included three subgroups: nine patients with Korsakoffs syndrome (subgroup KORS); six patients with amnesia secondary to an encephalitis (subgroup PE, presumed herpes simplex encephalitis in all cases); and six patients (subgroup MIX) with amnesia resulting from a number of other aetiologies. Three patients in the MIX subgroup had amnesia as a result of rupture and repair of an anterior communicating artery aneurysm.

TABLE I
A. Summary Characteristics for the Total Amnesic and Total Control Groups

Group	Age	FIQ	VIQ	PIQ	WRMTW	WRMTF
Amnesic (n = 21)	44.93 (14.95)	102.15 (13.34)	103.76 (14.28)	98.80 (11.16)	33.29 (6.17)	32.38 (5.31)
Control (n = 15)	45.38 (13.76)	104.87 (11.07)	107.60 (13.97)	100.13 (14.16)	47.47 (1.46)	43.47 (2.72)

B. Psychometric Details of the Three Amnesic Subgroups

Subgroup	Age	FIQ	WRMTW	WRMTF	WMQ	MWMI	WCSTcat	WCSTpe	FAS	CE
KORS (n = 9)	52.22 (13.17)	108.11 (13.95)	34.44 (7.20)	33.00 (5.10)	86.22 (11.89)	12.93 (4.37)	3.88 (8; 1.96)	19.63 (8; 21.21)	33.00 (6; 12.79)	5.86 (7; 2.91)
PE (n = 6)	42.67 (15.17)	99.67 (14.79)	33.00 (6.23)	30.17 (5.57)	77.17 (9.79)	9.21 (3.08)	5.60 (5; 0.89)	12.60 (5; 9.53)	31.00 (12.12)	8.50 (5.61)
MIX (n = 6)	37.83 (9.39)	94.40 (5; 4.34)	31.83 (5.04)	33.67 (5.61)	74.60 (5; 4.45)	9.50 (5; 2.35)	4.17 (1.47)	27.00 (19.51)	24.50 (13.69)	9.60 (5; 3.78)

Notes: Standard deviations are shown in parentheses; where two numbers are given, the first refers to the reduced N for that test. KORS, Korsakoff subgroup; PE, post encephalitic subgroup; MIX, mixed aetiology subgroup; VIQ, PIQ, FIQ, verbal, performance, and full-scale scores of the WAIS; WRMTW and WRMTF, Warrington's word and face recognition tests; WMQ, memory quotient; MWMI, modified Wechsler memory index; WCSTcat and WCSTpe, category and perseverative error scores on the Wisconsin Card Sorting Test; FAS, verbal fluency score; CE, cognitive estimates score.

One patient had amnesia as a result of rupture and repair of a posterior cerebral artery aneurysm. A Computed Tomography (CT) scan indicated that this patient had predominantly right medial temporal lobe damage. One patient had amnesia as a result of meningitis and a Magnetic Resonance Imaging (MRI) scan of this patient showed bilateral medial temporal lobe damage including the hippocampus and amygdala and also very slight more general atrophy. The last patient in this subgroup had amnesia resulting primarily from a left-sided thalamic infarct. MRI information was also available for one patient from the PE subgroup who showed bilateral hippocampal lesions which also affected the posterior end of the amygdala, although the volume of this structure was in the normal range. The parahippocampal gyrus may also have been very slightly damaged, but its volume was not markedly less than a group of young normal subjects.

Table IA shows the mean age for the total amnesic group together with mean scores on the Verbal, Performance, and Full Scales of the Wechsler Adult Intelligence Scale (WAIS), and scores on the Warrington Recognition Memory Test (WRMT, Warrington, 1984). This group of 21 amnesics was matched to a group of 15 control subjects in terms of age, t (34) = .09, and the three IQ scale scores of the WAIS (all t's less than 1). However, the total amnesic group scored significantly worse than the control subjects on both the word and face versions of the WRMT, words t (34) = 8.70 p < .001, faces t (34) = 7.41 p < .001. Table IB shows the mean ages and psychometric scores for the KORS, PE, and MIX amnesic subgroups. Two measures were derived from the Wechsler Memory test, a memory quotient (WMQ), and a modified Wechsler memory index (MWMI), which is the sum of the raw scores on the Logical Memory, Visual Reproduction, and hard pairs of the Paired Associates subtests. A group of control subjects of a similar age to the amnesic group had a mean score of 22.2 and a standard deviation of 3.5 on this measure (Mayes, MacDonald, Donlan et al., 1992). Also shown in Table IB are mean scores on the following measures of frontal lobe functioning: Wisconsin Card Sorting Test, number of categories (WCSTcat) and number of perseverative errors (WCSTpe); the FAS test of verbal fluency; and Shallice and Evans' (1978) Cognitive Estimates test (CE). One way ANOVAs revealed that there were no significant differences between amnesic subgroups on any of these variables, although it is clear that the Korsakoff patients were older and tended to have a milder memory impairment (WMQ and MWMI scores).

Stimuli

Stimuli were selected from the 85 colour discs of the Famsworth-Munsell 100 Hue Test. Two white wooden trays, of dimensions ó inches long by 2 inches wide, were used to display the colour discs, one of which contained partitions to hold the four choices of the recognition test. A camera shutter mechanism was also used to ensure accurate target exposure times below 2 seconds duration.

Although computerised administration would have increased the precision of the timing of presentations and reduced any experimenter bias, the present study required portable equipment which could be easily transported to subjects' homes if necessary. Good colour laptop computers were not available at the time of the study and so for this practical reason we decided to use the original Farnsworth-Munsell colour discs.

Design

Four hues (i.e. one target and three distractors) were used on each trial. Each set of four hues was matched for hue distance, with each consecutive pair of hues in a set being separated by four others. For example, the four hues numbered 1, 6, 11, and 16 comprised one set. A total of 85 unique combinations of four hues could be constructed in this way from the total set, and 80 of these were selected for the experiment. Although each set was a unique combinaton of hues, there was obviously some overlap between sets in the hues that were used. Most hues were actually used four times in the course of the experiment, although each served only once as a target colour. Care was taken to ensure that targets were balanced across the four positions of the hue spectrum defined by hue sets.

The 80 hue sets were arranged into sixteen blocks of five, and pairs of these were used to measure recognition performance at each of the delays. Thus, only 50 out of the total 80 hue sets were used to derive performance measures, but the remainder were necessary to ensure that recognition performance for each subject at the shortest delay could be titrated to within the desired window of 75%-85% correct. Each block of five hue sets comprised four which were consecutive and non-overlapping. For example, the following hue sets comprised one of the experimental blocks: 1, 6, 11, and 16; 21, 26, 31, and 36; 41, 46, 51, and 56; 61, 66, 71, and 76; 8, 13, 18, and 23. Blocks of hue sets were balanced across delays in the same way for each group.

Procedure

Subjects were tested individually in a series of short experimental blocks, and each of these comprised one of the blocks of five hue-sets. That is, for each experimental block five trials using the same delay were given. Experimental blocks were separated by rest periods of about ten minutes. At least ten blocks were given to each subject, with the number of additional blocks depending on subjects' performance on the shortest delay performance titration procedure. These shortest delay blocks were always given first in order to set the target exposure for all the longer delay blocks. For the four longer delays, test-order was counterbalanced.

Initial exposures were 1 second and 6 seconds respectively for the control and amnesic groups. Each subject was given two blocks at the shortest delay using the appropriate exposure. If the subject reached the criterion of 80% correct then that exposure was used for all subsequent blocks, but if their score was outside of these limits the shortest delay blocks were repeated with the exposure time adjusted up or down as appropriate. The number of repeat blocks with adjusted exposure times was the same for both control and amnesic groups.

An experimental trial consisted of the following stages. Out of view of the subject, the experimenter selected the target hue from the set of four for that trial. This was placed on a white tray and the subject alerted that the trial was about to begin. Subjects were informed that they would be required to memorise a single coloured disc for a short period and that they would then be asked to select that colour from a set of four. The target hue was then exposed to the subject for a period of between .5 and 16 seconds. For exposure periods of

up to 2 seconds a camera shutter mechanism was used, whereas for longer exposures timing was by a stop-watch. Delays began at the end of the stimulus exposure and were also timed using a stop-watch. During the delay subjects were engaged in conversation to minimize rehearsal. A colour processing distractor task was not used because this would have produced interference as well as prevented rehearsal and we did not wish to produce interference.

As already indicated, the stimuli were specially chosen so as to prevent the effective use of verbal strategies. At the end of the delay, the subject viewed another white tray, divided into four partitions each containing one of the four discs comprising the hue set.

Data Analysis

A split-plot factorial design was used. Pathology (amnesic vs. normal for one analysis and KORS vs. PE vs. MIX for a second analysis) was the between-subjects factor, and delay was the within-subjects factor, with five levels: 7, 15, 30, 60, and 120 seconds (7SEC, l5SEC, 30SEC, 60SEC, 120SEC). For these analyses delay was measured from stimulus offset as it was assumed that this was the point at which equivalent learning of the stimulus had been reached by the amnesic and control subject groups.

As the amnesic patients were given longer stimulus exposure in order to match their performance with that of the controls at the initial delay, delay between stimulus onset and initial test was longer for the amnesic than control group. If the assumption made in the Introduction was not valid, and subjects started forgetting before stimulus offset, it is possible that this difference in delay between groups may have led to an underestimation of the rate of forgetting of the amnesic patients. In order to investigate rate of forgetting when the delay between stimulus onset and initial test was approximately equivalent for the controls and amnesics, an ANOVA compared the number of correct recognition judgements achieved by the amnesic patients at the 7 seconds and 15 seconds delays (measured from stimulus offset) with those achieved by the controls at the 15 seconds and 30 seconds delays (measured from stimulus offset). These delays will be referred to as the first and second delays for each group. Given that the mean stimulus exposure time for the amnesics was 7.6 seconds whereas that for the controls was 1.6 seconds, the mean stimulus onset to first delay was 14.6 seconds for the amnesics and 16.6 seconds for the controls in this analysis. It should be noted that the difference between the first and second delays is shorter for the amnesic than the control group and so this analysis represents a conservative test.

RESULTS

In what follows, where means are given in the text, these are followed by standard deviations in parentheses. The first set of analyses compared the groups on the mean exposure times required to bring performance up to the criterion level of 75-85% correct. This revealed a significant difference between the total amnesic group, 7.57 seconds (3.17), and total control group, 1.60 seconds (0.63), t (22.19, separate variances estimate) = 8.40 $p < .0005$. However, a one-way ANOVA revealed that the differences between the amnesic subgroups were unreliable, $F (2, 18) < 1$, KORS, 8.33 seconds (3.12); PE, 7.50 seconds (4.32); MIX, 6.50 seconds (1.87).

Two dependent variables were used in the main analyses. First, the number of correct recognition judgements, and secondly, an error score. The error score was simply the number of steps, in terms of colour hues, that the incorrect recognition choice was from the target. For all the analyses reported below identical results were found for the two dependent variables, so only the analyses based on the number correct measure are reported.
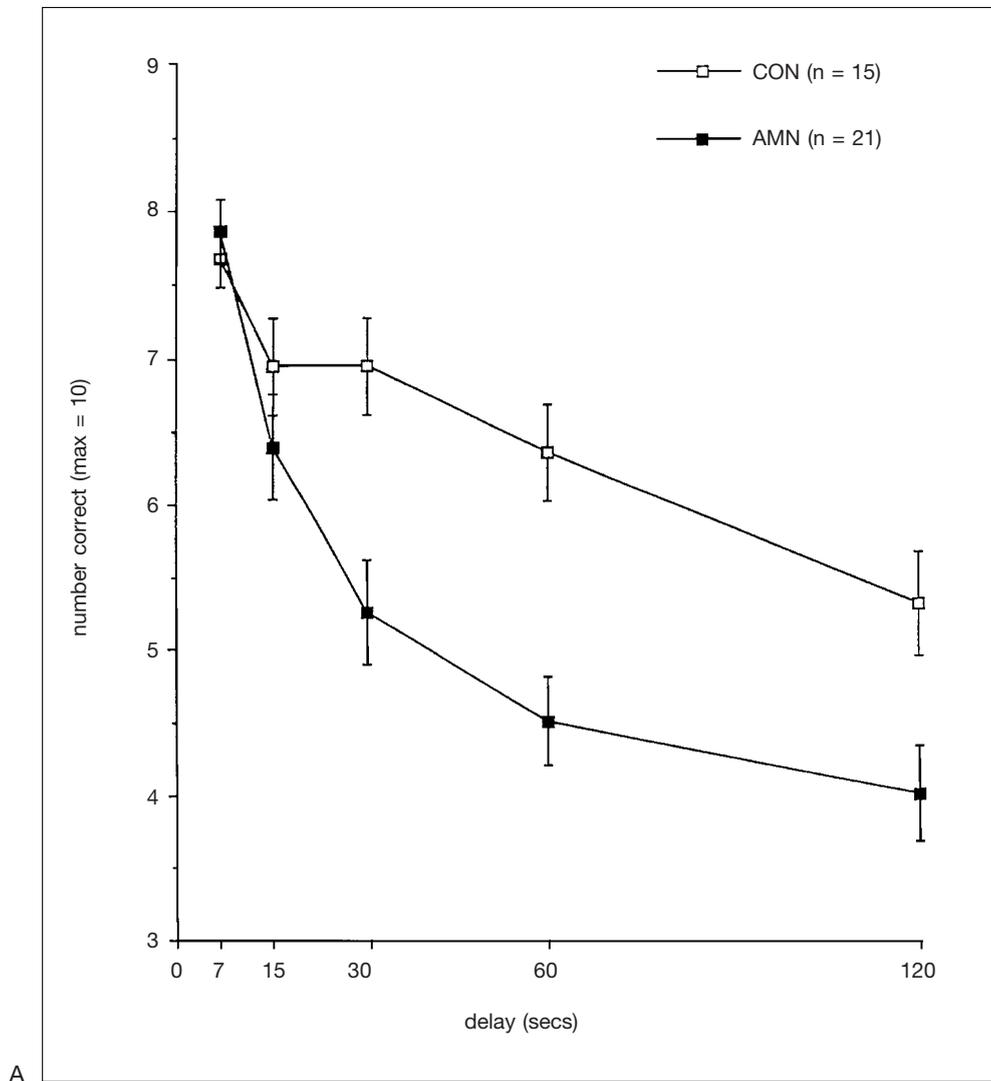
Fig. 1 – *Correct recognition performance as a function of increasing delay: A. Total amnesic group (AMN) and matched control group (CON); B. Relative performance of the three amnesic subgroups: Korsakoff (KORS); Post-encephalitic (PE); and mixed aetiology (MIX).*

The total amnesic group was compared to the matched control group using a 2-way ANOVA with repeated measures on the second factor (delay: 7, 15, 30, 60, and 120 seconds). This revealed significant main effects for both group and delay: averaged across delays, the control group remembered significantly more colours, 6.63 (1.43), than the total amnesic group, 5.58 (2.00), F (1, 24) = 12.60 p = .001; and, averaged across groups, performance declined significantly as delay increased, 7SEC: 7.78 (.90), l5SEC: 6.61 (1.50), 30SEC: 5.94 (1.71), 60SEC: 5.25 (1.63), 120SEC: 4.50 (1.58), F (4, 136) = 41.80, p < .001. Post hoc tests (Newman-Keuls) showed that all differences between delays were significant.
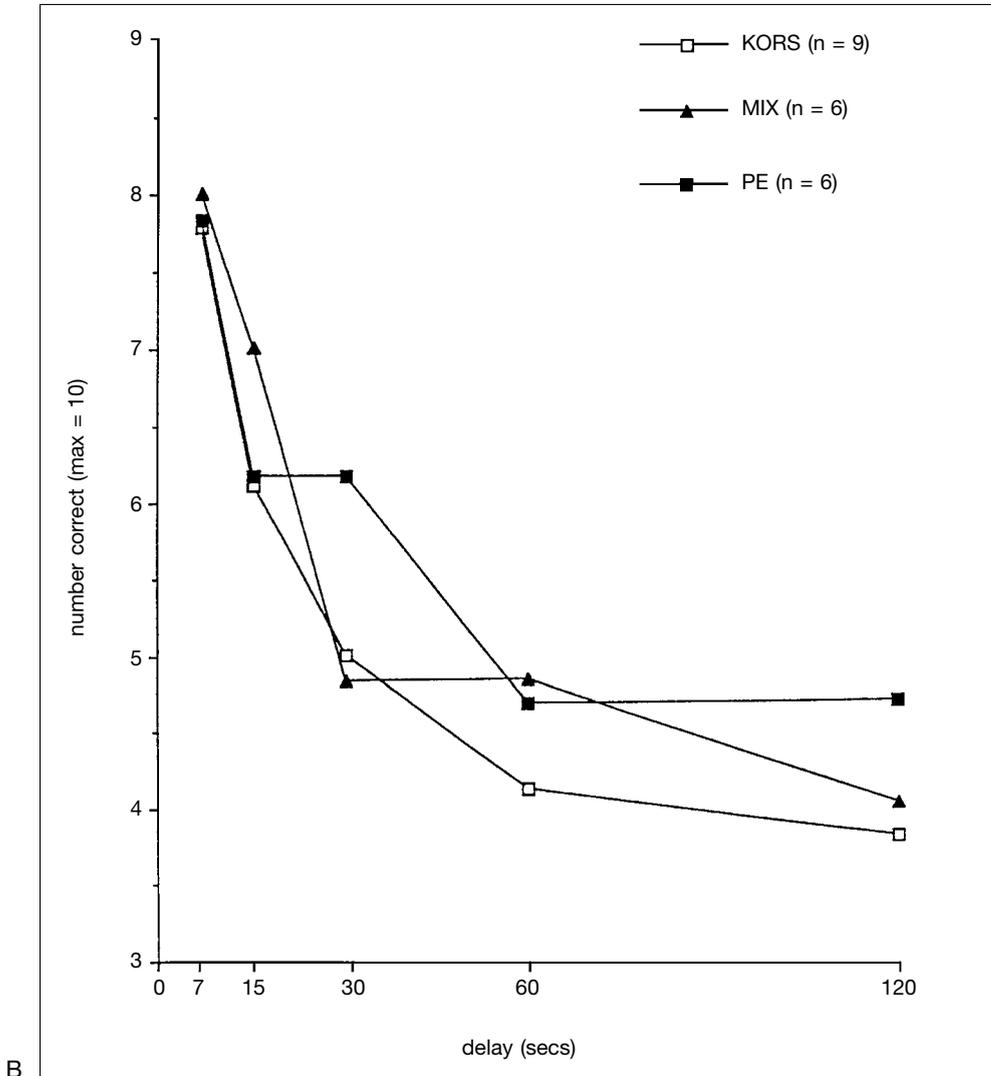
Fig. 1

There was also a significant group by delay interaction, $F_{(4, 136)} = 4.68$, $p = .001$, which is shown in Figure 1A and indicates that the rate of loss of colour information is much faster in the amnesic group. Post hoc tests showed that the difference between groups was not significant at 7SEC and 15SEC, but at 30SEC and thereafter the amnesics performed significantly worse than controls.

The profiles of forgetting shown by each of the groups were examined to investigate the group by delay interaction further. For each group a repeated measures ANOVA was performed with delay (7, 15, 30, 60, and 120 seconds) as the repeated measure. Both groups showed a significant effect of delay (controls, $F_{(4, 56)} = 8.639$, $p < .05$, and amnesics, $F_{(4, 80)} = 38.17$,

p < 0.0005). However, post hoc tests (Newman-Keuls) showed that the profile of forgetting over the 2 minute period was quite different for the controls and the amnesics. Amnesic patients showed nine significant drops in performance which occurred between all but the longer two delays (60SEC and 120SEC) ie. they showed significant forgetting during the first minute, but little thereafter. By contrast, the control subjects only showed five significant drops in performance which occurred between 7SEC and 60SEC and between each of the four shortest delays and 120SEC. This result suggests that the controls were only showing a small drop in performance over time so that this only became significant when delays separated by at least a minute were compared, but that the drop in performance continued during the whole two minute period.

As a further and more conservative test of whether amnesic and control subjects show different forgetting rates soon after learning we compared amnesic and control performance at just the shortest (7SEC) and the longest (120SEC) delays. This reduced the influence of any possibly artifactual data points at intervening delays which may have contributed to the group by delay interaction obtained when all delays were included. The analysis used a 2-way ANOVA with group as the between subject variable and delay (7SEC and 120SEC) as a repeated measure. This revealed a significant group by delay interaction, $F (1, 34) = 8.933$, $p < 0.01$, which, consistent with the previous analyses, indicates that the amnesic patients showed accelerated forgetting over the two minute period taken as a whole.

The 2-way ANOVA with repeated measures on the second factor (delay: 7, 15, 30, 60, and 120 seconds) was repeated without the control group and with the total amnesic group divided into the three aetiological subgroups. This revealed a significant main effect for delay, 7SEC: 7.86 (1.01), 15SEC: 6.38 (1.63), 30SEC: 5.29 (1.71), 60SEC: 4.48 (1.40), 120SEC: 4.09 (1.55), $F (4, 72) = 35.107$, $p < .001$. However, the group main effect was non-significant and, importantly, the group by delay interaction was also unreliable (both F's < 1). Therefore, there was no evidence that the amnesic subgroups forgot at different rates. These data are shown in Figure IB, which reveals that the KORS group initially forgot faster than the PE group, which in turn forgot at a faster rate between the second pair of delays, with the same cross-over pattern for the next two pairs of delays. However, it should be stressed that there were no significant differences between the two groups at any of these delays.

An additional ANOVA compared the number of correct recognition judgement achieved by the amnesic patients at 7SEC and l5SEC with those achieved by the controls at l5SEC and 30SEC. This comparison allowed an investigation of rate of forgetting when the delay between stimulus onset and initial test was approximately equivalent for the controls and amnesics. Collapsed across delay the ANOVA showed no significant effect of group ($F (1, 34) = 0.274$, $p > .05$). However, there was a significant effect of delay ($F (l, 34)= 10.171$, $p < 0.005$) such that, collapsed across group, performance was worse at the second delay and a significant group by delay interaction ($F (1, 34) = 7.265$, $p < .05$) was also revealed. T-tests between delay 1 and delay 2 for each subject group showed that whereas the amnesic group showed a significant difference in performance between the two delays ($t (20) = – 4.601$,

p < 0.001), the difference between performance on the two delays for the controls was unreliable (t (14) = 0.0, p < 1.0).

A correlational analysis was run to determine whether the abnormally fast forgetting of the amnesic group was associated with measures of frontal lobe damage. The difference score between 15SEC and 30SEC was used as the main index of forgetting, because amnesics' forgetting was abnormally fast during this period. The psychometric measures used in the analysis included the following: FAS, WCSTcat, WCSTpe, and CE scores. There were no significant correlations between the 15SEC-30SEC forgetting index and the measures of frontal lobe functioning (ranging from r = .01 for the CE correlation, and r = .31 for the FAS correlation).

DISCUSSION

The present study provides clear evidence of abnormally fast forgetting in both temporal lobe and diencephalic amnesic patients over delays of up to 120 seconds. This faster than normal forgetting was found both in the analyses in which delay was measured from stimulus offset and in an additional analysis which attempted to match the groups with respect to the delay between stimulus onset and initial test. Thus the accelerated forgetting of the amnesic patients is unlikely to be a result of the point from which the retention interval was measured. Even when forgetting was analysed using only the shortest and longest delays, the patients showed faster forgetting, which strongly suggests that the effect does not depend on the temporary blip in the controls' forgetting function. The analysis of amnesia subgroups showed that this abnormally fast forgetting is a general feature of amnesia with no apparent differences between aetiological subgroups. The data showed clearly that patients with encephalitis due to Herpes simplex infection, which is presumed to result in damage to the medial temporal lobes, were no different in their forgetting characteristics than a group of subjects with presumed diencephalic damage due to Korsakoff's syndrome. Not only were subgroup differences statistically unreliable, but there was also no evidence of a trend suggestive of faster forgetting in the PE subgroup compared with the other subgroups.

The results of the present study are consistent with Holdstock, Shaw and Aggleton (1995). Their study also used a recognition memory paradigm but avoided potential problems produced by manipulating stimulus exposure by using a matching manipulation which involved the amnesic patients completing an easier version of the task than controls. Using this procedure both amnesic patients who had medial temporal lobe damage and those with diencephalic damage showed abnormally fast forgetting of abstract designs over a period of seconds.

It is interesting to note that the control subjects seem to forget colour information more rapidly than normal subjects in other studies forget more typical material such as words. This rapid loss of the colour information is probably due to the very fine discriminations subjects are required to make in the task. Future research needs to determine whether the rapid forgetting observed in the present experiment generalises to other sorts of complex

materials e.g., faces or abstract shapes and, if so, why precision-matching recognition is associated with such rapid forgetting. The results also suggest the hypothesis, which needs testing, that amnesic patients show a memory deficit at shorter delays for information which is forgotten at a fast rate by healthy people as in the present study.

The results of the present study contrast both with results of studies which have used the Huppert and Piercy task and those which have used the Brown-Peterson task. The weight of evidence at the present time suggests that amnesic patients do not show accelerated forgetting on either task. By contrast, we found abnormally fast forgetting in both medial temporal lobe amnesia and amnesia due to Korsakoff's syndrome. The design of our task differed from the Huppert and Piercy and Brown-Peterson tasks in a number of ways which could have contributed to the differences in the results found in our and previous studies. Considering the Huppert and Piercy task first, a major difference with our design was that we tested recognition of single items rather than multi-item lists which allowed us to control for a problem of the Huppert and Piercy task which has been thought to result in underestimation of forgetting rate in amnesic patients (Mayes, 1986). Also, we used material that was forgotten faster by controls than that used in Huppert and Piercy studies which has been pictures and sentences. A further difference was the range of delays across which memory performance was assessed. Typically the shortest delay which has been used in previous studies employing the Huppert and Piercy procedure is ten minutes which contrasts with the delays of 7 to 120 seconds used in the present study. Considering the standard Brown-Peterson task, the present task differs from it in the following ways: in the present task performance was measured by forced-choice recognition rather than recall, an initial delay of 7 seconds was used rather than zero seconds which addressed problems of ceiling effects, a matching manipulation was used at the shortest delay and the distractor activity was not in the same modality as the target material. The discrepancy between our results and Huppert and Piercy and Brown-Peterson results will have to be explained by future research.

Like previous published work investigating amnesic forgetting, we have used rate of forgetting to refer to the absolute loss of information as a function of time (delay), we will refer to this as the absolute forgetting rate measure. A contrasting view has been brought to our attention by a reviewer which is that rate should be measured using a relative measure which is derived by expressing the amount lost at any one delay as a percentage of the total amount lost at the final delay tested. We have not used this measure in the present paper for three reasons. First, the relative measure of rate is unstable: Depending on the time point at which one decides to stop (or start) sampling performance, different (i.e. opposite) conclusions may be drawn. This is not the case with the absolute measure we have used. Second, the relative measure does not address the hypothesis we wished to investigate. The experiment was designed to determine whether, given a common starting point at which amnesic and control performance was closely matched, the amount of information retained after certain fixed intervals would differ between amnesic and matched healthy control subjects. If more information is lost over time by one subject group than

the other this will lead to differences in the absolute amount of information recognized at longer delays. This pattern of results will present as a significant group by delay interaction, which is exactly what we found. Third, the use of the relative measure would not allow a direct comparison between our results and the results of previous published studies on rate of forgetting in amnesia as these studies have all used the absolute forgetting rate measure.

Given the results of our study, attention will now be focused on possible explanations of the findings. There are a number of possible interpretations, three of which can be excluded and we will briefly discuss these first.

The first explanation that we believe can be excluded is that accelerated forgetting is related to frontal lobe damage. This explanation stems from the work of Leng and Parkin (1989) who recently reported that performance by a group of Korsakoff subjects on the Brown-Peterson task correlated significantly with measures of frontal lobe dysfunction. In the present study frontal lobe dysfunction was not significantly correlated with rate of forgetting measures suggesting that frontal lobe dysfunction was not influencing our results. The difference between our finding and that of Leng and Parkin (1989) may be due to the extent to which temporal discriminations are necessary for successful task performance. Successful performance on the Brown-Peterson task is thought to depend on the ability to retrieve and identify the target as the one most recently presented from several alternatives (Baddeley, 1990; Greene, 1992). As frontal lobe dysfunction is known to be associated with problems in recency judgements (Milner, Petrides and Smith, 1985; Shimamura, Janowsky and Squire, 1990), then the results found by Leng and Parkin (1989) would be predicted if recency judgements are necessary for successful performance on the Brown-Peterson task. As the present study used a recognition paradigm in which there was no overlap in the colours that served as targets in any one experimental block, successful performance of the task would probably not have required temporal discrimination of response alternatives.

Interference is an accepted cause of accelerated forgetting in normal subjects therefore another potential explanation is that abnormally fast forgetting in the amnesics could have resulted from excessive sensitivity to interference (Warrington and Weiskrantz, 1974). Like the first potential explanation, we think that this possibility can be excluded. The view that explicit memory in amnesics is more affected than explicit memory in normal people by proactive interference has arisen because amnesics typically show more memory disruption from interference than do their control subjects in an AB-AC paradigm. It has been argued by Mayes et al. (1987), however, that amnesics only show greater sensitivity to interference when their memory performance depends primarily on implicit memory whereas their control subjects' performance depends mainly on explicit memory. Mayes and his colleagues found that amnesics showed similar A-B and A-C performance levels and the same high levels of disruption from proactive interference regardless of whether direct (recall the response word that was shown with this stimulus word, i.e., cued recall) or indirect (give the first association that this word brings to mind) memory instructions were used. In contrast, control subjects showed better memory and effectively no interference effect when direct memory instructions were used, and equivalent performance

and levels of interference to the amnesics when indirect memory instructions were used. These findings strongly suggest that, with this task, the amnesics were mainly using implicit memory even when direct memory instructions were given, and that, both in them and their controls, sensitivity to interference was a function of reliance on implicit memory. In direct memory tasks (free recall and recognition) where implicit memory probably has much less of an effect on performance and performance of amnesics and controls has been matched in order to examine forgetting rate, we have found that interference, produced by encoding large amounts of material very similar to the target material, led to closely equivalent levels of memory disruption in amnesics and controls when free recall and recognition of story material (Isaac and Mayes, submitted) and recognition of faces (Mayes et al., 1994) were examined. In both studies, the amnesics' memory was disrupted to an insignificantly smaller degree than that of their controls, and the effect on forgetting rate of both groups was very small despite the high similarity of the interfering and target materials. As the present study also used recognition tests and was of a similar design to these two studies, interference is extremely unlikely to have caused the accelerated forgetting shown by our amnesic patients. Furthermore, in the present experiment, there was little overlap of the sets of hues which were used within each block of trials and a 10 minute interval between blocks of trials so the possibility for interference was minimal.

The final explanation which can be excluded concerns only the results of the patients with Korsakoff's amnesia. This is that the results of the KORS group are due to a visual sensory deficit involving colour discrimination which has been reported in Korsakoffs syndrome (Mair, Doty, Kelly et al., 1986). However, it is difficult to see how this could explain the abnormally fast forgetting observed for the KORS subgroup in the present study. First, a sensory discrimination deficit would be expected to impair recognition performance to the same degree at all delays. Secondly, the distance between hues forming the recognition sets was greater than the mean deviation error score on the Farnsworth-Munsell test reported by Mair et al. (5 versus 3.51), that is, the hues should have been clearly discriminable even to the KORS subgroup. Thirdly, as Mair et al. (1986) pointed out, the problem with hue discriminations was not an isolated deficit but part of a multi-modal sensory impairment. According to these authors, the most likely explanation for the sensory deficits they observed is a generalised impairment in attention which in turn could be linked to damage to "nonspecific" thalamic nuclei and/or to reduced levels of noradrenaline, both of which are associated with Korsakoff's syndrome. Given the matching procedure adopted in the present study, any underlying attentional deficits would have been compensated for, and we can confidently state that the abnormally fast forgetting observed in the KORS group represents a specific problem with memory processes as opposed to sensory/attentional processes.

Elimination of the three explanations discussed above leaves three further interpretations of the results which are all possible at the present time. The first is that there is an impairment in short-term or working memory for the colour stimuli. The duration of verbal STM has been estimated by experimental methods to be 2 seconds or less (Muter, 1980) when rehearsal is not possible and

if similar mechanisms underlie STM for other types of materials, it is unlikely that the present task is tapping STM provided subjects were not rehearsing the stimuli. However, some might suggest that the use of conversation as a distractor activity during the delays did not adequately prevent subjects from rehearsing and that the amnesic patients were showing abnormally fast forgetting from working memory because they did not rehearse the information as well as controls or were unable to do so. It could be argued that the use of a more demanding and controlled task in the delay would have eliminated this possibility. However, it is difficult to select a distractor task that is appropriate but which does not cause direct interference. A task involving colour material would be highly likely to cause retroactive interference. Indeed, Davidoff and Ostergaard (1984) have shown a large drop in recognition performance after only a few seconds when a distractor using similar colours to the target material was interposed between presentation and test. This contrasted with the small drop in perforrnance that followed a verbal distractor task. This effect is highly likely to have been due to interference which is greater with more similar material (Baddeley, 1976). As we wanted to avoid interference we decided not to use a distractor which employed colour material, but to use the interesting and flexible task of conversation as the distractor. We believed that this would be as effective at preventing rehearsal as a more controlled verbal task. This belief was confirmed by pilot work. When conversation as a distractor was compared with the more demanding, but less interesting, task of semantically categorising words as fast as possible, there was no indication of a differential effect of the two tasks on colour recognition at a 30 second delay. Debriefing of pilot subjects also suggested that they not only found it too difficult to image the colours, but that although, at encoding, they sometimes gave verbal labels to the studied colours, like "leaf green", these were pretty unhelpful.

Although we cannot exclude the possibility that conversation allowed the control subjects to rehearse the stimuli at least verbally more than the amnesics, we think this unlikely for two further reasons. First, Holdstock et al. (1995) found that when rehearsal was possible control subjects showed no forgetting across delays even when the task was very difficult and required a very fine discrimination to be made at recognition (controls scoring only 65 percent at the 3 second delay). This finding would predict that if effective rehearsal was occurring in the present study the performance of control subjects would not decline over delay. This was clearly not the case. In addition, we aimed to minimise the effectiveness of any verbal rehearsal by using as stimuli the set of Farnsworth-Munsell hues which do not have unique lexical referents in the English language and by selecting sets of these which were close in hue distance so that colour name could not aid target recognition.

The second possible interpretation is that the appearance of abnormally fast forgetting in the amnesic patients could be due to the differential contributions across delays of intact STM and LTM that is equally impaired at different delays. This combination of intact and impaired memory systems would produce the amnesic pattern, if performance at the shortest delay is a function of STM as well as LTM, but at longer delays it depends only on LTM. The more memory performance at a delay is determined by LTM than STM, the more impaired the

amnesic patients will be. Although plausible, we consider this explanation to be an unlikely account of our results. This is because if STM for colour information has a similar duration to STM for verbal material, given the estimated duration of verbal STM (Muter, 1980), we would not expect STM to be contributing to memory performance at delays as long as 15 seconds where amnesic performance is still not significantly impaired.

The third possible interpretation suggests that the accelerated rate of forgetting of the amnesic patients results from a deficit in the consolidation of information into LTM. Mayes (1995) offers a similar explanation but more specifically postulates a deficit in the consolidation of complex associations. Here we are not justified in making a distinction between complex and simple associations as the nature of the associations required for colour memory are uncertain. We consider the deficit in consolidation to be one of "initial consolidation", which is defined as the "stabilization of long-term memory during the first few hours of learning" (Abel et al., 1995). This has been distinguished from processes occurring over a period of several months or years which have been termed "later transformation", and these have been postulated to "reflect the transfer of the memory trace from one brain site, such as the hippocampus, to another, such as the cortex" (Abel et al., 1995). We are postulating a disruption of initial consolidation as only this can easily explain the accelerated forgetting that occurs in the patients. Given that it has been shown that the formation of synapses following induction of LTP occurs in minutes rather than seconds (Swain et al., 1995), it is unlikely that initial consolidation would be complete within 15 seconds which is the longest delay at which patient and control performance is matched. The consolidation deficit explanation predicts that as performance becomes more dependent on LTM, and the period over which initial consolidation has been active increases, the amnesic patients would become increasingly impaired. This decline in performance would continue until initial consolidation is complete after which the amnesic patients would show a consistently poor level of performance compared with controls, but would not show accelerated loss of that information from the LTM achieved. This interpretation assumes that at the delay at which the amnesics still show insignificantly impaired recognition (15 seconds in the present study) STM is no longer contributing to memory performance. It also assumes an intermediate term memory, which comes into existence immediately but lasts longer than STM, and which is relatively normal in amnesia. This kind of memory is necessary to explain the shape of the forgetting curve and without it normal and amnesic memory would suddenly drop and then later recover. As we found comparable results for all three of our amnesic subgroups, the results would suggest that both medial temporal lobe and midline diencephalic structures are involved in this initial consolidation process.

At the present time it is not possible to determine which of the above three explanations provides the best account of the results because of the uncertainty surrounding the characteristics of STM for colour. Although it seems reasonable to assume that there is STM for colour, its properties such as its duration, whether or not it is impaired in amnesia and the tasks which would be necessary to tap it are currently unknown. As a result we do not know whether the delays

we have used are tapping STM or longer term memory. Determining the duration of STM is not an easy task. It would not seem possible to determine STM duration by lesion studies or by experimental studies using healthy control subjects, but it is possible that functional imaging may help address this issue. It seems plausible to hypothesise that STM involves the continued activation of the neural system that represents the information being remembered (Goldman-Rakic, 1994). If so, it would be possible, using functional MRI, to assess how long the representing neurons remain active during unrelated distracting activity after the stimulus has been removed by measuring over 30 to 60 second samples. When the signal returns to baseline, the activity of the neurons would be considered to have ceased and STM to be no longer functioning (this approach is illustrated by the work of Kato, Erhard, Takayama et al., 1997).

In summary, our study provides unequivocal evidence for abnormally fast forgetting of recognition memory in amnesic patients of several different aetiologies over delays of up to 120 seconds. The nature of the stimuli allowed us to produce a task which was of sufficient difficulty so that control subjects were not performing at ceiling even at very short delays. This allowed us to look at the performance of the amnesic patients at delays shorter than the 10 minutes from which forgetting from LTM has traditionally been investigated. At the present time, although three interpretations of the results are possible, in our view available evidence makes the consolidation deficit hypothesis more likely. Resolution depends on removing the uncertainty surrounding the duration and other features of STM for colours. The results could be explained by impaired STM or working memory for colour, differential contributions of intact STM and LTM which is equally impaired across delays or impairment of initial consolidation of information into LTM. Further research focusing on the characteristics of STM for colour is necessary to reveal which of these is the most appropriate interpretation.

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