RETROSPLENIAL AMNESIA

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SUMMARY

A 39-year-old man developed retrograde and anterograde amnesia following haemorrhage from an arteriovenous malformation situated near the splenium of the corpus callosum. MRI studies demonstrated damage to the splenium, and to a region containing the retrosplenial cortex and the cingulate bundle. The fornix was anterior and inferior to the site of maximal damage, but may have been involved; the stria terminalis was probably spared. Structures known to be important in memory but spared by the lesion included the hippocampus, thalamus, and basal forebrain. The retrosplenial cortex receives input from the subiculum and projects to the anterior thalamus, thus providing an alternative route between hippocampus and thalamus. Perhaps more importantly, medial temporal structures involved in memory receive anterior thalamic input directly via the cingulate bundle and indirectly through a relay in the retrosplenial cortex. We suggest that this thalamocortical portion of Papez' circuit may be important in memory, and that lesions of the cingulum and retrosplenial cortex may cause amnesia by disrupting this pathway.

INTRODUCTION

In 1977, Heilman and Sypert described a patient who developed severe amnesia in association with the growth and removal of a pilocytic astrocytoma near the trigone of the lateral ventricles. These authors postulated that the memory disturbance was caused by involvement of the fornix bilaterally. They recognized that fornix lesions in humans had not consistently been associated with amnesia; however, they argued that, first, cases of alleged normal memory following fornix transection were not well studied neuropsychologically and, secondly, lesions of the posterior fornix interrupt connections between the fornix and thalamus that would be spared by lesions of the columns of the fornix. Authors arguing against the importance of the fornix in memory have discounted Heilman and Sypert's evidence on the grounds that only surgical observation was available to document the extent of the lesion, and that other structures, such as the temporal stem or the posterior hippocampus, might have been involved by the tumour or by surgery (Horel, 1978).

We now report a patient with amnesia associated with haemorrhage from an

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arteriovenous malformation located between the splenium of the corpus callosum and the trigone of the left lateral ventricle. For convenience, we have referred to this area as the retrospinal region, although the actual retrosplenial cortex is only one of the structures in this region. Neuroradiological studies in our case demonstrate no lesions in the hippocampus, temporal stem, or stria terminalis, and it is possible that the lesion did not involve the fornix. It unquestionably destroyed the cingulate bundle and retrospinal cortex, structures that were also involved in the case reported by Heilman and Sypert. These two structures have strong anatomical connections with structures known to be important in memory, raising the possibility that lesions restricted to the retrosplenial area can be associated with amnesia.

CASE REPORT

On March 15, 1984, T.R., a 39-year-old right-handed college-educated accountant, awoke with a severe headache. He was confused, and pyrexial, with a stiff neck and decreased memory. Lumbar puncture yielded blood-stained CSF. He was transferred to the Shands Teaching Hospital, University of Florida. His past medical history and family history were noncontributory. Nine months before the onset of his illness he had moved from California to a responsible post in the offices of a large corporation in Florida.

On admission (March, 15) his temperature was 38° C, and his blood pressure 118/90 mmHg. He had moderate meningism, was somnolent and orientated only for person. He recalled only 1 of 3 objects in 5 min, and had difficulty performing the serial 7s test. Spontaneous speech, comprehension and repetition were normal. He had a right homonymous hemianopia, full extraocular movements and normal pupillary responses. The rest of the cranial nerves were normal. He had no abnormal motor signs or alteration in reflexes and pain sensation was intact.

Urine and blood studies, including clotting studies, were normal. A CT scan showed blood in the ventricular system and an intracerebral haemorrhage just lateral to the splenium on the left (fig. 1). An angiogram showed an arteriovenous malformation (AVM) both above and below the splenium in the pericallosal cistern and the cistern of the velum interpositum, extending into the medial aspect of the trigone of the left lateral ventricle (fig. 2). Feeding vessels arose from interhemispheric branches of the left anterior and posterior cerebral arteries, and from choroidal branches of the left posterolateral choroidal artery. He was treated with dexamethasone, cimetidine, phenytoin and analgesics. He slowly became more alert. Repeat CT scans on March 21 and 29 demonstrated disappearance of the intraventricular blood and partial resolution of the intracerebral haemorrhage. There was slight ventricular enlargement.

Fifteen days after admission, he was alert and cooperative. He was orientated for place, person and year, but was unsure of the month. He remembered 1 of 3 objects after distraction, but did not recall the other 2 even with multiple choice. He had no recollection of the events of his illness, and he was very uncertain about time relationships before his illness; for example, he could not remember if he had moved to Florida before or after Christmas. Spontaneous speech was fluent, without errors, and repetition and naming were normal. He made occasional errors on calculations, but right-left orientation and finger naming were normal.

He had signs of a mild callosal disconnection. Although he made only occasional errors reading aloud and reading comprehension for short commands was normal, he sometimes failed to read correctly items in his left visual field. His writing was normal with his right hand; he made occasional errors with his left hand, but could type correctly with the left hand the same sentences that he had difficulty writing. He had some difficulty drawing with the left hand, but he was not apraxic with either hand.

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FIG. 1. Axial CT scan taken on the day after the haemorrhage demonstrating an intracerebral haemorrhage medial to the trigone of the left lateral ventricle, with extravasation into the ventricle.



FIG. 2. Anteroposterior subtraction view of a left carotid angiogram, demonstrating the arteriovenous malformation arising from posterior branches of the anterior cerebral artery.

Visuospatial abilities were intact. With his right hand, he could draw a cube and a rudimentary map of Florida, and could correctly indicate the location of major cities. He performed the cancellation task normally with either hand. He had a partial right superior quadrantanopia to confrontation, and variable left visual field extinction on bilateral simultaneous stimulation. He could accurately count fingers on either side, and he could name colours in either visual field. Extraocular movements and optokinetic nystagmus were normal, as was the remainder of his neurological examination.

On April 5 the AVM was removed through a left occipitoparietal craniotomy. After several bridging veins along the interhemispheric fissure were divided, the left occipital lobe was retracted laterally, exposing the splenium. The posterior pericallosal artery was followed to the AVM, which was surrounded by an old haematoma. Using microdissection techniques the AVM was removed. The fornix was not directly visualized during the operation.

He remained somnolent postoperatively. A CT scan showed intraventricular blood with increased ventricular size. On April 6 ventriculostomy was performed. Three days later he was still somnolent, but could name objects placed in his left hand and was not apraxic. He then became unresponsive. The ventriculostomy continued to drain blood-stained CSF. He was treated for suspected meningitis, although only one CSF culture was positive (anaerobic streptococcus). He gradually improved, and by April 23 he was alert, but had a more extensive retrograde amnesia than preoperatively. He was discharged on April 28. On June 15, formal visual field testing demonstrated a partial right superior homonymous quadrantanopia. Eleven months after his haemorrhage he tried returning to work on a limited basis, but he was unable to perform his previous work because of memory limitations. Although improving, he has significant memory deficits 2 yrs after his haemorrhage.

Neuropsychological testing

Formal neuropsychological testing was performed preoperatively, and several times during the next 20 months (April 24, 1984; June, July, and October, 1984; November, 1985; January, 1986). Details of the most comprehensive testing are given in the Appendix. We will first consider testing performed preoperatively and within 4 months of surgery. During preoperative and early post-operative testing, he was alert, attentive, and socially appropriate. With the exception of an increase in the span of his retrograde amnesia from 9 months preoperatively to 4 yrs postoperatively, differences between preoperative and early postoperative testing were minimal.

On the Wechsler Adult Intelligence Scale—Revised (Wechsler, 1981), his verbal IQ was 118 preoperatively and 115 postoperatively; his memory quotient of 84 preoperatively was significantly reduced compared with his verbal IQ. All language and language-related functions were intact, as was performance on tasks associated with frontal-subcortical functions (i.e., Verbal Fluency (Benton, 1968; Lezak, 1976), Proverb Interpretations, Stroop, Visual-Verbal Test (Siegel, 1957)). The single exception was poor performance on the Wisconsin Card Sorting test (Milner, 1963; Robinson *et al.*, 1980): abnormal preoperative performance on this test was thought at the time to reflect difficulty in remembering the categories that he was sorting.

In contrast to these preserved functions, he had a profound amnesia. Retrograde amnesia was present at the onset of his illness. He could recall none of the events surrounding his illness, and he thought he still worked in another state. His retrograde amnesia transiently increased postoperatively to encompass more than 4 yrs: he did not remember the birth of his second child (aged 4 yrs), believing he had only one child. His remote memory, however, appeared intact, as assessed by the Albert Remote Memory Battery (Albert *et al.*, 1979). At 6 months postoperatively, his retrograde amnesia had shrunk back to a span of 9 to 12 months.

His anterograde amnesia was profound. For at least 6 months after the onset of his illness, he had virtually no recall of episodic personal information. By lunch he could not recount reliably what he had done that morning. When asked how he felt, he would say that he had improved dramatically from the day before, that everything yesterday was a 'fog', and that he had now reached a 'new level of consciousness and clarity'. Formal assessment of recent memory confirmed this clinical impression: he was severely impaired across all verbal memory tasks, both with auditory and visual presentations. Story recall of the Logical Memory stories from the Weschler Memory Scale—I (Wechsler, 1945) was 6 immediately (average normal = 10), but zero after a 30 min delay (average normal = 8): no information was recalled and there was no overt recognition that stories had previously been presented. Paired associate learning from the Wechsler Memory Scale was impaired and none of the hard associates were learned. He was similarly impaired on word list learning tasks (California Verbal Learning Test (Delis *et al.*, 1983)), recalling no more than 5 of 16 words. After a 30 min delay, both cued and free recall of the California word list were zero.

In contrast, he performed much better on nonverbal recent memory tasks, such as the Milner facial memory task (Milner, 1968), and the Benton visual recognition task (Benton, 1974). Immediate and delayed recall of the Rey Osterreith figure (Lezak, 1976), however, was abnormal.

Follow-up testing

Tests performed 7 months after his haemorrhage showed improvement in his memory quotient and paired associate learning. Twenty to 22 months after his haemorrhage, his full scale IQ had increased to 125, but his memory quotient remained more than 20 points below his verbal IQ, indicating isolated memory impairment. He recalled none of the logical memory stories on the Wechsler memory test after a 30 min delay, and on the California Verbal Learning Test he recalled only 3 of 16 words after distraction without delay, and zero of 16 with a 30 min delay, and his performance on the Peterson-Peterson auditory consonant trigram memory task was at least as impaired at 22 months as it had been 3 to 4 months after the haemorrhage.

DISCUSSION

T.R.'s amnesia is similar to the classic amnesic syndromes described with bitemporal or diencephalic lesions (Butters and Miliotis, 1985). He had severe anterograde amnesia: he initially was disorientated, and remembered very little about daily events. His anterograde amnesia began to improve about 5 months after the haemorrhage, so that by 7 months he was usually orientated as to place and day, but even after 2 years he was unable to carry on the responsibilities of his previous job, and performed poorly on standardized tests of recent memory. He also demonstrated a circumscribed retrograde amnesia. Although he did well on some tests of remote memory (the Albert Remote Memory test and the Florida recent/remote memory battery), he had difficulty recalling with appropriate clarity and specificity personal memories for events occurring in the past 4 years, as demonstrated on a personal autobiographical test of memory. Finally, he had remarkably intact general intellectual functions: there were no consistent deficits on tests of 'frontal lobe' function, and language, praxis, and visuospatial functions that did not rely on memory were intact.

T.R.'s memory deficit was to some extent material-specific. Verbal tasks showed a consistent deficit; while tests of nonverbal memory, except for the Rey-Osterreith Complex Figure Test, were performed normally. Poor performance on the Rey Osterreith Complex Figure task, however, is seen in both right and left hemispheredamaged patients. While T.R. had an abnormal rate of forgetting for verbal material, the rate of forgetting for nonverbal material was not significantly different from controls (*see* Appendix). The results are compatible with the proposal that the predominantly left-sided lesion in this case affected verbal more than nonverbal memory.

More detailed consideration of the results of neuropsychological testing of this patient are to be reported separately.

Localization

Although T.R. has a classic amnesic syndrome, extensive neuroradiological studies demonstrated that the lesions associated with his intracerebral haemorrhage spared the areas classically associated with amnesia: there were no lesions seen in the diencephalon (Victor *et al.*, 1971; Squire and Moore, 1979; Speedie and Heilman, 1982, 1983; Graff-Radford *et al.*, 1984; von Cramon *et al.*, 1985; Rousseaux *et al.*, 1986) or basal forebrain (Whitehouse *et al.*, 1981; Alexander and Freedman, 1983; Volpe and Hirst, 1983; Damasio *et al.*, 1985; Hepler *et al.*, 1985), and both medial temporal lobes (Scoville, 1954; Scoville and Milner, 1957; Cummings *et al.*, 1984; Zola-Morgan *et al.*, 1986) were free of evidence of a destructive lesion. The small lucency in the anterior hippocampus on the left (*see fig. 3D*) probably represents anomalous vessels: it remained lucent on T2 MRI studies (spin echo sequence: 2000 ms pulse repetition time; 90 ms echo delay), suggesting either calcium or blood, but it was not visible on the CT scan or



FIG. 3. MRI scan taken 9 months after the haemorrhage. These T_1 -weighted images were taken using a spin echo pulse sequence with a 500 ms pulse repetition time and a 30 ms echo delay. Sagittal (A), axial (B), and coronal (C) views through the lesion. D, coronal view in the plane of the anterior portion of the hippocampus, demonstrating a small lucency on the left (arrowed). This was one of serial 6 mm coronal cuts, and the abnormality was not seen on any other section. This area also appeared lucent on T_2 -weighted images.

angiogram. Coronal sections at 6 mm intervals encompassing the entire extent of the temporal lobes showed no other lesions. There were no lesions involving neocortex and subcortical damage was confined to the site of the principal lesion, plus a small lesion at the site of a temporary ventricular shunt in the right dorsolateral frontal lobe.

The CT study (fig. 1), the angiogram (fig. 2) and the MRI studies (fig. 3) all locate the pathology near the splenium of the corpus callosum. Two MRI scans, performed 3 and 9 months after the onset of his deficit (and 2 and 8 months after operation) demonstrated a defect in the splenium of the corpus callosum, and an

adjacent lesion posterolateral to the splenium, involving the cortex and subcortical white matter between the splenium and trigone of the left lateral ventricle. This is the location of the most posterior portion of the cingulate gyrus, which is composed principally of the retrosplenial cortex. In the white matter deep to this cortex runs the cingulate bundle. The neuroradiological studies lack the resolution to identify with certainty whether certain small structures, such as the fornix and stria terminalis, were damaged. In the ventricle, the fornix runs anterior and inferior to the site of maximal damage, but may have been damaged, and the hippocampal commissure, directly beneath the splenium may also have been involved. The stria terminalis, adjacent to the caudate nucleus in the inferolateral wall of the lateral ventricle, was lateral to the lesion, and was probably spared. There was no radiological or clinical evidence to suggest right hemisphere involvement.

Pathogenesis of T.R.'s amnesia

Damage to one or more of the above-mentioned structures is presumably responsible for T.R.'s amnesia. Memory loss sometimes occurs after section of the corpus callosum (Dimond et al., 1977; Bentin et al., 1984). This has been attributed to associated damage to the fornix and/or the hippocampal commissure, or to disconnection of the left hemisphere from the right medial temporal region in patients with dysfunctional left medial temporal structures (associated with seizures) (Ross, 1980). The first mechanism might apply to our patient, and will be discussed below. The second appears unlikely; there is little to suggest that our patient's left hippocampus was dysfunctional, and even if it were, a small lesion in the splenium would not be sufficient to disconnect the left hemisphere from the right hippocampus. Patients with cerebral infarctions in the distribution of the left posterior cerebral artery may also present acutely with amnesia (Geschwind and Fusillo, 1966; Damasio and Damasio, 1983). These patients sometimes have lesions in the splenium; however, when amnesic, they always have associated damage to medial temporal structures (Damasio and Damasio, 1983). Although their lesions often extend anteriorly to involve at least a portion of the hippocampus, they may also involve the retrosplenial cortex and cingulum, and the mechanism of memory loss may thus be similar to that in our patient.

Lesions of the bodies of the *fornix* (fig. 4, lesion 1) disconnect the hippocampus from the septum, anterior thalamus and mamillary bodies. Fornix lesions in monkeys produce mild memory impairment (Gaffan, 1974; Owen and Butler, 1981; Moss *et al.*, 1981; Carr, 1982; Bachevalier *et al.*, 1985*a*, *b*). Fornix lesions in humans have had variable effects on memory. There are reports of definite memory impairment after surgical section (Sweet *et al.*, 1959; Hassler and Riechert, 1957) or trauma (Grafman *et al.*, 1985), and there are also reports of normal memory after surgical section (Dott, 1938; Cairns and Mosberg, 1951; Garcia Bengochea *et al.*, 1954) or destruction by tumour (Woolsey and Nelson, 1975). The reports of normal memory have not included careful neuropsychological testing. Heilman and Sypert (1977) also pointed out that surgical section of the columns of the fornix



FIG. 4. Semidiagrammatic representation of Papez' circuit, indicating the position of the retrosplenial area in this circuit. 1 and 2 indicate sites of hypothetical lesions discussed in the text. A = anterior nuclei of thalamus; AL = anterior limbic region; CB = cingulate bundle; CC = corpus callosum; Cing = cingulate cortex; DM = dorsomedial thalamic nucleus; F = fornix; Hip = hippocampus (D = dentate, CA = Ammon's horn, S = subiculum); MB = mamillary bodies; PHG = parahippocampal gyrus; PR/PA = presubiculum and parasubiculum; RS = retrosplenial cortex; SE = septum.

(fig. 4, lesion 2) may spare memory because projections from the hippocampus directly to the anterior thalamic nuclei and to the septal area leave the fornix before the site of the lesion.

Since in both humans and animals, section of the fornix has been associated with at most mild impairment of memory, it would be difficult to ascribe the severe memory loss suffered by the patient of Heilman and Sypert (1977) to fornix lesions alone. The lesion in their patient, however, also involved the retrosplenial cortex and cingulum. It may be that combined lesions of the fornix and these latter areas produce a more profound amnesia than lesions restricted to the fornix. Finally, it should be kept in mind that while it is virtually certain that the cingulate bundle and retrosplenial cortex were involved by T.R.'s lesion, we have no proof that the fornix was affected. It is therefore possible that all the memory deficits in the present case might need to be explained by factors other than fornix damage.

The *retrosplenial cortex*, architechtonically transitional between the allocortex of the hippocampus, and the isocortex of the posterior cingulate cortex, lies in the isthmus of the gyrus fornicatus between the subiculum-presubiculum and the posterior cingulate cortex (area 23) (Vogt, 1976; Braak, 1980) (fig. 5). Anteriorly, it is contained in the inferior bank of the posterior cingulate gyrus; posteriorly, it extends onto the medial surface of the hemisphere, curves downwards behind the splenium of the corpus callosum, and ends at the calcarine fissure.

The connections of the retrosplenial cortex have not been studied extensively in



FIG. 5. The extent of the retrosplenial cortex on the medial aspect of the cerebral hemisphere is indicated by cross-hatching. *Inset* shows a partial coronal section at the level indicated by the dotted line. CC = corpus callosum. Spl = splenium.

primates, but it is clear from observations in various species that retrosplenial cortex is richly connected both to the anterior thalamic nuclei and to medial temporal structures. In the rabbit, the principal projections of the anteromedial and anterodorsal thalamic nuclei are to the granular retrosplenial cortex, while the anteroventral nucleus projects principally to the agranular retrosplenial cortex (Robertson and Kaitz, 1981; Jones, 1985). The lateral dorsal thalamic nucleus (considered to be one of the anterior thalamic nuclei) projects strongly to the cingulate cortex, agranular retrosplenial cortex, and presubiculum and parasubiculum (Jones, 1985). It has been demonstrated in monkeys and rodents that the retrosplenial cortex has reciprocal connections with subiculum and presubiculum and projects back to the anterior and lateral dorsal thalamus (Domesick, 1972; Rosene and Van Hoesen, 1977; Sorensen, 1980, Schwerdtfeger and Sarvey, 1983; Vogt and Miller, 1983). The fibres connecting the retrosplenial cortex with the thalamus and medial temporal lobe structures are carried in the *cingulate bundle* (Mufson and Pandya, 1984).

A lesion of the retrosplenial cortex and underlying cingulate bundle would therefore interrupt an alternative pathway from hippocampus to anterior thalamus (via the retrosplenial cortex). Perhaps more importantly, such a lesion would also interrupt input from the anterior and lateral dorsal thalamic nuclei to retrosplenial cortex and medial temporal lobe (fig. 4). This lesion might substantially disrupt communication between the anterior thalamus and hippocampus, and thus may have an effect similar to lesions in either of the structures that it disconnects.

ACKNOWLEDGEMENTS

This investigation was supported by the Memory Disorders Clinic Grant, State of Florida.

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(Received October 9, 1986. Revised March 17, 1987. Accepted April 13, 1987)

APPENDIX

SUMMARY OF NEUROPSYCHOLOGICAL TEST RESULTS

| Date of testing | 4/2/84 | 67/84 | 11/85-1/86 |
|--|----------|---------|------------|
| Time after haemorrhage | 18 days | 3–4 mos | 20-22 mos |
| Time after surgery | (preop.) | 2-3 mos | 19-21 mos |
| General intelligence | | | |
| WAIS-R | | | |
| Verbal IQ | 118 | 115 | 127 |
| Performance IQ | 86 | | 115 |
| Full Scale IQ | 101 | - | 125 |
| Memory | | | |
| Wechsler Memory Scale | | | |
| Memory quotient (MQ) | 84 | 80 | 101 |
| VIQ-MQ difference | 34* | 35* | 26* |
| Information | 3/6* | 4/6* | 5/6 |
| Orientation | 5/5 | 4/5 | 4/5 |
| Mental Control | 9/9 | 8/9 | 7/9 |
| Stories, immediate | 6 | 6 | 8 |
| Stories, delay | 0** | 0** | 0** |
| Digit span forward | 6 | 7 | 7 |
| Digit span backward | 5 | 4 | 5 |
| Visual Reproduction | | | |
| Immediate | 9/14 | 4/14* | 9/14 |
| 30 min delay | 0/14* | 2/14* | <u> </u> |
| Paired Associates | 6/21* | 10/21* | 16/21 |
| No. of hard items learned | 1* | 0** | 4 |
| Recent verbal memory | | | |
| Word Lists (California) | | | |
| Trial 1 (total 16 words) | 5 | 4 | 5 |
| Trial 5 (total 16 words) | 7** | 5** | - 8** |
| Total trials 1–5 | 19** | 23** | 34** |
| Free recall: immediate | 1++ | 0** | 3** |
| Cued recall: immediate | 4** | 5** | 8* |
| Free recall: 30 min delay | 0** | 0** | 0** |
| Cued recall: 30 min delay | 2** | 0** | 4** |
| Peterson-Peterson words (visual presentation | on) | | |
| Immediate | 100% | 100% | — |
| 20 s, no distraction | 100% | 100% | _ |
| 20 s, with distraction | 40%* | 60% | |
| 4 s, predistraction delay | 80% | 73% | — |
| Peterson-Peterson CCC (auditory presentat | ion) | | |
| Immediate | - | 100% | 100% |
| 3 s | — | 67%* | 75% |
| 9 s | _ | 53%* | 40%** |
| 18 s | _ | 47%* | 33%** |

EDWARD VALENSTEIN AND OTHERS

| Date of testing | 4/2/84 | 6-7/84 | 11/85-1/86 |
|---------------------------------------|--------------|---------------------|------------|
| Verbal rate of forgetting | | | |
| 5 min | | 90% | |
| lh | _ | 73%** | |
| 24 h | | 70%** | |
| 5 d | _ | 50%** | ` |
| Kimura Recurring Words | - | 38 | |
| Memory, recent, nonverbal | | | |
| Kimura Recurring Figures | 45 | 47 | |
| Milner Facial Memory | 9/12 | 9/12 | 9/12 |
| Benton Visual Recognition | 13/15 | | _ |
| Rey Osterreith Figure | | | |
| Copy (score, pc) | 34 (pc = 75) | 34 (pc = 75) | |
| Immediate recall pc | 2 (pc < 25) | 16 (pc < 25) | |
| Delayed recall pc | 2 (pc < 25) | 0 (pc < 25) | |
| Nonverbal rate of forgetting | | | |
| 10 min | _ | 77% (WNL) | - |
| 1 h | _ | 63%** | |
| 24 h | | 56% | |
| 5 d | | 60% | |
| Remote memory | | | |
| Albert Remote Memory (1920s-1970s) | | | |
| Public events recognition | | 65% | |
| Event recall | — | 44% | |
| Face recognition | _ | 58% | |
| Florida recent/remote (1974–1984) | | | |
| Recognition | _ | 85% | 95% |
| Recall | — | 80% | |
| Personal autobiographical (1980-1984) | _ | 4/10 (chance = 20%) | |
| Frontal and miscellaneous | | | |
| Wisconsin Card Sorting | ABNL | NI | |
| Controlled Verbal Fluency | 85th pc | 75th pc | 95th pc |
| Shipley Abstraction score | - | 36/40 | |
| Luria tasks: | | | |
| Contrasting motor programs | 100% | — | |
| Go-no go motor programs | 100% | - | |
| Recursive writing of 'mn' | WNL | _ | |
| WAIS similarities (scaled scores) | 18 | 18 | 18 |
| Boston naming | 100% | 100% | |

* > 1 SD from the means of normal controls; ** > 2 SD from the means of normal controls; \rightarrow = test not performed on that date: pc = percentile; WNL = within normal limits; ABNL = abnormal; NI = normal.

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