Transient global amnesia: neuropsychological dysfunction during attack and recovery in two "pure" cases

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SUMMARY Two patients with transient global amnesia are reported. Comprehensive neuropsychological evaluation, during the amnesic episode, as well as follow-up examinations on memory were performed. The course of the amnesia was exemplified by two comparable memory tests in different modalities. Partial retrograde amnesia and complete anterograde amnesia were demonstrated during the transient episode. Objective recovery was found to be slower than subjectively experienced, but test performance was completely normal one month after onset.

Bender¹ was the first to describe transient confusion and amnesia in 12 cases. Numerous reports followed and in 1964, Fisher & Adams² first reviewed the literature and labelled the syndrome transient global amnesia. Of the many reported cases with transient global amnesia only six³⁻⁷ had some neuropsychological assessment during the amnesic phase, one of which had been reexamined 1 week and 6 months later, the other 15 months later.^{5,7} Neuropsychological assessment during the episode and close follow-up examinations on patients with transient global amnesia are of special interest since neurological examination as well as other diagnostic methods such as EEG and CT scan are usually normal. Objective test results may contribute to differential diagnosis especially from confusion states of other origins.

We report two patients with transient global amnesia in whom comprehensive neuropsychological evaluation during the attack as well as repeated memory testing during recovery were possible. The course of the amnesia is exemplified by two comparable memory tests.

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Case 1

This was a 40-year-old healthy right-handed policeman and former high school teacher without vascular risk factors and with an uneventful medical history. During gymnastics he suddenly became confused. Back at work again he kept asking the same questions over and over in an attempt to orient himself. He called his wife who immediately brought him to the local physician who noticed no neurological abnormalities and prescribed co-degocrine mesylate. Back home, the patient complained about feeling a bruise in the back of his head. He then drove himself miles through heavy traffic to the hospital without difficulty. On admission neurological examination, EEG, CT scan and cerebro-vascular Doppler sonography were completely normal. On mental status examination, the patient was disoriented in time but not in space. He appeared confused and asked the same question over and over, but was able to maintain a coherent train of thought. His retrograde amnesia was of several months with only fragments in between and there was a complete anterograde amnesia. There was, however, no confabulation and the patient showed a completely normal social interaction and an adequate concern about his disability. He spontaneously reported difficulties in remembering the chronological order of events, for example he knew the colour and type of his car, but did not know whether he owned it at present or did previously. He was aware of being in the hospital because he remembered having watched a helicopter landing, but he was unable to indicate whether this event took place a few minutes ago, a few hours ago or on a previous day. This kind of defect was no longer apparent 72 hours after onset of the episode. Extensive clinical neuropsychological evaluation of higher cortical functions was performed on admission during the episode (2½ hours after onset): questions referring to autobiographical and public events revealed a retrograde amnesia of several months. Anterograde amnesia was

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tested with two comparable tests, the Rey auditory-verbal test and the Rey visual learning test.⁸ In the verbal test, 15 words are read to the patient, which he then has to repeat from memory. This is repeated 5 times (learning) and after 40 min, the patient has to recall as many words as possible. In the visual learning test, 15 figures are presented one at the time and the patient has to draw them. This is repeated 5 times and after 40 min, recall is requested. In the verbal task, the patient recalled 4, 6, 5, 6, 5 in the learning phase and recalled none after 40 min. In the visual task, he recalled 1, 2, 4, 2, 3 and later 5. Digit span forward was 7, backward 5, visual span was 6. The Rey complex Figure^{9 10} was copied without errors, but 90 min later, only three elements (out of 18) were recalled and the main structure of the figure was rotated by 90 degrees. He could draw a Necker cube without difficulty and there were no signs of buccofacial, ideomotor and ideational apraxia. Objects, colours and famous faces he recognised and named instantly. Spontaneous speech, comprehension and repetition were normal, so was writing and visual and auditory reading. Verbal and nonverbal fluency and the Stroop Color Word Test^{11 12} were normal.

Except for memory all other clinically evaluated higher cortical functions were normal. Therefore, the course of recovery was followed with two parallel versions of the two memory tests and the Rey complex Figure at 9, 24, 48, 72 hours, and 1 month after onset of the amnesic episode. The memory performance of all examinations is summarised in the table.

Nine hours after onset, the retrograde amnesia shrank to about 2 days prior to the episode. Verbal learning improved (5, 6, 5, 6, 7, 10), but there was no recall. Of the 15 figures he recalled only three. The copy and recall of the Rey complex Figure was practically identical to the first testing: it was still rotated and he remembered five elements and displaced three. Inquiry revealed still defective time perception. *Twenty-four hours past onset*, there was improved verbal learning (7, 11, 12, 13, 14), but recall was only nine. Of the 15 figures he now learned 8, 10, 13, 11, 13 and recalled later 11 correctly. Again, the Rey Figure was rotated. Time perception was still defective. *Forty-eight hours past onset*, there was amnesia for the length of

the episode (ca 3 hours). Verbal learning was 8, 9, 12, 12, 13, recall nine, visual learning was 9, 11, 14, 13, 13 and recall 14. Of the correctly copied Rey Figure he remembered eight elements and displaced one without rotation. *Seventy-two hours past onset*, verbal learning was 8, 9, 12, 14, 13 and late recall eight, visual learning was 13, 14, 15, 14, 15 and recall 14. *One month after onset*, all functions recovered: verbal learning was now 8, 13, 14, 14, 13, recall 12, visual learning was 14, 15, 15, 15, 15 and all 15 figures recalled later. Of the Rey Figure he recalled 15 elements and displaced only one element, without rotation. The amnesia for the length of the episode remained.

Case 2

This was a 60-year-old right-handed railway supervisor without vascular risk factors and with an uneventful medical history, except for transient global amnesia nine months prior to admission. He suddenly became confused while bending over while washing his car and he repeated questions over and over. His wife brought him immediately to the hospital for an EEG as she was instructed after the earlier attack in case of the recurrence of a similar event. On admission neurological examination, EEG, CT scan as well as cerebro-vascular Doppler sonography were normal. On mental status examination the patient was disoriented in time but not in space, he appeared confused and repeated the same questions. No confabulation was noted. His fragmentary retrograde amnesia was of several months and there was a complete anterograde amnesia. Social interaction and affective behaviour were normal. As in the first case, this patient also spontaneously reported to have no sense for time estimation, for example, he remembered his two daughters, knew their names, but was unable to say who was born first. He knew the facts of events but not the time of their occurrence, for example, he remembered having eaten breakfast, but not whether this was just a few minutes or a few hours ago despite his correct time calculation by consulting his watch. His disturbed time perception was no longer apparent after 3 days. Similar neuropsychological evaluation as in case 1 was performed during episode (2½ hours after onset): there was a fragmentary retrograde amnesia of several months. There was an

Table Summary of the memory defects during the episode and follow-up for case 1 and case 2

Time after onset		Case 1	Case 2
(during episode)	retrograde	present	present
	anterograde	defective verbal and visual learning and recall, in Rey Complex Figure 90° rotation	defective verbal and visual learning and recall, in Rey Complex Figure 90° rotation
		time perception defective	time perception defective
	immediate span	normal auditory-verbal and visuo-spatial span	normal auditory-verbal and visuo-spatial span
9 hr.	retrograde	present (improved)	present (improved)
	anterograde	no change, Rey Figure still rotated	no change, Rey Figure still rotated
		time perception still defective	time perception still defective
24 hr.	retrograde	present (improved)	present (improved)
	anterograde	no change	verbal learning improved
48 hr.	retrograde	amnesia for length of episode: ca.3 hr.	amnesia for length of episode: ca.6 hr.
	anterograde	improved, Rey Figure no longer rotated	improved, Rey Figure no longer rotated
		time perception defective	time perception defective
72 hr.	retrograde	amnesia for length of episode	amnesia for length of episode
	anterograde	learning intact, verbal recall still defective	learning intact, verbal recall still defective
		time perception normal	time perception normal
1 mon		total recovery except for remaining amnesia for length of episode.	total recovery except for remaining amnesia for length of episode.

Fig 1 Rey auditory verbal learning and recall and Rey visual learning and recall during the episode and follow-up in 2 cases with transient global amnesia.

anterograde amnesia: the Rey verbal learning for the five phases was 3, 3, 4, 4, 5 and there was no recall, and of the 15 figures he recalled none. Of the Rey Figure he copied 17 elements and slightly displaced four, recall was rotated by 90 degrees, four elements remembered and two displaced. Immediate span, language, praxis, gnosis, writing and reading were normal. Verbal and nonverbal fluency and the Stroop test were normal. Besides the amnesic syndrome, there were no other higher cortical defects. Therefore, only memory tests were performed after 9, 24, 48, 72 hours, and 1 month after onset, as in case 1. The memory performance of all examinations is summarised in the table.

Nine hours after onset, the retrograde amnesia shrank to about 1 month prior to the episode, all other functions were similar to the first testing. *Twenty-four hours after onset*, the retrograde amnesia shrank to the evening prior to the episode (ca. 15 hours). Verbal learning improved (8, 10, 14, 13, 14), recall was 14, visual learning was still poor (3, 5, 5, 5, 6), recall five. Of the correctly copied figure he recalled four elements and displaced one. *Forty-eight hours after onset*, amnesia left only for the length of the episode (ca. 6 hours). Verbal learning was 6, 8, 10, 10, 11 and late recall eight, visual learning was 9, 10, 13, 11, 10 and late recall 10. Of the correctly copied Rey Figure, he recalled

seven elements with the main structure still rotated. Time estimation was still insecure. *Seventy-two hours after onset*, verbal learning was 8, 10, 12, 9, 12 late recall nine, visual learning was 9, 11, 11, 10, 10, late recall 11. Of the Rey Figure he remembered five elements. *One month after onset*, the amnesia remained for the length of the episode. Of the 15 words he learned now 5, 8, 9, 11, 12 and recalled later 11, of the 15 figures he kept 13, 13, 13, 11, 12 and recalled 13. Of the Rey Figure he recalled 13 and displaced three elements.

Figure 1 illustrates the recovery course of the anterograde amnesia for the two comparable memory tests, the Rey auditory verbal test and the Rey visual learning test. In fig 2, the copy and recall with the 90 degree rotations of the Rey Complex Figure are illustrated.

Conclusions

Both our cases are strikingly "pure" examples of transient global amnesia displaying no neurological and neuropsychological abnormalities other than a defect in memory. Their test performance during the episode as well as their recovery course were almost identical, that is characterised by a sudden onset, a

Fig 2 Copy and recall (90 min later) of the Rey complex Figure during the transient global amnesia episode demonstrating a 90° rotation in recall.

retrograde amnesia with more severe impairment of recently acquired memories than older memories and a recovery within 2 days. The anterograde amnesia improved after the first day but normalised only after 1 month despite the subjective feeling of complete recovery after the first day. In both cases, however, a permanent amnesia for a few hours persisted. Moreover, for the first 72 hours from onset of the episode, both patients clearly remembered some events but complained of an inability to place them in chronological order. This failure to encode the temporal relations among events was recently discussed in detail by Hurst & Volpe.¹³ This defect alone could explain sufficiently the temporary failure to integrate new and retrieve old information during the episode as well as the confusional state. Consequently, we found a dissociation between impaired episodic memory, that is, memory for events that are tied to a temporal and spatial context, and an unaffected semantic memory, that is, memory for automatised knowledge such as intact

motor and social programs and the recognition of famous faces. Furthermore, both cases recalled the normally copied Rey Complex Figure in 90 degree rotation. In adult brain damaged patients, this specific kind of rotation seems not to be a direct perceptual defect and is most frequently observed in patients with lesions in the right temporal-basal area.¹⁰ Immediate span was intact as well as other higher cortical functions.

The results of our investigation show that except for the shortness of the episode, the pattern of memory disturbance in transient global amnesia is the same as that in other amnesic syndromes, and reasonably, the same neuroanatomical structures or pathways may be compromised. However, the present results also show that even with refined neuropsychological testing, complete recovery is possible. This differs from the findings of Mazzucchi *et al*¹⁴ who still obtained neuropsychological defects in follow-up investigations in 16 patients. On the other hand, by following the course of recovery especially

during the first days after onset, we could also demonstrate that complete normalisation of the retrograde amnesia (ca. after 2 days) and the anterograde amnesia (ca. after one month) takes in fact longer than subjectively experienced.

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