

Short Report

Transient global amnesia—a hippocampal phenomenon?

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SUMMARY A case of transient global amnesia of clear vascular aetiology is described. Results of neuropsychological testing carried out during the attack clarify the nature of the memory disorder and suggest that the critical region of ischaemia is the medial temporal area around the hippocampus. Follow-up testing suggests that no lasting memory impairment resulted.

The syndrome of transient global amnesia (TGA) has been increasingly documented in recent years but most reports rely on subjective description. Shuttleworth and Wise¹ and Gordon and Marin² provide the only clear description of neuropsychological testing performed during the attack. With the exception of studies by Mathew and Meyer³ and Steinmetz and Vroom,⁴ follow-up data are also lacking. The present study reports a case of TGA of vascular aetiology, fully documented and followed up with neuropsychological testing. It clarifies the nature of the memory disorder and the degree of its recovery.

CASE REPORT

A 35-year-old carpenter presented following two episodes of vertigo, dysarthria, right facial paraesthesiae, ataxia to the right and occipital headache. Clinical examination revealed grade 1 nystagmus to the right and marked incoordination of both right upper and lower limbs. No other abnormalities were evident.

Following resolution of the signs within 48 hours, he suffered a further episode of vertigo, nausea and vomiting, with right hand incoordination and blurred vision. His wife reported that he did not recognise her and was unaware of the reason for his admission to hospital. Neuropsychological examination was undertaken during this five-hour episode of transient global amnesia

and repeated the following day. Vertebrobasilar disturbance was suggested by the clinical picture and the results of a CAT scan, which revealed a right cerebellar infarct, and angiography, which showed a tight stenosis at the origin of the right vertebral artery. One month later the patient underwent right vertebral endarterectomy and was re-tested five weeks after this.

NEUROPSYCHOLOGICAL TESTING

The tests administered included a short form of the Wechsler Adult Intelligence Scale,⁵ the Wechsler Memory Scale,⁶ two verbal memory tests⁷ and two tests developed by Lhermitte and Signoret.⁸ The last named were learning tasks—one of the spatial arrangement of nine pictures, the other of a logical arrangement of nine geometric figures (blue, red and yellow circles, triangles and squares). Lhermitte and Signoret were able to differentiate qualitative features of the amnesic disorder in patients with lesions confined to the hippocampus from that of patients with mamillo-thalamic lesions on the basis of their performance on these tests. Colour naming, reading, writing, drawing and word-finding abilities were also assessed, since defects of these may result from infarction of the posterior cerebral arteries.⁹ Practice effects were eliminated by use of parallel test forms.

Results and discussion

During the amnesic episode the patient was friendly and co-operative, but totally confused and disoriented, showing much agitation. Several

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Table Results obtained on sequential neuropsychological testing

	During episode (12.9.79)	Following day (13.9.79)	Follow-up (13.11.79)
<i>WAIS</i>			<i>NHATS</i>
Information	12	14	14
Similarities	12	16	13
Digit span	9	10	9
Prorated verbal IQ	106	127	112
Picture completion	—	14	15
Block design	—	10	15
Object assembly	—	11	—
Prorated performance IQ	—	116	138
<i>WMS (Form I)</i>			<i>(Form II)</i>
Information	4	6	6
Orientation	1	5	5
Mental control	5	8	9
Memory passages	4 (no recall after half hour)	13 (good recall after half hour)	15
Digits total	10 (6/4)	11 (6/5)	10 (5/5)
Visual reproduction	4	9	11
Associate learning	4.5 (3-0, 1-0, 5-0)	14 (6-1, 6-2, 6-2)	20 (6-3, 6-4, 6-4)
Memory quotient	63	110	132
<i>Luria word list</i>			
Trial	1 2 3 4 5 6 7	1 2 3	—
No. of words recalled	4 5 3 7 2 4 2	6 8 10	—
Recall after 3 minutes	0	8	—
<i>Word triads</i>			
Cat-house-forest	correctly repeated	correctly repeated	
Night-needle-pie	correctly repeated	correctly repeated	
Recall 1	house-cat-needle	cat-house-forest	
Recall 2	night-needle-pie	night-needle-pie	
<i>Spatial arrangement</i>			
Trial	1 2 3 4 5 6 7 8	1 2 3	1
Number correct	0 2 0 1 3 2 1 0	8 9 9	9
Evocation (free recall)	0	8	9
Designation (pictures as cues)	0	9	9
Arrangement	1	9	9
<i>Logical arrangement</i>			
Trial	1 2 3 4 5 6 7 8	1 (described	1
Number correct	5 7 7 4 6 7 6 8	0 arrangement	0
Evocation	0 (got symbols correct	9 after first	9
Designation	5 but confused colour	9 trial)	9
Arrangement	3 arrangement)	9	9

minutes after being given a cup of coffee he said, "Whose coffee is that?" He was unable to describe the events of the past few days, indicating some degree of retrograde amnesia, but gave details of his job, family etc., without difficulty. He showed no confabulation and his reasoning powers were good. Colour-naming, reading and drawing were performed well; his writing was accurate but poor due to right hand inco-ordination and he had no word-finding difficulties.

He obtained exceedingly low scores on the Orientation, Mental Control, Logical Prose, Visual Reproduction and Associate Learning subtests of the WMS during the attack (table). In this and other memory tests he exhibited a profound inability to retain any new material, whether verbal or non-verbal, or presented visually or auditorily. This substantiates the findings of Shuttleworth and Wise.¹ However, his digit span was within normal limits, reflecting an intact immediate memory, and although there was some improvement in his general knowledge the next day, his memory for past information and con-

ceptual skills were relatively good.

The patient thus exhibited the characteristic pattern associated with the axial amnesic syndrome which commonly results from bilateral medial temporal lobe lesions. This was probably caused by posterior vascular insufficiency involving the hippocampo-mamillo-thalamic system.

The Spatial and Logical Arrangement tests proved sensitive enough to distinguish certain qualitative features of his memory problem. His total inability to learn the Spatial Arrangement contrasted with a vastly better performance on the Logical Arrangement test. On this task he was able to attain a significant reduction in errors by discovering the concept underlying the array. This suggests that his difficulty was not so much one of information processing (the logical coding of material for efficient storage) or selective retrieval (since he was not aided by cued recall), but a simple defect in the activation-consolidation process.

The findings of Lhermitte and Signoret⁸ suggest that this picture is seen in patients with purely hippocampal lesions. Those with mamillo-thalamic

lesions were unable to learn the logical array due to associated disconnection from the frontal lobes, which play a significant role in the formation of strategies for efficient coding and storage of information and selective retrieval. The patient's acute awareness of his difficulties and lack of confabulation on free recall also indicate a lack of thalamic involvement. It appears that the medial temporal regions around the hippocampus were most affected by ischaemia in the posterior circulation.

The poor performance on the Mental Control subtest of the WMS, resulting from lack of concentration and slowness, may have been due to ischaemia affecting the brain stem reticular formation. His subsequent improvement on tests of general knowledge and verbal abstraction also suggests some generalised depression of cerebral function during the episode.

The following day, the patient had no recollection of the period of and shortly preceding the amnesic attack. His memory had improved considerably, but he did show evidence of slight memory impairment, obtaining a Memory Quotient which was 17 points lower than his verbal IQ. He performed well on all other tests. However, further improvement was evident on follow-up testing nine weeks later, when he obtained a Memory Quotient of 132. During the intervening period the arterial stenosis had been corrected and he had suffered no further symptoms of vertebrobasilar insufficiency.

In conclusion, TGA apparently has the characteristics of an axial amnesia due to bilateral temporal lobe abnormality. In the present case it appears that this resulted from ischaemia affecting

the hippocampus, rather than the mamillo-thalamic region. Follow-up testing suggests that TGA may produce slight memory impairment shortly following the attack, but this may improve following treatment and the consequent resolution of symptoms.

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References

- 1 Shuttleworth EC, Wise GR. Transient global amnesia due to arterial embolism. *Arch Neurol* 1973; **29**:340–2.
- 2 Gordon B, Marin OSM. Transient global amnesia: an extensive case report. *J Neurol Neurosurg Psychiatry* 1979; **42**:572–5.
- 3 Mathew NT, Meyer JS. Pathogenesis and natural history of transient global amnesia. *Stroke* 1974; **5**(3):303–11.
- 4 Steinmetz EF, Vroom FQ. Transient global amnesia. *Neurology (Minneapolis)* 1972; **22**:1193–200.
- 5 Wechsler D. Wechsler Adult Intelligence Scale Manual. New York: Psychological Corporation, 1955.
- 6 Wechsler D, Stone CP. Wechsler Memory Scale. *J Psychol* 1945; **19**:87–95.
- 7 Luria AR. The Neuropsychology of Memory, Washington: V H Winston and Sons; 1976.
- 8 Lhermitte F, Signoret JL. Analyse neuropsychologique des syndromes amnesiques. *Rev Neurol (Paris)* 1972; **126**:161–78.
- 9 Benson DF, Marsden CD, Meadows JC. The amnesic syndrome of posterior cerebral artery occlusion. *Acta Neurol Scand* 1974; **50**:133–45.