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Impaired picture recognition in transient epileptic amnesia

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ABSTRACT

Transient epileptic amnesia (TEA) is an epileptic syndrome characterized by recurrent, brief episodes of amnesia. Transient epileptic amnesia is often associated with the rapid decline in recall of new information over hours to days (accelerated long-term forgetting — 'ALF'). It remains unknown how recognition memory is affected in TEA over time. Here, we report a systematic study of picture recognition in patients with TEA over the course of one week.

Sixteen patients with TEA and 16 matched controls were presented with 300 photos of everyday life scenes. Yes/no picture recognition was tested 5 min, 2.5 h, 7.5 h, 24 h, and 1 week after picture presentation using a subset of target pictures as well as similar and different foils.

Picture recognition was impaired in the patient group at all test times, including the 5-minute test, but it declined normally over the course of 1 week. This impairment was associated predominantly with an increased false alarm rate, especially for similar foils. High performance on a control test indicates that this impairment was not associated with perceptual or discrimination deficits.

Our findings suggest that, at least in some TEA patients with ALF in verbal recall, picture recognition does not decline more rapidly than in controls over 1 week. However, our findings of an early picture recognition deficit suggest that new visual memories are impoverished after minutes in TEA. This could be the result of deficient encoding or impaired early consolidation. The early picture recognition deficit observed could reflect either the early stages of the process that leads to ALF or a separable deficit of anterograde memory in TEA. Lastly, our study suggests that at least some patients with TEA are prone to falsely recognizing new everyday visual information that they have not in fact seen previously. This deficit, alongside their ALF in free recall, likely affects everyday memory performance.

1. Introduction

Transient epileptic amnesia (TEA) is a syndrome of mesial temporal lobe epilepsy (TLE), characterized by brief, recurrent episodes of transient amnesia occurring as a result of epilepsy. During these episodes, declarative memory is impaired, while other cognitive functions remain intact [1].

In addition to interictal (between attacks) deficits of *remote* autobiographical [2–4] and topographical memory [2], almost half (44%) of patients with TEA describe interictal deficits in the retention of *recently* acquired memories [2,5–7]. This persistent interictal memory deficit is typically more troublesome for patients with TEA than are the occasional, brief amnesic episodes caused by seizures [8]. Patients with TEA describe this interictal memory deficit as an 'evaporation' of memories for recent events [8]. Systematic assessment of their free recall and cued recall over time corroborates this complaint, demonstrating a pattern of normal or near-normal recall over standard delays, followed by a rapid decrease in verbal, visual, and real-life event recall over days to weeks [2,5–7,9]. Recent studies show that this 'accelerated long-term forgetting' (ALF) in TEA can become apparent as early as 3–8 h postacquisition, without any requirement for intervening sleep [10,11]. It is hypothesized that ALF is associated with impairment of later consolidation processes [2,5,7,11,12], although it remains possible that it is the result of an early deficit occurring close to the time of memory acquisition [11,13].

Transient epileptic amnesia has been associated with subtle atrophy in the hippocampus and in perirhinal and orbitofrontal cortices, the volumes of which correlate with anterograde memory performance in TEA, including free recall and recognition [14,15]. However, no structural correlates have been found for forgetting rates over extended periods (i.e., ALF), hinting that ALF in TEA may reflect physiological disruption of memory networks by subclinical epileptiform activity [14,15].

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The behavioral TEA study reported here focused on visual *recognition memory*, which can be impaired in TEA [2,6]. Visual recognition memory is important in daily life for identifying faces, objects, or scenes that the patient has previously encountered, and any deficit in recognition memory is likely to contribute towards real-life memory difficulties experienced by the patient. Indeed, patients with TEA sometimes complain of difficulties in recognizing people or places that they have encountered before [2,16]. However, unlike free recall and cued recall, visual recognition memory has not been examined in detail and systematically over time in TEA.

The two previous studies on recognition memory in TEA included standardized recognition tests but revealed mixed and inconclusive findings: Manes et al. [6] showed that while patients with TEA and controls did not differ significantly in forced-choice story recognition after 30 min, the patients performed significantly worse than the controls on the same test after a delay of 6 weeks, thus suggesting that recognition can be affected by ALF. In contrast, however, the patients with TEA performed as well as the controls at recognizing 4 presented figures, both after 30 min and after 6 weeks. It is of note that performance was at ceiling on this figure recognition test, likely owing to the very small set of stimuli applied and the repetition of the 30-minute test stimuli at the 6-week juncture.

Unlike Manes et al. [6], Butler et al. [2] revealed a subtle, albeit significant, recognition deficit in patients with TEA over standard delays in clinical tests of story recognition (WMS-III logical memory, [17]) and word and face recognition (recognition memory test, [18]). However, since these tests were not repeated after longer delays, it is unclear how these subtle early deficits fared over time in the patients with TEA relative to the controls. The study also included a 3-week recognition test for 15 words and 10 visual designs that had been learned to a 90% criterion and tested via free recall at earlier test intervals in a subgroup of patients who performed normally on standard memory tests at a 30-minute delay [19]. These recognition tests revealed a 3-week recognition deficit in TEA for words [5,19], but not for visual designs, for which performance was high in both groups. Given that the two 3-week tests differed not only in modality but also in test type (words: yes/no; designs: forced choice), the basis of these mixed long-term recognition memory findings is unclear. Moreover, since no recognition testing was conducted for these words and designs after standard (30 min) delays, it is unclear to what extent 3-week recognition differed from baseline, and thus whether or not the 3-week recognition deficit in TEA reflected ALF.

In order to address this gap in the TEA recognition memory literature, we examined in a systematic and controlled way picture recognition memory in patients with TEA by (i) applying *the same test* over *standard, intermediate, and long delays*, (ii) using a *sufficiently large pool of items* to avoid ceiling effects, (iii) probing a *different subset of items* at each juncture to avoid repeated testing, and (iv) including as foils images that were similar to the target pictures as well as foils that were different from the target pictures, to allow for the teasing apart of detail memory and gist memory.

2. Materials and method

2.1. Participants

Seventeen patients with TEA and 18 controls were recruited for the study. One patient and two controls could not complete the study because of unavailability and were therefore excluded from the analysis. All 16 included patients met the diagnostic criteria for TEA [20]: (i) a history of recurrent, witnessed episodes of transient amnesia; (ii) intact cognitive functions (aside from memory) during typical episodes, as judged by a reliable witness; and (iii) evidence for a diagnosis of epilepsy based on one or more of the following: epileptiform abnormalities on EEG, concurrent onset of other clinical features of epilepsy (such as lip smacking or olfactory hallucinations), and clear-cut response to anticonvulsant therapy. Moreover, all patients complained of ALF, and 11 (69%) of these 16 patients showed ALF on a verbal cued recall test, which is reported in full in Hoefeijzers et al. [11]. All patients were on anticonvulsant monotherapy, and, at the time of testing, had been free from overt seizures for at least six months. Finally, all participants spoke English as their first language and had no symptoms of psychiatric disturbance.

Table 1 gives clinical information for the 16 patients with TEA, including the grounds for the diagnosis of TEA and the presence/absence of ALF in the verbal cued recall test.

The 16 patients and 16 controls were matched for age and education and were assessed on a battery of cognitive tests: the National Adult Reading Test (NART) [21] and the Wechsler Abbreviated Scale of Intelligence (WASI) similarities and matrix reasoning subtests [22] were used to assess general intelligence. The Wechsler Memory Scale — III (WMS-III) logical memory test (immediate and 30-minute delayed recall of story A only) [17] and delayed recall of the Rey–Osterrieth Complex Figure Test [23] were used as anterograde memory measures. The copy of the Rey–Osterrieth Complex Figure Test also was applied as a measure of visuospatial perception. The FAS letter fluency test [24,25] was used to examine executive function. Lastly, mood was assessed using the Hospital Anxiety and Depression Scale (HADS) [26]. The demographics and neuropsychology test performance of the participants are provided in Table 2.

As shown in Table 2, the patients with TEA performed well on all psychometric tests administered. The control group outperformed the patient group in the NART, resulting in a subtle, albeit significant, group difference in NART-predicted verbal IQ ($t(30) = -2.188$, $p = 0.037$, $r = .37$). Moreover, although relatively low, the HADS — depression score of the patient group was higher than that of the control group, resulting in a significant group difference ($t(30) = 3.028$, $p = 0.005$, $r = .48$). Both differences are discussed further in the Results section and in the Discussion section.

The study was approved by the National Health Service (NHS) Scotland A Research Ethics Committee and by the Psychology Research Ethics Committee of the University of Edinburgh. Informed consent was obtained from each participant according to the Declaration of Helsinki [27].

Testing took place at the participants' homes during five sessions. The experiment began between 10am and 11am on day 1.

2.2. Picture recognition test

2.2.1. Test material

Five hundred color photos were obtained from Getty Images (www.gettyimages.co.uk). In order to increase relevance to real-life memory, all selected pictures represented complex everyday life scenes such as activities or objects (e.g., people, work, sports, domestic activities, plants, tools, and animals; see example pictures in Fig. 1 and Supplementary Fig. A). Familiar places, buildings, or famous persons were avoided. Pictures were presented in e-prime as 24-bit (color depth) pictures (portrait or landscape) in bitmap format (.bmp).

Three hundred of the 500 pictures were selected for picture presentation — the *presentation pictures*, and 400 of the 500 pictures were selected for subsequent picture recognition testing — the *recognition test pictures* (see Fig. 1 and Supplementary Fig. A). Of the 400 recognition test pictures, 200 were presentation pictures. The remaining 100 presentation pictures were not used in the recognition test. Instead, we selected for each of these 100 pictures a similar equivalent — the *similar foils*. These 100 similar foils differed from the presentation pictures with regard to minor, though visible, changes in the background or by having different characters in the scene (i.e., same character(s) with changed background scene or different character(s) with the same background scene, see Fig. 1 and Supplementary Fig. A for an example). Lastly, the remaining 100 recognition test pictures were different