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Long-term memory deficits in temporal lobe epilepsy

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ABSTRACT

Memory complaints and deficits are common in patients with epilepsy, especially temporal lobe epilepsy (TLE), where memory-related brain structures are directly involved in the epileptic process. In recent years, substantial progress has been made in delineating memory impairment in TLE, challenging the traditional neuropsychological approach of the disorder. In particular, several lines of evidence have suggested that, beyond the apparent deficit demonstrable by standardized neuropsychological evaluations, TLE may also negatively interact with long-term memory, producing considerable loss of information of the patient's autobiographical history and an inability to maintain newly acquired information over a period of time. These observations have led to the development of innovative assessment techniques, and prompted a new domain of investigation focused on the relationships between interictal epileptiform activities and the integrity of anatomo-functional systems. The present paper reviews the available evidence for long-term memory deficits in TLE with respect to remote and very long-term memory, and discusses their putative pathophysiological mechanisms and the developing potential strategies to improve memory functioning.

1. Introduction

Memory complaints and deficits are commonly seen in patients with epilepsy, especially temporal lobe epilepsy (TLE), where memory-related brain structures are directly involved by seizures. Numerous studies and reviews have delineated the clinical neuropsychology of TLE, particularly with respect to lateralization of seizures within the classic visual/verbal memory framework and in comparisons of pre- and post-surgery memory performance, in attempts to reliably predict postsurgical outcomes [1]. The material-specific model

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of memory relies on the idea that, in right-handers, the left temporal lobe sustains verbal memories, while the right temporal lobe sustains non-verbal memories (for a recent review, see Willment and Golby [2]). However, over the years, neuropsychological and neuroimaging studies have progressively challenged this model. For instance, non-verbal deficits have been less consistently associated with right TLE than verbal memory has been with left TLE, and aberrant lateralization of activation patterns have been demonstrated during material-specific memory tasks in functional neuroimaging studies.

In recent years, additional issues challenging the traditional neuropsychological approach to TLE have emerged. First, the severity of memory complaints has not been consistently captured by standardized neuropsychological assessment, with many TLE patients performing at average levels or above [3]. Standardized memory tests typically assess the ability to retain new information over relatively short (from 20 min to 1 h) delays [4], whereas several lines of evidence suggest that TLE may also interfere with long-term consolidation, with successfully memorized information after short delays progressively fading over periods of days or weeks. Second, paralleling the progress in our understanding of declarative memory organization, the possibility that TLE memory deficits might be analyzed beyond the scope of the verbal/visual dichotomy has been raised [5], leading to the development of complementary assessment paradigms (for reviews, see Bell and Giovagnoli [6] and Butler and Zeman [7]). Finally, characterization of memory impairment in seizure-free patients has stimulated the development of a new area of research, focusing on the relationships between interictal activities and functional deficits.

Thus, the present report reviews all the available evidence on long-term memory deficits in TLE and discusses some of their putative pathophysiological mechanisms, along with potential strategies aimed at optimalizing memory functioning.

2. Remote memory in TLE

Remote memory deficits can have a considerable impact on psychological well-being and may represent one of the main complaints of TLE sufferers [8]. However, while the ability to acquire new information (anterograde memory) has been investigated in detail in TLE, relatively few studies have focused on remote memory, which is multifaceted, comprising memories encoded in the relatively distant past [7,9], with episodic and semantic components. Episodic memory is typically autobiographical, involving the recollection of personally experienced events through ''mental time travel'' (or ''autonoetic awareness'') [10]. Semantic memory enables the recollection of declarative facts, and includes personal (for example, what schools were attended) and public (such as facts about famous people) knowledge.

In a review of the literature, Butler and Zeman [7] gathered 18 case reports and 13 group studies reporting pronounced retrograde memory impairment in the context of epilepsy. As emphasized by the authors, the study of remote memory poses several methodological challenges [7], and the magnitude of the deficits has proved to be variable. Overall, however, almost all studies have demonstrated remote memory deficits in TLE patients involving, to various extents, episodic memory, personal semantics and public knowledge. Some studies revealed impairment of autobiographical memory throughout the entire lifetime [11,12], whereas others reported a circumscribed deficit limited to the past 5 years [13]. Such reduced vividness of autobiographical recollection has also been associated with loss of temporally specific details [14]. Viskontas et al. [11] found autobiographical memory deficits but with intact personal semantics, whereas others have reported additional impairment for public events [15,16] or a disproportionate loss of public semantics compared with autobiographical memory [17–19]. Recent studies have also demonstrated evidence of general semantic knowledge disorders in TLE patients (for a review, see Jaimes-Bautista et al. [20]).

Transient epileptic amnesia (TEA) is a late-onset, treatment-responsive form of TLE in which approximately two-thirds of patients spontaneously complain of autobiographical memory impairment [21,22] (for a review, see Butler and Zeman [7]). In the typical form of TEA, the main manifestation of seizures consists of acute amnesia episodes usually lasting < 1 h, during which other cognitive functions remain intact. In these cases, several lines of clinical, electrophysiological and neuroimaging evidence point towards a seizure focus originating in the medial temporal lobes (MTLs), particularly within the hippocampal formation [7,23,24]. Despite successful resolution of seizures, most patients complain of persistent autobiographical memory loss; case and group studies of TEA have indicated normal [5,24] or mildly impaired personal semantic memory [22,25], but marked episodic autobiographical memory loss [5,22,24,26–29]. Despite knowing substantial information about their past, patients are typically unable to recollect the specific contextual details that characterize each life event. While subtle deficits of public semantic memory have been highlighted in some reports [25], general semantic knowledge is largely preserved overall [5,24,27].

Some studies have attempted to identify the potential factors interacting with retrograde memory in epilepsy [11,12,16,30–32]. Several variables have been examined, such as demographics and epilepsy characteristics, the antiepileptic drugs (AEDs) used and psychosocial factors, but no correlation between epilepsy characteristics and autobiographical memory performance has conclusively been determined. Correlations with non-personal semantics (remote memory for public events and general knowledge) have suggested the negative effects of persistent postoperative seizures [31], yet no effects from duration of epilepsy [16,31] and seizure frequency [16]. In addition, contradictory findings have been reported regarding the impact of the lifetime number of generalized tonic-clonic seizures [30–32], age at epilepsy onset [12,16,31,32] and duration of epilepsy [31]. Several factors may account for these inconsistencies, including the difficulty of accurately estimating seizure frequency, an emphasis on patients with medically intractable seizures, and the possible contribution of other confounding factors such as mood status and general intelligence.

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Two studies have found a positive correlation between remote semantic loss and the number of AEDs taken [31,32]. However, interpretation of this finding may itself be confounded by the close relationship between medication regimen and epilepsy severity. In addition, in a group study of TEA patients, profound autobiographical memory loss was evidenced prior to treatment and with low-dose monotherapy [22].

Mood disturbance is commonly associated with an ''overgeneral'' recollection of public and autobiographical events [33]. Therefore, the possibility that mood changes associated with epilepsy contribute to producing retrograde memory impairment should be considered. In the Mosbah et al. [24] series of TEA patients, around one-third of them had a past or current history of depression. However, Butler et al. [22] found no evidence of a difference in either past or present diagnoses of major depression or generalized anxiety, and no correlation between scores on the Hospital Anxiety and Depression Scale and retrograde memory impairment in patients with TEA.

One explanation often proposed to account for autobiographical memory deficits in TLE is that clinical or subclinical activity propagates from MTL structures to the neocortical autobiographical memory network, thereby disrupting the memory trace [7,25] (for related accounts, see Gallassi et al. [34] and Mendes [35]). Evidence from both case (patient R.G. [28]) and group studies [5] supports the idea that autobiographical memory deficits in TEA reflects storage impairment. For example, R.G.'s performance did not benefit from relevant verbal and visual cues, thus suggesting permanent memory loss. However, an anecdotal case of recovery of lost personal memories following episodes of *déjà-vu* has been reported [36], raising the possibility that autobiographical memory deficits may also, at least partially, be a consequence to impaired retrieval.

Few studies to date have investigated the neural basis of autobiographical memory deficits in TLE. Addis et al. [37] found considerable neural differences during autobiographical recollection between patients with left TLE and intractable seizures vs. control subjects. Milton et al. [38] revealed a reduction of activity in the MTL and other key regions of the autobiographical memory network in TEA, together with reduced effective connectivity between the MTL and neocortical regions of the network. Finally, using 18F- fluorodeoxyglucose positron emission tomography (18F-FDG PET) in TEA patients, medial temporal clusters were found to correlate not only with standard memory performance, but also with autobiographical memory measures [24].

In summary, these studies in general suggest that remote memory impairment is a frequent feature of TLE. Autobiographical memory has been the most investigated component, and such deficits appear to be rather more associated with the anatomical typology of the epileptic focus (especially when originating from the hippocampal formation and interconnected structures) than with other epilepsy characteristics and demographic variables.

3. Accelerated forgetting

In ''typical'' cases of amnesia following brain insults, anterograde memory impairment is usually demonstrated within seconds or minutes of information exposure [39,40]. Over the past two decades, however, a different pattern of amnesia has been identified in TLE patients, who demonstrate normal learning and retention of information over relatively brief delays, but an accelerated rate of forgetting over days or weeks. This slowly developing form of amnesia, only apparent over extended periods of time, has been coined ''long-term amnesia'' [41] or ''accelerated long-term forgetting'' (ALF), and provides evidence of a prolonged multistage consolidation process. This issue has been examined in a detailed review that pooled seven case reports and 10 group studies [7]. ALF was demonstrated after variable delays, ranging from 24 h [40,42] to 8 weeks [43]. With the longest intervals, recall was severely impaired in most cases, with several patients being unable to even remember the context of learning. Recognition memory was at times better preserved [39,41,42,13], but was never completely spared. Only three studies [44–46] found no differences in the long-term forgetting rate between patients and controls. However, since then, several group studies have confirmed the frequent occurrence of ALF in TLE, with subjective complaints of ALF reported in about 50% of TEA patients [22,24]. Nevertheless, ALF is not specific of TEA and may also be evidenced in the broader context of TLE [47].

ALF is also variable with respect to the time window during which the forgetting happens, which can range from hours [48] to weeks [5,24,29,49]. In a recent study, ALF was tested in 18 patients with TLE after 30 s, 10 min, 1 day and 1 week of learning [50]. Forgetting of verbal material was found to be progressively quicker over the course of a week, whereas visual material was more quickly forgotten (in the first 10 min of learning), but with comparable forgetting rates thereafter [51].

The cognitive basis of ALF in TEA has remained a matter of debate. It has been suggested that ALF results from a subtle encoding deficit that goes undetected during learning and early retention, but ultimately leads to accelerated forgetting [52]. This hypothesis was recently evaluated in a group of TEA patients and controls. Yet, despite careful matching for performance on encoding and early recalls, TEA patients exhibited significant ALF after just a 1-week delay [49]. It is also unlikely that ALF is the result of a retrieval deficit, as shown by studies relying on procedures facilitating retrieval, such as recognition tasks. After long-term delays, TEA patients fail to show normal recognition performance in most studies [5,43,49]. It has thus been proposed that ALF most likely reflects a deficit of very long-term memory consolidation [13] and possibly of memory reconsolidation [5,53], a process thought to play a major role in long-term memory [54–56].

In the absence of standardized tests, clinicians and researchers have elaborated their own materials and procedures for assessing forgetting over extended time delays. The mixed findings in studies of ALF may therefore be explained by differences in methodological approaches, and the difficulties encountered when comparing normal and pathological forgetting (for a review and recommendations, see Geurts et al. [57] and Elliott et al. [58]). It is recommended that, after careful matching of groups for age and intellectual ability, both verbal and non-verbal tests should be used in combination with recall and recognition procedures, especially when assessing long-term memory over periods of

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weeks. In addition, experimental manipulations should be made to equate initial learning, and to avoid ceiling and floor effects while minimizing opportunities of rehearsal of test material. While such methodological issues have not always been taken into consideration, the consistent demonstration of ALF supports the robustness of this particular memory disorder.

ALF affects memory for real-life events [5,51,59] as well as laboratory stimuli (short stories, word lists, paired associations, Rey complex figure tests, picture recognition), is specific to declarative memory [60], and is commonly found using both verbal and visual materials [61] (for exceptions, see Manes et al. [29] and Blake et al. [43]). However, ALF may not be global but material-specific, that is, selective of certain kinds of memory such as episodic and spatial. This issue was addressed in two studies from our group that demonstrated dissociated long-term memory performance with impaired long-term consolidation of contextually bound material (including episodic and spatial memory), yet preserved context-free information (semantic knowledge and single items) [5,24].

Nevertheless, the neurobiological basis of ALF in TLE remains incompletely resolved. Several mechanisms have been suggested, including the cognitive effects of AEDs, clinical and subclinical epileptic activity, structural lesions and disrupted sleep [6,7,62,63]. However, the idea that AEDs might account for ALF is unlikely, as the phenomenon typically predates the onset of treatment [5,7,22,26]. In addition, most studies demonstrating ALF in TLE have included drug-free patients [53] or those using low doses of a single AED regimen. Moreover, improved ALF has occasionally been observed following treatment initiation [40,64] and recurrent clinical seizures are not a prerequisite of ALF, as it has been demonstrated in seizure-free patients [5]. Thus, it has been hypothesized that ALF could be the result of ongoing subclinical epileptiform activity [65,66] that impedes the building and stabilization of memory traces [67].

To date, no study has convincingly documented the neural basis of ALF, while the implication of MTL structures is a plausible hypothesis, given their well-known involvement in declarative memory. In the study by Mosbah et al. [24], medial temporal clusters correlated with long-term anterograde memory scores, suggesting a potential contribution of MTL dysfunction to long-term consolidation memory disturbances (such as ALF).

TLE patients are also more prone to display epileptiform abnormalities during sleep electroencephalography (EEG) recordings, raising the issue of the relationship between sleep and ALF. Sleep has long been assumed to play a major role in memory consolidation, possibly by increasing the resistance of memory traces to interference via reactivation mechanisms [68,69]. Thus, the possibility that subclinical epilepsy-related activity during sleep may disrupt some of the fundamental mechanisms involved in memory (re)consolidation has been raised, as also suggested by animal studies [70,71]. However, the available data remain inconclusive on this specific issue. While some studies have suggested that ALF is not caused by disruption of sleep-dependent memory consolidation [62,72–74], a recent study has shown that the amount of slow-wave sleep correlates negatively with sleep-related memory benefits, thereby suggesting that slow-wave sleep might negatively impact memory retention in patients with ALF [75].

In summary, TLE patients appear to be heterogeneous with regard to ALF, which is an inconsistent feature of TLE; it also may involve specific types of material within declarative memory (as shown by categorical dissociation in some patients) and may arise after different delays from encoding (some patients exhibit ALF after hours, others after days or weeks). Thus, ALF most likely reflects dysfunction of a multicomponent process of consolidation of memory, starting from the disruption of early tagging of information to long-term storage, possibly via sleep-dependent synaptic reorganization and plasticity.

4. A role of interictal spikes in memory disturbances?

Interictal spikes (ISs) are defined as high-amplitude ($> 50\ \mu V$) fast EEG transients usually followed by a slow wave lasting several hundreds of ms. In intracellular recordings, this pattern is associated with an intracellular discharge, a paroxysmal depolarizing shift, characterized by a rapid sequence of fast action potentials superimposed on a slow depolarizing potential. ISs are followed by a refractory period lasting a few seconds and generally attributed to a post-spike inhibitory phenomenon [76]. It has also been suggested that, under some circumstances, IS genesis may be linked to abnormal GABAergic neuron recruitment, which could have negative consequences on normal functioning and cognition [77]. Kleen et al. [78] showed that the appearance of ISs in the hippocampus decreased performance on recognition tasks (the delayed match-to-sample task) in a rat pilocarpine model of TLE. ISs may also be associated with altered physiological networks. In pilocarpine rats, Chauvière et al. [79] showed that performance of a spatial memory task was impaired before the occurrence of epileptic seizures. The impairment correlated with a decrease in theta activity in the hippocampus, known to be critical for memory processes, but not to interictal-like activities. In humans, several studies have found a direct link between ISs and memory impairment [80–82], and the timing with which the ISs arose in the hippocampus during the task seems critical, although the issue nevertheless remains controversial. Indeed, Kleen et al. [81] found impaired memory performances only when the ISs appeared during retrieval and maintenance periods, whereas Horak et al. [82] found impaired performances when ISs occurred during encoding and recall.

Interictal epileptic activities may also prevent memory processes during sleep, in particular, during non-REM (rapid eye movement) sleep known to activate epileptic networks (see Halasz [83] for a review). During non-REM sleep, the transfer of information from the hippocampus to neocortex takes place, mediated by specific activities such as hippocampal spindles and ripples, a process that is probably a key mechanism of long-term memory consolidation [84], whereas the occurrence of ISs within the hippocampus probably has a deleterious impact on this information transfer, as they are negatively correlated with hippocampal spindles [85]. Thus,

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ISs may disrupt the sleep-related hippocampal-neocortical dialogue necessary for long-term consolidation of declarative memory by impeding the sustained retention of both newly acquired and remote information [86].

5. Impact of AEDs on memory functioning in TLE

Following the onset of treatment, a subjective improvement of memory is sometimes reported [8,21,22], but remains inconsistent. With regard to objective measures, few studies have provided direct comparisons of pre- and post-treatment memory performance in TLE. In a study of three TEA patients, a persistent decline in remote autobiographical memory was reported despite full resolution of amnestic seizures [87]. In a subset of our present patients for whom longitudinal data were available, no significant improvement was found on standard neuropsychological measures obtained before treatment and 1 year after the onset of treatment. However, an improvement of autobiographical memory performance was noted for the most recent period, suggesting a positive drug-related effect on accelerated forgetting [24]. Similar findings were reported in a case study following AED administration [64].

6. Impact of a non-pharmacological intervention on memory functioning in TLE

Several non-pharmacological protocols to alleviate the memory disturbances associated with epilepsy have been evaluated. Some studies focusing on psychosocial failures [88–90] have suggested that generic psychological support or psychotherapy may help to overcome psychosocial handicaps and seizure-related distress [91,92]. Cognitive rehabilitation targeting specific cognitive impairments has been tried in addition to clinical care. However, the lack of homogeneity in methods of treatment and outcome indicators prevents reliable comparisons of the results and general conclusions (for a recent evidence-based review, see Farina et al. [93]).

Memory rehabilitation in clinical practice refers to a range of approaches, including guidance in the use of external memory supports, and the use of mental and mnemonic strategies to directly improve memory functioning. Most interventions include an educational component aimed at increasing awareness of the various factors involved in memory functioning, such as mood status, physical exercise and sleep. However, memory-training studies in epilepsy have been criticized for their inability to demonstrate generalization effects, while a recent study assessed whether engaging in traditional memory training and/or an online cognitive program could improve memory performance in 77 patients with TLE [94]. Nevertheless, such studies have shown that memory training is associated with enhanced memory function: improvement was most evident in association with traditional memory techniques and there was no evidence that an online training program had specific benefits, although it was associated with positive changes in mood ratings.

These results indicate that traditional memory rehabilitation techniques can help to reduce the burden of memory impairment in TLE [94], although positive changes were not universal and larger studies are now required to explore the factors associated with successful outcomes. Illman et al. [95] found that patients with TLE are able to monitor the status of their memory systems, suggesting that, while recollection is impaired in TLE, metacognition-based strategies may be explored to optimalize memory function [96].

Finally, the effects of transcranial direct-current stimulation (TDCS) on depression and memory dysfunction in 37 TLE patients have also been examined. TDCS was found to be a safe and well-tolerated non-pharmacological approach to improve depressive symptoms. However, memory function remained unchanged following stimulation sessions.

7. Conclusion

The present report has reviewed the large body of evidence that TLE produces memory impairment with distinctive characteristics and features, with a particular impact on remote personal memory and very long-term storage of information. This form of memory disorder appears to be closely associated with the anatomical focus of epilepsy, especially in cases originating from MTL structures. Although the pathophysiological mechanisms have yet to be established, the role of interictal activities in impeding the sleep-related hippocampal-neocortical dialogue needed to produce a stable and durable trace of declarative memory should now be investigated.

Disclosure of interest

The authors declare that they have no competing interest.

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