Neuropsychologia 48 (2010) 921–932

Contents lists available at ScienceDirect

Neuropsychologia

journal homepage: www.elsevier.com/locate/neuropsychologia

Memory, metamemory and their dissociation in temporal lobe epilepsy

Charlotte E. Howard[a], Pilar Andrés[b,*,1], Paul Broks[a,2], Rupert Noad[c,2], Martin Sadler[d,2], Debbie Coker[d,2], Giuliana Mazzoni[e,3]

[a] School of Psychology, University of Plymouth, United Kingdom
[b] Department of Psychology, University of the Balearic Islands, Cra de Valldemossa, Km 7,5, 07122 Palma de Mallorca, Spain
[c] Department of Clinical Neuropsychology, Derriford Hospital, Plymouth Hospitals NHS Trust, United Kingdom
[d] Department of Neurology, Derriford Hospital, Plymouth Hospitals NHS Trust, United Kingdom
[e] Department of Psychology, University of Hull, United Kingdom

ARTICLE INFO

Article history:
Received 15 July 2009
Received in revised form
12 November 2009
Accepted 14 November 2009
Available online 22 November 2009

Keywords:
Metamemory
Judgement-of-Learning
Feeling-of-Knowing
Temporal-lobe epilepsy

ABSTRACT

Patients with temporal-lobe epilepsy (TLE) present with memory difficulties. The aim of the current study was to determine to what extent these difficulties could be related to a metamemory impairment. Fifteen patients with TLE and 15 matched healthy controls carried out a paired-associates learning task. Memory recall was measured at intervals of 30 min and 4 weeks. We employed a combined Judgement-of-Learning (JOL) and Feeling-of-Knowing (FOK) task to investigate whether participants could monitor their memory successfully at both the item-by-item level and the global level. The results revealed a clear deficit of episodic memory in patients with epilepsy compared with controls, but metamemory in TLE patients was intact. Patients were able to monitor their memory successfully at the item-by-item level, and tended to be even more accurate than controls when making global judgements.

1. Introduction

Epilepsy is a chronic and common neurological disorder characterised by recurrent seizures. Temporal-lobe epilepsy (TLE), the most common form of epilepsy, is associated with cell loss in the hippocampus and the surrounding areas resulting in memory difficulties such as episodic memory impairments, as well as impairments in long-term consolidation and remote memory (see Bell & Giovagnoli, 2007; Leritz, Grande, & Bauer, 2006 for reviews).

Several studies have shown discrepancies between subjective reports of memory problems and objective measures from neuropsychological tasks in TLE patients (Gleissner, Helmstaedter, Quiske, & Elger, 1998; Thompson & Corcoran, 1992; Vermeulen, Aldenkamp, & Alpherts, 1993). For example, some studies have shown that TLE patients present with memory complaints, but perform adequately when assessed objectively with standardised memory tasks (Gallassi, Morreale, Lorusso, Pazzaglia, & Lugaresi, 1988; Hermann, Wyler, Steenman, & Richey, 1988; O'Shea, Saling, Bladin, & Berkovic, 1996; Thompson & Corcoran, 1992). Three main factors have been suggested to explain these underestimations of memory in TLE patients. The first is the existence of accelerated forgetting (AF) which has been attributed to the presence of seizures during the retention period. Since seizures affect the consolidation process, immediate recall should not be affected by TLE, and therefore, only delayed recall tasks would show differences in memory performance between controls and TLE patients. Blake, Wroe, Breen, and McCarthy (2000; also see Mameniskiene, Jatuzis, Kaubrys, & Budrys, 2006) showed for example significant differences between TLE patients and controls at delayed recall for complex verbal material for which the initial level of encoding was equated between TLE patients and controls. AF, however, is far from being a constant feature of TLE, and numerous studies have shown equivalent differences between TLE patients and controls in immediate and delayed recall (Bell, Fine, Dow, Seidenberg, & Hermann, 2005; Bell, 2006; Giovagnoli, Casazza, & Avanzini, 1995; Helmstaedter, Hauff, & Elger, 1998).

The second factor refers to the presence of mood disturbances (anxiety and depression), which interfere with the subjective perception of memory performance, leading to underestimations (Baños et al., 2004; Elixhauser, Leidy, Meador, Means, & Willian, 1999; Giovagnoli, Mascheroni, & Avanzini, 1997; Vermeulen et al., 1993).

The third factor is a specific deficit in metamemory. Metamemory plays a central role in human learning through development

* Corresponding author.
E-mail address: pilar.andres@uib.es (P. Andrés).
[1] Pilar Andrés was the Principal Supervisor during the realisation of this project at the University of Plymouth.
[2] These authors facilitated the access to the patients at Derriford Hospital.
[3] Giuliana Mazzoni was a Visiting Professor at the University of the Balearic Islands during the writing up of this paper.

0028-3932/$ – see front matter © 2009 Elsevier Ltd. All rights reserved.
doi:10.1016/j.neuropsychologia.2009.11.011

(Flavell & Wellman, 1977), and a deficit in this set of processes has been proposed as a major contributor to episodic memory dysfunction in several populations (e.g., Light, 1991; Shimamura & Squire, 1986).

Metamemory is one component of metacognition, which can be broadly defined as the knowledge about one's own cognitive abilities. According to Nelson and Narens (1990), metamemory, in common with other metacognitive processes, is comprised of two key processes: 'monitoring' and 'control'. Monitoring refers to the collection of information and awareness about one's memory processes, including encoding, level of knowledge, retrieval, and performance outcome, whereas control acts as a self-regulation process, activating and directing these same cognitive processes. One example of control processes is allocating sufficient time to studying material for a successful later recall. Depending on the difficulty of the material, different amounts of study time should be allocated, with more time allocated to more difficult items which are closer to the recall threshold (see Son & Metcalfe, 2000 for review). Memory monitoring is usually measured with tasks such as Judgements-of-Learning (JOL) and Feeling-of-Knowing (FOK), which provide an indicator of self-awareness of one's own memory ability.

As applied to clinical populations, the hypothesis is that poor memory can result from inadequate metamemory monitoring, inadequate metamemory control, or both. Deficits in metamemory have been observed in some types of neurological patients, but not in others (see review by Pannu & Kaszniak, 2005). In the case of Alzheimerĭs Disease, it has been proposed that the loss in episodic memory experienced by some patients can be explained by the observed impairment in metamemory functions, and in particular by the deficit in metacognitive monitoring (e.g., Correa, Graves, & Costa, 1996; McGlynn & Kaszniak, 1991; Souchay, 2007). However, it has also been shown that a metamemory deficit is not an obligatory trait of AD, and that some patients can also show unimpaired metamemory ability (Moulin, Perfect, & Jones, 2000). In that context, Cosentino, Metcalfe, Butterfield, and Stern (2007) have recently shown that AD patients who have poor awareness of memory loss show poor monitoring processes whereas patients who are aware of their memory loss demonstrate metamemory that is comparable to healthy older adults. It has also been shown that patients with severe anterograde amnesia can produce accurate metamemory (Feeling-of-Knowing) judgements. Thus, impaired metamemory accuracy is not an obligatory feature of amnesia (Shimamura & Squire, 1986).

A relationship has been suggested between metacognition and executive processes (Fernandez-Duque, Baird, & Posner, 2000; Shimamura, 2000; Souchay, Isingrini, & Espagnet, 2000). Neuroimaging (Kikyo, Ohki, & Miyashita, 2002; Maril, Simons, Mitchell, Schwartz, & Schacter, 2003) and neuropsychological (Janowsky, Shimamura, & Squire, 1989; Modirrousta & Fellows, 2008; Schnyer et al., 2004; Vilkki, Servo, & Surma-aho, 1998; Vilkki, Surma-aho, & Servo, 1999) studies have confirmed a primary role of the prefrontal cortex in metamemory processing. Although deficits in the prefrontal areas seem to be more likely to be linked to metamemory problems, there are reasons to predict that patients with TLE would present with metamemory deficits too. First, executive functions in general are sustained by a diffuse neural network rather than by only prefrontal areas (Andrés, 2003; Collette & Van der Linden, 2002). Second, in a study in which the neural correlates of FOK judgements were assessed in a face-name association task, Kikyo and Miyashita (2004; see also Schnyer, Nicholls, & Verfaellie, 2005) showed activations within temporal lobe regions when making FOKs on higher-order information processing of face images or semantic information processing of the to-be-remembered person. Additionally, Modirrousta and Fellows (2008) showed an interesting dissociation between impaired FOK judgements and intact JOLs in patients with prefrontal damage. Pannu, Kaszniak, and Rapcsak (2005) and Schnyer et al. (2004) also showed important dissociations in frontal patients, with some metamemory tasks (for example, FOKs) impaired and others (for example, JOLs) within normal range. These findings suggest that JOL accuracy is likely to be dependent on other areas than the prefrontal cortex, for example the temporal cortex. Thirdly, it has been shown that patients with early Alzheimer's disease, who, like TLE patients, suffer from hippocampal and temporal atrophy, present with metamemory deficits (see Souchay, 2007 for a review). Finally, several studies have documented that cognitive dysfunction in TLE affects functions supported by the frontal cortex such as mental flexibility and inhibition (Corcoran and Upton, 1993; Hermann et al., 1996; Martin et al., 2000). More specifically, Hermann, Seidenberg, Haltiner and Wyler (1991; also see Keller, Baker, Downes, & Roberts, 2009) postulated that executive impairment in TLE patients could result from the "spread of temporal lobe hypometabolism to the thalamus secondarily affecting the frontal lobe" or possibly the direct "spread of temporal lobe hypometabolism to the frontal lobe" (p. 1214). This has lead to the 'nociferous cortex hypothesis', postulating that there are electrophysiological abnormalities in distal extratemporal brain regions in TLE that affect executive functions. It is therefore likely that metamemory processes, intimately related to executive functions (Fernandez-Duque et al., 2000; Shimamura, 2000; Souchay et al., 2000) are also disrupted in TLE patients.

Although scarce, some neuropsychological studies have looked at metacognitive deficits in TLEs. In two studies, Prevey, Delaney and Mattson (1988) and Prevey, Delaney, Mattson and Tice (1991) concluded that TLE patients present a deficit in metacognitive monitoring. Prevey et al. (1988) conducted two experiments in which metamemory functioning was explored at encoding and retrieval in TLE patients and controls. In Experiment 1, participants were presented with two memory span tasks consisting of lists of single syllable nouns (verbal task) and non-meaningful geometrical shapes (visual task). Lists were of increasing length, from 1 to 10 items per list, and after learning each list, participants were instructed to provide a yes/no judgement as to whether they thought they could remember the words/non-meaningful geometric shapes in the list in the order presented. The results showed that TLE patients anticipated that they would perform just as well as the controls, but in fact, they performed less well than the controls on the recall tasks. It was also noted that the site of the lesion (left–right) mediated prediction accuracy depending on the experimental materials used (verbal/non-verbal).

In Experiment 2 participants were asked to make FOK judgements on general information questions they had previously answered incorrectly, by providing a 'yes' or 'no' response as to whether they would be able to recognise the correct answer from a range of six alternatives. Although also in this case the authors conclude that monitoring is impaired in TLE patients, the results are actually not clear, and depend on which measure of FOK accuracy is used. Gamma correlations, which are the most commonly used measures of relative metamemory accuracy (the ability to discriminate between which items will or will not be recalled and whether judgements are predictive of actual performance) showed no significant differences between controls and patients, either left or right. Only when proportion of positive FOK recognitions was used to assess accuracy, TLE patients resulted to be less accurate than controls and apparently overestimated their memory abilities. This measure reflects the proportion of correctly recognised items over the total number of items for which positive (yes) recognition was predicted. In the assessment of relative metamnemonic accuracy, this conditional probability measure has long been abandoned in favour of the use of Gamma, owing to the influential paper by Nelson (1984) in which Gamma was demonstrated to be superior to a number of other measures of association, including scores