Recall and recognition memory in amnesia: Patients with hippocampal, medial temporal, temporal lobe or frontal pathology

Michael D. Kopelmana,∗ Peter Brighta,1 Joseph Buckmana, Alex Fraderaa, Haruo Yoshimasu a,2 Clare Jacobson a, Alan C.F. Colchesterb

a King’s College London, Institute of Psychiatry, London, United Kingdom
b Kent Institute of Medicine and Health Sciences, University of Kent, Canterbury, Kent, United Kingdom

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Abstract

The relationship between recall and recognition memory impairments was examined in memory-disordered patients with either hippocampal, medial temporal, more widespread temporal lobe or frontal pathology. The Hirst [Hirst, W., Johnson, M. K., Phelps, E. A., & Volpe, B. T. (1988). More on recognition and recall in amnesics. Journal of Experimental Psychology: Learning, Memory, & Cognition, 14, 758–762] technique for titrating exposure times was used to match recognition memory performance as closely as possible before comparing recall memory scores. Data were available from two different control groups given differing exposure times. Each of the patient groups showed poorer recall memory performance than recognition scores, proportionate to the difference seen in healthy participants. When patients’ scores were converted to Z-scores, there was no significant difference between mean Z-recall and Z-recognition scores. When plotted on a scatterplot, the majority of the data-points indicating disproportionately low recall memory scores came from healthy controls or patients with pathology extending into the lateral temporal lobes, rather than from patients with pathology confined to the medial temporal lobes. Patients with atrophy extending into the parahippocampal gyrus (H+) performed worse than patients with atrophy confined to the hippocampi (H−); but, when H− patients were given a shorter exposure time (5 s) and compared with H+ at a longer exposure (10 s), their performance was virtually identical and did not indicate any disproportionate recall memory impairment in the H− group. Parahippocampal volumes on MRI correlated significantly with both recall and recognition memory. The possibility that findings were confounded by inter-stimulus artefacts was examined and rejected. These findings argue against the view that hippocampal amnesia or memory disorders in general are typically characterised by a disproportionate impairment in recall memory. Disproportionate recall memory impairment has been observed in a number of published cases, and the reason for the varying pattern obtained across hippocampal patients requires further examination.

Keywords: Amnesia; Memory disorders; Hippocampus; Medial temporal lobes; Recognition; Recall

1. Introduction

It is widely accepted that recognition memory reflects a combination of a familiarity judgement and a degree of recollective processes (Giovanello & Verfaellie, 2001; Jacoby, Toth, & Yonelinas, 1993; Mayes, Holdstock, Isaac, Hunkin, & Roberts, 2002). However, there is considerable controversy concerning the effects of amnesia upon recall and recognition memory, respectively. One view is that hippocampal amnesia, including cases of developmental amnesia, is specifically characterised by a disproportionate impairment in recall memory, whereas recognition memory is preserved. A second view is that amnesic or memory-disordered patients in general manifest disproportionate recall memory impairment. A third view is that amnesia, including that which follows focal hippocampal pathology, produces a proportionate impairment in both recall and recognition memory. This controversy relates to views of

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hippocampal function—whether the hippocampi are involved in encoding/retrieval processes in general, or whether they contribute specifically to the contextual/associative/relation memory processes which characterise recollection. This, in turn, relates to whether recollection (recall) and familiarity (recognition) should be viewed as ‘redundant’ processes (recollection incorporates whatever happens in familiarity plus further operations), ‘independent’ (different but overlapping operations) or ‘exclusive’ processes (different and non-overlapping).

On the basis of a meta-analysis of single case and small group studies of memory-disordered patients, Aggleton and Shaw (1996) (see also Aggleton & Brown, 1999) argued that patients with pathology within the hippocampi, fornices, mamillary bodies, mamillo-thalamic tract or anterior thalami showed impairments on verbal and visual recall but not recognition memory. In such patients with damage to what they called the ‘extended hippocampal circuit’, memory based on familiarity judgements (recognition) was intact, whereas recall memory, involving recollection of contextual features, such as time and spatial location, was impaired. They argued that combined hippocampal and parahippocampal (including entorhinal and perirhinal) lesions were required to produce an impairment in familiarity-based or recognition memory. However, there was a ‘floor’ effect in the recall scores of the subjects with larger lesions in their meta-analysis, making interpretation difficult.

There are other cases, which provide support for this hypothesis. Vargha-Khadem, Gadian, Watkins, and Connelly (1997) described three patients with a developmental amnesia for everyday events, resulting from brain injuries in infancy or early childhood. These patients showed a pronounced loss of hippocampal volume bilaterally, and their neuropsychological test performance revealed impairments on verbal and visual recall but not recognition memory, the latter being tested with material that included lists of words, non-words, familiar faces and unfamiliar faces. These findings suggested that, whilst recall of episodic memories was impaired as a result of these patients’ hippocampal pathology, recognition memory and semantic memory were spared. More detailed evidence in support of this in one of these cases was published by Baddeley, Vargha-Khadem, and Mishkin (2001), using the Doors and People Test battery (Baddeley, Emslie, & Nimmo-Smith, 1994). Moreover, Mayes et al. (2002); (Holdstock et al., 2002; Mayes et al., 2004) have described in detail an adult-onset patient, Mayes et al. (2002); (Holdstock et al., 2002), and she was also impaired at recognition memory tests, whether tested by forced-choice or yes/no. These authors argued that amnesic patients’ performance to that of healthy subjects in two different ways on a recognition memory test, the amnesic group’s recall scores were disproportionately impaired, relative to the controls. Giovanello and Verfaellie (2001) employed a very similar design to that of Hirst et al. (1986, 1988), finding that they replicated Hirst et al.’s result in one task, but not the other. These authors argued that amnesic patients and healthy participants performed the two tasks in different ways, and that this was consistent with a differential impairment of recollective memory in the amnesic patients.

The third view – namely, that (verbal and visual) recall and recognition memory are proportionately impaired in amnesia – has been advocated by Squire and colleagues in a series of publications (Haist, Shimamura, & Squire, 1992; Manns, Hopkins, Reed, Kitchener, & Squire, 2003; Manns & Squire, 1999; Reed & Squire, 1997; Stark, Bayley, & Squire, 2002; Stark & Squire, 2003). These authors have argued that patients with damage thought to be limited to the hippocampal region consistently show impairments on tests, such as the Recognition Memory Test, especially if a delay is introduced (Reed & Squire, 1997), the Doors and People Test (Manns & Squire, 1999), the recognition component of the Rey Auditory Verbal Learning Test (Manns et al., 2003), as well as on a wide variety of other recognition memory tests, whether tested by forced-choice or yes/no recognition procedures (Reed & Squire, 1997; Stark & Squire, 2003). Consistent with these findings, Kopelman and Stanhope (1998) used a variant of Hirst et al. (1988) technique, matching performance on recognition memory testing and avoiding
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