# **Retrograde amnesia and malingering** Keith G. Jenkins<sup>a</sup>, Narinder Kapur<sup>c</sup> and Michael D. Kopelman<sup>b</sup>

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#### Purpose of review

Malingered anterograde amnesia is a phenomenon that has been exhaustively studied, whereas research on retrograde amnesia has tended to focus upon functional and organic accounts of impairment. The present review explores studies relevant to extending the malingering paradigm to retrograde amnesia.

#### **Recent findings**

In the period reviewed, very little work has directly addressed the area of malingered retrograde amnesia. Researchers have tended to explain apparent 'anomalies' in memory performance or individual presentation, as manifestations of unconscious or psychological distress-mediated behaviour. In contrast, research with offenders claiming amnesia for their crimes has emphasized that malingered retrograde amnesia can be identified with relevant assessment methods. Brain imaging work too has begun to clearly describe the associated neural processes that underlie deception. It appears that the necessary coalescence of insights from clinical neuropsychology, brain imaging and neurology has reached a critical moment.

#### Summary

Current and previous studies are reviewed that addresses the assessment of malingered retrograde amnesia and evidences that a critical moment has been reached.

#### Keywords

functional, malingering, retrograde amnesia

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## Introduction

The growth of research identifying malingered or feigned cognitive impairment has proceeded at a near exponential rate over the past 2 decades. In parallel with the evolution of increasingly litigious societies in Europe and North America, research has been increasingly called upon when providing expert testimony. Neuropsychologists have tended to focus upon means to identify individuals where claimed memory impairment is out of proportion to expectations for a given severity of brain injury. The resultant measures have generally been based upon tasks in which learning of newly presented information is assessed, typically within a forced-choice recognition format enabling identification of belowchance levels of performance or empirically derived cut-off scores, for example, Tombaugh's [1] Test of Memory Malingering (TOMM) and Green et al.'s [2] Word Memory Test (WMT). Below-chance performance on any measure is seen as supporting the identification of malingered impairment. However, how far such a pattern of performance might remain unconscious, and consequently not be synonymous with 'malingering', remains open to investigation. Moreover, not all individuals consciously simulating memory impairment may fake so obviously and coaching directed at avoiding below-chance performance has been found to reduce the sensitivity of symptom validity tests [3]. Importantly, none of the currently available measures of memory that have been developed to identify malingered impairment appears to have addressed the means to examine claims of memory loss for past events, as occurs when individuals represents themselves as having acquired wide-ranging memory loss after injury, or when claiming the loss of memories for a time period that encompasses the alleged commission of a criminal act [4,5].

Remote memory refers to the store of knowledge and information held by an individual that is created through their past learning and experience, that is, preceding an injury, the onset of a disease or a specific event. Impairment of this memory system is most typically referred to as retrograde amnesia. Individuals can present with retrograde amnesia as a consequence of acquired brain injury, degenerative conditions, psychological factors (functional retrograde amnesia) or some combination of the preceding. Kapur [6] identified six factors that have contributed to the relative neglect of retrograde amnesia: symptoms of retrograde amnesia were less frequently reported by patients, and appeared less disabling than other memory impairment when present; caregivers and clinicians may have been less aware of a patient's retrograde amnesia; there were fewer retrograde amnesia tests available for psychologists; causes of retrograde amnesia were beyond experimenter control; few cerebral pathologies presented with focal or isolated symptoms of retrograde amnesia; and retrograde amnesia was comparatively neglected in research with animals, functional imaging and computational modelling when compared with anterograde amnesia (impairment of new learning). More recently, Lah and Miller [7] reviewed currently available retrograde amnesia measures. Although identifying a range of tools or techniques, including the dead/alive test [8], autobiographical memory interview (AMI) [9], the cued recall technique [10] and the use of famous faces-based measures [11], there was no discussion of how retrograde amnesia measures could be affected by, or used to identify, malingered impairment.

## Assessment issues

Zago *et al.* [12], in reviewing the 1926 case of the 'Collegno Amnesic', noted that relatively few psychological investigations addressed the malingering of retrograde amnesia. Two recent books by Rogers [13] and Larrabee [14] provide extensive coverage of work addressing the detection of malingered memory impairment. However, apart from some coverage of symptom validity test-based approaches to assess claimed amnesia for criminal acts, neither provides coverage of retrograde amnesia.

Henning-Fast et al. [15] described a follow-up study of an individual with selective functional retrograde amnesia. Their study illustrates again the lack of available testbased methodologies to investigate claimed retrograde amnesia. Although stating that there '... was some concern that NN might be feigning his amnesia ... ' (p. 2994), they relied upon observations of everyday function made by family and medical staff to assess validity of claimed amnesia. Fujiwara et al.'s [16•] multiple case study of functional retrograde amnesia made three points regarding the difficulty of assessing malingering: standard tests of malingering assess anterograde not retrograde amnesia; patients with functional retrograde amnesia often relearn their past quickly, creating difficulty disentangling information relearned from that remembered; and even if using standard symptom validity measures, how can conscious and unconscious feigning be discriminated?

Fujiwara *et al.*'s [16<sup>•</sup>] discussion provides an excellent illustration of the difference there appears to be in how chance memory test performance is interpreted by psychologists. They described how study participants scored below chance on the famous events measure, though accepting that such performance is usually taken as proof of simulation, they preferred an interpretation incorporating combined conscious and unconscious factors, for

example, '... implicit use of "forgotten" memories ...' (p. 42). Even when describing the performance of G.H., who performed perfectly on an item contained within the claimed amnesic period, and became distressed when made aware of that fact, they felt it reasonable to conclude '... that a highly emotional reaction would be an ill-suited attempt to cover deliberate malingering and considering the multitude of her additional psychiatric symptoms, we favour the psychogenic rather than the conscious alternative ...' (p. 42). Such an interpretation is in some contrast to that best represented by the title of Green *et al.*'s [17] study, 'Effort has a greater effect on test scores than severe brain injury in compensation claimants', in which deliberate conscious processes are viewed as dominant.

In her detailed case study, Mackenzie Ross [18] examined organic and functional causes for retrograde amnesia. Complexities were illustrated clearly by the concurrent presence of significant evidence for emotional disorder and instability, for example, a probable history of abuse in childhood, the experience of abusive adult relationships and premorbid lengthy treatment for depression, alongside evidence for organic factors, for example, MRI brain scan indication of small vessel disease and a mild head injury history. However, of relevance to the subject of this review was the evidence of feigning, for example, forwards digit span of 2 and backwards 0, a score of 20 out of 50 on the recognition memory test words [19], and inconsistency across separate neuropsychological assessments, for example, from 1994-1996 digit span deteriorated, as did recognition memory test score. It was concluded that memory loss had '... become exaggerated over time. Unfortunately, it is not possible to determine the extent to which unconscious (hysteria) or conscious (malingering) factors underlie ...' (p. 536). Distinguishing whether behaviour is consciously or unconsciously mediated is a key challenge in exploring malingered retrograde amnesia, and brain imaging work has begun to address the closely related issue of deliberate (conscious) deception.

# **Brain imaging perspectives**

The use of functional brain imaging provides an alternative means of exploring malingered memory impairment, but how far has it addressed malingered retrograde amnesia? Spence *et al.* [20] reviewed cognitive neurobiological accounts of deception based upon neuroimaging studies and offered an account of the performance differences between honest response and lying that were observed. Deception is dependent upon activation of brain areas within prefrontal cortex and key structures (ventrolateral prefrontal cortex, dorsolateral prefrontal cortex) are observed to be involved across a number of studies. Browndyke *et al.* [21] explored the neural substrates of simulated memory impairment using visual stimuli and a symptom validity test that drew heavily upon the TOMM [1]. Results supported previous work, showing greater brain activity with deception, focused upon prefrontal cortex and associated with slowed response latency. The posited link between executive function focused upon prefrontal cortex as the neurobiological substrate of deception raises the interesting question as to whether individuals with executive dysfunction following a brain injury are able to deceive effectively. A key role for executive function in deception and memory is also relevant to work on retrograde amnesia in which patterns of performance ordinarily taken as evidence of malingering are instead attributed to unconscious processes or emotional disturbance. Fujiwara et al. [16•] described psychological stress as causing executive dysfunction that can be causally linked to retrograde amnesia and Kopelman [22] has linked impaired executive control with dysfunctional autobiographical retrieval. Imaging studies that concurrently explore deception, retrograde amnesia and consciousness may be needed.

# Functional retrograde amnesia and malingering

For the specific situation of amnesia for criminal offences, Pyszora et al.'s study [5] of all those sentenced to life imprisonment in England and Wales in 1994, found 29% of the total sample claimed amnesia for the offence at trial. Characteristics of this group were that they were significantly older than nonamnesic offenders, more likely to have a previous history of psychiatric disorder and/or substance abuse/dependency and a history of 'blackouts' (transient memory loss not related to neurological disease). At 3-year follow-up, 33% of the amnesic sample had a complete return of memory, 26% partial return and 41% no return. At the time of trial, only 2.4% of amnesic offenders were suspected of feigning their memory loss. At 3-year follow-up 1.9% claimed that they had feigned their amnesia. The characteristics of this group comprising four offenders, for example, whether there were features apparent on preconviction assessments that may have identified their feigning were unfortunately not reported.

Ardolf *et al.* [23] in the United States suggested a much higher rate of malingered neurocognitive dysfunction based upon the Slick *et al.* [24] criteria and assessments of negative response bias, with over 89% of male criminal defendants positive on at least one measure. Much of their sample had histories of alcohol and substance misuse as also found by Pyszora *et al.* [5]. However, crucially for comparing the studies, it is not clear whether the Ardolf *et al.* sample claimed amnesia for their offence or, as it appears from the study, they were a different subgroup of offenders who claimed more generalized neurocognitive impairment.

Kritchevsky et al. [25] in their description of 10 cases of functional amnesia, partially addressed the issue of malingering because one of their participants, R.W., later admitted to having malingered his amnesia (a very rare example of a confirmed case). Characteristic of this performance was the lack of any recall of well formed episodic memories, an unusual pattern of good recall and recognition of famous faces but poor recall and recognition of public events, and the lowest performance of all participants on a test of anterograde amnesia (story recall). R.W. was the only participant not to have a significant premorbid psychiatric history. However, R.W. did not have the lowest scores on all study measures and does not appear to have been identified until 'confessing'. Kritchevsky et al. [25] still preferred an unconscious mechanism account for the poor performance of other participants, avoiding a malingering account by suggesting that in cases of functional amnesia the more a test measures common-sense notions of memory, the worse is performance.

Lack of research systematically exploring the use of measures of retrograde amnesia and patterns of performance when impairment is malingered was partially addressed by Jenkins [26]. His study incorporated individuals with moderate-severe acquired brain injury and compared performance on measures of retrograde amnesia with normal controls and participants instructed to simulate memory impairment. Several measures were used, the AMI [6], the dead/alive test [5] and newly constructed measures using photographic stimuli measuring familiarity, naming and recognition. The results suggested that, as with measures of anterograde amnesia, it may be possible to refine retrograde amnesia measures to provide cut-off scores highlighting when memory performance is unlikely to be solely attributable to brain injury/dysfunction.

The AMI samples personal semantic memories (e.g. information such as school attended, names of friends) and autobiographical incidents (e.g. something that happened at school) across the lifespan. Jenkins [26] found that performance of impairment simulators on the AMI was distinguished from that of an injured group with sensitivity of 70% and specificity of 100% using a cut-off score of less than 12 out of 21 on personal semantic items from early adult life. Furthermore, 80% of simulators scored below the AMI cut-off indicative of memory impairment compared with just 45% of the brain injured. Jenkins concluded that his results not only suggest a need for clinicians to be more aware of the possibility of remote memory impairment following acquired brain injury, but also the tendency of simulators to exaggerate such impairment. There is a need to replicate Jenkins's findings and to extend them to incorporate the comparative performance of individuals with functional retrograde

amnesia. The challenge for assessments of remote memory is not only how to differentiate individuals simulating impairment from those with brain injury related memory loss, but also to differentiate simulators from individuals with authentic memory loss arising from emotional conflict or trauma (functional retrograde amnesia).

Using the dead/alive test, Jenkins found that it provided a sensitive (70%) and specific (95%) means of differentiating memory impairment simulators. Simulators identified significantly fewer names as familiar to them and made significantly fewer correct dead/alive judgements. However, extending use of this measure as an indicator of the validity of assessed impairment is complicated by findings from other studies in which scores on dead/alive have fallen within the simulator range described by Jenkins, for example Cipolotti et al. [27]. Given this, Jenkins recommended that use of dead/alive as part of the assessment of impairment validity needs to be restricted to where there is no apparent organic pathology that might lead to a genuine retrograde amnesia. Moreover, as with the AMI, performance of individuals with authentic functional (emotionally-induced) retrograde amnesia needs to be differentiated. Jenkins's [26] forced-choice recognition measure using photographs of famous faces and varying the number of famous 'targets' across stimuli so that one, two or three targets out of four faces in total were viewed was also able to differentiate simulator, brain-injured and control groups, and determine group membership for individuals. There was a large individual variability in scores and some overlap between braininjured and simulating participants. However, an overall sensitivity of 60% with specificity of 90% was found.

Jenkins's study [26] is perhaps the first to employ methods used commonly in developing measures of malingered anterograde amnesia to explore retrograde amnesia, employing a known groups design that compares simulators with genuinely impaired individuals. His study also highlighted the need to consider remote memory impairment and its potential effects when working with individuals who have acquired brain injury, where the focus of memory assessment has typically been upon anterograde impairment.

# Conclusion

This review set out to report upon recent studies that address the malingering of retrograde amnesia. However, appropriate searches using both *Medline* and *PsycINFO* revealed that there was no study specifically addressing this topic published in the period of interest. Consequently, the present review has attempted to incorporate other relevant recent research that has a bearing upon its key focus. The study of Jenkins [26] seeking to incorporate relevant tests and methodology into the assessment of retrograde amnesia and malingering has also been described. Clearly, a great deal remains to be done to develop standardized measures of retrograde amnesia, including exploration of their use in relevant groups in order to identify cut-off scores or patterns of performance that can assist in understanding the causes of an individual's retrograde amnesia. Reliability in distinguishing organic, functional and malingered impairment needs to be established, and the acceptance of 'malingering' as the most likely explanation for some cases of retrograde amnesia requires greater consideration.

#### References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

of special interestof outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 686).

- 1 Tombaugh TN. Test of Memory Malingering. North Tonawanda, New York: Multi Health Systems; 1996.
- 2 Green P, Lees-Haley PR, Allen LM III. The Word Memory Test and the validity of neuropsychological test scores. J Forens Neuropsychol 2002; 2:97–124.
- 3 Verschuere B, Meijer E, Crombez G. Symptom validity testing for the detection of simulated amnesia: not robust to coaching. Psychol Crime Law 2008; 14:523–528.
- 4 Kopelman MD. Invited review: disorders of memory. Brain 2002; 125:2152– 2190.
- 5 Pyszora NM, Barker AF, Kopelman MD. Amnesia for criminal offences: a study of life sentence prisoners. J Forens Psychiatry Psychol 2003; 14:475–490.
- 6 Kapur N. Syndromes of retrograde amnesia: a conceptual and empirical synthesis. Psychol Bull 1999; 125:800-825.
- 7 Lah S, Miller L. Effects of temporal lobe lesions on retrograde memory: a critical review. Neuropsychol Rev 2008; 18:24–52.
- 8 Kapur N, Ellison D, Smith M, et al. Focal retrograde amnesia following bilateral temporal lobe pathology: a neuropsychological and magnetic resonance study. Brain 1992; 115:73–85.
- 9 Kopelman MD, Wilson BA, Baddeley AD. The autobiographical memory interview. Suffolk, England: Thames Valley Test Company; 1990.
- 10 Crovitz HG, Schiffman H. Frequency of episodic memory as a function of their age. Psychon Bull 1974; 4:517–518.
- 11 Bright P, Buckman J, Fradera A, et al. Retrograde amnesia in patients with hippocampal, medial temporal, or frontal pathology. Learn Mem 2006; 13:545–557.
- 12 Zago S, Sartori G, Scarlat G. Malingering and retrograde amnesia: the historic case of the Collegno Amnesic. Cortex 2004; 40:519–532.
- 13 Rogers R, editor. Clinical assessment of malingering and deception. 3rd ed. New York, London: The Guilford Press; 2008.
- 14 Larrabee GJ, editor. Assessment of malingered neuropsychological deficits. Oxford University Press; 2007.
- 15 Henning-Fast K, Meister F, Frodl T, et al. A case of persistent retrograde amnesia following a dissociative fugue: neuropsychological and neurofunctional underpinnings of loss of autobiographical memory and self-awareness. Neuropsychologia 2008; 46:2993-3005.
- Fujiwara E, Brand M, Kracht L, *et al.* Functional retrograde amnesia: a multiple
  case study. Cortex 2008; 44:29–45.

Very detailed review illustrating the key issues of how evidence typically indicative of malingering is re-interpreted.

- 17 Green P, Rohling ML, Lees-Hayley PR, Allen LM. Effort has a greater effect on test scores than severe brain injury in compensation claimants. Brain Inj 2001; 15:1045–1060.
- 18 Mackenzie Ross S. Profound retrograde amnesia following mild head injury: organic or functional? Cortex 2000; 36:521–537.
- 19 Warrington EK. Recognition Memory Test manual. Windsor, UK: Nelson Publishing Company; 1984.

- 20 Spence SA, Hunter MD, Farrow TFD, et al. A cognitive neurobiological account of deception: evidence from functional neuroimaging. Philos Trans R Soc Lond B 2004; 359:1755-1762.
- 21 Browndyke JN, Paskavitz J, Sweet LH, et al. Neuroanatomical correlates of malingered memory impairment: event-related fMRI of deception on a recognition memory task. Brain Inj 2008; 22:481–489.
- 22 Kopelman MD. Focal retrograde amnesia and the attribution of causality: an exceptionally critical review. Cognit Neuropsychol 2000; 17:585–621.
- 23 Ardolf BR, Denney RL, Houston CM. Base rates of negative response bias and malingered neurocognitive dysfunction among criminal defendants referred for neuropsychological evaluation. Clin Neuropsychol 2007; 21:899–916.
- 24 Slick DJ, Sherman EMS, Iverson GL. Diagnostic criteria for malingered neurocognitive dysfunction: proposed standards for clinical practice and research. Clin Neuropsychol 1999; 13:545–561.
- 25 Kritchevsky M, Chang J, Squire LR. Functional Amnesia: clinical description and neuropsychological profile of 10 Cases. Learn Mem 2004; 11:213– 226.
- 26 Jenkins KG. The assessment of simulated memory impairment following brain injury [dissertation]. Institute of Psychiatry. King's College: University of London; 2009.
- 27 Cipolotti L, Shallice T, Chan D, et al. Long-term retrograde amnesia ... the crucial role of the hippocampus. Neuropsychologia 2001; 39:151-172.