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Effects of Temporal Lobe Lesions on Retrograde Memory: A Critical Review

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Abstract Scientific interest in retrograde memory has grown considerably over the last two decades. Studies have demonstrated that several brain regions, including the temporal lobes, participate in the formation, maintenance and storage of past memories. Different patterns and extents of retrograde memory deficits have been recorded in patients with temporal lobe lesions. It has been argued that the pattern of retrograde memory deficits is determined not only by the side and specific location of lesion within this region, but also by methodology, medical variables (such as etiology or seizure history), and coexisting cognitive deficits. In this paper we will review published case and group studies of patients with temporal lobe lesions to evaluate the impact of these factors on retrograde memory.

Keywords Consolidation theory · Multiple trace theory · Temporal gradients · Epilepsy · Brain lesions · Lobectomy

Introduction

Retrograde memory is a term used to describe the ability to recall or recognize a range of information, such as public or personal facts, and events that were encountered during one's lifetime. Often, retrograde memory is used to specify memory for events that happened or information that was acquired before the onset of a traumatic event or acute neurological condition. Of course, for some neurological disorders, such as dementia or epilepsy, the exact time of onset of neurological abnormality may be difficult to establish. In such cases, the terms "retrograde memory" and "memory for the past" are often used interchangeably.

Until recently, retrograde memory disorders have been relatively neglected by neuropsychological researchers due to a number of factors. As pointed out by Kapur (1999), patients relatively infrequently complain about retrograde memory difficulties, caregivers/clinicians may be less aware of retrograde memory deficits, there are numerous methodological difficulties linked to the measurement of retrograde memory, and very few standardized tests are available for its assessment. Nevertheless, scientific interest in retrograde memory and its neurological underpinnings has grown considerably over the last two decades. Deficits in retrograde memory have been recorded in patients with lesions involving frontal (e.g., Baddley and Wilson 1986; Levine et al. 1998; O'Connor and Lafleche 2004), posterior cerebral (Hunkin et al. 1995) or thalamic regions (Barbizet et al. 1981; Hodges and McCarthy 1993; Isaac et al. 1998; Luchelli et al. 1995; Markowitsch et al. 1993; Miller et al. 2001; Nichelli et al. 1988; Stuss et al. 1988), but most attention has been paid to the consequences of temporal lobe lesions (Table 1). Retrograde memory deficits of different severity and type have been reported in patients with lesions in different locations, and the way in which lesion site influences the pattern of retrograde memory loss has been considered by many authors.

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Table 1 Published retrograde memory studies in patients with temporal lobe lesions

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
Scoville & Milner (1957) (also reported in Marslen-Wilson & Teuber, 1975; Corkin, 1984; further findings in Steinvorth, Levine & Corkin, 2005)	Epilepsy, bilateral TL Case HM	Bilateral mesial temporal: including hippocampi (Surgical report)	Public and autobiographical (unclear from description): clinical interview	Public and autobiographical: retrograde amnesia for a few years prior to the operation TG: found
Dimsdale, Logue & Piercy (1964) (autopsy results reported in Chan, Revesz, & Rudge, 2002; further findings in Warrington & Duchen, 1992)	Epilepsy, right TL Single case Case NT	Right temporal lobectomy (Surgical report)	Not explicitly stated (questioning)	"Profound retrograde amnesia extending over a period of years." pp 295. No spontaneous recall of the 10 years of life preceding the surgery. TG: not studied/specified
Goldberg et al. (1981)	HI Single case	Bilateral temporal: convexial and mesial Midbrain: ventral tegmentum to the ponto-mesencephalic junction (CT)	Public: Boston retrograde amnesia battery (event and people knowledge) Autobiographic: clinical observations	Public and autobiographic: impaired TG: not studied
Cermak & O'Connor (1983)	HSE Single case	Bilateral temporal: hippocampal and cortical lesions (CT)	Public: retrograde amnesia battery (famous people and events 30s to 70s) Autobiographical: individually tailored questions based on information obtained from relatives; cued recall	Public: impaired across decades and materials TG: not found Autobiographic: questions – preserved memories from the distant past; cued recall – preserved semantic, but not episodic information from recent and distant past TG: found
Damasio, Eslinger, Damasion, Van Hoesen, & Cornell (1985)	HSE Single case	Bilateral mesial temporal: hippocampi and amygdala Bilateral mesial frontal (CT)	Public: Boston famous faces test (famous people 20's to 70's) Autobiographic: interview, family photo albums	Public: impaired across decades TG: not found Autobiographic: impaired across lifespan TG: not found
Duyckaerts, et al. (1985)	Hodgkin's disease Single case	Bilateral almost complete neuronal loss restricted to hippocampus and amygdala (Autopsy)	Public and autobiographical (clinical interviews) concerning recent retrograde information	Public and autobiographical: appeared preserved for three years prior to illness onset TG: not examined
Zola-Morgan, Squire, & Amaral (1986)	Ischemia Case RB	Bilateral mesial temporal: the entire CA1 field of the hippocampi Minor pathology in other brain regions (e.g., right postcentral gyrus, left globus pallidus,	Public: Boston famous faces test (famous people 20's to 70's), television programs test (programs broadcasted between 1963 and 1977) Autobiographical: recall of specific	Public: some evidence of difficulties in detailed recall of recent public events and T.V. programs TG: found for events Autobiographical: intact TG: not applicable

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
		left internal capsule) (Autopsy)	events (Crovitz procedure)	
De Renzi, Liotti, & Nichelli (1987)	Encephalitis Case LP	Left temporal lobe: amygdala, uncus, hippocampus, hippocampal gyrus and the anterior part of the fusiform gyrus (MRI)	Public: questionnaires - famous events ('66-'85), famous people Autobiographical: 20-item questionnaire, based on information obtained from relatives	Public: severe impairment in recall of public events and famous people TG: not studied Autobiographical: intact TG: not applicable
Ellis, Young, & Critchley (1989)	Epilepsy and right TL Case KS	Right TL: hippocampus, amygdala and laterally to the Sylvian fissure (Surgical report and CT)	Public: famous people, famous voices, famous animals, famous buildings, old product names Autobiographical: observations	Public: impaired knowledge of famous people, animals, products and buildings TG: not studied Autobiographical: impaired memory for people TG: not studied
Barr, Goldberg, Wasserstein, & Novelly (1990)	TL for epilepsy N = 12 (6 right & 6 left)	Right or left TL: hippocampus, amygdala, parahippocampal gyrus, uncus and 4 cm of the dominant lateral neocortex or 51/2 of the non-dominant neocortex (Surgical reports)	Public: famous faces test, television test, GBRMB Autobiographical: autobiographical knowledge from the GBRMB	Public: left TL – impaired on the famous faces and events TG – not found Autobiographic: left TL – impaired TG: not studied
Kapur, Ellison, Smith, McLelland, & Burrows (1992)	HI Case LT	Bilateral temporal: anterior polar and middle temporal regions; right – anterior parahippocampal gyrus, hippocampi otherwise spared; Some frontal pole pathology (MRI)	Public: famous scenes, dead/alive, famous faces, famous names Autobiographical: structured interview based on information obtained from LT's fiancé; cue words recall	Public: impaired memory for famous scenes and people TG: not found Autobiographical: impaired recall of events and semantic details (less severe than events) TG: not found
McCarthy & Warrington (1992) (also reported in Warrington & McCarthy, 1988; see also McCarthy, Kopelman and Warrington, 2005)	HSE Case RFR	Bilateral medial temporal, extending laterally into the temporal cortex on the right (CT)	Public: famous faces identification, semantic knowledge, name completion and familiarity Autobiographical: semantic details and events – family photographs and list of friends names provided by RFR's wife	Public & autobiographical: impaired knowledge about events and preserved knowledge about people TG: not found
O'Connor, Butters, Miliotis, Eslinger & Cermak (1992) (also reported in Eslinger, Damasio, Damasio, & Butters, 1993)	HSE Case LD	Bilateral temporal: right – hippocampus, amygdala, parahippocampal gyrus, temporal pole and temporal neocortex; left – portion of the parahippocampal gyrus Right parietal occipital junction and part of the parietal lobe; left	Public: transient events test Autobiographical: semantic details and events – autobiographical events schedule, free association test	Public: impaired recall for news events for 6 years prior to the onset of illness TG: found Autobiographical: semantic – some difficulties; events – severely and temporally unlimited impairment TG: not found

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
		posterior ventromesial frontal lobe, insula (CT & MRI)		
Yoneda, Yamadori, Mori, & Yamashita (1992)	HSE Single case	Bilateral temporal (Initial SPECT) Left temporal (SPECT, 2 months later)	Public: clinical interview Autobiographical: interview based on information collected from friends and relatives	Public and autobiographical: retrograde amnesia for a period of 1 year preceding the onset of illness TG: found
Warrington & Duchen (1992) (initially reported by Dimsdale, Logue, & Piercy, 1964; subsequent autopsy results reported in Chan, Revesz, & Rudge, 2002)	Epilepsy and right TL Case NT (previously presented in table)	Bilateral temporal: right TL (no pathology found in resected hippocampus); left temporal – extensive pathology found in hippocampal formation; Some evidence of recent cortical infarctions Bilateral temporal: right TL - no pathology) Left TL– confirmed pathology restricted to the hippocampus"…in the absence of evidence of significant pathology in the neocortex." (Autopsy in 2002, 25 years post surgery)	Public: test of public faces and events Autobiographical: interview	Public: profound, temporally unlimited retrograde amnesia TG: not found Autobiographical: able to provide basic information going back to her school days, had difficulties elaborating and providing details TG: not found
Babinsky, Maier, Calabrese, Markowitsch, & Gehlen (1994)	Arachnoid cyst (untreated) Single case	Left temporal pole (CT)	Public: famous events test Autobiographical: semantic details and events – AMI	Public: intact TG: not applicable Autobiographical: semantic – intact; episodic – impaired for recent events only TG: found
Kapur et al. (1994) (also reported in Parkin & Hunkin, 1991)	Radionecrosis (secondary to nasopharyngeal carcinoma treatment) Case TJ	Bilateral temporal: inferior and anterior regions, white matter, but sparing the hippocampi (MRI)	Public: recall and recognition of famous events, dead or alive Autobiographical: semantic details and events – AMI, Crovitz procedure	Public: famous events and personalities – impaired TG: not found Autobiographical: semantic – preserved; events – normal for childhood and recent life, impaired for early adult life; TG: not found
Yoneda, Mori, Yamashita, & Yamadori (1994)	HSE Case 1	Bilateral temporal lobe atrophy, more prominent on the left (MRI)	Public and autobiographical: detailed interview about social and personal history before HSE onset	Public and autobiographical: retrograde impairment for 5 years before HSE onset TG: found
	HSE Case 2	Bilateral mesial temporal atrophy (MRI)		Public and autobiographical: retrograde impairment for 3 years before HSE onset TG: found

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
	HSE Case 3	Bilateral mesial temporal: atrophy restricted to the hippocampal formation (MRI)		Public and autobiographical: retrograde impairment for 1 year before HSE onset TG: found
	HSE Case 4	Bilateral mesial temporal: atrophy restricted to the hippocampal formation (MRI)		Public and autobiographical: intact TG: not applicable
	HSE Case 5	Left temporal: hippocampal formation (MRI)		Public and autobiographical: retrograde impairment for 1 year before HSE onset TG: found
Hokkanen, Launes, Vataja, Valanne, & Iivanainen (1995)	Viral encephalitis Single case	Left temporal (SPECT & EEG)	Autobiographical: semantic details and events – AMI	Impaired recall of recent autobiographical semantic details and events TG: not assessed
Kartsounis, Rudge, & Stevens (1995) (see also Cipolotti et al., 2001)	Ischaemia, convulsion Case VC	Bilateral: limited to CA1 and CA2 fields of the hippocampi (MRI)	Public: facts and events -famous faces identification and naming, famous events identification Autobiographical: semantic details and events – AMI, Galton-Crovitz, Family Photographs	Impaired recall of recent and distant public and autobiographical memories TG: not found
Schnider, Bassetti, Gutbrod, & Ozdoba (1995)	Systemic lupus erythematosus Single case	Bilateral temporal: hippocampi (MRI)	Public: politicians' names Autobiographical: semantic details and events (AMI)	Public and autobiographical: impaired recall for a period of 10 to 15 years TG: found
Calabrese et al. (1996)	HSE Single case	Right frontotemporal: anterior amygdala and hippocampus, ventral branches of the uncinate fascicle (MRI) Frontotemporal hyper perfusion (SPECT)	Public: famous faces test Autobiographic: semantic details and events – AMI, Crovitz technique	Public: difficulties in recall of information from a recent past TG: found Autobiographical: semantic details – some impairment; incidents – severely and extensively impaired TG: not found
Eslinger, Easton, Grattan, & Van Hoesen (1996) (see also Eslinger, 1998)	HSE Case EK	Bilateral temporal: left – polar, mesial, anterolateral and inferior regions; right: inferior mesial and polar Left – occipiotemporal gyri, anterior insula, posterior	Public: famous names and famous faces	Public: famous names – severe loss of retrieval and familiarity; famous faces – preserved TG: not found

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
		venromesial frontal & basal forebrain (MRI)		
	HSE Case DR	Right temporal: polar, mesial, inferior and anterolateral Right lateral and mesial occipitotemporal gyri, anterior insula and minimal ventromesial frontal (MRI)		Public: Mild difficulties in retrograde memory tasks TG: not found
Kapur et al. (1996)	HI and post traumatic epilepsy Case GR	Bilateral temporal: left – some atrophy of the posterior hippocampus, inferolateral, spreading into the left parietal lobe; discrete right temporal – the inferior temporal gyrus (MRI) Left temporoparietal (PET)	Public: famous faces, famous names, dead or alive Autobiographical: semantic details and events - structured interview based on the information obtained from relatives and AMI	Public: famous faces and names – marked impairments TG: not studied Autobiographical: impaired recall of events TG: not found
	HI Case SP	Bilateral temporal: left – major temporal lobe atrophy; right – some evidence of the damage to the right uncus/hippocampus (MRI)	Public: verbal news events (1960's to 1990's) Autobiographical: structured interview covering past personal events and AMI	Public: impaired across all life periods TG: not found Autobiographical: semantic and episodic impaired TG: not found
Mattioli et al. (1996)	HI Case MEB	Bilateral temporal: hippocampi and cingulate cortex (PET)	Public: public events questionnaire Autobiographical: structured interview	Public: impaired (at a chance level) TG: ? not found (period covered not specified) Autobiographical: severely impaired, except for childhood and adolescence TG: found
Rempel-Clower, Zola, Squire, & Amaral (1996)	Ischemia Case GD	Bilateral temporal: limited to the CA1 field of hippocampi; Left thalamus and globus pallidus: minimal damage (Autopsy)	Public: famous faces and public events tests Autobiographical: single-word cues	Public: impaired TG: not found Autobiographic: intact TG: not applicable
	Seizures and alcohol abuse Case LM	Bilateral temporal – CA1, CA2, CA3 fields, dentate gyrus, entorhinal cortex; Lateral occipitotemporal cortical		Public: impaired TG: found Autobiographical: impaired TG: found

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
		infarcts, small metastasis in the frontal, parietal, temporal and occipital cortices and in the basal ganglia (Autopsy)		
	Hypoxemia Case WH	Bilateral temporal: CA1, CA2, CA3 fields, dentate gyrus, subiculum, entorhinal cortex; Pontine infarct and widespread perivascular enlargements in the white matter and striatum (Autopsy)		Public: impaired TG: found Autobiographical: extensive deficits TG: found
Vertsichel, Cohen, & Crochet (1996)	CVA Single case	Left temporal: lateral and mesial occipito-temporal cortex Thalamus: posterolateral (MRI)	Public: face naming, face/name comprehension, naming to description, face-name matching	Public: impaired recall of famous people's names TG: not studied
Yasuda, Watanabe, & Ono (1997)	Meningioma (3 surgeries and radiotherapy) Case MN	Bilateral temporal Right basal frontal lobe and mesial occipital lobe (MRI)	Public: public events (since 1944), famous people Autobiographical: free recall of autobiographical events, family photographs	Public: severely impaired memory for public events, moderately impaired memory for famous people TG: not studied Autobiographical: intact TG: not applicable
Eslinger (1998) (Cases DR and EK also presented in Eslinger, et al., 1996)	Tumour (resected) Case PP	Left temporal: inferior polar excision, sparing of the hippocampus (Surgical notes and MRI)	Autobiographical: semantic details and events - AMI	Autobiographical: intact TG: not applicable
	CVA Case RS	Left mesial temporal Left occipital (MRI)		Autobiographical: semantic and episodic – mild deficit in recall of recent information TG: found
	HSE Case AD	Left temporal: extensive damage – polar, anterolateral and hippocampus Basal forebrain and insula (MRI)		Autobiographical: semantic – temporally unlimited impairment; events – normal recall TG: not found
	HSE Case DR (previously presented in table)	Right temporal: extensive damage – polar, anterolateral and hippocampus, occipitotemporal gyri Right basal forebrain & insula (MRI)		Autobiographical: intact TG: not applicable

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
	HSE Case JL	Right temporal: extensive damage – polar, anterolateral, hippocampal formation, amygdala, lateral and mesial occipitotemporal gyri (MRI)		Autobiographical: intact TG: not applicable
	Anoxia (due to status epilepticus) Case MR	Bilateral mesial temporal: involving primarily the hippocampus (MRI)		Autobiographical: semantic and episodic deficits more noticeable in recall of recent information TG: found
	HSE Case PD	Bilateral mesial temporal: involving primarily the hippocampus and amygdala (MRI)		Autobiographical: semantic and episodic deficits more noticeable in recall of recent information TG: found
	HSE Case EK (previously presented in table)	Bilateral temporal: left – polar, anterolateral and mesial structures; right – inferior polar, sparing mesial (MRI)		Autobiographical: semantic and episodic deficits more noticeable in recall of remote information TG: found
Hirano & Noguchi (1998)	HSE Case YK	Bilateral temporal: restricted to hippocampi (MRI)	Public: public events test (1950's – 1980's) Autobiographical: semantic details and events – Crovitz technique, AMI	Public: defective recall of recent, but not remote events TG: found Autobiographical: semantic – impaired for recent period only; episodic – temporally extensive impairment TG: found for semantic only
Reed & Squire (1998) (for Case AB, see also Bayley, Hopkins and Squire, 2003)	Cardiac arrest Case AB	Damage restricted to hippocampal region (CT reported in Schmolck, Kensinger, Corkin, & Squire, 2002)	Public: new vocabulary, public events, famous faces, famous names Autobiographical: semantic details and events – AMI, Galton-Crovitz technique	Public: temporally limited (<10y) for information prior to the onset of amnesia TG: found Autobiographical: intact for the period prior to the onset of amnesia TG: not applicable
	Memory deterioration, unknown cause Case LJ	Bilateral temporal: reduced hippocampal volumes (MRI)		Public: temporally limited (<10y) for information prior to the onset of amnesia TG: found Autobiographical: semantic details and events – intact for information preceding the onset of anterograde amnesia TG: not applicable

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
	HSE Case EP	Bilateral temporal: predominantly mesial, but also some fusiform gyrus involvement (MRI)		Public: severe impairment in retrograde memory for people and events TG: not found Autobiographical: impaired for semantic details and events, except for a childhood period TG: found
	HSE Case GT	Bilateral temporal: extensive lesions including mesial region (MRI)		Public: severe impairment in retrograde memory for people and events TG: not found Autobiographical: semantic details and events – impaired TG: not found
Kapur & Brooks (1999)	Viral encephalitis Case BE	Bilateral mesial temporal: restricted to the hippocampi (MRI)	Public: dead or alive Autobiographical: semantic details and events – AMI, structured interview, shared experiences test	Public: impaired recognition of recently famous people TG: found Autobiographic: semantic details – intact; events – impaired recall of incidents for a period of 2 years prior to the onset of illness TG: found for events only
	HSE Case LC	Bilateral temporal: hippocampi and left uncus/entorhinal cortex (MRI)	Public: dead or alive, verbal news events Autobiographical: semi-structured interview	Public: people and events – difficulties in recognition of recent information TG: found Autobiographic: some loss for the year preceding the illness TG: found
Fujii, Yamadori, Endo, Suzuki & Fukatsu (1999)	HSE Single case	Bilateral mesial temporal: 2/3 of the hippocampal formation (MRI)	Public: memory for public events, dead or alive Autobiographical: semantic details and events – structured interview and AMI	Public: impaired knowledge of public people and events for the last 10 years TG: found Autobiographical: semantic details and events – impaired for the last 10 years TG: found
Kay, & Hanley (1999) (also reported in Hanley, Young, & Pearson 1989)	HSE Case BD	Right temporal: anterior end of the inferior longitudinal fasciculus (CT)	Public: a range of tasks assessing biographical knowledge	Public: selective impairment of biographical knowledge of people (but not other living things) and inability to recognize people other than his wife TG: not studied
Kopelman, Stanhope, & Kingsley (1999) (volumetric studies	Group study of patients with temporal lobe	Mesial temporal – significant reduction in volumes (MRI)	Public: news events test Autobiographical: semantic details	Public: impaired recall of recent but not remote news events TG: found

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
subsequently reported in Kopelman et al., 2003)	lesions HSE (9) Hypoxia (4) TLE (1)	In HSE: Hypometabolism throughout the temporal lobes: bilateral (1), predominantly right (3), predominantly left (3), left unilateral (2) (PET)	and events – AMI	Autobiographical: sematic and episodic – impaired (group by time interaction not significant) TG: not found
Tanaka, Miyazawa, Hashimoto, Nakano, & Obayashi (1999)	HSE Case HK	Bilateral temporal: pole and anterior parts of the lateral and mesial temporal structures, more extensive on the left (MRI)	Public: famous faces, famous events, TV programs Autobiographical: factual and episodic – AMI, personal photographs	Public: markedly impaired recall of public information across decades TG: not found Autobiographical: semantic and episodic – impaired TG: not found
Viskontas, McAndrews, & Moscovitch (2000)	TLE and TL N = 25 (11 right and 14 left)	Unilateral mesial lesions Additional discrete extratemporal lesions in 3 patients (Radiological – scan type not defined; or surgical reports)	Autobiographical: semantic details and events – AMI	Autobiographical: semantic – intact; episodic – impaired (no group by time interaction) TG: not found
Miceli et al. (2000)	HI Case APA	Left temporal – middle and inferior temporal gyri, anterior portion of the fusiform gyrus (temporal pole, lingual gyrus, parahippocampal gyrus and hippocampus spared) (MRI)	Public: famous events, famous people (naming and semantic information)	Public: events inadequately reported; people: impaired naming and semantic descriptions TG: not studied
Papps, Best, & O'Carroll (2000)	HI Case HA	Left temporal – extensive neocortical (except for the small part of the inferior temporal sulcus) Left parietal (MRI)	Autobiographical: semantic details and events – AMI	Autobiographical: semantic and episodic – preserved recall of distant and recent life, but impaired recall for the time period when the lesion was sustained TG: not found
Reinvang, Nielsen, Gjerrstad, & Bakke (2000)	Tubercular encephalitis Case (KE)	Bilateral mesial temporal Right frontal and deep white matter (MRI)	Public: true/false judgement of media events Autobiographical: semantic details and events – AMI, true/false judgement (concurrent ERP recording)	Public: impaired, but some evidence of memory for media events (true/false) TG: not found Autobiographical: semantic – preserved, episodic – impaired (true/false – some implicit memory for personal episodes) TG: not found
Stanhope & Kopelman (2000)	Herpes encephalitis Case DJ	Left temporal lobe: severe atrophy atrophy and hypometabolism: mesial and anterolateral structures	Public: events (Famous News Events test) and famous faces (modelled on the test by Warrington &	Public: difficulties in recall of recent but not distant events; anomia for people TG: gentle temporal gradient found for recall &

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
		(MRI, quantitative & PET)	McCarthy, 1988) Autobiographical: semantic details and events - AMI	recognition of events and for cued recall of famous names in response to faces Autobiographical: semantic and episodic: difficulties in recall of distant, but not recent memories TG: reversed temporal gradient
Stefanacci, Buffalo, Schmolck, & Squire (2000) (see also Reed & Squire, 1998)	HSE Case EP (previously presented in table)	Bilateral temporal: mesial and lateral Bilateral rostral fusiform gyrus, insula and left parietal (MRI)	Public: recall and recognition of public events, famous faces and famous names Autobiographical: semantic details and events – AMI	Public: impaired recall of facts and events TG: not found Autobiographical: semantic details and events – impaired recall of recent information TG: found
Cipolotti, et al. (2001) (see also Kartsounis et al., 1995)	Ischaemia, convulsion Case VC (previously presented in table)	Bilateral: limited to CA1 and CA2 fields of the hippocampi and left amygdala (MRI) Right thalamus and possible right parietal decreased tracer uptake (PET)	Public: dead or alive test, famous public events questionnaire, famous faces (recall and forced choice), famous people names and familiarity test Autobiographical: semantic details and events - AMI	Public: impaired, except for famous people names familiarity test (some preserved implicit knowledge) TG: not found Autobiographical: semantic: impaired, episodic: impaired TG: not found
Haslam, Cook, & Coltheart (2001) (also reported in Haslam, Cook, & McKone, 1998)	HSE Case TG	Bilateral temporal lobe: mesial and polar extending into the lateral cortex on the right (MRI)	Public: biographical knowledge about famous people Autobiographical: semantic details and events – AMI, modified Crovitz technique	Public: Impaired, but better access to information about famous people from their names than from their faces TG: not studied Autobiographical: semantic – impaired for the last 20 years, episodic – extensive impairment TG: found for semantic only
Westmacott, Leach, Freedman, & Moscovitch (2001) (also reported in Tulving, Schachter, McLachland & Moscovitch, 1988)	HI Case KC	Bilateral mesial temporal: hippocampi Right occipital and left fronto-parietal lesion (MRI)	Autobiographical: semantic details and events – family photographs and interview based on information collected from other family members	Autobiographical: semantic –intact; episodic – temporally extensive impairments TG: not found
Seidenberg et al. (2002)	TLE N = 21 (11 right & 10 left)	Unilateral hippocampal damage (MRI)	Public: famous faces (recognition, naming, semantic identification)	Public: left TLE – impaired famous face naming; right TLE – impaired face recognition, semantic identification and naming TG: found for the left TLE only
Viskontas, McAndrews, & Moscovitch (2002)	TLE /TL N = 24 (12 right & 12 left)	Unilateral mesial temporal Additional discrete extratemporal lesions in 3 patients Radiological – scan type not	Public: famous individuals (familiarity, sematic and naming; 1970's to 1990's)	Public: right TLE/TL impaired familiarity judgement (face but not name), left and right TLE/TL impaired recall of semantic information and names TG: found for the familiarity judgement only

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
		defined; or surgical reports		
Westmacott, & Moscovitch (2002) (see also Westmacott et al., 2001)	HI Case KC (previously presented in table)	Bilateral temporal: hippocampi Right occipital and left fronto-parietal (MRI)	Public: memory for famous names and English vocabulary terms (specifically designed tasks)	Public: selective impairments of recent names and words TG: found
Bayley, Hopkins & Squire (2003) (see also Bayley, Hopkins, & Squire, 2006) (Cases EP and AB also reported in Reed & Squire, 1998; Schmolck, Kesinger, Corkin, & Square, 2002; Stefanacci, Buffalo, Schmolck, & Squire, 2000)	Unknown aetiology Case LJ	Bilateral hippocampal and parahippocampal volume reduction of 28% and 15% (MRI)	Autobiographical: semantic and episodic detail (Crovitz procedure and AMI)	Autobiographical: recall of semantic details and events from childhood comparable to controls TG: not applicable
	Cardiovascular disease Case MJ	Bilateral hippocampal and parahippocampal volume reduction of 10% and 3% (MRI)	Autobiographical: semantic and autobiographical detail (Crovitz procedure and AMI)	Autobiographical: recall of semantic details and events from childhood comparable to controls TG: not applicable
	Drug overdose and respiratory arrest Case GW	Bilateral hippocampal and parahippocampal volume reduction of 45% and 15% (MRI)	Autobiographical: semantic and episodic detail (Crovitz procedure and AMI)	Autobiographical: recall of semantic details and events from childhood comparable to controls TG: not applicable
	Viral encephalitis Case EP (previously presented in table	Bilateral: hippocampal volume reduction of 90%, all of the entorhinal and perirhinal cortex, much of the parahippocampal cortex (20% L, 60% R), anterior fusiform (40% L, 53% R), lateral temporal cortex of 19% and insula of 13% (MRI)	Autobiographical: semantic and episodic detail (Crovitz procedure and AMI)	Autobiographical: recall of semantic details and events from childhood comparable to controls TG: not applicable
	Cardiac arrest Case JRW	Bilateral hippocampal and parahippocampal volume reduction of 29% and 0% (MRI)	Autobiographical: semantic and autobiographical detail (Crovitz procedure and AMI)	Autobiographical: recall of semantic details and events from childhood comparable to controls TG: not applicable
	Drug overdose	Bilateral: hippocampal and	Autobiographical: semantic and	Autobiographical: recall of semantic details and

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
	and respiratory arrest Case RS	parahippocampal volume reduction of 40% and +3% (MRI)	episodic detail (Crovitz procedure and AMI)	events from childhood comparable to controls TG: not applicable
	Viral encephalitisGP	Bilateral: all of the hippocampal region, all of the entorhinal and perirhinal cx, much of the parahippocampal cortex (77% L, 17% R), anterior fusiform (40% L, 53% R), lateral temporal cortex (1cm of fusiform, inferior, middle, and superior temporal gyri), insula. (MRI)	Autobiographical: semantic and episodic detail (Crovitz procedure and AMI)	Autobiographical: recall of semantic details and events from childhood comparable to controls TG: not applicable
	Cardiac arrest Case AB (previously presented in table)	Unable to undergo MRI Damage restricted to the hippocampal region (CT reported in Schmolck, Kensinger, Corkin, & Squire, 2002)	Autobiographical: semantic and episodic detail (Crovitz procedure and AMI)	Autobiographical: recall of semantic details and events from childhood comparable to controls TG: not applicable
Grewal (2003)	CVA Case study	Left TL: hippocampus, parahippocampus, subcortical white matter into the occipital lobe. (MRI, MRA, SPECT)	Public: facts and events Autobiographical: semantic details and events (AMI)	Public: impaired facts and events, but some recollection of very distant information (>4 decades ago) TG: found Autobiographical: semantic – preserved, episodic – impaired TG: not found
Lah, Lee, Grayson, & Miller (2004)	TL N = 30 (15 left, 15 right)	Unilateral TL (Neurosurgical reports)	Public: famous people (Australian Remote Memory Battery – Famous Faces recall and recognition, Public Fluency - Names) and events (Australian Remote Memory Battery – Famous Events recall and recognition; Public Fluency – Events) Autobiographical: personally familiar names (Autobiographical Fluency – Names) and events (Autobiographical Fluency – Events)	Public: sematic – left TLE recalled a smaller number of faces, episodic- left and right TLE recalled and recognised a significantly smaller number of events TG: not found Autobiographic: semantic – TL recalled a smaller number of semantic details, episodic – below controls, but not statistically significant TG: not found
Buchanan, Tranel &	Anoxia	Bilateral damage restricted to the	Autobiographical: events (Top 5	Autobiographical: events – patients with lesions that

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
Adolphs (2005)	N = 6 HSE N = 4	hippocampus (8) + additional mesial and lateral temporal (2) (MRI)	emotional memory interview, Crovitz procedure with affective leaden words)	involved hippocampus, mesial and lateral structures (2) recalled a smaller proportion of unpleasant memories TG: not found (instead reminiscence bump found)
McCarthy, Kopelman & Warrington (2005) (also reported in McCarthy & Warrington, 1992)	HSE Case RFR (previously presented in table)	Bilateral mesial (hippocampus, parahippocampal, ento- perirhinal) Right TL: antero-lateral (MRI)	Public: facts (vocabulary and person knowledge) events recall and recognition (pictures of famous news events and 38 item questionnaire) Autobiographical: semantic details and events – clinical interviews and AMI	Public: mildly reduced vocabulary knowledge, relatively preserved knowledge of people; impaired memory for famous events. TG: not found Autobiographical: semantic details and events – impaired; semantic better then events TG: not found
Steinvorth, Levine & Corkin (2005) (also reported in Scoville & Milner, 1957; see also Corkin 2002)	TL Case HM (previously presented in table)	Bilateral TL: mesial lesions (½ of the rostrocaudal extent of the hippocampal formation, entorhinal cx, amygdala, temporal polar cx) and atrophy (ventral perirhibal, parahippocampal, posterior hippocampus), anterior medial temporal cortex & white matter underlying antero-lateral temporal lobes (MRI, Corkin, Amaral, Gonzalez, Johnson & Hyman, 1997)	Public: facts (new vocabulary, facts about public events) and events (Public Events Interview) Autobiographical: semantic details and events (AMI, Autobiographical Interview)	Public: semantic – mild difficulty, episodic – unimpaired TG: not found Autobiographical: semantic – impaired, episodic – severely impaired TG: not found
Bayley, Hopkins, & Squire (2006). (all cases included in the mesial temporal group also reported in Bayley, Hopkins & Squire 2003)	Case Series 1: Mesial temporal (MTL) N =5 (previously presented in table) Case Series 2: Mesial temporal & neocortical (MTL +) N = 3 Not reported here, as cortical lesions were diffuse	MTL See patients RS, GW, JRW, EP & GP in Bayley, Hopkins & Squire 2003	Autobiographical: semantic details and events (Crovitz procedure and AMI)	Results for MTL Case Series already reported in Bayley, Hopkins & Squire 2003

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
Bright, Buckman, Fradera, Yoshimasu, Colchester & Kopelman (2006)	Mesial temporal (MTL) N =5 Hypoxia (N = 3) encephalopathy (N = 1), complex partial seizures N = 1	Bilateral atrophy confined to the hippocampi and parahippocampal gyri (MRI)	Public: Famous Faces (recall, forced-choice recognition, familiarity, 60 to 99) and News Events (recall, forced-choice recognition, 60 to 99) Autobiographical: events (modified AMI, progressive prompts)	MTL Public: no deficits TG: not applicable Autobiographical: trend to recall a smaller number of events from the most distant past ($p < 0.01$), no significant difference for the intermediate past and significantly smaller number of events from the recent past TG: partially found
	Mesial & lateral temporal (MLTL) N = 7 Encephalitis (N = 5), encephalitis & TLE (N = 1), abscess & seizures (N=1).	Bilateral mesial and anterolateral cortical damage; anterolateral lesions predominantly left in 3/7, predominantly right in 1/7 (MRI)		MLTL Public: faces – significantly below controls on recall across decades, TG: not found events – intact TG: not applicable Autobiographical: significantly below controls across life span TG: not found
Buchanan, Tranel & Adolphs (2006)	TL N = 23 (12 left, 11 right)	Unilateral TL: including the hippocampus, amygdala and surrounding cotices (MRI)	Autobiographical: events (Top 5 emotional memory interview, Crovitz procedure with affective words)	Autobiographical: events – right TL – impaired recall (number and intensity) of unpleasant events TG: not found (instead reminiscence bump found)
Lah, Lee, Grayson, & Miller (2006)	TLE N = 29 (15 left, 14 right)	Unilateral TLE (MRI & video EEG)	Public: famous people (Australian Remote Memory Battery – Famous Faces recall and recognition, Public Fluency - Names) and events (Australian Remote Memory Battery – Famous Events recall and recognition; Public Fluency – Events) Autobiographical: personally familiar people (Autobiographical Fluency – Names) and events (Autobiographical Fluency – Events)	Public: people and events – left TLE group recalled significantly smaller number of names and events and impaired on event recognition memory TG: not found Autobiographical: names – left and right TLE groups recalled fewer names than controls, events - right TLE group had reduced recall TG: not found
Lambert, Swain, Miller & Caine (2006)	Mixed aetiology (8 left, 11 right)	Unilateral temporal lobe lesions (MRI/CT)	Public: famous people (recognition, naming, semantic identification of faces, recognition and semantic identification of names)	Public: right TL group impaired at recognising a famous face from amongst distractors; left TL group impaired at naming the faces; neither group impaired at providing semantic information for faces

Table 1 (continued)

Authors	Etiology	Lesion details (technique for determining)	Retrograde memory area assessed (test/paradigm)	Results
				recognised as famous TG: not studied
Tranel & Jones (2006)				
	Mixed aetiology N = 11	Mesial temporal lesions 5 bilateral, 3 left, 3 right (MRI/CT or volumetric analyses for 5 patients with cerebral anoxia	Autobiographical: semantic details and events (Iowa Autobiographical Memory Questionnaire)	Autobiographical: semantic details and events intact TG: not applicable
Voltzenlogel, Despres, Vignal, Steinhoff, Kehrli, & Manning, (2006)	TLE N = 38 (19 left, 19 right)	Unilateral TLE (video EEG & MRI or PET)	Public: famous people (Famous Faces, Dead/Alive) and events (Public Events Test and Famous Scenes Test) Autobiographical: semantic details and events (AMI and shortened version of the modified Crovitz test)	Public: names - left TLE < right TLE < NC Dead/Alive: left TLE < right TLE & NC events - left TLE< right TLE <NC TG: not studied Autobiographical: semantic details – no significant difficulties TG: not applicable events - left TLE < right TLE < NC TG: not studied
Hepner, Mohamed, Fulham & Miller (2007)	CVA Case SG	Bilateral mesial temporal extending to retrosplenial regions (MRI, PET)	Public: names and events (Australian Remote Memory Battery – Famous Faces & Famous Events recall and recognition Autobiographical events and semantic details (AMI, modified Crovitz technique)	Public: names – intact; events - mild impairments for childhood and mid-life TG: not applicable for names, not found for events Autobiographical: poor recall of recent retrograde events (~3 years prior to CVA) TG: Found

AMI = Autobiographical Memory Interview (Kopelman, Wilson, & Baddley, 1989); CVA = cerebrovascular accident; Goldberg-Barnett remote memory battery (GBRMB); HI = head injury; HSE = herpes simplex encephalitis; TG = temporal gradient; TL = temporal lobectomy; TLE = temporal lobe epilepsy; MLTL = mesial and lateral temporal lobe

One of the main findings arising from the cognitive psychology literature of normal (non-neurological) participants is related to the temporal distribution of memories. It suggests that certain periods of autobiographical memory may be preferentially preserved (i.e., for review, see Rubin et al. 1986). When free recall of autobiographical events in response to cue words has been sampled, these studies have indicated that recollections come primarily from the most recent year of life. In older participants only (over 50 years of age) recall of events from the ages of 10 to 30 exceeded the recall of events from the preceding and subsequent decades. In patients with neurological lesions, on the other hand, "temporal gradients" have sometimes been noticed in the recall of past autobiographical events, referring to the pattern of retrograde memory deficit in which recent memories are more impaired than distant memories. In statistical terms, when patients are compared to control subjects, this should be apparent as an interaction between group and the time from which the memory dates. Much debate in the literature has been devoted to whether temporal gradients are seen after focal hippocampal lesions. Presence versus absence of a temporal gradient after a hippocampal lesion in recollection of autobiographical events has been used for validation of Consolidation Theory (Squire et al. 1984; for discussion see Bayley et al. 2003) and Multiple Trace Theory (MTT, Nadel and Moscovitch 1997; for discussion see Moscovitch et al. 2005), respectively. To date, cases supporting both positions have been put forward in the literature, and these will be reviewed here.

Besides the presence/absence of hippocampal damage, there are many other factors that seem to influence the pattern and extent of retrograde memory loss after a temporal lobe lesion. For example, several authors have considered the impact of lesion side (e.g., Barr et al. 1990; Gainotti 2007; Eslinger 1998; Lah et al. 2006; Lambert et al. 2006; Markowitsch 1995; Seidenberg et al. 2002; Viskontas et al. 2002) and others have suggested that the methods of assessing and/or scoring retrograde memory influence the patterns of results (e.g., Nadel et al. 2000; Steinvorth et al. 2005). In many patients with retrograde memory deficits, additional cognitive impairments, such as deficits in anterograde memory, executive functioning or word finding have been documented. As a result, a question about the extent to which retrograde memory deficits are secondary to other cognitive impairments has been raised (D'Esposito et al. 1996; Greene et al. 1995; Kopelman 1991; Lah et al. 2004, 2006; Mayes et al. 1997; Rubin and Greenberg 1998; Schmidtke and Vollmer 1997; Shimamura and Squire 1986; Worthington 1999). Medical factors, such as lesion etiology (Jefferies and Lambon Ralph 2006), seizure frequency, and pharmacotherapy have also been noted to affect memory for the past (Bergin et al. 2000; Lah et al. 2004, 2006).

In this paper, we will review the literature published in English on retrograde memory deficits in patients with focal temporal (or predominantly temporal) lesions, excluding studies of patients with neurodegenerative disorders (i.e., dementias). We will also limit the review to studies that included investigation of either autobiographical memories, memories for public events, or memories for people who were famous during the patient's lifetime. Table 1 presents all the studies we found that met these criteria on this topic to date. The aim of the review is to consider how lesion site, lesion side, methodology, level of functioning in other cognitive domains and medical factors influence the pattern of retrograde memory deficits seen in patients with temporal lobe lesions.

Site of Lesion Within the Temporal Lobes

Considering the Role of the Hippocampus

There is little doubt that the hippocampus plays a pivotal role in memory formation (acquisition and retention of new information) since it has been repeatedly demonstrated that bilateral, mesial temporal damage results in anterograde amnesia (e.g., Kapur and Brooks 1999; Kartsounis et al. 1995; Rempel-Clower et al. 1996; Scoville and Milner 1957; Zola-Morgan et al. 1986). Most researchers agree that interaction between the hippocampal system and the neocortex is necessary for permanent memory storage (Alvarez and Squire 1994; Muree 1996; Squire 1992, Nadel and Moscovitch 1997). Whether, when and which memories become independent of the hippocampus is currently an issue of a considerable scientific debate. On the one hand, consolidation theorists (Alvarez and Squire 1994; Squire 1992; Squire et al. 1984) propose that the hippocampus has a time-limited role in memory formation and retrieval. They claim that in the early stages of memory formation, the hippocampal complex is essential for storage and retrieval of memories. The process of consolidation is gradual, involves ongoing interaction between the hippocampus and the neocortex and takes years to complete. At completion, the neocortex alone can maintain memories and mediate their retrieval. Hence, Consolidation Theory predicts that isolated hippocampal damage will result in a temporally-limited retrograde memory deficit for all types of material, whereas damage to the temporal lobe cortex in addition to the hippocampus will result in more temporally extensive retrograde memory loss.

On the other hand, Nadel and Moscovitch (1997) have proposed a multiple-trace theory (MTT), which suggests a slightly different role for the hippocampus in the maintenance of autobiographical event memory (see Moscovitch et al. 2005 for a recent review). According to MTT,

memories for autobiographical events are created rapidly and entail the formation of a code by the hippocampus and memory traces in other brain regions. This code, formed in the hippocampus, binds bits of information stored in other brain regions into a unique scene or specific episode. Not only the formation, but also the maintenance and reconstruction of autobiographical events require an interaction between the hippocampus and other brain regions indefinitely. According to the MTT "...the full re-experiencing of an event, will always be dependent on the hippocampus" (Moscovitch et al. 2005, pp. 43). The MTT also states, however (similarly to Consolidation Theory), that the greater the damage to the hippocampal complex [including the hippocampal formation (hippocampus proper, the dentate gyrus and the subiculum) and related mesial temporal structures (the entorhinal cortex, the perirhinal cortex and the parahippocampal gyrus)], the more temporally extensive the resulting retrograde memory deficit for autobiographical events will be. Furthermore, similar to Consolidation Theory, the MTT proposes that the hippocampus and mesial temporal regions are involved only temporarily in the formation of semantic memories; according to both theories, once established, these memories can exist independently of the hippocampus.

The well-known amnesic patient HM, who underwent a bilateral mesial temporal lobe resection at the age of 27, represents a unique and important case for the argument concerning the temporal extent of the hippocampus' role in autobiographical event memory. Interestingly, data from HM has been used to support both the Consolidation Theory and the MTT. Early postoperative interviews of HM indicated a patchy retrograde memory deficit limited to the three years prior to his surgery (Scoville and Milner 1957). (Given that no preoperative data were available, it is possible that poor memory for this period might also have preceded surgery as a result of his preoperative seizures and/or the underlying neuropathology.) Studies of HM conducted two decades later using a more structured approach to the evaluation of retrograde memory revealed more extensive, but still time-limited (8 to 10 years prior to his operation) retrograde memory deficits for a range of autobiographical and public information (Corkin 1984; Marslen-Wilson and Teuber 1975). Finally, in the most recent study, conducted nearly 50 years postoperatively (Steinvorth et al. 2005), HM's recall of specific details of autobiographical events from his life prior to surgery was found to be severely impaired and no temporal gradient was found in recall of information from this period. This new evidence of a temporally extensive deficit for details of specific autobiographical events (contrary to previous findings) was attributed to a change in methodology. The authors proposed that the new methodology allowed more complete evaluation of the amount of detail recalled and degree to which life events were re-experienced during recall (Steinvorth et al. 2005).

Given HM's dense anterograde amnesia, however, it seems possible that the passage of time between the assessments of HM's retrograde memory also contributed to the differences in the findings. In support of this hypothesis, in a different patient with anterograde amnesia following an episode of encephalitis in 1971 (Case SS), Cermak and O'Connor (1983) noticed a difference in the results obtained on consecutive assessments of retrograde memory. SS's retrograde memory was assessed on two occasions, only one year apart (in 1980 and again in 1981), using the same set of questions concerning famous public events from five decades (1930s to 1970s). While in both instances SS's results were much below the results of the normal control subjects, the pattern (temporal gradient) was very different. On first assessment, SS's scores showed a temporal gradient, declining at a steady rate from the 1930s to the 1960s, with a sharp drop in the 1970s. Just one year later, on second assessment, he obtained similar scores across the four decades from the 1930s to the 1960s and then a drop for the 1970s. Although Cermak and O'Connor examined memory for past public rather than autobiographical events, their work raises concerns that the observed change in HM's results may also be secondary to the passage of time. More than five decades have passed since the initial interview was conducted by Scoville and Milner (1957) and over three decades have passed since the subsequent structured examination that was carried out by Marslen-Wilson and Teuber (1975). Given this very long period of time, and HM's inability to acquire new memories (which we would presume to be normally involved with evocation and rehearsal of old memories), it seems likely to us that he could well have forgotten some information, leaving him at the end of his life with no detailed autobiographical memories for his most distant past.

Fujii et al. (2000) reviewed published cases of patients with retrograde amnesia with acute or subacute onset and bilateral, predominantly temporal lesions (confirmed by CT, MRI or autopsy) from the previous 20 years. In the domain of autobiographical memory, recall of episodes seemed to be related to the extent of the lesion in the mesial region. Temporally-limited deficits in recall of recent autobiographical events were found in the majority of patients with damage restricted to the hippocampus proper. Patients with more extensive damage (involving other aspects of the hippocampal complex) and especially those whose lesions involved neocortical structures had more temporally-extensive deficits in their ability to recall autobiographical episodes. Autobiographical *semantic* memory (memory for autobiographical facts or details) was generally found to be less impaired, and the severity of autobiographical semantic memory impairment did not seem to be related to the extent of the mesial

lesion. Similarly, findings in the domain of retrograde memory for public knowledge indicated a shorter period of retrograde amnesia in patients with damage limited to the mesial temporal structures compared to patients with additional temporal cortical damage.

Considering the vital importance of the presence or absence of a temporal gradient in autobiographical event memories in weighing Consolidation Theory versus MTT, in Table 1 we present the authors' conclusions about this issue. Unfortunately, in some of the publications, the conclusions were based on a single patient's ability to recall events from their own life in the context of a clinical interview, with few or no comparison subjects (e.g., Damasio et al. 1985; Kapur et al. 1992; Scoville and Milner 1957; O'Connor et al. 1992; Yoneda et al. 1994). Even when a control group of subjects has been tested and data are presented, in many cases, standard deviations are not provided, making it difficult to verify the reported presence or absence of a temporal gradient (e.g., Barr et al. 1990; Cermak and O'Connor 1983; Rempel-Clower et al. 1996). In most of the published cases, no statistical analysis has been applied to look at the interaction between the two independent variables of importance: group (presence/absence of a hippocampal lesion) and time from which the memory dates. Given the recent development of valid statistical procedures to compare single cases to small groups of control subjects (e.g., Crawford and Howell 1998; Crawford and Garthwaite 2002) it would be helpful to apply such statistical procedures in the future in order to verify conclusions about temporal gradients.

Nonetheless, if we consider the authors' interpretation of their own findings, we can make the following observations. In Table 1, if we look for studies in which autobiographical event memory was measured, we find 53 *different* single cases[1] and 10 groups[2]. Of these, 52 (83%) showed a deficit in autobiographical event memory. In some of the studies in which autobiographical memory was found to be defective, the temporal profile of the impairment was not investigated, but 46 of those found to be impaired *could* be classified as either showing or not showing a temporal gradient. Of these, slightly less than half (n=21) demonstrated a temporal gradient and the others (n=25) demonstrated deficits that were not temporally graded. If we add the six novel (i.e., not included elsewhere in the table) cases reported in Bayley, Hopkins and Squire (2003) (who were studied because they had mesial temporal lesions and who all showed normal recall of childhood events) as cases with a temporal gradient, then the ratio becomes closer to equal at 27:25! (Given this even split, it is easy to see how both sides of the MTT/Consolidation argument have accumulated support.)

In most of the studies described in Table 1, however, the patients had lesions that involved both the mesial structures and more extensive temporal cortical regions. If we consider only the 16 instances from the table where the individual cases (n=14) or groups (n=2) seemed to have lesions that were limited to the mesial temporal region, then we find 11 with impaired autobiographical event memory in whom the temporal profile was examined[3]. Ten of these showed a temporal gradient, with recent memories more impaired than remote ones, and only one (Case YK, Hirano and Noguchi 1998) showed a temporally pervasive impairment. This ratio clearly adds support to the Consolidation theory, which proposes that the hippocampal region remains important to autobiographical event recall for a limited period of time. Somewhat surprisingly (given the predictions of both the Consolidation Theory and MTT), 11 of the patients or groups in Table 1 had *no* significant deficits in autobiographical event memory. Of these, the majority (n=9) had evidence of hippocampal involvement[4] and two had temporal lobe lesions that seemed to spare this region (Yasuda et al. 1997; Eslinger 1998 [case PP]).

Turning our attention now to studies of memory for famous events and famous people, 49 different single cases and 9 group studies were identified. Forty three of them performed measures of famous events and 42 used measures of knowledge of famous people. As was the case for autobiographical event memory, the majority of studies found deficits in public knowledge (93% of those tested on events and 95% of those tested on faces). Hence, impairments in memory for famous events and/or faces were slightly more common than impairment in autobiographical event memory. In the studies where public knowledge was tested and the possibilities of temporal gradients were explored, the results were similar to those for autobiographical memory. In approximately half of the cases,

[1] Not counting HM, who has demonstrated a different pattern of retrograde memory impairment over the years, and not counting the 6 new cases presented in Bayley et al. 2003, who had only very remote memory tested

[2] Counting two groups in the Bright et al. 2006 study, but not counting the group from the Buchanan et al. 2005 study because quality not quantity of memories was studied.

[3] (Duyckaerts et al. 1985; Yoneda et al. 1994 [cases 2–5]; Schnider et al. 1995; Eslinger 1998 [cases MR and PD]; Hirano and Noguchi 1998; Reed and Squire 1998 [cases AB and LJ]; Kapur and Brooks 1999 [cases BE and LC]; Fujii et al. 1999; Bright et al. 2006 [MTL group]; Tranel and Jones 2006)

[4] (Zola-Morgan et al. 1986; De Renzi et al. 1987; Yoneda et al. 1994 [case 4]; Rempel-Clower et al. 1996 [case GD]; Eslinger 1998 [cases DR and JL]; Reed and Squire 1998 [cases AB and LJ]; Tranel and Jones 2006)

temporal gradients were found for both famous people and famous events. Again, if we consider only the instances from the table where the individual cases (n=12) or groups (n=2) had lesions that were limited to the mesial temporal region[5], then the distribution with regard to temporal gradients changes. In *all* cases reporting impairment (8/11 impaired for famous events, 7/8 impaired for famous people), a temporal gradient was seen. Thus, when damage is limited to mesial temporal lobe structures, retrograde memory deficits are frequently seen for autobiographical events, public events and famous people and when these occur, they are almost always time limited, rather than temporally extensive.

Effects of Extra-Mesial Temporal Lobe Lesions

Given that both Consolidation Theory and MTT propose a role for the neocortex in the storage of long-term memories, one might also expect deficits in retrograde memory as a consequence of temporal lobe neocortical lesions that spare the hippocampus. There are at least two cases reported in the literature with large, acute temporal neocortical lesions that spared the mesial structures. The first, TJ, developed memory difficulties following radiotherapy for nasopharyngeal carcinoma (Kapur et al. 1994). His MR scans revealed radionecrosis involving neocortex (especially anterior and inferior regions) and white matter in the temporal lobes, bilaterally. In the domain of retrograde memory, TJ showed defective recall of public information (famous personalities and famous events), but preserved recall of both distant and recent autobiographical facts and incidents. The second case, HA, sustained a severe head injury in early adulthood (in 1944), then developed psychosis and temporal lobe epilepsy in 1971 (Papps et al. 2000). Her MRI showed extensive unilateral (left) temporal and parietal damage (cystic encephalomalacia) involving the neocortex (MRI findings). Her left hippocampus, parahippocampus and enthorhinal cortex were reportedly preserved. On the assessment that was conducted in 1997/1998, her autobiographical memory was intact for childhood, and recent semantic and episodic material (as measured by the Autobiographical Memory Interview (AMI), Kopelman et al. 1990). She had difficulties, however, recalling personal semantic and (to a lesser degree) personal episodic information from her early adulthood, which coincided with the time period of the head injury. The authors suggested that it was possible that during this period her ability to lay down new memories was temporarily disrupted.

Additional studies have emphasized the role of the lateral temporal cortex in memory for sematic details related to public events, famous people, or vocabulary words, and its relatively limited importance to autobiographical event memory. Two patients studied by Hodges et al. (1992), who had focal temporal lobar atrophy, were given the AMI. Case FM, who had atrophy limited to the left neocortex (sparing the hippocampus) and Case JL, with bitemporal (predominantly polar) atrophy that seemed to spare at least the left mesial region, both demonstrated impairment of memory for autobiographical semantic details (from all life periods), memory for famous people and vocabulary words but preservation of autobiographical event memory. Together with the previously discussed studies, these cases raise the possibility that mesial temporal structures may be sufficient for (but not necessary to) the recall of autobiographical facts and events from the distant past. That is, mesial temporal structures may contain more than binding codes for remote autobiographical facts and events. Alternatively, brain regions outside the temporal cortex may help to store autobiographical memories. Links between the hippocampus, retrosplenial area and frontal lobes may be particularly important for these sorts of memories.

Markowitsch (1995), for example, has proposed that anterolateral prefrontal and temporal polar cortices are necessary for retrieval of information from long-term memory. According to him, functions of the prefrontal cortex include effortful initiation of recall and temporal sequencing of information, while the anterior temporal cortex provides the connection to the posterior cortical regions where engrams are stored. Kroll et al. (1997) examined two patients with bilateral frontotemporal lesions and one patient with extensive bilateral frontal lesions. They concluded that combined frontal and temporal polar damage, but not prefrontal damage alone, results in significant retrograde memory impairments. Whether isolated temporal polar damage (with no prefrontal damage) can result in retrograde memory deficits remains a question. Further consideration of the roles played be other cortical regions in the support of autobiographical memory is beyond the scope of this paper.

Of the cases from Table 1 in which the lesion extended beyond the mesial temporal region, the prevalence of a deficit in memory for public events (32/33) or famous faces (33/34) was very high. Furthermore, in these instances where a deficit was found and temporal gradient was examined, in most instances, the deficit was temporally pervasive (for events: 67% of cases; for famous people: 74% of cases), indicating that the temporal neocortex is particularly important for the storage of public semantic information.

[5] (Duyckaerts et al. 1985; Yoneda et al. 1994 [cases 2–5]; Schnider et al. 1995; Hirano and Noguchi 1998; Reed and Squire 1998 [cases AB and LJ]; Kapur and Brooks 1999 [cases BE and LC]; Fujii et al. 1999; Seidenberg et al. 2002; Bright et al. 2006 [MTL group])

Side of Lesion

Much of the research on retrograde memory has been conducted on patients with bilateral temporal lesions. Studies of patients with predominantly unilateral temporal lesions suggest that they are likely to present with milder deficits affecting only some of the retrograde memory domains. Based on a review of single case reports and functional neuroimaging studies of normal subjects performing tasks involving episodic and autobiographical memory recall, Markowitsch (1995) proposed that the left hemisphere may be essential for retrieval of semantic information from long-term memory and the right hemisphere may subserve episodic memory retrieval. An alternative theory with regard to the role of the left and right temporal lobes in the representation of semantic knowledge comes from the study of patients with focal temporal lobar atrophy/semantic dementia (Snowden et al. 2004). These investigators have proposed a model of semantic memory comprising a single interconnected network, with dedicated brain regions representing modality specific information. That is, the left temporal lobe is more important for the representation of words (including names) and the right temporal lobe for pictorial stimuli (including faces) (Howard and Patterson 1992; Snowden et al. 2004).

When testing patients with verbal material or when asked to name pictures of people, most studies of patients with unilateral temporal lobe lesions have revealed deficits in the retrieval of retrograde semantic information in patients with left sided lesions (Babinsky et al. 1994; Barr et al. 1990; De Renzi et al. 1987; Eslinger 1998; Hokkanen et al. 1995; Kopelman et al. 1999; Lah et al. 2004, 2006; Miceli et al. 2000; Verstichel et al. 1996). This has been found both for public information, such as the names of famous people or facts about public events, and for autobiographical information, including both facts and events.

The right temporal lobe is also crucial for the retrieval of some types of retrograde memories. When the ability to choose previously-known famous faces from amongst distractors has been investigated, right temporal-lobe lesions cause a deficit, whereas left temporal lesions do not (Lambert et al. 2006; Seidenberg et al. 2002; Viskontas et al. 2002). Group studies involving patients with right or left temporal lobe epilepsy or temporal lobectomy have indicated that the right temporal lobe is important for recalling details related to famous world events (Lah et al. 2004) as well as autobiographical events (Lah et al. 2006; Viskontas et al. 2000), so that patients with either right- or left-sided lesions were impaired relative to control subjects. In contrast, these same right-temporal patients in the Lah et al. studies performed normally when asked to recall names of people, suggesting that their deficit was specific to event memory. Kopelman et al. (1999) found that patients with right (but not left) temporal lobe lesions recalled a significantly smaller number of autobiographical incidents and famous news events, however, inspection of the lesion details given in that study reveals that the patients considered to have right temporal lesions had more extensive damage (with evidence of bilateral lesions in some cases) compared to patients with the left temporal lesions. Hence, defective recall of autobiographical incidents in their right-temporal cases possibly reflects extent-of-lesion (or bilateral lesion) effects. Finally, Buchanan et al. (2006) examined recall of emotionally charged (positive, neutral and negative) autobiographical episodes in patients who had undergone left- or right-sided temporal lobectomies. Right-temporal lobectomy patients recalled a significantly smaller number of unpleasant autobiographical events, while showing a tendency to recall a larger number of pleasant memories compared to left- temporal lobectomy and control groups, raising the possibility that the right temporal lobe is important for recall of episodic information with specific (unpleasant) emotional valence.

Overall, the above-described results provide some support for Markowitsch's (1995) proposal that the left hemisphere is more important than the right for retrieval of many types of semantic retrograde memories. Some aspects of semantic knowledge (e.g., the recognition of faces as familiar), however, seem to depend more on the right temporal lobe. The degree to which modality of material determines these side-of-lesion effects has rarely been explored (but cf. Lambert et al. 2006 and Snowden et al. 2004) and merits further attention. Both the left- and right-temporal lobes seem to be important for the retrieval of episodic material. The studies reviewed here, however, suggest that a greater deficit in retrieval of episodic memory after right than after left-sided lesion is only found when lesions extend beyond the right-temporal lobe or when emotionally unpleasant events are to be recalled.

Impact of Other Cognitive Skills on Retrograde Memory

Group studies of neurological patients have shown that performance on tests of retrograde memory is associated with other cognitive skills, such as anterograde memory, object naming ability, and executive functioning. In many patients with temporal lesions, retrograde memory deficits appeared in the context of severe anterograde amnesia (Cipolotti et al. 2001; Eslinger 1998; Eslinger et al. 1996; Hirano and Noguchi 1998; Kapur and Brooks 1999; Kartsounis et al. 1995; Oxbury et al. 1997; Rempel-Clower et al. 1996; Schnider et al. 1995; Warrington and Duchen

1992; Warrington and McCarthy 1988; Yoneda et al. 1994; Zola-Morgan et al. 1986). However, there are also some patients with temporal lobe lesions who display retrograde memory impairments but no (or only mild) anterograde memory deficits (Babinsky et al. 1994; Eslinger 1998; Fujii et al. 1999; Hokkanen et al. 1995; Kapur et al. 1994, 1996; Yoneda et al. 1992), suggesting that retrograde memory deficits are not simply a result of retrieval difficulties. Indeed, Kopelman and Kapur (2001) pointed out that in patients with anterograde amnesia there is a poor correlation between anterograde and retrograde memory loss. On the other hand, studies of patients with temporal lobe epilepsy and patients who underwent unilateral temporal lobectomy (Barr et al. 1990; Bergin et al. 2000; Lah et al. 2004, 2006) have found significant correlations between some retrograde memory tests and measures of verbal anterograde memory. Further analyses, however, revealed that in most instances anterograde memory deficits could not account for difficulties in retrograde memory (Lah et al. 2004, 2006).

Even though language deficits are frequently found in patients with dominant temporal lobe lesions and tests used for examination of retrograde memory predominantly require verbal responses, little work has been done to explore the relationship between language skills, such as naming ability, and retrograde memory. Cases with retrograde memory impairment and preserved naming have been reported in patients with different etiologies (Miceli et al. 2000; Schmidtke and Vollmer 1997), but double dissociation has not been found. Instead, in patients with temporal lobe epilepsy, Barr et al. (1990) found a significant correlation between object naming and famous-face naming, and Lah et al. (2004, 2006), found significant correlations between many retrograde memory measures (famous face naming, memory for details of famous events and autobiographical memory for names) and object naming (Boston Naming test) scores. Further analyses in the Lah et al. studies showed that deficits in many (but not all) aspects of retrograde memory were accounted for by underlying naming rather than anterograde memory problems.

Evidence that executive functioning skills may play a role in retrograde memory has also been presented in the literature, mainly in patients with frontal lobe lesions (e.g., Della Sala et al. 1993; Mangels et al. 1996). Executive functioning impairments may cause difficulties in strategy selection and organization of mental search, resulting in poor recall of retrograde memories. While executive skills are not primarily mediated by the temporal lobes, executive skills are sometimes reduced in patients with temporal lobe epilepsy (i.e., Corcoran and Upton 1993; Hermann et al. 1988; Strauss et al. 1993) or temporal lobectomy (Martin et al. 2000), possibly due to the propagation of seizures of temporal lobe origin causing disruption in adjacent (frontal) brain regions. Our own work has indicated that performance on some measures of executive functioning (i.e., word fluency) is reduced in these patients and that their scores on letter fluency tasks is positively correlated with their performance on tests of retrograde memory (Lah et al. 2004, 2006).

Methodological Factors

There is some evidence that the pattern of deficits seen in conjunction with a temporal lobe lesion depends on the methodology used to assess retrograde memory. Initial information about retrograde memory in patients with temporal lobe lesions came from studies that used clinical procedures, such as clinical interviews (i.e., Duyckaerts et al. 1985; Scoville and Milner 1957), observations (i.e., Goldberg et al. 1981) or (unspecified) questioning (i.e., Dimsdale et al. 1964). Over the last few decades a number of standardized procedures have been developed for assessment of past public and autobiographical memories.

The first structured questionnaire for examination of remote memory for public events and famous people was developed by Elizabeth Warrington and her colleagues (Warrington and Sanders 1971; Warrington and Silberstein 1970). The test included recall and multiple choice versions. The content of the test was based on the major news that was being published in the London Times Review of the Year in the period from 1930 to 1968. An attempt was made to limit the questions to events that were finite and significant at the time. It soon became apparent, however, that this test was specific for the UK culture, resulting in similar batteries being developed in other countries (i.e., Boston Remote Memory Battery, Albert et al. 1981, 1979; Australian Remote Memory Battery, Shum and O'Gorman 2001). Also, in each case, when these tests are re-used in later years, new material needs to be added and new normative data needs to be collected, as subject matter that is initially well known can quickly fade to insignificance and vice versa. These tests, which have been used for investigations of retrograde memory in clinical populations, allow for between-studies comparison. For example, using the Boston Remote Memory Battery, Damasio and colleagues (1985) found evidence of temporally extensive (spanning five decades) deficits in recall and recognition of famous people and events in their patient DRB. The patient had a history of herpes simplex encephalitis, following which he developed an amnesic syndrome. His CT scan revealed bilateral lesions of the mesial temporal structures (hippocampus and amygdala), orbitofrontal cortex, basal forebrain gray matter, and anterolateral temporal lobes. Zola-Morgan et al. (1986)

used the same test when examining their patient RB who developed severe, persistent anterograde memory difficulties following an ischemic event secondary to cardiac disease. In contrast to the patient DRB, RB had no deficits in memory for famous people and events when compared to the control subjects, except for public events that preceded his illness for 3 years or less. This patient died five years after the ischaemic event. Histopathological examination of his brain revealed distinct, but discrete lesions which obliterated the entire CA1 field of the hippocampus, bilaterally. There was little evidence of brain pathology outside this region. Together, these two studies, which used the same methodology, suggest that temporally extensive retrograde memory deficits for public events and famous people, such as those found in DRB, are unlikely to be secondary to CA1 lesions. In other words, the results provide further evidence that CA1 has a temporally limited role in memory for information related to public events and famous people.

In addition to structured questionnaires, techniques for examination of memory for particular types of information related to public events or famous people have been developed, including such measures as the Dead/Alive test (Kapur, Ellison, Smith, McLelland, & Burrows) or memory for movie titles (Miller et al. 2003). In the former, the subject is given the names of persons and asked to judge whether they are dead or alive. In the latter, one real movie title is presented amongst distractors and must be recognized. Photographs are often employed when testing recognition and naming of famous people (e.g., Lambert et al. 2006; Seidenberg et al. 2002; Viskontas et al. 2002). We developed the Public Fluency Test for Names and Events (Lah et al. 2004, 2006) by modifying the technique initially developed for examination of autobiographical memory by Dritschel, Williams, Baddeley and Nimmo Smith (1992). In the Public Fluency Test for Names, subjects are given 90 s each to recall names of people who were famous during their lifetime from three different public domains (politics, sports, entertainment). In Fluency for Public Events, they are given 90 s to recall public events that took place during their lifetime. In our studies of retrograde memory that involved patients with temporal lobe epilepsy and patients with temporal lobectomy, structured sets of questions regarding famous events and famous faces (Australian Remote Memory Battery, 2001) and public fluency tasks were used in parallel. Across methodologies, defective recall of famous people's names was found in patients with left (but not right) temporal lobectomy or temporal lobe epilepsy. Again, regardless of methodology, recall of past events was found to be deficient in patients with left (but not right) temporal lobe epilepsy and in patients who underwent temporal lobectomy, independently of resection side. This high concordance of results suggests that the public fluency technique has sufficient sensitivity to detect deficits in retrograde memory for famous faces and events. The advantage of the fluency technique is that it is much faster to administer than the questionnaire. The disadvantage is that it does not allow for temporal gradients to be examined.

In the domain of autobiographical memory, there are three main methods used for assessment: The Crovitz and Schiffman technique (1974), Autobiographical Memory Interview (AMI, Kopelman et al. 1990) and Autobiographical Fluency Test (AFT, Dritschel et al. 1992). The Crovitz and Schiffmann technique assesses autobiographical event memory, while AMI and AFT evaluate autobiographical memory for both events and semantic details.

Many studies have used the Crovitz and Schiffman (1974) technique, which was developed by modifying the Galton cued-recall method. A subject is given one word (noun) at a time and asked to recall a specific event from his/her past. Traditionally, a subject is allowed to recall an event from any period of his/her life. The responses are given scores from 0 (generic response) to 3 (a specific autobiographical episode containing information about specific time and place). Using this approach (and scoring method), Zola-Morgan et al. (1986) found that the previously-mentioned patient, RB's recall of past autobiographical events (overall score and temporal distribution) was comparable to that of control subjects. Such findings suggested that the hippocampus plays little role in recollection of past personal events. The same test and scoring method were used by Moscovitch and Melo (1997) to test amnesic patients (with thalamic, diencephalic or mesial temporal lesions). The patients' recall of past autobiographical events was significantly below that of the control group. The between-group difference increased dramatically, however, when results were re-scored using a new technique developed by Moscovitch and colleagues (Moscovitch et al. 1999). Moreover, a mild temporal gradient that was observed initially was eliminated after re-scoring. In a follow-up study, the amnesic subjects were asked to recall two personally-experienced events from each of the following life periods: childhood, adolescence, early adulthood, middle age, and recent past. When a traditional three-point scoring system was used, loss of personal episodic memories was seen in all amnesic patients, and there was some evidence of a temporal gradient with older memories being seemingly better preserved compared to the recent ones. When the new scoring procedure was implemented, the magnitude of the difference (relative to the control subjects) increased from 15–20% to 50%. Moreover, the slight temporal gradient that was evident with the original scoring system was no longer present. Thus, methodology and scoring techniques have been

purported to play a crucial role in explaining the presence or absence of deficits and temporal gradient in retrograde memory for autobiographical events in patients with mesial temporal lesions (i.e., Fujii et al. 2000).

In the 1990s, the AMI, a structured interview for examination of autobiographical memory became commercially available (Kopelman et al. 1990). The ease of use, clear structure, standardization and availability of normative data are probably some of the key features that made this instrument a test of choice in many studies that have been published since its release. Eslinger (1998) and Fujii et al. (2000), however, raised concerns about the sensitivity of the AMI, as again, a maximum of three points is awarded for recall of an autobiographical event. Other criticisms include its cultural and social biases. For example, detailed questions are asked about the participant's wedding and offspring. Anyone without these personal experiences is asked the same questions about a relative's wedding and/or offspring, which may be less well remembered. It is possible that this could bias against patients with chronic neurological problems in group studies.

At around the same time, a third method (AFT) was developed for examination of autobiographical memories (Dritschel et al. 1992), purported to eliminate ceiling effects since participants are encouraged to recall a large number of responses rather than very detailed descriptions. Greene et al. (1995) compared results from the AMI and AFT-Events in a study of patients with early Alzheimer's dementia. Patients were impaired on both measures and both domains (semantic and episodic) across the whole of the life span, but whereas the AMI event recall indicated a temporal gradient, the AFT-Events did not. Concerns were also raised about AFT-Events failing to require recall of detailed contextual event information, resulting in recall of semanticized rather than episodic information. Greene et al. (1995) further explored the relationship between personal semantic and autobiographical incidents within the tests and between the tests (AMI and AFT) using correlational and factor analyses. Within the tests, the correlations between measures of personal semantic information and autobiographical incidents were moderate and significant. Between the tests, recall of autobiographical incidents from the AMI correlated significantly with the recall of incidents on the AFT, but not with the recall of personally-relevant names on the AFT. Recall of personal semantic memories from the AMI and AFT were also significantly correlated. A factor analysis revealed two factors, one that included recall of personal semantic information from the AFT and AMI and another that encompassed recall of autobiographical incidents again from the AFT and AMI. Another way to address whether the two conditions (Names and Events) of the AFT measure different components of autobiographical memory is to examine the pattern of group differences. We used the AFT in our studies of patients with temporal lobectomy and temporal lobe epilepsy (Lah et al. 2004, 2006); between-group differences were found in recall of autobiographical incidents (AFT-Events), but not for personal semantic memories (AFT-Names). Together, the results described above give evidence that the AFT is comparable to the AMI in assessing two separable components of autobiographical memory (personal semantic and autobiographical incidents).

In summary, a number of different techniques have been developed to examine retrograde memory. It is crucial to keep in mind that when using tests of retrograde memory for information in the public domain, normative comparison values need to be collected contemporaneously with the patient data, and from subjects who are matched for educational and cultural background. There is also some evidence that the presence of a temporal gradient in autobiographical event recall depends on the amount of detail elicited and/ or the scoring technique employed.

Impact of Medical Variables

One aspect of a patient's medical history that may play a role in retrograde memory is the presence/absence of a seizure disorder. Many of the patients with temporal-lobe lesions included in Table 1 had a history of seizures. Kapur (1997) points out that seizures themselves might disrupt acquisition and consolidation of memories. Our studies of patients with unilateral temporal lobe epilepsy have revealed temporally extensive deficits in memories for the past (Lah et al. 2004, 2006), with little evidence of sparing of memories that predated the onset of a seizure disorder, both for recall and recognition memory. These results suggested that the onset of a seizure disorder interfered with previously stored memories, not only with the acquisition and consolidation of new memories. It is also possible that the underlying pathological process, which may pre-date the seizure onset, has disrupted memory storage in these patients. With regard to HM, as previously mentioned, there was some evidence that his remote autobiographical memories deteriorated over time. It seems possible that his ongoing postoperative seizures might be contributing to this loss.

Furthermore, there is evidence that variables related to a seizure disorder also influence retrograde memory. In a study that included patients with temporal, extratemporal, and primary generalized epilepsy, Bergin et al. (2000) found that patients with a high frequency of complex partial seizures (six or more complex partial seizures per month) or generalized convulsions (>10 per year) had poorer memory for past public events compared to patients with less frequent seizures. In patients with temporal lobe epilepsy, we found that a young age of seizure onset (<14 years) and

polytherapy (as opposed to monotherapy) were associated with greater difficulties in recall of famous and autobiographical events, respectively (Lah et al. 2006). In those who underwent temporal lobectomy, medication status (some versus no anticonvulsants) seemed to influence recall of autobiographical semantic details, with patients who were still taking an anticonvulsant having significantly lower scores even for remote memories.

Almost all of the group studies presented in Table 1 involved patients with a history of temporal lobe epilepsy. Whether or not these patients had undergone temporal lobectomy does not seem to have much influence on the profile of the retrograde memory impairment (c.f., Viskontas et al. 2000). Interestingly, none of these group studies (where patients are selected for locus of lesion rather than memory complaint) has attempted to determine the prevalence of significant retrograde memory deficits in patients with temporal lobe epilepsy by counting the number with scores falling below normative cut-offs. This would give us a good indication of the likelihood of impairment for these sorts of patients. Also, by examining the memory profiles of individual patients within the groups in light of their age of seizure onset, one might glean further information about the temporal extent of memory loss caused by the onset of seizures.

Of the single case studies in Table 1, approximately 50% had encephalitis. The other most common etiologies were head injury, ischaemia/CVA and anoxia/hypoxia (approximately 12% each). In most instances patients with encephalitis and in some cases those with head injury, ischaemia or hypoxia also had a history of seizures. Thus, although it is likely that the pathological change to the temporal lobes in these patients is largely responsible for their retrograde memory deficits, the extent to which the seizures, per se, served to disrupt long-term memory storage is a question that remains unanswered. This could be addressed by studying retrograde memory particularly in patients with temporal lobe lesions who have no history of seizures.

Conclusion

There is little doubt that the temporal lobes play a significant role in retrograde memory. Studies of patients who sustain acute, focal mesial temporal lesions have demonstrated that the hippocampus has a time-limited role in the retrieval of autobiographical event memory as well as semantic knowledge related to famous events or famous people. Temporally extensive deficits, however, seem more likely to be found after focal mesial temporal lobe lesions if one uses tasks that require recall of more elaborate details or the re-experiencing of an event. With respect to laterality, studies have shown that the left temporal lobe plays a significant role in retrieval of retrograde semantic details (both public and autobiographical). Results from patients with lesions limited to the right temporal lobe are rare, but suggest that the right temporal lobe, along with other parts of that hemisphere, contribute to the retrieval of autobiographical event memory. The right temporal lobe may be particularly important for recall of unpleasant autobiographical events.

Group studies have revealed that patients' performance on retrograde memory tasks may be affected by deficits in other cognitive skills, such as anterograde memory or executive functioning, but particularly by word generation skills such as those measured by object naming tasks. When they have been evaluated, different medical factors, such as seizure history and presence of anticonvulsant medication, have also proved to have a significant influence. For example, a history of early onset and grand mal seizures has been associated with poorer retrograde memory. Finally, we suggest that lesion etiology also has a significant influence, with hippocampal sclerosis and/or temporal lobe epilepsy, encephalitis and possibly anoxia and head injury more likely to cause deficits than stroke. All these factors need to be weighed in future studies.

More studies of patients with intact hippocampi and acute lateral temporal lesions would be useful to investigate further the role of mesial and lateral temporal structures in retrograde memory. In addition, given Markowitsch's proposals, whether or not temporal polar structures are damaged should be considered and reported. In all future cases, statistical consideration of temporal gradients should be applied.

References

Albert, M. S., Butters, N., & Brandt, J. (1981). Patterns of remote memory in amnesic and demented patients. *Archives of Neurology*, *38*, 495–500.

Albert, M. S., Butters, N., & Levin, J. (1979). Temporal gradients in the retrograde amnesia of patients with alcoholic Korsakoff's disease. *Archives of Neurology*, *36*, 211–226.

Alvarez, P., & Squire, L. (1994). Memory consolidation and the medial temporal lobe: A simple network model. *Proceedings of the National Academy of Science*, *91*, 7041–7045.

Babinsky, R., Maier, B., Calabrese, P., Markowitsch, H. J., & Gehlen, W. (1994). A traumatic temporal lobe pathology and autobiographical memory. *European Neurology*, *34*, 290–291.

Baddley, A. D., & Wilson, B. (1986). Amnesia, autobiographical memory, and confabulation. In D. C. Rubin (Ed.), *Autobiographical Memory* (pp. 225–253). Cambridge: Cambridge University Press.

Barbizet, J., Degos, J. D., Louarn, F., Nguyen, J. P., & Mas, J. L. (1981). Amnesié per lesion ischémique bi-thalamique. *Revue Neurologique*, *137*, 415–424.

Barr, W. B., Goldberg, E., Wasserstein, J., & Novelly, R. A. (1990). Retrograde amnesia following unilateral temporal lobectomy. *Neuropsychologia*, *28*, 243–255.

Bayley, P. J., Hopkins, R. O., & Squire, L. R. (2003). Successful recollection of remote autobiographical memories by amnesic patients with medial temporal lobe lesions. *Neuron*, *38*, 135–144.

Bayley, P. J., Hopkins, R. O., & Squire, L. R. (2006). The fate of old memories after medial temporal lobe damage. *The Journal of Neuroscience*, *26*, 13311–13317.

Bergin, P. S., Thompson, P. J., Baxendale, S. A., Fish, D. R., & Shorvon, S. D. (2000). Remote memory in epilepsy. *Epilepsia*, *41*, 231–239.

Bright, P., Buckman, J., Fradera, A., Yoshinmasu, H., Colchester, A. C., & Kopelman, M. D. (2006). Retrograde amnesia in patients with hippocampal, medial temporal, temporal lobe, or frontal pathology. *Learning & Memory*, *13*, 545–557.

Buchanan, T. W., Tranel, D., & Adolphs, R. (2005). Emotional autobiographical memories in amnesic patients with medial temporal damage. *The Journal of Neuroscience*, *12*, 3151–3160.

Buchanan, T. W., Tranel, D., & Adolphs, R. (2006). Memories for emotional autobiographical events following unilateral damage to medial temporal lobe. *Brain*, *129*, 115–127.

Calabrese, P., Markowitsch, H. J., Durwen, H. F., Widlitzek, H., Haupts, M., Holinka, B., et al. (1996). Right temporofrontal cortex as critical locus for the ecphory of old episodic memories. *Journal of Neurology, Neurosurgery, and Psychiatry*, *61*, 304–310.

Cermak, L. S., & O'Connor, M. (1983). The anterograde and retrograde retrieval ability of a patient with amnesia due to encephalitis. *Neuropsychologia*, *21*, 213–234.

Chan, D., Revesz, T., & Rudge, P. (2002). Hippocampal, but not parahippocampal, damage in a case of dense retrograde amnesia: a pathological study. *Neuroscience Letters*, *329*, 61–64.

Cipolotti, L., Shallice, T., Chan, D., Fox, N., Scahill, R., Harrison, G., et al. (2001). Long-term retrograde amnesia...the crucial role of the hippocampus. *Neuropsychologia*, *39*, 151–172.

Corkin, S. (1984). Lasting consequences of bilateral medial temporal lobectomy: Clinical course and experimental findings in HM. *Seminars in Neurology*, *4*, 249–259.

Corkin, S. (2002). What's new with the amnesic patient H.M.? *Nature Reviews*, *3*, 153–160.

Corkin, S., Amaral, D. G., Gonzalez, R. G., Johnson, K. A., & Hyman, B. T. (1997). H.M.'s mesial temporal lesion: Findings from magnetic resonance imaging. *Journal of Neuroscience*, *17*, 3964–3979.

Crawford, J. R., & Garthwaite, P. H. (2002). Investigation of the single case in neuropsychology: Confidence limits on the abnormality of test scores and test score differences. *Neuropsychologia*, *40*, 1196–1208.

Crawford, J. R., & Howell, D. C. (1998). Comparing an individual's test score against norms derived from small samples. *Clinical Neuropsychologist*, *12*, 482–486.

Crovitz, H. G., & Schiffman, H. (1974). Frequency of episodic memory as a function of their age. *Bulletin of the Psychonomic Society*, *4*, 517–518.

Damasio, E. R., Eslinger, P. J., Damasio, H., Van Hoesen, G. W., & Cornell, S. (1985). Multimodal amnesic syndrome following bilateral temporal and basal forebrain damage. *Archives in Neurology*, *42*, 252–259.

Della Sala, S., Laiacona, M., Spinnler, H., & Trivelli, C. (1993). Autobiographical recollection and frontal damage. *Neuropsychologia*, *31*, 823–839.

De Renzi, E., Liotti, M., & Nichelli, P. (1987). Semantic amnesia with preservation of autobiographical memory. *Cortex*, *23*, 575–597.

Dimsdale, H., Logue, V., & Piercy, M. (1964). A case of persisting impairment of recent memory following right temporal lobectomy. *Neuropsychologia*, *1*, 287–298.

Dritschel, B. H., Williams, J. M. G., Baddeley, A. D., & Nimmo-Smith, I. (1992). Autobiographical fluency: A method for the study of personal memory. *Memory and Cognition*, *20*, 133–140.

Duyckaerts, C., Derouesne, C., Signoret, J. L., Gray, F., Escourolle, R., & Castaigne, P. (1985). Bilateral and limited amygdalohippocampal lesions causing a pure amnesic syndrome. *Annals of Neurology*, *18*, 314–319.

D'Esposito, M., Alexander, M. P., Fischer, R., McGlinchey-Berroth, R., & O'Connor, M. (1996). Recovery of memory and executive function following anterior communication artery aneurism. *Journal of the International Neuropsychological Society*, *2*, 565–570.

Ellis, A., Young, A., & Critchley, E. M. R. (1989). Loss of memory for people following temporal lobe damage. *Brain*, *112*, 1469–1483.

Eslinger, P. J. (1998). Autobiographical memory after temporal lobe lesions. *Neurocase*, *4*, 481–495.

Eslinger, P. J., Damasio, H., Damasio, A. R., & Butters, N. (1993). Nonverbal amnesia and asymmetric cerebral lesions following encephalitis. *Brain and Cognition*, *21*, 140–152.

Eslinger, P. J., Easton, A., Grattan, L. M., & Van Hoesen, G. W. (1996). Distinctive forms of partial retrograde amnesia after asymmetric temporal lobe lesions: Possible role of the occipitotemporal gyri in memory. *Cerebral Cortex*, *6*, 530–539.

Fujii, T., Moscovitch, M., & Nadel, L. (2000). Memory consolidation, retrograde memory and the temporal lobe. In F. Boller, & J. Grafman (Eds.) *Handbook of Neuropsychology, 2nd Edition, 2,* 223–250.

Fujii, T., Yamadori, A., Endo, K., Suzuki, K., & Fukatsu, R. (1999). Disproportionate retrograde amnesia in a patient with herpes simplex encephalitis. *Cortex*, *35*, 599–614.

Gainotti, G. (2007). Different patterns of famous people recognition disorders in patients with right and left anterior temporal lesions: A systematic review. *Neuropsychologia*, *45*, 1591–1607.

Goldberg, E., Antin, S. P., Bilder, R. M., Gerstmann, L. J., Hughes, J. E. O., & Mattis, S. (1981). Retrograde amnesia: Possible role of mesencephalic reticular formation in long-term memory. *Science*, *213*, 1392–1394.

Greene, J. D. W., Hodges, J. R., & Baddley, A. D. (1995). Autobiographical memory and executive function in early dementia of Alzheimer type. *Neuropsychologia*, *33*, 1647–1670.

Grewal, R. P. (2003). Severe amnesia following a unilateral temporal lobe stroke. *Journal of Clinical Neuroscience*, *10*, 102–104.

Hanley, J. R., Young, A. W., & Pearson, N. (1989). Defective recognition of familiar people. *Cognitive Neuropsychology*, *6*, 179–210.

Haslam, C., Cook, M., & Coltheart, M. (2001). "I know your name but not your face": Explaining modality-based differences in access to biographical knowledge in a patient with retrograde amnesia. *Neurocase*, *7*, 189–200.

Haslam, C., Cook, M., & McKone, E. (1998). Memory for generalities: Assess to higher-level categorical relationships in amnesia. *Cognitive Neuropsychology*, *15*, 401–437.

Hepner, I., Mohamed, A., Fulham, M., & Miller, L. A. (2007). Topographical, autobiographical and semantic memory in a patient with bilateral mesial temporal and retrosplenial infarction. *Neurocase*, *13*, 97–114.

Hermann, B. P., Wyler, A. R., & Richey, E. T. (1988). Wisconsin Card Sorting Test performance in patients with complex partial seizures of temporal lobe origin. *Journal of Clinical and Experimental Neuropsychology*, *10*, 467–476.

Hirano, M., & Noguchi, K. (1998). Dissociation between specific personal episodes and other aspects of remote memory in a patient with hippocampal amnesia. *Perceptual and Motor Skills*, *87*, 99–107.

Hodges, J. R., & McCarthy, R. A. (1993). Autobiographical amnesia resulting from bilateral paramedian thalamic infarction. *Brain*, *116*, 921–940.

Hodges, J. R., Patterson, K., Oxbury, S., & Funnell, E. (1992). Semantic dementia: Progressive fluent aphasia with temporal lobe atrophy. *Brain*, *115*, 1783–1806.

Hokkanen, L., Launes, J., Varaja, R., Valanne, L., & Iivanainen, M. (1995). Isolated retrograde amnesia for autobiographical material associated with acute left temporal lobe encephalitis. *Psychological Medicine*, *25*, 203–208.

Howard, D., & Patterson, K. (1992). *Pyramids and Palm Trees: Access from pictures and words*. Bury St. Edmunds (UK): Thames Valley Test Company.

Hunkin, N. M., Parkin, A. J., Bradley, V. A., Burrows, E. H., Aldrich, F. K., Jansari, A., et al. (1995). Focal retrograde amnesia following closed head injury: A case study and theoretical account. *Neuropsychologia*, *33*, 509–523.

Isaac, C. L., Holdstock, J. S., Cezayirli, E., Roberts, J. N., Holmes, C. J., & Mayes, A. R. (1998). Amnesia in a patient with lesions limited to the dorsomedial thalamic nucleus. *Neurocase*, *4*, 497–508.

Jefferies, E., & Lambon Ralph, M. A. (2006). Semantic impairment in stroke aphasia versus semantic dementia: a case-series comparison. *Brain*, *129*, 2132–2147.

Kapur, N. (1997). Autobiographical amnesia and temporal lobe pathology. In A. J. Parkin (Ed.), *Classic Case Studies in the Neuropsychology of Memory* (pp. 37–62). Hove, UK: Lawrence Erlbaum Associates Ltd.

Kapur, N. (1999). Syndromes of retrograde amnesia: A conceptual and empirical synthesis. *Psychological Bulletin*, *125*, 800–825.

Kapur, N., & Brooks, D. J. (1999). Temporally specific retrograde amnesia in two cases of discrete bilateral hippocampal pathology. *Hippocampus*, *9*, 247–254.

Kapur, N., Ellison, D., Parkin, A. J., Hunkin, N., Burrows, E., Sampson, S., et al. (1994). Bilateral temporal lobe pathology with sparing of medial temporal lobe structures: Lesion profile and pattern of memory disorder. *Neuropsychologia*, *32*, 23–38.

Kapur, N., Ellison, D., Smith, M., McLellan, L., & Burrows, E. H. (1992). Focal retrograde amnesia following bilateral temporal lobe pathology: A neuropsychological and magnetic resonance study. *Brain*, *115*, 73–85.

Kapur, N., Scholey, K., Moore, E., Barker, S., Brice, J., Thompson, S., et al. (1996). Long-term retention deficits in two cases of disproportionate retrograde amnesia. *Journal of Cognitive Neuroscience*, *8*, 416–434.

Kartsounis, L. D., Rudge, P., & Stevens, J. M. (1995). Bilateral lesion of CA1–CA2 fields of the hippocampus are sufficient to cause a severe amnesic syndrome in humans. *Journal of Neurology, Neurosurgery and Psychiatry*, *59*, 95–98.

Kay, J., & Hanley, J. R. (1999). Person-specific knowledge and knowledge of biological categories. *Cognitive Neuropsychology*, *16*, 171–180.

Kopelman, M. D. (1991). Frontal dysfunction and memory deficits in the alcoholic Korsakoff Syndrome and Alzheimer-type dementia. *Brain*, *114*, 117–137.

Kopelman, M. D., & Kapur, N. (2001). The loss of episodic memories in retrograde amnesia: Single-case and group studies. *Philosophical Transactions of The Royal Society of London B*, *385*, 1409–1421.

Kopelman, M. D., Stanhope, N., & Kingsley, D. (1999). Retrograde amnesia in patients with diencephalic, temporal lobe or frontal lesions. *Neuropsychologia*, *37*, 939–958.

Kopelman, M. D., Wilson, B. A., & Baddley, A. D. (1989). The autobiographical memory interview: A new assessment of autobiographical and personal semantic memory in amnesic patients. *Journal of Clinical and Experimental Neuropsychology*, *11*, 724–744.

Kopelman, M. D., Wilson, B. A., & Baddeley, A. D. (1990). *The Autobiographical Memory Interview*. Suffolk, England: Thames Valley Test Company.

Kopelman, M. D., Lasserson, D., Kingsley, D. R., Bello, F., Rush, C., Stanhope, N., et al. (2003). Retrograde amnesia and the volume of critical brain structures. *Hippocampus*, *13*, 879–891.

Kroll, N. E. A., Markowitsch, H. J., Knight, R. T., & von Cramon, D. Y. (1997). Retrieval of old memories: The temporofrontal hypothesis. *Brain*, *120*, 1377–1399.

Lah, S., Grayson, S., Lee, T., & Miller, L. (2004). Memory for the past after temporal lobectomy: impact of epilepsy and cognitive variables. *Neuropsychologia*, *42*, 1666–1679.

Lah, S., Lee, T., Grayson, S., & Miller, L. (2006). Effects of temporal lobe epilepsy on retrograde memory. *Epilepsia*, *47*, 615–625.

Lambert, N. A., Swain, M. A., Miller, L. A., & Caine, D. (2006). Exploring the neural organization of person-related knowledge: Lateralisation of lesion, category specificity and stimulus modality effects. *Neuropsychology*, *20*, 346–354.

Levine, B., Black, S., Cabeza, R., Sinden, M., McIntosh, A., Toth, J., et al. (1998). Episodic memory and the self in a case of isolated retrograde amnesia. *Brain*, *121*, 1951–1973.

Luchelli, F., Muggia, S., & Spinnler, H. (1995). The "Petites Madelines" phenomenon in two amnesic patients. Sudden recovery of forgotten memories. *Brain*, *118*, 167–183.

Mangels, J. A., Gershberg, F. B., Shimamura, A. P., & Knight, R. T. (1996). Impaired retrieval from remote memory in patients with frontal lobe damage. *Neuropsychology*, *10*, 32–41.

Markowitsch, H. J. (1995). Which brain regions are critically involved in the retrieval of old episodic memory? *Brain Research Reviews*, *21*, 117–127.

Markowitsch, J. J., vonCramon, Y., & Schuri, U. (1993). Mnestic performance profile of a bilateral diencephalic infarct patient with preserved intelligence and severe amnesic disturbance. *Journal of Clinical and Experimental Neuropsychology*, *15*, 627–652.

Marslen-Wilson, W. D., & Teuber, H. L. (1975). Memory for remote events in anterograde amnesia: Recognition of public figures from news photographs. *Neuropsychologia*, *13*, 347–352.

Martin, R. C., Sawrie, S. M., Faught, E., Kuzniecky, R. I., Gilliam, F. G., Edwards, R., et al. (2000). Investigation of executive function change following anterior temporal lobectomy: Selective normalization of verbal fluency. *Neuropsychology*, *14*, 501–508.

Mattioli, F., Grassi, F., Perani, D., Cappa, S. F., Miozzo, A., & Fazio, F. (1996). Persistent post-traumatic retrograde amnesia: A neuropsychological and (18F) FDG PET study. *Cortex*, *32*, 121–129.

Mayes, A. R., Daum, I., Markowitsch, H. J., & Sauter, B. (1997). The relationship between retrograde and anterograde amnesia in patients with typical global amnesia. *Cortex*, *33*, 197–217.

McCarthy, R. A., & Warrington, E. (1992). Actors but not scripts: The dissociation of people and events in retrograde amnesia. *Neuropsychologia*, *30*, 633–644.

McCarthy, R. A., Kopelman, M. D., & Warrington, E. K. (2005). Remembering and forgetting of semantic knowledge in amnesia: A 16-year follow-up investigation of RFR. *Neuropsychologia*, *43*, 356–372.

Miceli, G., Capasso, R., Daniele, A., Esposito, T., Magarelli, M., & Toumaiuolo, F. (2000). Selective deficit for people's names following temporal damage: An impairment of domain-specific conceptual knowledge. *Cognitive Neuropsychology*, *17*, 489–516.

Miller, L. A., Caine, D., Harding, A., Thompson, E. M., Large, M., & Watson, J. D. G. (2001). Right medial thalamic lesion causes isolated retrograde amnesia. *Neuropsychologia*, *39*, 1037–1046.

Miller, L. A., Caine, D., & Watson, J. D. G. (2003). A role for the thalamus in memory for unique entities. *Neurocase*, *9*, 504–514.

Moscovitch, M., & Melo, B. (1997) Strategic retrieval and the frontal lobes: evidence from confabulation and amnesia. *Neuropsychologia*, *35*, 1017–1034.

Moscovitch, M., Yaschyshyn, T., Ziegler, M., & Nadel, L. (1999). Remote episodic memory and retrograde amnesia: was Endel Tulving right all along? In E. Tulving (Ed.), *Memory, Consciousness and the Brain: The Tallin Conference* (pp. 331–345). New York: Psychology Press.

Moscovitch, M., Rosenbaum, R. S., Gilboa, A., Addis, D. R., Westmacott, R., Grady, C., et al. (2005). Functional neuroanatomy of remote episodic, semantic and spatial memory: a unified account based on multiple trace theory. *Journal of Anatomy*, *207*, 35–66.

Muree, J. M. J. (1996). A model of amnesia and consolidation of memory. *Hippocampus*, *6*, 675–684.

Nadel, L., & Moscovitch, M. (1997). Memory consolidation, retrograde amnesia and the hippocampal complex. *Current Opinion in Neurobiology*, *7*, 217–227.

Nadel, L., Samsonovich, A., Ryan, L., & Moscovitch, M. (2000). Multiple trace theory of human memory: Computational, neuroimaging, and neuropsychological results. *Hippocampus*, *10*, 352–368.

Nichelli, P., Bahmanian-Behbahani, G., Gentilini, M., & Vecchi, A. (1988). Preserved memory abilities in thalamic amnesia. *Brain*, *111*, 1337–1353.

O'Connor, M., Butters, N., Miliotis, P., Eslinger, P., & Cermak, L. S. (1992). The dissociation of anterograde and retrograde amnesia in a patient with herpes encephalitis. *Journal of Clinical and Experimental Neuropsychology*, *14*, 159–178.

O'Connor, M. G., & Lafleche, G. M. C. (2004). Retrograde amnesia in patients with rupture and surgical repair of anterior communicating artery aneurism. *Journal of the International Neuropsychological Society*, *10*, 221–229.

Papps, N. P., Best, J. J., & O'Carroll, R. E. (2000). Semantic memory functioning and the left temporal lobe. *Neurocase*, *6*, 197–192.

Parkin, A. J., & Hunkin, N. M. (1991). Memory for loss following radiotherapy for naso-pharyngeal cancer. *British Journal of Psychology*, *30*, 349–357.

Reed, J. M., & Squire, L. R. (1998). Retrograde amnesia for facts and events: Findings from four new cases. *The Journal of Neuroscience*, *18*, 3943–3954.

Reinvang, I., Nielsen, C. S., Gjerstad, L., & Bakke, S. J. (2000). Isolated retrograde amnesia: Evidence for preservation of implicit memory. An event-related potential investigation. *Neurocase*, *6*, 423–433.

Rempel-Clower, N. L., Zola, S. M., Squire, L. R., & Amaral, D. G. (1996). Three cases of enduring memory impairment after bilateral damage limited to the hippocampal formation. *Journal of Neuroscience*, *16*, 5233–5255.

Rubin, D. C., & Greenberg, D. L. (1998). Visual memory-deficit amnesia: A distinct amnesic presentation and etiology. *Proceedings of the National Academy of Science of the United States of America*, *95*, 5413–5416.

Rubin, D. C., Wetzler, S. E., & Nebes, R. D. (1986). Autobiographical memory across the lifespan. In D. C. Rubin (Ed.), *Autobiographical Memory* (pp. 202–221). Cambridge: Cambridge University Press.

Schmidtke, K., & Vollmer, H. (1997). Retrograde amnesia: A study of its relation to anterograde amnesia and sematic memory deficit. *Neuropsychologia*, *35*, 505–518.

Schmolck, H., Kensinger, E., Corkin, S., & Squire, L. R. (2002). Semantic knowledge in patient H. M. and other patients with bilateral medial and lateral temporal lobe lesions. *Hippocampus*, *12*, 520–533.

Schnider, A., Bassetti, C., Gutbrod, K., & Ozdoba, C. (1995). Very severe amnesia with acute onset hippocampal damage due to systemic lupus erythematosus. *Journal of Neurology, Neurosurgery and Psychiatry*, *59*, 644–646.

Scoville, W. B., & Milner, B. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery and Psychiatry*, *20*, 11–26.

Seidenberg, M., Griffith, R., Sabsevitz, D., Moran, M., Haltiner, A., Bell, B., et al. (2002). Recognition and identification of famous faces in patients with unilateral temporal lobe epilepsy. *Neuropsychologia*, *40*, 446–456.

Shimamura, A. P., & Squire, L. R. (1986). Korsakoff's Syndrome: A study of the relation between anterograde amnesia and remote memory impairment. *Behavioural Neuroscience*, *100*, 165–170.

Shum, D. H., & O'Gorman, J. G. (2001). A test of remote memory for use in Australia. *Australian Journal of Psychology*, *53*, 36–44.

Snowden, J. S., Thompson, J. C., & Neary, D. (2004). Knowledge of famous faces and names in semantic dementia. *Brain*, *127*, 860–872.

Squire, L. R. (1992). Memory and hippocampus: A synthesis from findings with rats, monkeys and humans. *Psychology Review*, *99*, 195–231.

Squire, L. R., Cohen, N. J., & Nadel, L. (1984). The medial temporal region and memory consolidation: a new hypothesis. In H. Weingartner & E. Parker (Eds.), *Memory Consolidation* (pp. 185–210). NJ: Erlbaum, Hillsdale.

Stanhope N., Kopelman M. D. (2000). Art and memory: A 7-year follow-up of herpes encephalitis in a professional artist. *Neurocase*, *6*, 99–110.

Stefanacci, L., Buffalo, E. A., Schmolck, H., & Squire, L. R. (2000). Profound amnesia after damage to the medial temporal lobe: Aeuroanatomical and neuropsychological profile in patient E. P. *The Journal of Neuroscience*, *15*, 7024–7036.

Steinvorth, S., Levine, B., & Corkin, S. (2005). Medial temporal lobe structures are needed to re-experience remote autobiographical memories: evidence from H.M. and W.R. *Neuropsychologia*, *43*, 479–496.

Strauss, E., Hunter, M., & Wada, J. (1993). Wisconsin Card Sorting performance: Effects of age of onset of damage and laterality of dysfunction. *Journal of Clinical and Experimental Psychology*, *15*, 896–902.

Stuss, D. T., Guberman, A., Nelson, R., & Larochelle, S. (1988). The neuropsychology of paramedial thalamic infarction. *Brain and Cognition*, *8*, 348–378.

Tanaka, Y., Miyazawa, Y., Hashimoto, R., Nakano, I., & Obayashi, T. (1999). Post-encephalitic focal retrograde amnesia after bilateral anterior temporal lobe damage. *Neurology*, *53*, 344–350.

Tranel, D., & Jones, R. D. (2006). Knowing "what" and knowing "when". *Journal of Clinical and Experimental Neuropsychology*, *28*, 43–66.

Tulving, E., Schachter, D. L., McLachland, D., & Moscovitch, M. (1988). Priming of semantic autobiographical knowledge: A case study of retrograde amnesia. *Brain and Cognition*, *8*, 2–20.

Verstichel, P., Cohen, L., & Crochet, G. (1996). Associated production and comprehension deficits for people's names following left temporal lesion. *Neurocase*, *2*, 221–234.

Viskontas, I. V., McAndrews, M. P., & Moscovitch, M. (2000). Remote episodic memory deficits in patients with unilateral temporal lobe epilepsy and excisions. *The Journal of Neuroscience*, *20*, 5853–5857.

Viskontas, I. V., McAndrews, M. P., & Moscovitch, M. (2002). Memory for famous people in patients with unilateral temporal lobe epilepsy and excision. *Neuropsychology*, *16*, 472–480.

Voltzenlogel, V., Despres, O., Vignal, J-P., Steinhoff, B. J., Kehril, P., & Manning, L. (2006). Remote memory in temporal lobe epilepsy. *Epilepsia*, 1329–1336.

Warrington, E. K., & Duchen, L. W. (1992). A re-appraisal of a case of persistent global amnesia following right temporal lobectomy: A clinico-pathological study. *Neuropsychologia*, *30*, 437–450.

Warrington, E. K., & McCarthy, R. A. (1988). The fractionation of retrograde amnesia. *Brain and Cognition*, *7*, 184–200.

Warrington, E. K., & Sanders, H. I. (1971). The fate of old memories. *Quarterly Journal of Psychology*, *23*, 432–442.

Warrington, E. K., & Silberstein, M. (1970). A questionnaire technique for investigating very long term memory. *Quarterly Journal of Experimental Psychology*, *22*, 508–512.

Westmacott, R., Leach, L., Freedman, M., & Moscovitch, M. (2001). Differential patterns of autobiographical memory loss in semantic dementia and medial temporal lobe amnesia: A challenge to consolidation theory. *Neurocase*, *7*, 37–55.

Westmacott, R., & Moscovitch, M. (2002). Temporally graded semantic memory loss in amnesia and sematic dementia: Further evidence for opposite gradients. *Cognitive Neuropsychology*, *19*, 135–163.

Worthington, A. (1999). Dysexecutive paramnesia: Strategic retrieval deficits in retrospective and prospective remembering. *Neurocase*, *5*, 47–57.

Yasuda, K., Watanabe, O., & Ono, Y. (1997). Dissociation between semantic and autobiographical memory: A case report. *Cortex*, *33*, 623–638.

Yoneda, Y., Mori, E., Yamashita, H., & Yamadori, A. (1994). MRI volumetry of medial temporal lobe structures in amnesia following herpes simplex encephalitis. *European Neurology*, *34*, 243–252.

Yoneda, Y., Yamadori, A., Mori, E., & Yamashita, H. (1992). Isolated prolonged retrograde amnesia. *European Neurology*, *32*, 340–342.

Zola-Morgan, S., Squire, L. R., & Amaral, D. G. (1986). Human amnesia and the medial temporal lobe region: enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus. *Journal of Neuroscience*, *6*, 2950–2967.