Disorders of memory

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Different types of memory

Fractionation of memory systems

- Short-term & working memory
- Long-term memory
  - Explicit memory: With conscious recall
    - Semantic memory: Facts and general knowledge
    - Episodic memory: Personally experienced events
  - Implicit memory: Without conscious recall
    - Procedural memory: Motor and cognitive skills
    - Priming: Enhanced identification of objects or words
Clinical measures of STM

Forwards digit span (verbal) and spatial span (visuospatial) provide an index of STM capacity

How many things (digits or spatial locations) can you remember?
At what sequence length (‘span’) of digits or tapped spatial locations does performance fail?
Patient studies reveal double dissociations

Between verbal deficits from left posterior lesions and visuospatial deficits from right hemisphere damage

Patient KF (Shallice & Warrington, 1970) with a left parietal lesion had a digit span of only 2 but normal spatial span on Corsi blocks and normal long-term memory

By contrast, patient ELD (Hanley et al, 1991) with a right hemisphere lesion had an impaired spatial span but normal digit span and long-term memory
Working memory

*Manipulation of STM contents*

**Executive control**

mechanisms manipulate contents of STM, e.g., reverse or backward digit span test where people are required to recall a sequence of numbers in reverse

‘Slave’ storage systems

Crystallized LTM (long term memory) systems
Frontoparietal systems

*Implicated in working memory tasks with one influential view being*

**Parietal cortex**
- Maintenance / storage

**Prefrontal cortex**
- Manipulation & monitoring of information in STM
- E.g. updating contents as in N-back tasks or reading them in reverse order as in reverse span tasks

*BUT now clear that it is difficult to dissociate differences in function between parietal and prefrontal regions*
Short-term and working memory

Summary 1

- **STM** is often used to refer to *passive storage* over *seconds*
- **Working memory** refers to *executive control* over material stored in STM
- STM stores are ‘modality specific’, e.g. visuospatial material may be held in a separate store (*visuospatial sketchpad*) to verbal material (*phonological loop*)
- Early focal lesion and functional imaging studies suggested that STM stores reside in *posterior parietal cortex* (visuospatial material in the right parietal cortex and verbal material in the left) whereas executive control systems reside in *dorsolateral prefrontal cortex*. This division may be an over-simplification but can be helpful.
Short-term and working memory

Summary 2

► Verbal STM **storage capacity** is measured at the bedside using **digit span** (how many numbers in a sequence can be recalled)

► Visuospatial STM **storage capacity** is measured using the **Corsi blocks** (how many spatial locations in a sequence can be recalled)

► **Verbal working memory** (storage plus executive control) is measured by using **reverse digit span** (how many numbers in a sequence can be recalled in reverse order)

► **Visuospatial working memory** is measured by using **reverse Corsi blocks** (how many location in a sequence can be recalled in reverse order)
Fractionation of Long term memory

*Into episodic and semantic memory*
How do we test for episodic memory?

In the clinical assessment

**Anterograde verbal memory**

- Ask patient to recall details of very recent events, e.g. what happened to them in the last few days or how they got to the clinic
- You can tell them something about your own interests at the beginning of the interview and ask about these later
- Name and address recall (e.g. in Addenbrooke’s Cognitive Examination – next slide)
- Formal tests: story recall or word list learning tests (e.g. Rey Auditory Verbal Learning Test (RAVLT) or California Verbal Learning Test (CVLT)).

**Anterograde non-verbal memory**

- Rey-Osterrieth Complex Figure recall from memory (after a delay)
- Recognition Memory Test (RMT) subset for faces: recall of a series of photographs of faces

Hodges (2007) *Cognitive Assessment for Clinicians* 2nd ed
Verbal episodic memory

For example by asking patient to remember a name and address

- Free recall is harder than recognition when people have to choose between alternative possibilities
- In the Addenbrooke’s Cognitive Examination, participants learn a name and address over 3 trials (encoding) and are then asked to recall (retrieve) it at the end of the test. If they can’t recall elements, they are given recognition test (choose between possible alternatives)

**MEMORY**

- Tell: “I'm going to give you a name and address and I'd like you to repeat the name and address after me. So you have a chance to learn, we’ll be doing that 3 times. I'll ask you the name and address later.”
- Score only the third trial.

<table>
<thead>
<tr>
<th>1st Trial</th>
<th>2nd Trial</th>
<th>3rd Trial</th>
</tr>
</thead>
</table>
| Harry Barnes  
73 Orchard Close  
Kingsbridge  
Devon |           |           |
|           |           |           |
Verbal episodic memory

For example by asking patient to remember a name and address

- In the Addenbrooke’s Cognitive Examination, participants learn a name and address over 3 trials (encoding) and are then asked to recall (retrieve) it at the end of the test. If they can’t recall elements, they are given recognition test (choose between possible alternatives)

<table>
<thead>
<tr>
<th>Memory</th>
<th>Memory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ask “Now tell me what you remember about that name and address we were repeating at the beginning”</td>
<td>Memory [Score 0-7]</td>
</tr>
<tr>
<td>Harry Barnes 73 Orchard Close Kingsbridge Devon</td>
<td></td>
</tr>
<tr>
<td>……………… …………………………………</td>
<td></td>
</tr>
<tr>
<td>……………… …………………………………</td>
<td></td>
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<tr>
<td>……………………………………………</td>
<td></td>
</tr>
<tr>
<td>……………………………………………</td>
<td></td>
</tr>
<tr>
<td>Memory [Score 0-5]</td>
<td></td>
</tr>
</tbody>
</table>

This test should be done if the subject failed to recall one or more items above. If all items were recalled, skip the test and score 5. If only part was recalled start by ticking items recalled in the shadowed column on the right hand side, and then test not recalled items by telling the subject “ok, I’ll give you some hints: was the name X, Y or Z?” and so on. Each recognised item scores one point, which is added to the point gained by recalling.

<table>
<thead>
<tr>
<th>Memory</th>
<th>Memory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jerry Barnes 37 Orchard Place Oakhampton Devon</td>
<td>Harry Barnes 73 Oak Close Kingsbridge Dorset</td>
</tr>
<tr>
<td>recalled</td>
<td>recalled</td>
</tr>
</tbody>
</table>
Non-verbal episodic memory
For example by asking patient to remember a complex figure

- Rey-Osterrieth complex figure immediate copying and then 10-15 mins later
How do we test for episodic memory?

*In the clinical assessment*

**Retrograde memory**

- Famous news or sports events from preceding months, years and decades: e.g., wars, scandals, political events, disasters
- Remote personal autobiographical memory
- Formal test: Autobiographical Memory Interview

Hodges (2007) *Cognitive Assessment for Clinicians* 2nd ed
Patient HM
Had surgery for intractable epilepsy in 1953 and was left with a profound disorder of episodic memory

Severe anterograde amnesia

Couldn’t learn new information such as events, names or even find his new home. Language ‘frozen’in 1950s so new words introduced into the lexicon, e.g. jacuzzi, meant nothing to him.
Patient HM

Had surgery for intractable epilepsy in 1953 and was left with a profound disorder of episodic memory.

Severe but graded retrograde amnesia

Could still recall childhood memories and jobs in teens and twenties, but difficulty remembering events that occurred in the years immediately preceding surgery.

Retrograde memory loss extended back 11 yrs.
Temporal gradient in retrograde amnesia (Ribot’s law)

First in, last out | earliest memories survive best

Butters & Cermak 1986
Consolidation hypothesis of hippocampal role in memory

‘Standard model’ proposes initially hippocampal-cortical interactions are required but eventually transfers to cortex.

So lesions of hippocampus won’t erase old memories which are consolidated and robustly represented in the cortex. Hence graded retrograde amnesia after hippocampal lesions.

But hippocampal lesions would prevent consolidation of new memories. Hence severe anterograde amnesia in HM.

Frankland & Bontempi (2005) Nat Rev Neurosci
Medial temporal involvement in Alzheimer’s disease

High resolution imaging at 7 Tesla

24 yr old healthy person

72 yr old with mild AD

Dickerson & Eichenbaum (2010) Neuropsychopharmac Rev
Regional spread of Alzheimer pathology

Earliest regions to show tau pathology (neurofibrillary tangles) is entorhinal cortex | Braak staging

A  NFT stages I + II

B  NFT stages II + IV

C  NFT stage III

D  NFT stage VI
Mild cognitive impairment (MCI)

Prodromal Alzheimer’s disease in many cases

Figure 2. Coronal MRI Scans from Patients with Normal Cognition, Mild Cognitive Impairment, and Alzheimer’s Disease.

The arrows depict the hippocampal formations and the progressive atrophy characterizing the progression from normal cognition (Panel A) to mild cognitive impairment (Panel B) to Alzheimer’s disease (Panel C).
Mild cognitive impairment (MCI) and prodromal Alzheimer’s

MCI Patients have an annual risk of ~10% conversion to diagnosis of Alzheimer’s disease

Figure 2. Annual rates of conversion from mild cognitive impairment (MCI) to dementia over 48 months.
**DSM-5 criteria**

*For mild neurocognitive disorder*

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**Box 2 | Diagnostic criteria for mild neurocognitive disorder**

A. Evidence of modest cognitive decline from a previous level of performance in one or more cognitive domains (complex attention, executive function, learning and memory, language, perceptual–motor, or social cognition) based on:

1. Concern of the individual, a knowledgeable informant, or the clinician that there has been a mild decline in cognitive function; and
2. A modest impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.

B. The cognitive deficits do not interfere with capacity for independence in everyday activities (that is, complex instrumental activities of daily living such as paying bills or managing medications are preserved, but greater effort, compensatory strategies, or accommodation may be required).

C. The cognitive deficits do not occur exclusively in the context of a delirium.

D. The cognitive deficits are not better explained by another mental disorder (for example, major depressive disorder or schizophrenia).

Subjective cognitive impairment (SCI) / decline (SCD)

Most cases are not prodromal Alzheimer's but nevertheless SCI is associated with increased risk of developing AD

- Individuals perceive decline in memory and/or other cognitive abilities relative to their previous level of performance, in the absence of objective neuropsychological deficits
- Increased risk of developing dementia, but exactly what is that risk?
Acute onset amnesia

- **MRI: focal DWI lesions in the lateral hippocampus (CA1) 48–72 h after acute phase**
  - **EEG without pathology**
  - **Transient global amnesia**

- **MRI normal or chronic lesion in the temporal lobe**
  - **EEG: epileptiform activity**
  - **Clinically: other ictal signs**
  - **Transient epileptic amnesia**

- **MRI: DWI lesion with vascular distribution (thalamus, posterior cerebral artery)**
  - **Stressful, emotional event in the past**
  - **Possible psychogenic amnesia**

- **Situation-specific psychogenic amnesia or global psychogenic amnesia**
  - **Knowledge of personal identity is often impaired**
  - **Remote memory often more strongly affected**
  - **Anterograde memory is comparatively preserved**
  - **Herpes simplex encephalitis**

- **Memory impairment, focal neurological signs**
  - **MRI: abnormalities in temporal lobe, limbic system**
  - **CSF: HSV-PCR**
  - **Herpes simplex encephalitis**

- **Headache, fever, delirium, confusion, epileptic fits, focal neurological signs, hallucinations**
  - **MRI: abnormalities in temporal lobe, limbic system**
  - **CSF: pleocytosis**
  - **Autoimmune limbic encephalitis**

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- **Acute onset amnesia**
  - **Duration: 1 h to <24 h**
  - **Transient global amnesia**

- **Duration: <1 h Frequency: >1 per year**
  - **Memory impairment, focal neurological signs**
  - **EEG: epileptiform activity**
  - **Clinically: other ictal signs**
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  - **EEG without pathology**
  - **Transient global amnesia**

Barsch & Butler (2013) Nat Rev Neurol
Transient global amnesia (TGA)

*Episode of amnesia resolving within 24hrs*

- Witnessed anterograde amnesia without clouding of consciousness or loss of personal identity
- Cognitive impairment limited to amnesia. No focal physical neurological signs or features of epilepsy. No recent head trauma or seizures.
- Resolution within 24hrs. Recurrence unusual but may occur in 6-10%
- Mild headache / nausea / dizziness might be present during attack
- **Memory during attack:** Episodic memory tested with recall of word list or complex figures shows dense anterograde amnesia. Performance on tests of retrograde amnesia variable but typically shows Ribot’s gradient, particularly with respect to autobiographical details. Spatial memory deficits for learning and retrieval too. Semantic memory spared
- **Memory after attack:** Usually good but there is some debate on this. After recovery a dense amnesic gap for events that occurred during the attack itself often persists
A 68-year-old woman presented following an episode of witnessed sudden-onset memory loss after lifting a heavy plant while gardening. Her husband’s description of the event suggested that there was retrograde and anterograde amnesia with repetitive questioning. Speech, motor function and vision were unaffected. The episode spontaneously resolved after 2 h. Her medical history included hypertension and hypercholesterolaemia controlled with lisinopril and atorvastatin. On admission she was fully conscious and alert and neurological examination was normal. A 12-lead ECG and haematological investigations were normal. We made a clinical diagnosis of transient global amnesia (TGA).

Brain diffusion-weighted MRI at 48 h postevent showed focal bilateral medial temporal lobe high signal with restricted diffusion (figure 1). Other sequences were normal. These findings had resolved on repeat imaging 23 days later. The patient has remained asymptomatic since then.

TGA is a benign amnestic syndrome, usually lasting less than 24 h. It is characterised by a sudden onset of transient anterograde and retrograde amnesia without focal neurological signs or seizure features, and with other intellectual abilities preserved.

Our patient’s neuroradiological findings are consistent with similar previous reports of reversible hyperintensities on diffusion-weighted MRI within 48 h of symptom onset in 8–33% of TGA patients. These findings have intensified debate around the likely aetiology of TGA. The proposed aetiologies for TGA include migraine, venous flow abnormalities, focal ischaemia and epilepsy. Along with being potential aetiologies of TGA, these conditions are also differential diagnoses for patients with an amnestic syndrome. Despite the MRI changes in a subset of patients with TGA, the diagnosis remains clinical. In terms of management, reassurance is sufficient.

Acknowledgements
This paper was reviewed by Adam Zeman, Exeter, UK.

MRI 48hrs after symptom onset
A) DWI
B) Restricted diffusion on ADC maps

Changes had resolved on repeat imaging 3 weeks later

Transient epileptic amnesia (TEA)

Recurrent episodes associated with brief episodes of amnesia but with long term consequences

- Recurrent, witnessed episodes of transient amnesia (often during waking)
- Cognitive functions other than memory as judged by a reliable witness appear normal, e.g. speech, finding way around a house, even driving may be intact
- Evidence of epilepsy: concurrent suggestive clinical features (olfactory / gustatory hallucinations, automatisms such as lip smacking), abnormal EEG or clear-cut response to anticonvulsant therapy

Memory during attack: Patients have difficulty laying down new memories (anterograde amnesia) and also retrieving past events (retrograde amnesia) BUT in contrast to TGA, anterograde deficit is often partial. >40% patients say they can recall “not being able to remember”. They may repetitively ask questions but this is not as consistent as in TGA.

Memory after attack: Most cases have significant ongoing difficulties with either remote autobiographical memory, accelerated long term forgetting (ALF) after days, spatial navigation / topographical amnesia.
Accelerated long-term forgetting in TEA

Standard neuropsychological testing of episodic memory might test recall after 15 or 30 minutes, not days
Autobiographical memory deficit in TEA

Gradient of deficit following Ribot’s law

Complete retrograde amnesia but intact anterograde memory

*Jason Bourne in the ‘Bourne Identity’ has no idea who he is or what he did*
Psychogenic amnesia

Profound retrograde amnesia for personal events but often with intact anterograde memory

- Amnesia without any cause found on investigation
- Often associated with highly stressful life event as precipitant
- There may be a period of wandering or ‘psychogenic fugue’
- **Memory during attack:** Patient may not recall or their own name or be able to recognize family members. They may have no knowledge of who they are, where they live or what they do. They may have no knowledge of famous news events across their lifespan. But often have no difficulty remembering new information, e.g. from clinical staff. In fact they may be able to ‘relearn’ about themselves
- **Memory after attack:** Most cases have full recovery
**Psychogenic amnesia vs TGA or TEA**

*Some important tell-tale differences*

**IN BOTH**
- Can be preceded by precipitating stress/significant life-event
- Standard investigations (routine EEG, CT, MRI) can be normal

**DIFFERENTIATION**
- Loss of personal identity in fugue, never in TGA
- Repetitive questioning in TGA/TEA, seldom in fugue/psychogenic amnesia where there may be ‘la belle indifférence’
- Other symptoms/signs, e.g. sensorimotor in TEA, wandering in fugue
- ‘Temporal gradient’ of retrograde amnesia in TGA/TEA vs. ‘reversed gradient’ in psychogenic amnesia

Kopelman (2003) *Practical Neurol*
Transient amnesia syndromes

Table 1 Distinguishing clinical features of the transient amnesic syndromes.

<table>
<thead>
<tr>
<th>Feature</th>
<th>Transient epileptic amnesia</th>
<th>Transient global amnesia</th>
<th>Psychogenic amnesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical age of onset</td>
<td>50–70 years</td>
<td>50–70 years</td>
<td>Any age</td>
</tr>
<tr>
<td>Past medical history</td>
<td>None</td>
<td>Migraine</td>
<td>‘Organic’ transient amnesia; substance abuse; psychiatric illness</td>
</tr>
<tr>
<td>Precipitants</td>
<td>Waking</td>
<td>Cold water; physical exertion; psychological stress</td>
<td>Minor head injury; stress; depression</td>
</tr>
<tr>
<td>Ictal memory profile</td>
<td>Anterograde and retrograde amnesia showing within-patient variation; patient might later partially recall attack; retrograde procedural memory intact</td>
<td>Profound anterograde amnesia including repetitive questioning; variable retrograde amnesia; intact nondeclarative memory</td>
<td>Highly variable: often profound retrograde amnesia with loss of personal identity; relatively preserved anterograde memory; variable procedural memory</td>
</tr>
<tr>
<td>Duration of amnestic episode</td>
<td>Usually &lt;1 h but can last much longer (days)</td>
<td>Typically 4–10 h</td>
<td>Days or months</td>
</tr>
<tr>
<td>Recurrence</td>
<td>Mean frequency 13 attacks per year</td>
<td>Rare</td>
<td>Rare</td>
</tr>
<tr>
<td>Postictal and interictal memory</td>
<td>Accelerated forgetting; remote autobiographical memory loss; topographical amnesia</td>
<td>Grossly intact, but subtle deficits might persist for several months</td>
<td>Variable: patient might be able to ‘relearn’ the past</td>
</tr>
<tr>
<td>Other features</td>
<td>Olfactory hallucinations; orolimentary automatisms; brief loss of responsiveness</td>
<td>Headache and/or nausea</td>
<td>Focal ‘neurological’ symptoms or signs, such as hemiparesis</td>
</tr>
</tbody>
</table>
Amnesia with encephalitic syndromes

Viral encephalitis

Insausti et al (2013) PNAS | Squire’s group
Amnesia with encephalitic syndromes

Paraneoplastic limbic encephalitis

Antibody
- Anti-Hu antibody (ANNA-1)
- Anti-CV 2 antibody (CRMP5)
- Anti-Ma antibody (Ma 1, Ma 2)
- Antiamphiphysis antibody
- ANNA-3
- PCA-2

Associated Cancers
- Small-cell lung cancer
- Small-cell lung cancer, thymoma, testicular germ-cell tumor
- Testicular cancer, lung cancer, breast cancer
- Small-cell lung cancer, breast cancer
- Small-cell lung cancer
- Small-cell lung cancer

**Associated here with carcinoid tumour involving thymus**

Daffner et al (2008) NEJM
Focal MTL signal change with amnesia and seizures

LGI1 antibody mediated limbic encephalitis

Pertsov et al (2013) Brain
Long-term hippocampal atrophy in LGI1 ab encephalitis

CA3 atrophy on 7T MRI

Barsch & Butler (2013) Nat Rev Neurol

Miller et al (2017) Brain
Long-term cognitive outcome in LGI1 ab encephalitis

Most prominent deficit is in episodic verbal memory

Butler et al (2017) JNNP
Acute onset amnesia

- **Duration: 1 h to <24 h**
  - MRI: focal DWI lesions in the lateral hippocampus (CA1) 48–72 h after acute phase
  - EEG without pathology
  - Transient global amnesia

- **Duration: <1 h**
  - Frequency: >1 per year
  - MRI normal or chronic lesion in the temporal lobe
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  - Transient epileptic amnesia

- **Memory impairment, focal neurological signs**
  - MRI: DWI lesion with vascular distribution (thalamus, posterior cerebral artery)
  - Stressful, emotional event in the past
  - Strategic insult, ‘amnesic stroke’

- **Situation-specific psychogenic amnesia or global psychogenic amnesia**
  - Knowledge of personal identity is often impaired
  - Remote memory often more strongly affected
  - Anterograde memory is comparatively preserved
  - Possible psychogenic amnesia

- **Headache, fever, delirium, confusion, epileptic fits, focal neurological signs, hallucinations**
  - MRI: abnormalities in temporal lobe, limbic system
  - CSF: HSV-PCR
  - Herpes simplex encephalitis

- **MRI: abnormalities in temporal lobe, limbic system**
  - CSF: pleocytosis
  - Underlying malignancy, onconeural antibody-positive
  - Autoimmune limbic encephalitis

**Barsch & Butler (2013) Nat Rev Neurol**
Confabulation

False memories without conscious knowledge of their falsehood

Often a plausible, but imaginary, recollection of an event or sometimes a grand account of personal life

After orbitofrontal / ventromedial prefrontal cortex damage and in Korsakoff’s syndrome
Wernicke-Korsakoff syndrome

Non-alcohol related causes

Table 2 Precipitating illness in cases of Wernicke-Korsakoff syndrome not related to alcohol

<table>
<thead>
<tr>
<th>Precipitating illness</th>
<th>n (%)</th>
<th>Male</th>
<th>Female</th>
<th>Gender not reported</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal tract disease or surgery</td>
<td>213 (34)</td>
<td>78</td>
<td>135</td>
<td>0</td>
</tr>
<tr>
<td>(Bariatric surgery)</td>
<td>69 (11)</td>
<td>11</td>
<td>58</td>
<td>0</td>
</tr>
<tr>
<td>(Cancer)</td>
<td>54 (9)</td>
<td>29</td>
<td>25</td>
<td>0</td>
</tr>
<tr>
<td>(Obstruction)</td>
<td>25 (4)</td>
<td>12</td>
<td>13</td>
<td>0</td>
</tr>
<tr>
<td>(Pancreatitis)</td>
<td>11 (2)</td>
<td>6</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>(Crohn’s disease)</td>
<td>6 (1)</td>
<td>0</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>(Other)</td>
<td>48 (8)</td>
<td>20</td>
<td>28</td>
<td>0</td>
</tr>
<tr>
<td>Hyperemesis gravidarum</td>
<td>115 (18)</td>
<td>0</td>
<td>115</td>
<td>0</td>
</tr>
<tr>
<td>Dietary insufficiency, starvation or vomiting</td>
<td>106 (17)</td>
<td>59</td>
<td>47</td>
<td>0</td>
</tr>
<tr>
<td>Leukaemia or cancer of lymphoid system</td>
<td>36 (6)</td>
<td>17</td>
<td>19</td>
<td>0</td>
</tr>
<tr>
<td>Intravenous feeding or hyperalimentation</td>
<td>29 (5)</td>
<td>10</td>
<td>11</td>
<td>8</td>
</tr>
<tr>
<td>Psychiatric disorders</td>
<td>13 (2)</td>
<td>4</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>(Schizophrenia spectrum)</td>
<td>7 (1)</td>
<td>4</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>(Anorexia nervosa)</td>
<td>6 (1)</td>
<td>0</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Dialysis</td>
<td>11 (2)</td>
<td>7</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>HIV/AIDS</td>
<td>10 (2)</td>
<td>7</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Other or unspecified</td>
<td>90 (14)</td>
<td>34</td>
<td>55</td>
<td>1</td>
</tr>
</tbody>
</table>
Korsakoff’s syndrome
Amnesia due to thiamine deficiency

Haemorrhage then atrophy of mamillary bodies
### Theories of confabulation

*Propose a disorder of source monitoring or a deficit in strategic retrieval of memories*

<table>
<thead>
<tr>
<th>Temporality account</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confusion over when they were exposed to information</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source monitoring account (not dissimilar to temporality accounts)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inability to distinguish the source of different memories</td>
</tr>
<tr>
<td><em>e.g.</em> “I went on a flight to New York by Concorde last week and then embarked on a cruise to the Caribbean, before dining with the King of Lesotho”</td>
</tr>
<tr>
<td>The patient might have memories of seeing such events on television sometime in the past, even though he never did these himself</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Strategic retrieval hypothesis</th>
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<tbody>
<tr>
<td>Monitoring the quality of retrieved information fails in confabulators</td>
</tr>
</tbody>
</table>
Episodic memory

**Summary 1**

- **Episodic memory** refers to consciously recalled personal experiences and specific events that happened in the past.

- **Retrograde memory** is memory for distant events in the past while **anterograde memory** is memory for newly learnt material.

- The **hippocampus** and associated medial temporal lobe structures play a key role in episodic memory.

- **Confabulation** refers to false memories without conscious knowledge of their falsehood.
Episodic memory

Summary 2

- **Anterograde memory** is often tested by getting the patient to learn a name and address, and asking them to recall it minutes later.

- Alternatively, you can tell them something about your own interests at the beginning of the consultation and ask them to recall these at the end.

- Neuropsychologists test **verbal** anterograde memory with story recall or word list learning. **Visual** anterograde memory is often tested with recall of a complex figure.

- **Retrograde memory** is assessed by asking the patient to recall past personal, news or sport events. It might be necessary to corroborate some of the information.

- More formally, retrograde memory can be tested using the Autobiographical Memory Interview.
Fractionation of Long term memory

Into episodic and semantic memory
Testing semantic knowledge

Pyramid and Palm Tree Test

3 Picture Version

Pyramid
Palm Tree

3 Word Version

Pyramid and Palm Trees Test – which one of the two lower items goes with the upper item?
Testing semantic knowledge

Naming and asking about the use of an object
Ask the subject to repeat:

- 'All that glitters is not gold'

Ask the subject to name the following pictures:

- Using the pictures above, ask the subject to:
  - Point to the one which is associated with the monarchy
  - Point to the one which is a marsupial
  - Point to the one which is found in the Antarctic
  - Point to the one which has a nautical connection
Semantic dementia

Atrophy of left temporal pole
**Semantic dementia**

*Semantic hub in left temporal pole?*

By contrast, the distributed-plus-hub view posits that, in addition to these modality-dependent representations of different aspects of knowledge, there are also 'hub' regions that associate different domains of knowledge (such as objects, actions, and temporal information) and that support a more abstract level of semantic processing. The distributed-plus-hub view differs in at least two respects from the distributed-only view. First, it suggests that, for example, there may be multiple specialized convergence regions: one that encodes associations between shape and object name, and so on. Second, it suggests that abstract away from modality-specific attributes, the neural network for semantic processing relies.

Central function of semantic memory is to generalize across concepts that have similar semantic significance. To some extent, this is apparently organized in a particular way. For example, man-made objects, the zone that links object shape and name, and so on. Similarly, because animals move in characteristic ways, there is presumably a special salience for knowledge of animals. These two central aspects of the convergence-zone hypothesis make it a favorite regarding the neuroanatomical basis for this aspect of the convergence-zone hypothesis make it a favorite regarding the neuroanatomical basis for this aspect of semantic processing.

With a more centralized view, we ask to what extent the resulting conclusions are consistent with, or challenged by, evidence from functional neuroimaging studies of central function of semantic memory. By, evidence from functional neuroimaging studies of central function of semantic memory is widely considered to be consistent with the idea that the cortical semantic network is organized around a single convergence zone.' If so, are they caused by relatively focal brain damage? And, if so, are they caused by relatively focal brain damage? We therefore start with two important empirical questions.

1. Such a generalized impairment. We therefore start with two important empirical questions.
2. By contrast, the distributed-only perspective, no form of focal brain damage would be expected to engender a generalized impairment. We therefore start with two important empirical questions.
Semantic memory

Summary 1

- **Semantic memory** refers to recollection of facts, concepts and general knowledge about the world.
- The **left temporal pole** is considered to be a critical brain region for semantic memory.
Semantic memory

At the bedside semantic memory can be assessed by asking the patient to name objects or line drawings of objects, and then asking them to explain what they are or what they are used for. For example, you might point to a telephone or a watch, or a stethoscope.

Alternatively, give the name of an object and ask them to explain its use.

Neuropsychologists test semantic memory more formally using tasks that probe semantic knowledge.

For example, in the Pyramid and Palm Trees Test patients have to say which of the two choices are closer semantically to the target object.
Reading

*General textbooks*