Neuropsychological evidence: more recent neuropsychological studies, reviewed by Ranganath & Blumenfeld (2005), do not appear so clear cut as earlier studies. When memory of patients with medial temporal lobe damage was tested using novel visual objects, impairment was observed after only a few seconds, suggesting that the patients exhibited a short-term as well as long-term memory deficit. Similarly, when patients with supposedly selective short-term memory deficits were tested using novel words, their short-term recall impairment was found to extend to long-term memory. Ranganath & Blumenfeld argued that the selective memory preservation seen in previous studies may have occurred because simple, over-learned stimuli were used (e.g., digits, words). Perhaps these types of very familiar stimuli become represented in other areas of the brain, resulting in “selective” memory impairments that are more apparent than real.

Neuroimaging evidence: again, evidence is not as clear-cut as previously proposed. Ranganath & D’Esposito (2001) asked participants to actively maintain a novel face in memory for 7s, and then decide whether a second presented face was the same or different to the first. Activation was observed in the medial temporal lobe during the short-term active maintenance period, suggesting that this region may be involved in both short-term and long-term memory.

The episodic buffer:

1) Neither long term memory nor the phonological loop seem sufficient for sentence span, suggesting that another store is needed for this process. (Especially as the visuospatial sketchpad is not suitable and the central executive is an attentional control system with no intrinsic storage capacity.) Baddeley et al. (1987) questioned why memory span for meaningful sentences can be 15-16 words, vastly exceeding normal phonological loop capacity. The traditional explanation is that information from long-term memory is used for “integrating words into meaningful ‘chunks’”. However, Baddeley & Wilson (2002) reported a number of densely amnesic patients, with grossly impaired long-term memory, who nevertheless showed normal immediate memory for passages of prose comprising ~25 words. Also, patient PV had a word span of one word, but a sentence span of 5. These results suggest that memory for meaningful sentences is not reliant on just long term memory or the phonological loop.

2) Considering that the visuospatial sketchpad is poor at serial recall, the effects of articulatory suppression on visual recall are much less than you would expect. Similarly, patients with impaired short term phonological memory have greater recall for visual digits. This suggests that visual and verbal memory must be combined somewhere.

Furthermore, the visuospatial sketchpad is good at storing a single complex pattern but is not suited to serial recall, thus you would expect articulatory suppression to have devastating effects on memory span for visually-presented material. However, Baddeley et al. (1984) noted that articulatory suppression reduces memory span for visually-presented material, but does not eliminate it as the phonological loop model predicts. Furthermore, if such an effect were due to the visuospatial sketchpad, you would expect there to be very large effects of visual similarity, but such effects are actually very small. Moreover, patients with impaired short-term phonological memory, with an auditory span of only 1 digit, can typically recall 4 digits with visual presentation. Chincotta et al. (1999) studied memory span for Arabic numerals and digit words, observing that a numeral advantage effect was eliminated with performance of a concurrent spatial task. These findings
can show selective anterograde and retrograde episodic impairments, sparing semantic memory.

5. On the basis of this evidence, Tulving (1998) concluded that episodic and semantic memory are separate, and proposed a process-specific relationship between memory systems: serial encoding (S), parallel storage (P), independent retrieval (I). The SPI model predicts that preserved semantic memory should be possible in the absence of a functioning episodic memory system, but not vice versa.

6. However, patients with early-stage Semantic Dementia suffer profound degradation of semantic knowledge with intact episodic memory. Thus, Graham et al (2000) proposed that perceptual information might be able to feed directly into episodic memory. To test this, they compared episodic memory in patients with Semantic Dementia for perceptually identical and perceptually different pictures. Episodic memory was normal when the perceptual representation was the same at study and test but was impaired when the perceptual representation could not be relied on. Graham et al. argued that episodic memory typically relies upon multiple inputs from perceptual and semantic systems, and, in the absence of meaningful semantic input, perceptual information alone can be sufficient to support successful episodic memory.

Squire (1992) suggested that long-term memory comprises several separate memory systems.

Explicit memory tasks might include word recognition, cued recall or free recall. Implicit memory tasks might include fragment completion, word stem completion or degraded word naming.

Evidence for a dissociation between explicit and implicit memory:

- Jacoby & Dallas (1981) found that deeper levels of processing improved explicit memory but had little effect on implicit memory.
- Jacoby & Dallas also found that changing modality between study and test had no effect on explicit memory, but significantly reduced implicit memory.
- Graf et al. (1984) found that patients with amnesia were impaired on all the explicit memory tests, but showed no deficit on implicit memory.
- Gabrieli et al. (1995) reported one patient, MS, whose lesion affected occipital cortex. Patient MS performed as well as controls at explicit memory, but was severely impaired on the implicit memory task.

Evidence for the separation of episodic and semantic memory:
Alzheimer’s disease – most common form of neurodegenerative disease, which impacts on medial and later lateral temporal lobes and is associated with profound amnesia, and later semantic and other deficits

Semantic dementia – relatively rare neurodegenerative disease, associated with progressive atrophy of anterior temporal lobe(s) and selective impairment of semantic memory, later spreading to other regions and associated functions.

A number of stroke patients have been reported who exhibited impairments in semantic knowledge only when tested through one modality. For example, McCarthy & Warrington (1988) reported patient TOB, whose deficit was only apparent when tested using words, performing well with pictures. Conversely, Warrington & McCarthy (1994) reported patient DRS, who was very impaired at picture naming, but was accurate at naming from verbal description. These results led these authors and others to propose that there are multiple semantic memory systems in the brain, any one of which can be damaged selectively.

However, Hodges et al. (1995) reported a longitudinal study of naming performance in patient JL, who suffered from semantic dementia, and who demonstrated progressive deterioration of semantic knowledge, with the pattern of his naming errors suggesting gradual “pruning” back of the semantic tree. Progressively worsening deficits are seen on tasks that tap semantic knowledge through all modalities of input and output, with consistency of errors across tasks. Thus, the deficits in semantic dementia seem unlikely to reflect degradation of multiple semantic systems. On the basis of such data, Caramazza et al. (1990) proposed that there is a unitary semantic store, and that the apparent modality-specific effects observed in some stroke patients reflect disruption to one of the access routes to the unitary semantic store. Consistent with this view, Vandenberghe et al. (1996) examined activity in healthy volunteers during performance of semantic memory tasks that used either words or pictures. They found evidence of a common semantic system for words and pictures.

Evidence from semantic dementia indicates that the anterior temporal lobe is the critical area for semantic memory, but some studies of stroke patients suggest that different categories of knowledge may be represented in different brain regions. For example, Warrington & McCarthy (1983) reported patient VER, who was selectively impaired at naming pictures of nonliving things. Conversely, Warrington & Shallice (1984) reported 4 patients who were selectively impaired at tasks tapping semantic knowledge about living things. Some studies reporting category specific semantic deficits have been criticized because the different categories used differed on factors like frequency, familiarity, age of acquisition, etc, but Caramazza & Shelton (1998) controlled for all these factors and still found category specific deficits in patient EW. Warrington & Shallice (1984) proposed that category-specific semantic disorders could be explained by a sensory-functional distinction. According to this view, living things are defined more by their sensory properties (colour, shape, taste, etc), whereas nonliving things are defined more by functional properties (what they are used for).
Psychologists use neuropsychological theories of cognitive function when assessing patients, e.g. models of memory, and are trained to notice how difficulties in different domains of memory present in patients in a clinical setting. Interviews, observations and neuropsychological tests are all used to define the patient’s memory problems. Since specific memory and cognitive problems are associated with specific neurological disorders, once we have a profile of the cognitive problems the patient has, we can make a diagnosis. We also bear in mind that mood and various other factors may also be contributing to the patient’s difficulties, and never use neuropsychological tests alone when making diagnoses or formulating a patient’s problems.

3. The assessment process

The patient is interviewed to find out about the current problems they are experiencing, and their partner, spouse or close friend or relative are also interviewed separately. During these interviews the patient’s level of premorbid functioning (i.e. their expected level of performance) is established, by asking questions about their educational and occupational achievements and by using a test of premorbid functioning, which is usually a reading test (except in patients with language disorders). The patient is carefully observed during the discussion - specific symptoms may become apparent that may represent an organic brain impairment, such as aphasia or memory difficulties. Training makes it easier for psychologists to identify the difference between memory or cognitive problems caused by organic causes, and those caused by psychological distress.

People with episodic memory deficits have problems recollecting recent or remote episodic events, such as the visit to the neurologist who referred them, a recent holiday or current news events or what is happening in a soap opera that they watch avidly. Likewise, they find it difficult to recollect material presented to them after a delay, even if they paid careful attention when they were presented with the stimuli.

On the other hand, people with short-term memory problems describe attentional problems, e.g. walking into a room and forgetting why they are there for, forgetting what someone has just said to them, or forgetting to meet someone somewhere. Such patients tend to describe these incidents in some detail, demonstrating an intact episodic memory. They are poor at encoding information in tests, but the information that they do encode is well-recalled later.

Patients with semantic dementia have lost their semantic knowledge. As a result they may use the wrong object when carrying out a task. They have difficulty word-finding and therefore they may be hesitant or speak slowly and use circumlocutions to describe things (because they cannot recall the words to use). They perform poorly on tests of object naming and semantic associative knowledge, e.g. they cannot tell you that a pyramid is associated with a palm tree, rather than a fir tree.

Once the interview is over, a battery of tests are specifically chosen for the patient, based on their hypothesised deficits. The tests cover a broad range of cognitive functions but tend to focus on the areas of most concern. Tests are carried out across five domains of cognition: memory, attention, visuospatial functioning, language and executive functioning. The patient's performance on standardised tests is compared with their expected performance to look for a discrepancy.

There are many factors that may influence performance:

- Test anxiety, which particularly affects tests of attention and memory