

The hippocampus is the site of memory essay



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As Blakemore (1988) stated, “ without memory, we would be servants of the moment, with nothing but our innate reflexes to help us deal with the world. There could be no language, no art, no science, no culture. Civilisation itself is the distillation of human memory” (p.

277). This quotation rightly emphasises the importance of memory as a human faculty and highlights the importance attached to our understanding of this complex higher function. As pointed out by Mayes & Montaldi (1997), understanding of the human memory system has been revolutionised since the mid 70s by the emergence of brain imaging techniques. Furthermore, much of what is known today has come about from studies of memory failure, or more specifically the amnesic syndrome, which can occur as a function of brain damage, disease, or psychological trauma (Parkin, 2000). This technological revolution has allowed investigators to assess the location, degree, and kind of structural brain damage in patients who show relatively selective memory deficits (Mayes & Montaldi.

997). And, with this ‘ selectivity’ has come a better understanding of how memory cognitively functions and is laid out within the human brain. As Gazzaniga, Ivry and Mangun (1998) remark, it is now known that ‘ amnesia’ (general term meaning any loss of memory) can take on many forms, such as the inability to learn new things or a loss of previous knowledge, which can differ for short-term and long-term memory, including semantic knowledge, episodic knowledge, priming and procedural knowledge. It is also widely accepted that memory can be broken into the stages of encoding, storage and retrieval with memory failure being a possibility at anyone of these stages. This would deductively lead one to the idea that the lay out of

memory within the human brain must be multifarious enough to allow for one form of memory to be damaged without impairing all memory forms.

That is to say, the use of these better neuroradiological systems for measuring the structure and functions of the brain has allowed researchers to construe that memory is a “fantastic faculty with many faces” (Gazzaniga et al. 1998: p. 247). More specifically this goes hand in hand with the now widely accepted modular approach to memory, which views different structures within the brain as highly specialised for different memory functions (Ramachandran, 1998).

This knowledge has thus allowed researchers to appreciate that to declare the hippocampus as the site of memory greatly oversimplifies this higher cognitive function. The ensuing paper will therefore critically examine this statement, presenting the reader with evidence that will seek to rationalise why the hippocampus may have been declared the site of memory but also demonstrating, as Ramachandran (1998) points out, that to understand how memories are retrieved, pigeonholed or censored there is a need to look at how the hippocampus interacts with other brain structures. To begin with, much understanding of how memory functions has been gained from extrapolations of brain damaged individuals. One of the most influential findings to date is Scoville & Milner's (1957) case 'H.

M', who has now undergone over 35 years of study (Kolb ; Whishaw, 1999). H. M. suffered from severe epilepsy, and Scoville ; Milner (1957) described his case justifiable to the “frankly experimental operation” of bilateral medial temporal-lobe resection due to being totally incapacitated by his

seizures, which did not respond to maximum medication of various forms (p. 11).

The operation was considered a success in that H. M's 'incapacitating' seizures were reduced, however H. M. was left with a grave loss of recent memory (now described as anterograde amnesia). Scoville & Milner (1957) concluded, based on the case of H.

M. and 9 other patients who underwent bilateral medial temporal-lobe resection, that the findings pointed to the importance of the hippocampal region for normal memory function. More specifically, Scoville & Milner (1957) suggested that bilateral medial temporal-lobe resection in man resulted in persistent impairment of recent memory whenever the removal was carried far enough posteriorly to damage portions of the anterior hippocampus and hippocampal gyrus, thus inferring that the degree of memory loss was dependent on the extent of the hippocampal removal. It is important to note that, although Scoville & Milner's (1957) conclusions point strongly towards the importance of the hippocampus, only H. M's recent memory was impaired.

That is, subsequent study of H. M. has shown that his deficit in memory effected only his long term memory (e. g.

Wickelgren, 1868). What was disrupted was H. M's transfer of information form short-term to long-term memory. H. M.

had normal short-term memory with digit span abilities of a normal subject. However, unlike normal subjects, H. M. performed badly on digit span tests

where acquisition of new long term memories were required (Parkin, 2000). This supports the current view that the hippocampal region is critical for transfer of short-term memories into long-term ones. Evidence has also shown that other than being important in the formation of new memories (that is, the transfer of short-term memories into long-term ones) the hippocampus is crucial in the consolidation of long term memories (Gazzaniga et al.

, 1998; Thompson, 2000). That is to say, the hippocampal region is critical in the transition of information from short term to long term memory and the solidification of these long term memories (Gazzaniga et al. , 1998). From a biological perspective, Hebb (1949) describes consolidation as the process by which neurones become modified to produce new memories.

Evidence for temporal/hippocampal consolidation comes from patients who have undergone electro convulsive therapy to treat psychological disturbances such as severe depression (Squire ; Slater, 1983) Since the initial analysis of H. M. it has also been shown that amnesia seems to spare information that is based on rules or procedures, as contrasted with information that is data-based or declarative. This supports Squire's (1987) distinction between procedural memory, described as ' knowing how' memory/memory for actions, and declarative memory, described as ' knowing that' memory/memory for specific facts and events (Parkin, 2000). Many experiments have shown that amnesiacs are able to learn and remember certain perceptual-motor skills, including tracking and mirror tracing, frequently at a rate comparable to that of control subjects (e. .

Brooks & Baddeley, 1976; Cohen & Squire, 1978). Cohen & Squire (1980) have noted that this shows amnesiacs' can learn the encoding rules or procedures for acquiring skills, but can remember little or nothing of the information that results from applying them. This goes in favour of a modular view of memory as Cohen ; Squire (1980) proposed that perceptual-motor and pattern-analysing skills belonged to a class of operations governed by rules or procedures, which have memory characteristics different from those operations that depend on specific declarative, data-based material. Additional evidence for a modular set up of memory comes from work with normal/non-amnesic subjects, which has shown a dissociation between kinesthetic-motor memory from spatial and verbal memory on the basis of differences in forgetting rates and in susceptibility to interference (Posner, 1966). A division between semantic (memory of knowledge, facts or meaning without any reference to ' when' it was learnt) and episodic memories (memories for specific places or situations that occurred in the past) has also been shown through work carried out on amnesic patients (Thompson, 2000).

For example, Lhermitte ; Serdaru (1993) carried out tests on three amnesic patients who were submitted to 4 interviews over 12 days on 3 topics concerning places and itineraries that were very familiar to them before the onset of their amnesia. Their retrieved memories were compared to those of normal matched subjects, that is, subjects that would have had as similar as possible memories for the places and events as those of the amnesic patients (Lhermite ; Serdaru, 1993). The results showed a “ dramatic increase for memories of places and itineraries over sessions, and the

absence of improvement of autobiographical memories” (ibid, 1993: p. 1).

Lhermitte ; Serdaru (1993) concluded that the neuropsychological conditions needed for the memory activation for a day in one’s life are different from those for places, unless the often recalled event becomes a familiar story, in which case the autobiographical/episodic memory becomes a semantic memory. These findings support the hypothesis that semantic and episodic memory can be considered as two independent systems that may involve different underlying anatomical structures (Tulving, 1984).

These findings also support the idea that damage to the hippocampal region will not exclude all types of new memory formation as would be inferred if one was to take the view that the hippocampus was the sole site of memory. Furthermore, Gazzaniga et al. (1998) point out that as damage to the medial temporal lobe/hippocampal region does not wipe out most of the episodic and semantic memories formed over a life time it can be deduced that the hippocampus is not the “ repository of stored explicit knowledge” (p. 266).

The evidence thus far has shown the importance of the hippocampus in human memory as well as pointing to the existence of different forms of memory, which are not all controlled by a functional hippocampus. However, in addition to this line of confirmation comes strong evidence against the hippocampus as the sole site of memory from the existence of amnesias that are caused by damage to other regions of the neocortex outside of the medial temporal lobe (Parkin, 1997; Gazzaniga et al. , 1998; Kolb & Whishaw, 1999). For instance, progressive neurological diseases such as Alzheimer’s or herpes simplex encephalitis (involving viral infection of the brain) can cause dense retrograde amnesia, which is due to damage of the lateral cortex of

the anterior pole, involving damage to the entorhinal and perihippocampal cortex (Gazzaniga et al. , 1998).

Furthermore, cases of isolated retrograde amnesia have also been reported in the literature whereby the patients have dense retrograde amnesia but may still form new long-term memories (e. g. Miller, Caine, Harding, Thompson, Large, Watson 2001; DallaBarba, Mantovan, Ferruzza ; Denes 1997). Isolated retrograde amnesia is particularly associated to damage of the anterior temporal lobe, which is important for memory storage but not new memory acquisition (Miller et al. , 2001). Another form of amnesia discussed in the literature, is diencephalic amnesia.

Diencephalic amnesia emerges from damage to midline structures of the diencephalon where the prime structures are the dorsal medial nucleus of the thalamus and the mamillary bodies (Gazzaniga et al. 998). Damage to these midline sub cortical regions can be induced by strokes, tumours, and metabolic problems such as those brought on by Korsakoff's syndrome (Parkin, 1987). The leading area of evidence for this form of amnesia comes from research into Korsakoff's syndrome whereby patients suffer from an anterograde and retrograde amnesia associated with brain damage caused by a dietary deficiency of thiamine (Vitamin B-1), which the body uses to convert carbohydrates into energy.

Over time, thiamine deficiency can cause damage to several brain areas critical for memory including the thalamus, mammillary bodies and basal forebrain: This will, in rare cases, culminate in Korsakoff's disease, commonly caused by chronic alcoholism (Parkin, 1987). Korsakoff's patients have

degenerated diencephalons, although it is not yet known whether the dorsomedial thalamic nucleus, the mamillary bodies, or both are necessary for the patient's amnesia (Gazzaniga et al. , 1998). Nevertheless, damage to the diencephalon does produce amnesia and supports the view that the hippocampus is not the sole site of memory.

At this point it should also be mentioned that there is extensive literature showing the importance of the amygdala in memory for emotions, olfactory and visceral events and of arousal (Kolb & Whishaw, 1999). In a review of the current literature, Sarter and Markowitsch (1985) have shown that studies of animal and human memory show the involvement of the amygdala in memory processes associated with emotionally significant events. Therefore, if the amygdala makes any contribution to the amnesia of patients, it may be emotional in nature. In conclusion it would seem clear that the evidence put forward falsifies the statement that the hippocampus is the site of memory.

Admittedly the hippocampus does play a crucial role in forming new memories. However it has also been shown that other areas, such as the diencephalon, the perirhinal cortex, the amygdala and the basal forebrain also play critical roles in the functioning of human memory. It would thus seem more appropriate to declare that the hippocampus plays a crucial role in human memory as opposed to labelling it the ' sole' site of memory. This discussion has also shed light onto the complexity of human memory and human cognitive functioning in general. Therefore, as Ramachandran (1998) appropriately stipulates, the logical conclusion to be drawn from studies of amnesia is not that memories are actually stored in the hippocampus (as old

memories are preserved), but that the hippocampus is vital for the acquisition of new memory traces in the brain.