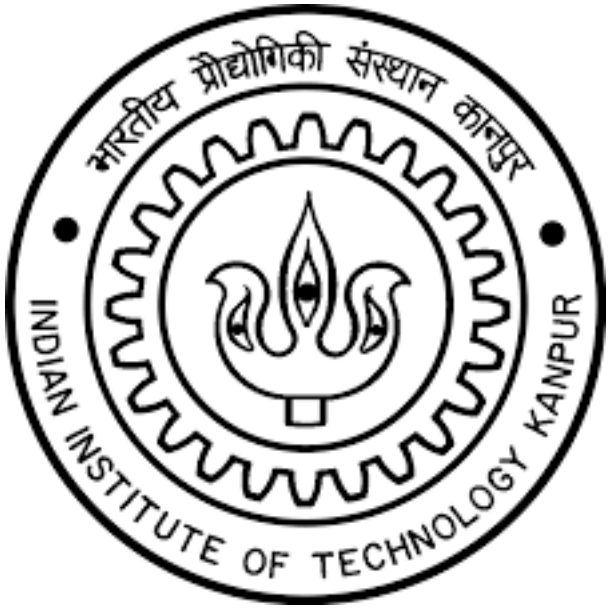

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Presents

Course Title:

Basic Cognitive Processes

By: Dr. Ark Verma,
Assistant Professor of Psychology,
Department of Humanities & Social Sciences,
IIT Kanpur

Lecture 38: Disorders of Memory

AMNESIA

- Amnesia is the name given to disorders of memory.
  - Normally involves forgetfulness, which goes beyond the everyday forgetting observed in normal people; to the extent that it may interfere with the activities of normal life.
  - A person suffering from amnesia may be quite unable to remember any recent events like the happenings of the current day or previous minutes, hours etc.
  - Amnesia therefore is a very disabling condition.

Causes of Amnesia

- Amnesias may arise from a number of different causes, which can be divided into two main groups:
  - **Organic Amnesias:** caused by some form of physical damage inflicted on the brain. For e.g. brain infections, strokes, head injuries, & degenerative disorders such as Alzheimer's disease.
  - Organic amnesias tend to be severe and disabling, & they are also irreversible in the majority of the cases because the brain lesions usually do not heal.

- **Psychogenic Amnesias** are caused by pychological factors and usually involve the temporary suppression of disturbing memories which are unacceptable to the patient at some subconscious level.

- Psychogenic amnesias can be disorientating & disruptive to the patient, but they are rarely completely disabling, & as there is no actual brain damage they are reversible and in most cases will eventually disappear.

- Four kinds of organic amnesias:
  - **Alzheimer's Disease (AD):** is a degenerative brain disorder which first appears as an impairment of memory but later develops into a more general dementia, affecting all aspects of cognition.
  - AD happens mostly in the senile population, affecting upto about 20% of the elderly.
  - AD is a basic neuro –degenerative disorder which involves tangling of neuronal fibres, something which has been shown to be responsible for other kinds of senile dementia as well.
  - However, because of the overlap with symptoms of general dementia, AD is not considered to be a pure form of amnesia & it becomes rather difficult to investigate the nature of memory damage in AD.

- **Korsakoff Syndrome:** is a brain disorder resulting usually from excessive alcoholism & being characterized by a memory impairment that affects both recent & past memories. First reported by Korsakoff (1987), it is one of the most studied forms of amnesia as it is not marred by additional complications of extensive dementia or retardation.

- **Hepres simplex encephalitis (HSE)** is a viral infection of the brain which can lead to severe amnesia. It has a relatively sudden onset as compared to the slow increase of degenerative diseases like dementias & AD.

- **Temporal Lobe Surgery:** a small number of patients become amnesic because of brain lesions caused by surgical procedures. Usually temporal love surgery can lead to amnesia. An example of the case was H.M. a amensia patient post surgical treatment for epilepsy.

- **Post – ECT amnesia:** Sometimes the use of electroconvulsive therapy for alleviating depression in patients ,ay also lead to amnesia. ECT involves administering electric shocks to the front of the patient's head. Patients experience periods of amnesia post the electric shock which may continue for longer periods as well. This has been extensively studied in order to evaluate the usefulness of the treatment & as a side – effect.

Amnesia: Long Term Memory

- One of the symptoms of the organic amnesic syndrome is the impairment of the Long Term Memory; i.e. organic amnesics will have difficulty in consolidating new information into their long term memory store, & also they often have problems retrieving old memories from storage.
- Interestingly, organic amnesics generally have an intact short term memory and are generally able to carry on a normal conversation, only unable to recall past events but able to talk about current things.

- Talland (1965) acrried out a study involving 29 Korsakoff patients, all significantly impaired on a whole battery of long term memory; but found that their scores on tests of short term memory were close to that of normal individuals.

- Others like Baddeley & Warrington (1970) also reporte dnormal STM span in Korsakoff patients; which led Pujol & Kopelman (2003) conclude that Korsakoff's patients showed normal performance on tests of both verbal & non −verbal STM.

Anterograde & Retrograde Amnesia

- AA: involves impairment of memory for events occurring since the onset of amnesia.
- RA: involves impairment of memory for events occurring before the onset of amnesia.

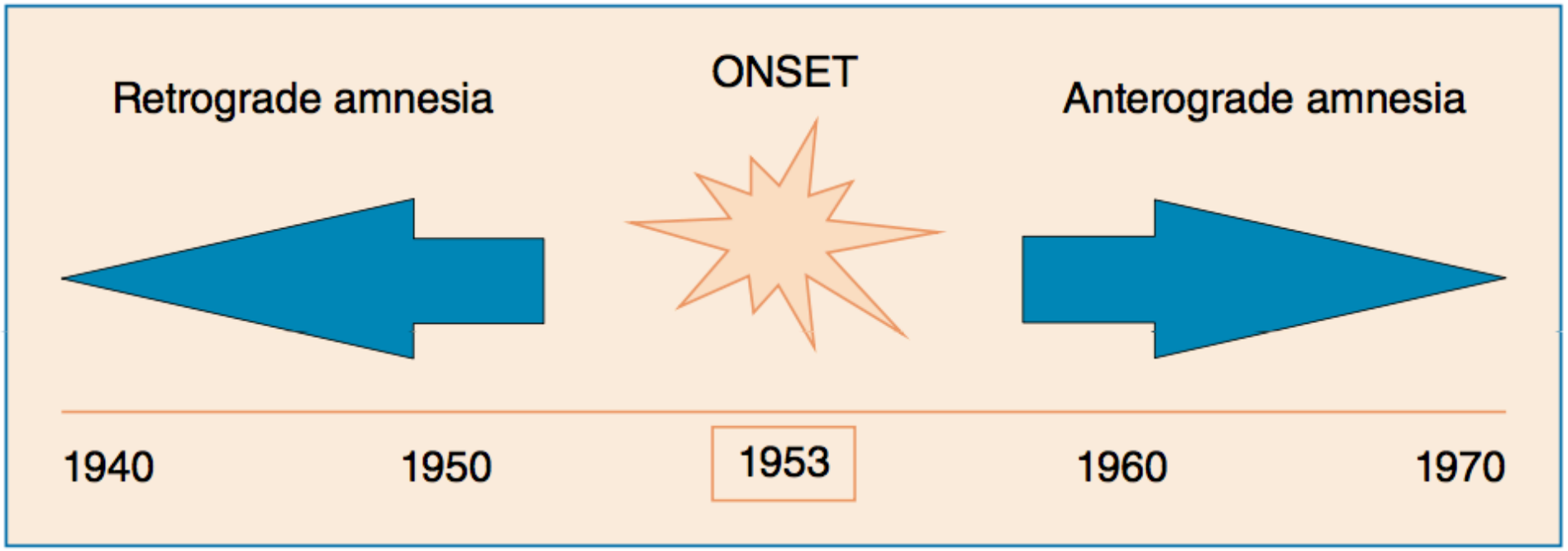

Figure 7.2 Anterograde and retrograde amnesia shown in relation to the moment of onset (in this case for patient HM).

Image: Groome et al., (2013). An Introduction to Cognitive Psychology: Processes & Disorders. *Psychology Press.* (Fig. 7.2; pp. 211).

- This distinction between AA & RA provides a way of distinguishing the locus of amnesia as either a learning or a retrieval disorder.
  - Wherein AA can be more indicative of a learning disorder while RA can be used to indicate a retrieval disorder. Obviously it is possible for a patient to have both the disorders; in which case both AA & RA will be observed.
  - An interesting observation was made by Ribot (1882) who concluded that RA of amnesic patients showed a temporal gradient where the degree of impairment with the recency of the event & childhood or most remote memories are somewhat spared. This has been referred to as ***Ribot's Law.***

Testing Retrograde & Anterograde Amnesia

- As AA is more of a learning disorder, testing is easier; as the patients can be simply given the task of learning new materials like words, stories, pictures etc. and then tested for the retrieval at a later time.

- Testing for RA, however is slightly complicated as testing has to be done for events & materials unknown to the tester.

- Tests for remote memory therefore can either involve testing of past public events or the test of past personal events which only the patient or close ones might know of.

  - Test of past public events is slightly easier as the events are known to a wide variety of people and similar test items can be given to many different people and hence it is possible to devise a standardized test with well specified performance norms. For e.g. the Boston Remote Memory Test (Albert et al., 1979)

  - Test of past personal events have test items pertaining to autobiographical information about the patient. However because this information can vary widely from individual to individual, the scores of these test are not easily comparable. Also it is difficult to check for the accuracy of the given responses.

Anterograde & Retrograde Impairment in Organic Amnesia

- Both Ribot (1882) & Korsakoff (1887) suggested that most amnesic patients have both AA & RA.
    - AA together with RA has been observed in dementing AD patients (wilson et al., 1981) & Korsakoff patients (Albert et al., 1979); also in patients with HSE.
    - Pujol & Koppelman (2003) have reported that in RA in Korsakoff patients can extend back to a period of 30 years or more before onset & shows a marked temporal gradient.

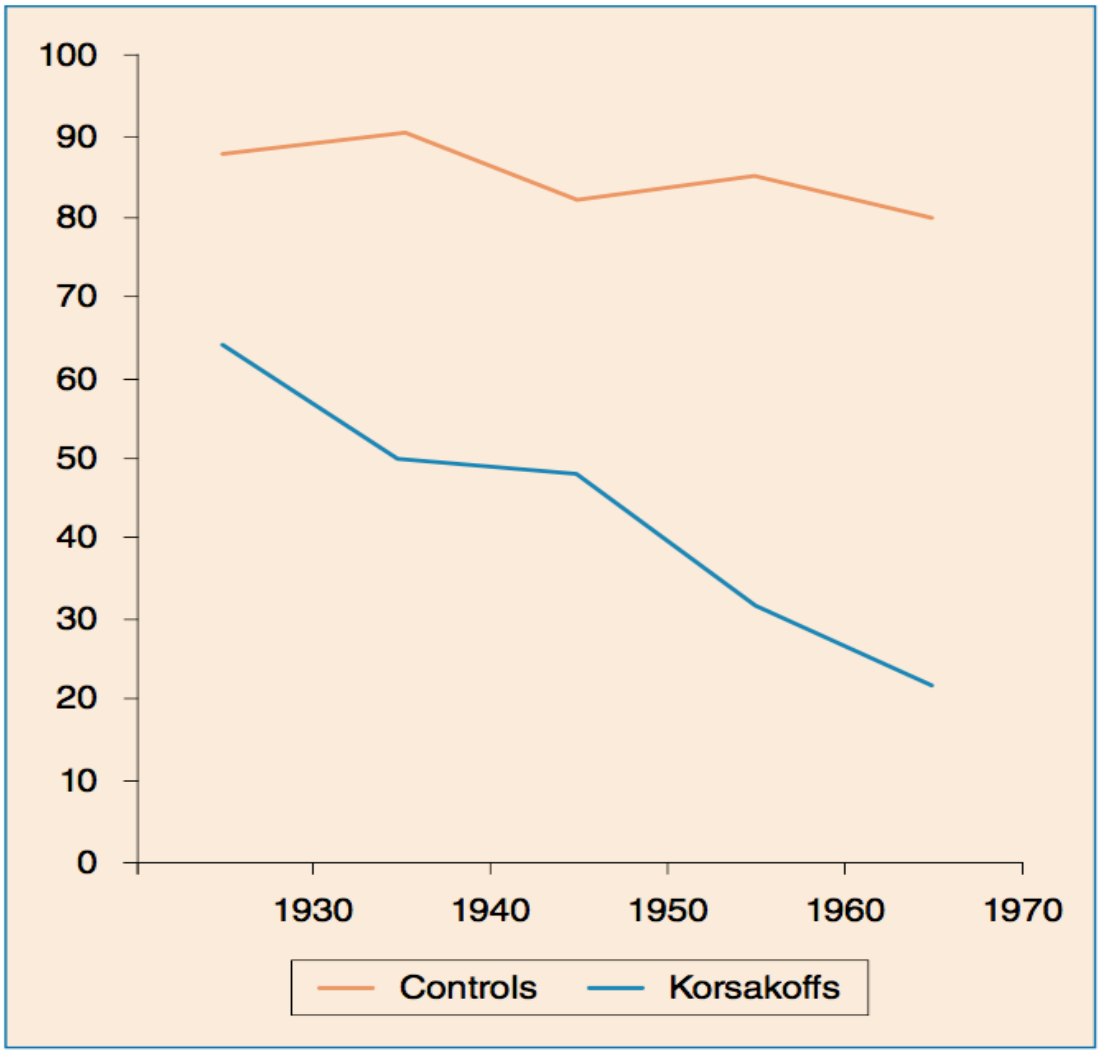

Figure 7.3 Memory performance for different periods from the past.

Source: Albert *et al.* (1979).

Image: Groome et al., (2013). An Introduction to Cognitive Psychology: Processes and Disorders. *Psychology Press.* (Fig. 7.3; pp. 213).

- However, the said pattern is not found everywhere, i.e. there could be some patients who show severe AA but have very limited RA. For e.g. H.M. had severe AA, but RA was only up to about 3 years before the onset.

- **FOCAL RA & AA**:

- few cases of focal AA (without RA) have been reported.
  - For e.g. Mair et al. (1979) studied two Korsakoff patients who had focal AA & Cohen and Squire (1981) also studied NA, a patient who had focal AA after an accident. NA's injury & few other studies have confirmed that damage in focal AA could be mostly restricted to the anterior thalamus (Kapur et al., 1996).
- focal RA cases are usually rare, but some patients with severe head injuries (Kapur et al., 1992), Hse infection (O'Connor et al., 1992) & following epileptic seizures (Sehm et al., 2011) have been reported to have focal RA.

- Focal RA has been associated with lesions to various brain areas, but most commonly it tends to involve lesions in the temporal cortex (Sehm et al., 2011).

- About the temporal gradient in RA:
  - Ribot (1882) reported that amnesics tend to retrieve older memories better than recent memories. The same has been confirmed in more recent studies (Brown, 2002).
  - A possible explanation is put forward by Squire (1982) is that older memories may be more durable because they have developed various retrieval routes over the period of years.
    - Squire (1992) suggest that the consolidation of a new memory may take several years to complete, and that it becomes increasingly resilient over time. - *standard model of consolidation.*
    - Moscovitch et al. (1999) proposes that memories recent events are more vulnerable as they are still held as individual episodic memories. These episodic memories combine to produce a semantic memory which is more lasting and does not depend on hippocampal activity with time.

- **Brain Lesions with AA & RA**
  - Several of the brain's regions have been implicated in cases of organic amnesia. To name a few, *temporal lobes, hippocampus, thalamus & the prefrontal lobes.*

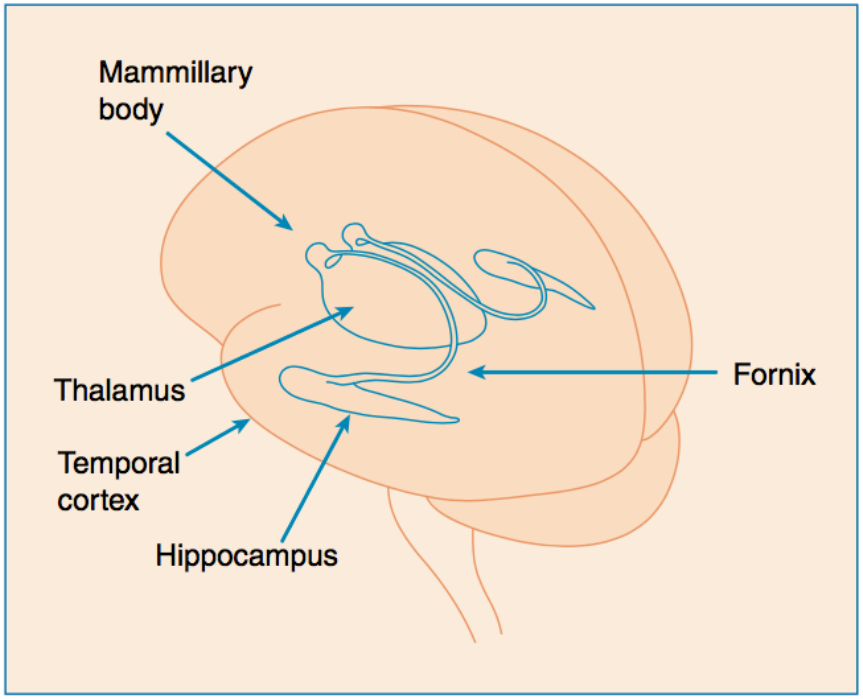

Figure 7.4 Brain structures involved in memory storage and consolidation.

Image: Groome et al., (2013). An Introduction to Cognitive Psychology: Processes & Disorders. *Psychology Press.* (Fig. 7.4; pp. 216).

- The temporal lobes contain the hippocampus, & this structure is of particular importance to the creation of new memories.
    - Surgical removal of the hippocampus an dparts of the medial temporal lobes of the patient HM was found to have a devastating effect on his memory, especially his ability to acquire and consolidate new memories (Scoville & Milner, 1957).
    - The temporal lobes & the hippocampus are aso damaged in case of HSE, AD. Though the lesions may get more extensive and cover right from the majority of the temporal cortex but the frontal brain regions as well.

- other areas of lesions could be the *diecephalon a region including the thalamus & mammilary bodies*. These areas are damaged in Korsakoff Syndrome patients (Victor et al., 1989; Reed et al., 2003) along with lesions in the anterior thalamic nuclei (Harding et al., 2000).

- Retrieval of past memories may involve different regions of the brain, most notably the *temporal & prefrontal cortex*.
  - Korsakoff patients have been shown to have retrieval problems associated with prefrontal lesions.
  - Koppelman (2001) established that in Korsakoff syndrom the severity of AA is related to thalamic damage, whereas the severity of AA is correlated with the extent of prefrontal damage.

- Finally, lesions in the temporal cortex have also found to be linked with retrieval problems (Reed & Squire, 1998),

- HSE patients whose lesions extend beyond the hippocampus to include large areas of the temporal cortex are usually found to exhibit severe RA in addition to their dense AA (Cermak & O' Connor, 1983).

- Stefanacci et al. (2001) reported that in HSE patients AA correlates with extent of hippocampal lesions whilst the RA extent correlates with lateral temporal lobe lesions.

Intact & Impaired Memory Systems

- **Motor Skills**: there is considerable evidence that motor skills are preserved in organic amnesics. Amnesics retain their previously learned motor skills & can also learn new skills post onset of amnesia. (remember H.M. learned mirror drawing).
  - HSE patient Clive W. retained most of his piano skills (both in conducting & playing); but is unaware of them. Another patient PQ also retained his ability to play the piano & also learn to play new pieces.

- Glisky et al. (1986) reported that amnesics could be successfully trained to carry out simple computer tasks withh time & patience; however there was little or no generalization of the learning observed.

- Cavaco et al., (2004) reported normal performance of 10 amnesic patients with temporal lesions caused by HSE on a range of motor skills including weaving, figure – tracing & target tracking.

- With these results in mind Cohen & Squire (1980) suggested that amnesics might have a preserved procedural memory but an impaired declarative memory.

- **Implicit Memory**: it has been shown that amnesics might still retain aspects of implicit memory like priming/conditioning wherein their behaviour may be influenced by previous experience, though they may not remember the specific experience.
  - E.g. Claparede (1911) pin – handshake experiment.
    - Warrington & Weiskrantz (1968) showed Korsakoff patients degraded pictures of common objects or words; starting with the most incomplete to the complete version & demonstrated that the Korsakoff patients exhibited an increase in their ability to identify the object after a few trials.
    - Graf et al., (1984) used the primiing of verbal material to demonstrate intact implicit memory in Korsakoff patients, in a task where subjects were presented with word fragments and asked to complete them. It was found that the patients responded with previously primed words.

- **Familiarity & Context based recollection**: Mandler (1980) suggested that familiarity & recollection represent two alternative routes to recognition.

  - Mandler believed tat a familiarity judgment is an automatic process, occurring without any conscious effort whereas recollection, is a controlled process requiring conscious effort and is deliberately carried out.

  - Studies have shown that organic amnesics retain the ability to detect familiarity of a previously encountered item, though they may find it difficult recollecting the context. For e.g. Clive W found his old friends familiar but could not locate them in memory.

- Huppert & Piercy (1976) devised an experimental procedure wherein he showed Korsakoff patients and Controls two sets of pictures, first set on day 1 & the second set on day 2.
  - Shortly after the presentation of the second set, the subjects were tested for their ability to judge whether particular pictures were familiar (previously presented /new).
  - Also, after the above they were asked to identify which pictures were shown on day 1 & which on day 2.

  - While the performance of the amnesics & control participants was equivalent on familiarity judgment; amnesics could not do well at all in the task where they had to identify the pictures presented on specific days.

- Huppert & Piercy (1978) further demonstrated that the recognition performance of the Korsakoff patients was mainly based on judgment of the general familiarity of pictures. The same has been confirmed in other studies as well (King et al., 2004; Gardiner et al., 2008).

- **Episodic & Semantic Memory**: ?
  - Episodic memory: memory for specific events in our lives
  - Semantic memory: store of knowledge acquired over time.

  - Tulving (1989) suggested that amnesics exhibit a selective impairment of episodic memory while their semantic memory stays intact.
    - General observation points out that indeed, amnesics usually retain a normal vocabulary despite their inability to remember any recent events in their lives.
    - However, other studies show that in some cases the semantic memory may be jeopardised too. For e.g. studies of Korsakoff patients have been shown to have severely impaired episodic memory along with semantic memory impairments.

- Further, Alzheimer patients are also generally found to show impairments of both episodic & semantic memory; though the former is more severely impaired.
  - Addis & Tippet (2004) reported that Alzheimer patients tend to suffer impaired autobiographical memory extending back over their entire lifespan, but theyalso exhibit limited semantic memory impairment.
- Overall, it can be said that both semantic & episodic memories are affected in most organic amnesics; leaving less support for Tulving's proposal.
  - Though at least a couple of known patients (K.C.; & Jon) were found to have almost no episodic memory (termed episodic amnesia) but relatively normal semantic memory (before amnesic onset).

- **Theories of Amnesia:**
- Encoding Deficit Theories:
  - Milner (1966) argues that HM's impairment was essentially a failure to learn new information, i.e. he was unable to consolidate memories from a temporary STM trace to a permanent LTM trace.
  - Similar explanations could also be thought to account for deficits such as those in the Korsakoff Syndrome. Basically, it could be argued that the apprent occurrence of RA in K patients might actually be an anterograde impairment that could not be detected in time & continued deteriorating.
  - However, the theory cannot explain the occurrence of RA. For e.g. in case of HSE patients having genuine RA with the precise date of onset.

- Retrieval Deficit Theories: Warrington & Weiskrantz (1970) proposed retrieval impairment as the basic deficit underlying organic amnesia.

  - A retrieval impairment could explain both the anterograde & retrograde components of amnesia, as it would explain a failure of the retrieval mechanism of both past & recent memories.
  - But, a retrieval based theory would predict equally severe AA & RA; though we know that amnesic patients suffer far more severe AA than RA, indicating that past memories are somehow more durable.
  - The retrieval based account can explain the temporal gradient in RA, by proposing that the earliest memories would be the most rehearsed as compared to more recent memories (Squire et al., 1984).

- However, the account cannot readily explain the variations in the severity of AA & RA between different patients and also why some amnesics have virtually no RA at all.

- Also, the account cannot explain how AA & RA impairments can sometimes occur in isolation (as in focal AA & RA).

- Separate Impairments of Encoding & Retrieval
  - As either of the accounts cannot explain the deficits in amnesia alone, it has been suggested that impairments of encoding & retrieval are independent of one another (Parkin, 1996).
  - The finding that impairments of learning & retrieval are associated with lesions in different areas of the brain lend support to this view.
    - It has been suggested that amnesics may exhibit both AA & RA because the brain regions mainly involved in eencoding & retrieval of information are physically qute close to each other and are extensively interconnected.
- In all, it can be concluded that most amnesics suffer from both AA & RA, though their relative severity may vary from patient to patient.

- The Standard Model of Consolidation
  - Squire (1992) that in addition to the STM consolidation process, a slower form of consolidation continues to strengthen the memory traces for two to tree years after its acquisition.
  - The trace therefore remains vulnerable for few years post input, until fully consolidated.
  - Squire proposes that this long – term consolidation process involves the hippocampus, which plays a role in the initial encoding of the new information as well it's consolidation over the period of few years.
  - Hence, the relatively limited RA in patients such as HM may result from the disruption of the slow consolidation process post hippocampus removal.

- Multiple Trace Theory
  - Moscovitch et al., (1999) suggests that each time an item is retrieved from memory, it creates a fresh memory trace & forms new connections.
    - In the years following the acquisition of a new memory trace; this process causes episodic memories to be bound together to create semantic memories.
    - The binding is assumed to be carried out in the hippocampus, but once the semantic memory is formed it becomes independent of the hippocampus.
    - The retrieval of the episodic memory however will always require the hippocampus.
  - This theory can explain the finding that hippocampal lesions disrupt episodic memories from all time periods, but only disrupt the most recently acquired semantic memories. Supporting evidence (Hm, WR). (Steinvorth et al., 2005).

- Other Impacts of Amnesia
    - *Impaired Declarative Memory*: Squire argues that organic amnesia is chiefly characterised by an impairment of declarative memory. Mandler (1989) argues that amnesia is essentially 'a disease of consciousness', since the memory functions which are damaged are those which require conscious retrieval.

- *Impaired Binding*: Cohen & Eichenbaum (1993) argue that the main feature of declarative memory is that it involves the creation of associative connections between memories – binding. In contrast, non – declarative memory seems to be restricted to the strengthening of a single response.
- Eichenbaum (2004) suggests that the hippocampus performs the associative binding function of declarative memory, whereas non – declarative memory involves the cortex & the cerebellum.
- This view has been supported by some imaging studies, for e.g. Rosenbaum et al., (1999) found that the amnesic patient KC (having hippocampal lesions) had severe retrograde amnesia for episodic memories & some autobiographical memories, but could still retrieve semantic memories before onset. They concluded that the main function of the hippocampus is to bind memories together &no just store information.

- *Impaired Perceptual Processing*: While the function of hippocampus is largely assumed to involve memory storage, animal studies have also suggested it's role in visual perception (Gaffan et al., 2001).

  - Graham et al (2008) have presented some evidence which suggests that the hippocampus may also have a perception function in humans leading to some of their troubles with memory in amnesic patients.

  - Graham et al., suggest that memory storage involves a network of perceptual representations which are distributed throughout the cortex & are controlled & activated by the hippocampus.

- Damage to the hippocampus would therefore impair the retrieval of old memories as well as the processing of new input.
- One problem of this theory is that studies of amnesic have mostly failed to identify a amajor perceptual impairment. For e.g. Hartley et al., (2007) tested four amnesics with focal hippocampal lesions & only two of them showed any perceptual impairment. Interestingly though, all four individuals were found deficient on spatial memory.
- Similarly, Lee et al., (2012) found evidence for the involvement of the hippocampus in the visual discrimination of complex scene stimuli.

Other Types of Memory Disorders

- *Impairment of Short Term Memory*:
  - Impairments of STM occur sometimes, but involve a quite different pattern of brain lesions to those found in typical organic amnesia.
  - AD patients may also show severe WM impairments, chiefly involving executive function (Storandt, 2008).

- *Concussion Amnesia*
  - People who suffer from a concussion on the head due to an accident or injury usually suffer from both RA & AA; whicch could be extensive in the beginning but diminshes slowly.
    - A footballer concussed by a collision on – field might find it difficult to remember events immediately preceding the collision.
    - Amnesias of the kind are known as ***concussion amnesia*** and fall into the category of post – traumatic amnesias, which includes all kind of closed – head injury.
  - Russel (1971) surveyed a large number of concussion victims to find that typically retrograde amnesia affected memories for a period extending only to a minute or two prior to the accident; but in rare cases could upto days or weeks.

- Characteristics of concussion amnesia resemble those of organic amnesia; for e.g. for the period immediately following the accident the patients is likely to show impairment on LTM tasks, but do ok on WM tasks.

- Although the effects of a concussion on memory are usually temporary, a minority of mild to moderate head injuries may leave a more lasting impairment – post concussive syndrome.

- *ECT & Memory Loss*:
  - While ECT has been shown to be useful in treating depression in some patients, these benefits are only temporary & therefore must be compared with possibilities of lasting brain damage caused by ECT.
  - In the period following an ECT shock, the patient typically shows both AA & RA (Squire et al., 1981), which might seem extensive initially but fades off gradually to leave only a fairly limited amnesia for the treatment period.
  - It seems however, that for most patients ECT caused only a limited impairment (Weeks et al., 1980; Meeter et al., 2011).

- A recent review of previous ECT studies (Read & Bentall, 2010) concluded that ECT treatment produces no lasting benefits but it does cause significant memory loss in some patients.

- Overall, a growing number of researchers argue that the limited benefits of ECT cannot be justified in view of its damaging effects on memory & cognition (Johnstone, 2003; Read & Bentall, 2010).

- *Frontal Lobe Lesions*

  - Patients with frontal lobe lesions often show some impairment of memory, though these tend to be rather different in nature to those associated with temporal lobe or thalamic lesions & then mainly seem to involve impaired retrieval.

  - More specifically, patients with frontal lobe lesions tend to have particular difficulty in retrieving contextual information (Parkin et al., 1995).
    - Possibly because they have an impairment of the central executive component of their working memory (Shallice, 1988).

- Another characteristic of patients with frontal obe lesions is a tendency to confabulation (Moscovitch, 1989), implying that the patient describes memories for events which did not really take place and are apparently invented.
  - Confabulation is also associated with impaired executive function & with the consequent loss of mental flexibility (Nys et al., 2004).
- Also, it must be noted that frontal lobe lesions may sometime co – exist with other types of lesion. For e.g. many Korsakoff patients have been found to have frontal lobe lesions along with diencephalic lesions (Shimamura et al., 1988) & these individuals often exhibit a marked tendency to confabulation and retrieval problems in addition to the more usual amnesic symptoms found in Korsakoff patients.

- *Memory Loss in the Normal Elderly*
  - There is some evidence for age – related decline in memory, though not readily detectable until the age of about 65 – 70.
    - The degree of impairments is usually not very great (Verhaeghen, 2011).
  - Studies have indicated that the norma elderly may show a decline in recall ability but not in recognition (craik & McDowd, 1987).
  - Elderly subjects tend to show a a deterioration of explicit memory; though their implicit memory remains unimpaired (Fleischman et al., 2004); seem to have problems in retrieving contextual information (temporal context, Parkin et al., 1995).

- Parkin & Walter (1992) demonstrated that elderly people were able to rcognise a familiar item but had poor recall of context.

- Interestingly, the amount of decline in context recognition correlated with measures of frontal lobe impairment (Tisserand & Jolles, 2003).

- A possible explanation of age – related memory decline may be that the elderly lose some their capacity for consciously controlled processing and attention & have to rely more on an automatic process (Craik & McDowd, 1987). Further, they also show decline in speed of processing (Salthouse, 1994).

- All in all, a recent meta – analysis concludes that the actual decline in executive function in the normal elderly is very slight (Verhaeghen, 2011).

- *Psychogenic Amnesia*
  - Some amnesias may occur without any evidence of brain lesions & maybe brought on by stress and are usually temporary (Khilstrom & Schachter, 2000).
  - These included loss of memory for past events, in other words retrogrde amnesia; anterograde amnesia is fairly unusual (Kopelman, 1995).
  - The pattern of impairment in psychogenic amnesias varies widely, from case to case.
  - Kopelman (2010) points out that psychogenic amnesias may be 'global' or 'situation specific'.

- The main feature which enables clinicians to distinguish between psychogenic & organic amnesias is that those of psychogenic origin do not usually match up with usual pattern of impairment found in organic amnesics.

Rehabilitation

- There are a number of ways in which the lives of organic amnesics can be significantly improved by helping them to cope with their impairment and helping them to function as effectively as possible within the limitations of their condition. - *Rehabilitation*

- *Maximising Memory Performance*

  - It might be useful to advise the amnesic patients to pay more attention to the input, to repeat what is said to them, to organize information & make meaningful associations between new input and the items already there in the memory.

  - Wilson (2004) suggests that amnesics learn one thing at a time, keep the input simple & avoid jargon or long words. Also they would perform better if their learning is not context – specific. For e.g. training imparted does not generalize to others unless specifically trained.

  - Amnesics also generally benefit through 'spaced' rather than 'massed' learning sessions.

- *External Memory Aids*

  - An option is to change the immediate environment & living conditions of the amnesic patients so as to reduce the dependence on memory. For e.g. by putting on big labels on cupboards, or labelling doors as a reminder of which is the kitchen or toilet.

  - Electronic or computerised aid system also have been devised to help out the amnesics by producing reminders to carry out a particular action at a particular time, by emitting a warning beep or alarm. An example was 'Neuropage' (Hersh and Treadgold, 1994).

- Another interesting device was the ‘Sensecam’ which makes use of digital camera attached to the user’s belt to take photos of events experienced each day in order to provide a reminder which can be viewed later on.

  - Loveday & Conway (2011) reported that Sensecam can significantly enhancee an amnesic’s personal memories.

  - Reviewing events recorded on Sensecam at te end of each day also helps to improve the vividness of recall, & helps to add context to an event. For e.g. by reminding the amnesic patient of the things they were doing & thinking, & their interactions with other people etc.

References

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