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Neuropsychological Generation of Source Amnesia: An Episodic Memory Disorder of the Frontal Brain

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Abstract

Source amnesia is an explicit memory (declarative) disorder, particularly episodic, where source or contextual information concerning facts is severely distorted and/or unable to be recalled. This paper reviews the literature on source amnesia, including memory distrust syndrome, and its accepted correlation with the medial diencephalic system and the temporal lobes, and the suggested linkage between the frontal lobes, including special interest with the prefrontal cortex. Posthypnotic induction was the first presentation of source amnesia identified in the literature. The Wisconsin Cart Sorting Test (WCST), Positron Emission Topography (PET), Phonemic Verbal Fluency Test, Stroop Color Word Interference Test, and explicit and implicit memory tests are defined and linked to empirical research on amnesiacs.

Introduction

One often remembers factual information yet forgets the contextual information related to the fact (i.e., when, where, and with whom the fact was learned). In the case of Jon, recollection of such contextual information was nearly impossible. At the age of 23, although born premature, Jon ponders the nature of his apparent ailment. Neuropathological findings suggest that one of Jon's brain centers is severely retarded, impairing one route of the human memory course - namely episodic memory (Baddeley, Vargha-Khadem et al. 2001). Such an occurrence represents a loss of source or contextual memory, a phenomenon referred to as source amnesia (Evans and Thorn 1966; Schacter, Harbluk et al. 1984). Research conducted in the past two decades has considerably advanced our comprehension of the human memory process, and consequently improved our understanding of the complex mechanisms of source amnesia.

However, until recently, most explanations for amnesia assumed that the condition was undivided and had a solitary functional deficit. Using various neurological imaging and executive function tests only available in the recent past, researchers now suggest that the frontal region of the brain, particularly the prefrontal cortex of the frontal lobes and the medial diencephalic system, is highly attributable to the storage and retrieval of contextual details, but plays a minor role in fact recollection. Therefore, the brain's functional systems for memory for facts and memory for contexts may be dissociated. In addition, the deterioration of these neuroanatomical organic structures due to "normal" aging may contribute to the degradation of contextual scanning ability, as well as information encoding and retrieval efficiency. Likewise, in subject populations where the anatomical structures are not fully mature (i.e., very young children), amnesic symptoms should be profound.

The purpose of this paper is to survey and synthesize the rich research on source amnesia gathered from the 1950s to the present, with a particular emphasis on the suspected and identified neurological centers associated with source memory. Source amnesia genesis and presentation in the posthypnotic environment is also of interest because the discovery of source amnesia's symptom of forgetting context-specific information first occurred in subjects under hypnosis. Finally, the common assessment tools are discussed, including neuroimaging techniques, implicit and explicit memory tests, the Wisconsin Card Sorting Test, Phonemic Verbal Fluency Test and Stroop Color Word Interference Test for prefrontal assessment.

Human Memory Process

Cellular and molecular studies of both implicit and explicit memory suggest that experience dependent modulation of synaptic strength and structure is a fundamental mechanism by which implicit and explicit memories are encoded and stored within the brain. Before discussing human memory concepts, it is worthwhile going in brief about memory formation in sensory neurons of Aplysia (snail) and hippocampal neurons of mouse. The eleven critical cellular and molecular mechanisms of memory storage identified are

1. Neurotransmitter release and short term strengthening of synaptic connections
2. Equilibrium between kinase and phosphatase activities at the synapse
3. Retrograde transport from the synapse to the nucleus
4. Activation of nuclear transcription factors
5. Activity dependent induction of gene expression
6. Chromatin alteration and epigenetic changes in gene expression
7. Synaptic capture of newly synthesized gene products
8. Local protein synthesis at active synapses
9. Synaptic growth and the formation of new synapses
10. Activation of pre existing silent synapses and
11. Self perpetuating mechanisms and the molecular basis of memory persistence

The location of the above events moves from the synapse (1-2) to the nucleus (3-6) and then back to the synapse (7-11).

There is great interest in how the brain can maintain the persistent neural activity encoding recent stimuli that is thought to be the basis of working memory (Fuster, 1988). Several theoretical mechanisms for the maintenance of persistent activity have been described (Durstewitz et al., 2000), including local recurrent feedback and intrinsic persistent activity on a single-cell basis. Recurrent excitation at the local circuit level has received the most attention, from Hopfield models through elaborated conductance unit networks (Durstewitz et al., 2000; Wang, 2001). Wilson and Cowan suggested that meaningful insight into the behavior of neural ensembles might be gained by a mean field approach describing the average rate of firing over some coherent population (Wilson and Cowan, 1972). Averaging on a local scale in combination with lateral connectivity described by a spatial kernel with wider support leads to a continuum, integrodifferential description of spatiotemporal neural activity (Wilson and Cowan, 1973). At the same time, there is now renewed interest in the concept that individual neurons might have some inherent ability to sustain persistent activity without recurrence. The remarkable finding that individual entorhinal cortical neurons can sustain graded persistent activity (Egorov et al., 2002) is the perhaps most striking example to date. At the intersection of these ideas is a model by Camperi and Wang (C-W) which explores the idea of bistability in individual neurons within an Amari-type (Amari, 1977) integrodifferential network model (Camperi andWang, 1998).

The biological basis for such bistability remains little explored. Ca2+ has been shown experimentally (Egorov et al., 2002) and theoretically (Fransen et al., 2002) to contribute to persistent firing of neurons, via upregulation of a nonspecific cation current. Neuromodulators linked to Ca2+ release or Ca2+ sensitive signaling cascades have been shown to increase synaptic NMDA currents (Seamans et al., 2001) and the persistent sodium current (Yang and Seamans, 1996), both of which might increase the efficacy of synaptic input. There is a fair amount of controversy over the effects of neuromodulators (Seamans and Yang, 2004).

Numerous theoretical accounts of human memory have differentiated memory for facts and memory for contexts (Tulving 1972; Schacter 1987). Tulving (1972; 1983) further defined these two declarative explicit memory concepts (in which information is consciously registered and recalled) into semantic memory, where general world knowledge not tied to specific events is stored, and episodic memory involving the storage of context specific information about personal experiences (i.e., time, location, and the surroundings of personal knowledge). Conversely, implicit memory (non declarative) may involve unconscious registration (lack of awareness during encoding), yet definite unconscious recollection (Schacter 1987). Skills and habits, priming, and classical conditioning all utilize implicit memory. The diagram

shows an abridged graphical representation of the human memory process and its general association with source amnesia (Figure 1).

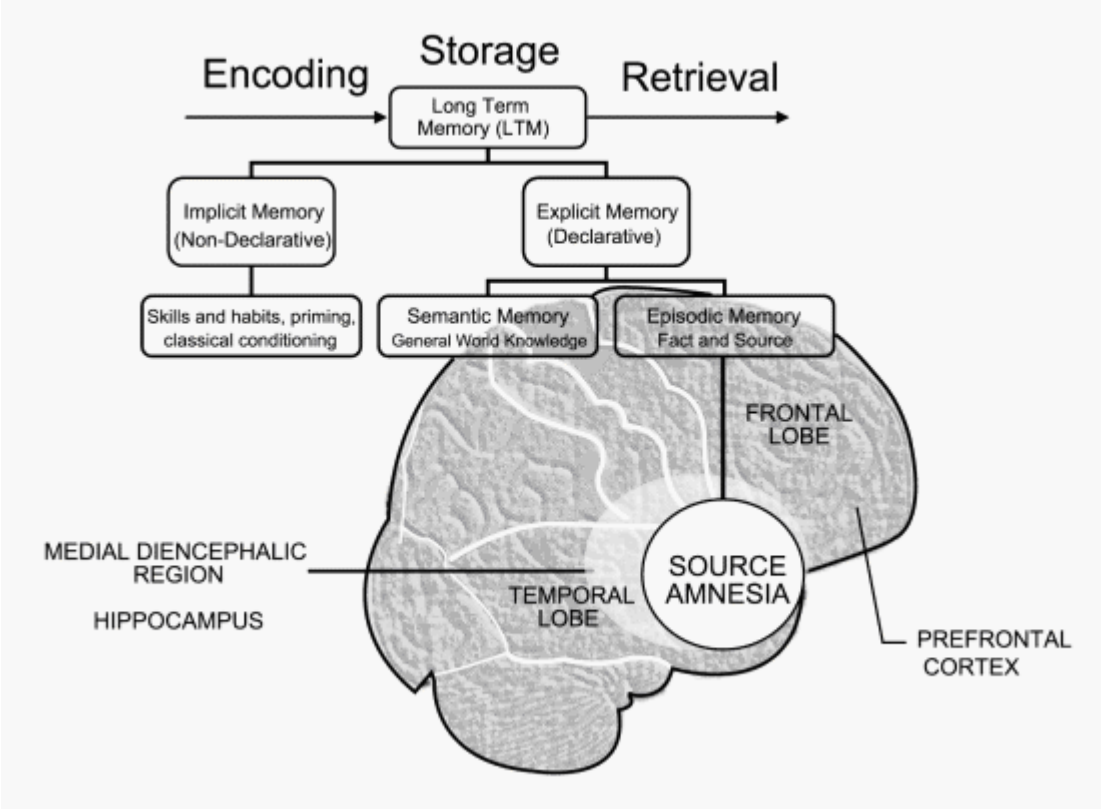

Figure 1: Source amnesia neuropsychological association diagram with partial information processing and long term memory organization chart.

An essential aspect of episodic memory includes date and time encoding in the subject's past. For such processing, the details surrounding the memory (i.e., where, when, and with whom the experience took place) must be preserved and are necessary for an episodic memory to form, otherwise the memory would be semantic (Bullock 1998). For example, one may posses an episodic memory of President John F. Kennedy's assassination, including watching Walter Cronkite announce Kennedy's death on TV. However, if the contextual details of this event were lost, the remaining would be only a semantic memory that John F. Kennedy was assassinated. The ability to recall episodic information concerning a memory is called source monitoring (Johnson, Hashtroudi et al. 1993), and is subject to distortion (Johnson, Hashtroudi et al. 1993; Goff and Roediger 1998) that may be lead to source amnesia.

Memory Distrust Syndrome

As source amnesia prohibits the recollection of context specific information surrounding facts in experienced events, there is also the inclusive case of confusion concerning the content or context of events, a highly attributable factor to confabulation in brain disease. Such confusion has been loosely termed memory distrust syndrome (Gudjonsson and MacKeith 1982). A person who suffers from memory distrust syndrome distrusts their memory and may be motivated to rely on external (non-self) sources. One known cause is people who are suffering from Obsessive Compulsive Disorder (OCD), where repeated relevant checking rituals is known to cause reductions in memory confidence, vividness and detail, resulting in memory distrust (Van den Hout and Kindt, 2003, 2004).

The propensity to accept information from external sources (i.e., an interrogator) based on the influence of susceptibility has led to well documented false confessions (Gudjonsson and MacKeith 1982; 1990; Gudjonsson 1992; 1996; McCann 1998; Gudjonsson, Kopelman et al. 1999; see Gudjonsson 2003 for a review). Moreover, the credibility of a witness that suffers from memory distrust syndrome is questionable. In a parallel situation, amnesic individuals may be more

susceptible to having their memory manipulated, perhaps performing non-advantageous acts at the "direction" of external sources, and experience difficulty differentiating between imaginary and real experiences. Given that source amnesia pathology is an identified and natural occurrence in the criminal law system, psychiatrists should perform assessment and identification measures to isolate this disorder in accused individuals and eyewitnesses.

Assessment and Identification Methods

Tests involving various interactions and responses are employed on subjects to determine the type of memory processes that are utilized, affected, or regulated. Intuitively, accessing such facets of human memory requires circumlocutory procedures since implicit memory is understood to be unconsciously registered and categorically involuntarily recalled. Explicit memory tests are rather straightforward, although variation exists within this subcategory. In assessing neurological function (and dysfunction), standard measure neuropsychological tests are utilized.

Explicit and Implicit Memory Tests

Different techniques are utilized to reveal and examine explicit and implicit memory. For explicit memory, there are four prominent methods for direct examination: free recall, cued recall, yes-no recognition, and forced-choice recognition (Leahey and Harris 2001). Free recall is devoid of any indicators or hints (i.e., the experimenter would ask questions like "Tell me about [a general topic]..."), whereas cued recall contains clues (i.e., a fill-in-the-blanks assessment). A side effect of free recall is the recall of less accurate information; however, a potential for bias is nonexistent but present in cued recall.

In the two recognition pathways, one must produce a response and recognize its validity. With yes-no recognition, information is presented to the subject and they must indicate whether it is valid with a "yes" or "no" response. Finally, the forced-choice recognition measure consists of a required choice among several options where only one is correct (i.e., multiple choice exams). Recent reports indicate that since recognition memory tests depend on familiarity, amnesiacs perform normally on yes-no recognition and forced-choice recognition (Hirst and et al. 1986; Aggleton and Shaw 1996) tests in equal proportions (Khoe, Kroll et al. 2000).

Recent studies of explicit memory have employed delayed non-matching to sample performance tasks to assess recognition memory for objects (Gaffan, Gaffan et al. 1984; Diamond, Zola-Morgan et al. 1989; Gow 1995). The task has three parts: a familiarization, delay, and test phase. During the familiarization process, the subject is presented with a sample object. After a period of delay, the experimenter presents a new object either to the left or right of the sample object. The subject's goal is to choose the novel object (i.e., the object that is not the sample), and they receive a reward if they are successful. Research has demonstrated that children enjoy novelty over consistency (Fantz 1964; Fagan 1970; Cohen and Gelber 1975), thus they find it easier to understand and proceed with delayed non-matching than matching to sample tasks (Brush, Mishkin et al. 1961; Gaffan, Gaffan et al. 1984). The more objects a subject correctly discriminates, the greater their recognition memory skill.

Implicit memory evaluations test memory more indirectly by examining performance on tasks that may obliquely reveal properties of remembrance. With word-fragment completion, implicit memory is measured by presenting a fragmented word (i.e., P_Y_H_L_G_) and asking the subject to identify the complete word. It should be noted that priming greatly affects task performance (i.e., presenting the phrase "mental-health" prior to the aforesaid fragmented word, "Psychology") (Leahey and Harris 2001).

In examining amnesiacs, the priming effect is quite noticeable in patients undergoing surgical procedures requiring general anesthesia. The patients were more likely to produce words of a given category if they had heard them while under anesthesia (Millar 1987; Charlton, Wang et al. 1993), even though there was no explicit conscious memory for the information heard under anesthesia.

Positron Emission Topography (PET)

Neurological imaging techniques have been used extensively in amnesiac studies to locate the neurological areas attributable to memory and cognitive functions. Positron Emission Tomography (PET) scans provide biochemical resolution of a patient's body without the use of invasive measures. While other imaging scans such as CT and MRI isolate organic anatomical changes in the body, PET scanners are capable of molecular biology detail (even prior to anatomical change)

via the use of radioisotopes highly metabolized in cancerous tissues. Regarding source amnesia neuropsychological research, the changing of regional blood flows in various anatomical structures (as a measure of the injected glucose emitter) can be visualized and relatively quantified with a PET scan (Cabeza, Kapur et al. 1997).

The three common tests used to assess executive function are

1. Wisconsin Card Sorting Test (WCST)
2. Phonemic Verbal Fluency Test
3. Stroop Color Word Interference Test

Wisconsin Card Sorting Test (WCST)

The Wisconsin Card Sorting Test (WCST), developed by Berg (1948), is the most common assessment tool for executive function (Bullock 1998). It assesses

1. Cognitive flexibility
2. Problem solving and
3. Response maintenance

The subject is presented with four model cards that differ in color, number, and shape. The subject is instructed to place the response cards (consisting of all possible combinations of shape, color, and number traits) directly below the model cards that they believe match. Feedback is given to the subject about whether they correctly associated the cards. If the subject fails to correctly match the cards according to the unstated guidelines, they must be able to alter their sorting strategy (Bullock 1998). The WCST model without the number attribute has been adopted for testing on very young children, thereby providing an extensive range for a potential population based on age.

Throughout the WCST procedure, the subject is not informed of the reasoning behind the experimenter's "right" or "wrong" feedback. Thus, a great level of ambiguity exists for novice and skilled subjects performing the task. The main calculating factor of the WCST is the number of preservative errors committed (when the subject can not alter the sorting criterion despite being given feedback that their selection is erroneous). The next important significant variable as an index of executive function is the total number of categories attained by the subject. A credited category occurs when the subject has correctly sorted a given number of successive cards (usually ten).

Although the WCST is an valuable indicator of prefrontal function (Nelson 1976), its sole use to discern a group of front damage patients from a control set is unreliable (Anderson, Damasio et al. 1991; Mountain and Snow 1993). It is because in addition to the activation of dorsolateral prefrontal cortex, it is found that activation of ventromedial and orbitofrontal prefrontal cortex, inferior parietal cortex, basal ganglia, tempero parietal association cortex, occipito temporal, temporal pole and occipital cortices also occur during performance of WCST. It is also unclear whether left or right prefrontal cortex is stimulated by WCST. But it is accepted that bilaterally intact prefrontal cortex, especially dorsolateral is necessary for normal WCST performance.

For the above reasons, WCST is a sensitive, but not a specific test to assess prefrontal function.

Phonemic Verbal Fluency Test

The Phonemic Verbal Fluency Test is another test used to assess executive function, especially of the frontal cortex. In this, the participants are instructed to say or write as many words as possible beginning with a specific letter. People with frontal lesions (especially left) perform poorly (Troyer et al., 1998). Like WCST, this test is very sensitive but not very specific for assessing functioning of Prefrontal lobes.

Stroop Color Word Interference Test

The Stroop Color Word Interference Test is another measure to assess frontal cortex function, especially selective attention (Blenner, 1993; Carter et al., 1995; Goodglass and Kaplan, 1979; Lezak, 1995; Macleod, 1991; Stuss et al.,

2001). In this test, participants are exposed to three sets of stimuli namely

1. Color words printed in black ink
2. Color patches or colored Xs or
3. Color words printed in incongruous colored ink (eg. The word red printed in blue ink)

Participants are requested to read the color words of the first sheet, colors on the second sheet and color of the INK on the third sheet. In the third task, the normal tendency to read the words, rather than the color of the ink in which the words are printed, elicits a significant slowing in reaction time (RT) call the "Stroop Effect" or the interference effect.

Persons with left frontal lobe lesions (especially of lateral and superior medial and not orbitofrontal) display significantly longer interference trial RTs than those with non frontal lobe lesion.

Etiology

Since source amnesia involves the lack of source memory recollection - although the fact itself is remembered - one would expect the prominent occurrence of source amnesia with pre-mature, deteriorated, or damaged neurological centers responsible for episodic memory. Such dysfunction may be due to encoding, retrieval, or storage processing errors, including inefficient source monitoring.

The prefrontal cortex has been associated with the processing of executive functions, including the organization of information; goal intended behavior, and planning and inhibition. Thus, performance on such functions has become an accepted indicator of prefrontal function (i.e., the WCST). In addition, recent investigations have attributed episodic memory to be another function of the prefrontal cortex even though the role of the prefrontal cortex is still disputed (Swick and Knight 1999; Heckers, Curran et al. 2000; Mayes and Montaldi 2001; Rossi, Cappa et al. 2001; Lee, Robbins et al. 2002; Wagner 2002).

Posthypnotic Induction

Posthypnotic source amnesia occurs when some fact is learned under hypnosis and the information is forwarded to the conscious or walking state, but the knowledge that it was learned under hypnosis is forgotten (contextual-specific information). In contrast, posthypnotic recall amnesia is used to describe similar hypnotic situations with the loss of factual information instead of contextual information (Thorn 1960).

In a typical posthypnotic source amnesia experiment, the subject is told the answers to previously unknown questions under hypnosis (i.e., Harare is the capital of Zimbabwe). The time, place, and experimenter-specific information are recorded (i.e., number of experimenters and which one informed the patient of the fact). After the hypnotic session, the awakened subject is questioned concerning the learned facts and the contextual specifics. In order to credit the subject with source amnesia, they must answer the questions correctly but fail to identify the source of the information.

Thorn's study (1960) implemented such hypnotic procedures and, most importantly, amnesia was never suggested to the subjects; thus, the measure was for spontaneous posthypnotic source amnesia. Based on his procedural conditions, Thorn demonstrated that source amnesia was an authentic effect of hypnosis and not an artifact of the demand characteristics of the situation (Cooper 1966). Furthermore, Cooper (1966) suggested source amnesia to patients (i.e., informed the hypnotized subject to forget the source of information) and noticed a greater frequency of source amnesia in the suggested than in the spontaneous subjects, further validating source amnesia as a genuine hypnotic phenomenon.

Evans and Thorn's (1963) early hypnotic research forged a path for current scientists to follow regarding the multifaceted machinery of source amnesia, and permitted Schater (1984) to systematically demonstrate source amnesia in amnesic patients.

Neuropsychology of Episodic Memory

Neuroanatomical evidence for the correlation of explicit memory - and hence semantic and episodic memory - to the temporal lobe dates back to the 1950s (Milner 1964). Since then it has been recognized that the medial diencephalic

structures, in addition to the temporal lobe, are involved with episodic memory (Diamond, Towle et al. 1994; Andreasen, O'Leary et al. 1995; Buckner, Petersen et al. 1995; Nyberg, Tulving et al. 1995; Cabeza, Kapur et al. 1997; Buckner, Koutstaal et al. 1998; Buckner, Koutstaal et al. 1998; Reed and Squire 1999). Through magnetic resonance imaging (MRI) (Alvarez, Zola-Morgan et al. 1995), two-choice recognition tasks (Reed and Squire 1999), and delayed non-matching to sample performance tasks (Diamond, Towle et al. 1994), source memory has been attributed to medial diencephalic system activity. The structures of the medial diencephalic system include the hippocampus, perirhinal cortex, parahippocampal cortex, entorhinal cortex, and direct connections to the limbic system (Kaut 2001).

The reliance of source memory on the prefrontal cortex (a division of the frontal lobes) has been recently suggested (Shimamura and Squire 1987; Janowsky, Shimamura et al. 1989; Shimamura and Squire 1991; Knowlton and Squire 1995; Squire and Knowlton 1995; Bullock 1998; Senkfor and Van Petten 1998; Wilding 1999; Drummey and Newcombe 2002). Patients with prefrontal damage have exhibited a higher propensity to source amnesia than normal controls (Schacter, Harbluk et al. 1984; Shimamura and Squire 1987; Janowsky, Shimamura et al. 1989).

Age Associated Source Amnesia: Neurological Maturation and Deterioration

It has been hypothesized that the frontal lobes fully mature between the ages of four to seven years old (Luria 1963) and, remarkably, is the last area of the brain to fully develop. Neuroimaging (PET) (Chugani 1987), electroencephalographs (EEG) (Thatcher 1991), and research on monkeys (Diamond, Zola-Morgan et al. 1989) have demonstrated that the frontal lobes mature relatively late. Therefore, on the premise of structure-function parallels in this area of neuroanatomy, very young children with pre-mature frontal lobes should display significance in source recollection errors and thus greater source amnesia than those with mature frontal lobes.

As predicted, Bullock's study (1998) showed that four-year old children showed a significant amount of source amnesia (as an index of the WCST). In addition, she found that eight-year olds (who have fully mature frontal lobes) exhibited "dramatically less" source amnesia than four-year olds. Aside from the associated frontal lobe maturation that occurs during this four-year interval, the episodic memory changes in childhood may be attributable to the simultaneous development of other neuroanatomical structures yet to be identified.
Although the frontal lobes are the last neuroanatomical region to fully mature (Bullock 1998) during the normal aging process, they are considered the first to deteriorate (Huttenlocher 1979; Haug, Barmwater et al. 1983; Raz, Torres et al. 1993). As people age, memory for context tends to deteriorate at a fast rate (Salthouse 1982; Craik 1983), while memory for facts declines relatively little (Salthouse 1982). Researchers have discovered that frontal lobe dysfunction correlates to the normal aging course neuroanatomically (Whelihan and Lesher 1985), neuropyschologically (Whelihan and Lesher 1985), and electrophysiologically (Woodruf 1985).

Prefrontal cortex correlation studies of the normal aging process have varied, but the majority of research has shown prefrontal association. For example, McIntyre and Craik (1987) and Craik (1990) showed a parallel correlation, while Spencer and Raz (Spencer and Raz 1994) found non-significant associations. However, studies using neuroimaging techniques (Kapur 1998; Rossi, Cappa et al. 2001) have mapped the prefrontal cortex to episodic memory processing (see Mayes and Montaldi 2001 for review). Thus, the variations in the relationships between the normal aging process and the prefrontal cortex may be credited to a lack of proper prefrontal assessment (i.e., the lack of standardization measures) and the nature of the subjects in the studies (i.e., variability in health status and cognitive abilities). However, Bullock (1998) found significant amounts of source amnesia in very young children, and identified prefrontal function linkage with source amnesia incidence on the basis of prefrontal cortex maturation. One can suggest that episodic memory, particularly memory for source, may rely on the prefrontal cortex in addition to the temporal regions and the medial diencephalic structures of the brain. However, it is possible that deteriorated (as in the course of the normal aging process) compared to pre-mature (i.e., very young children) frontal cortexes perform significantly different in memory tasks.

The prevalence for source amnesia has been well documented in elderly populations owing to the neurological deterioration of the frontal region, namely the medial diencephalic system and the frontal lobes, including special interest with the prefrontal cortex (see Figure 1 for diagram). However, most studies have indicated mild source amnesia in young children undergoing frontal lobe maturation, but only in direct comparison with newly fully developed patients (i.e., the eight year olds in Bullock's (1998) study). Thus, the organic deterioration of the frontal lobes in the process of normal aging has a greater influence on episodic memory than pre-mature lobes in young children. The differentiating mechanisms between the maturation and deterioration, if any, are unknown at this time.

Conclusion

The discussion done in this review is just the tip of the iceberg in the understanding of source amnesia and the day will definitely come in future whereby different treatment options for source amnesia become available. Until we understand the simple and complex neural circuitry, map the neurological centers responsible for episodic memory and thus source amnesia, and identify the pathways for neuro-biological and -psychological maturation, deterioration, and damage, source amnesia and related conditions will remain largely unsolved. However, at this stage, we have pieced together a portion of the puzzle and, remarkably, eradicated the old-fashion notion that amnesia is a unitary disorder. As an episodic memory disorder, source amnesia is attributable to the dysfunction of the normal memory process involving encoding, storage, and retrieval. Thus, a parallel exists between advancements in the human memory process and neuropsychology and our comprehension of source amnesia.

In future, it is fully expected that many more fascinating details of the molecular mechanisms responsible for memory formation and stabilization processes will come to light. Toward that end, the field will continue to benefit from the rich contribution of the diverse disciplines including psychology, physiology, pharmacology, cellular and molecular biology, biochemistry, genetics, biophysics and bioinformatics that have already shaped the study of memory during the last four decades. Although the ultimate goal of this multidisciplinary approach will be to explain, in molecular detail, how specific memories are encoded and maintained within the human brain, experiments in the different animal model systems, will continue to pave the way. The major question that now also needs to be addressed relates to the system properties of memory storage, such as how synaptic changes at different points in an explicit network, give rise to the storage capacity of the system. The long-term prospects are extremely exciting (Angel Barco, Craig H. Bailey and Eric R. Kandel. 2006).

Presently, on the clinical side, it has been found that amnesiacs in general perform better in verbal recall when the retroactive interference is diminished by placing them in a dark quiet room (Nelson Cowan, Nicoletta Beschin and Sergio Della Sala, 2004). Also it has been understood that recognition memory deficit in amnesia is attenuated under conditions that increased the salience of study-induced fluency possibly because memory for a past experience can be based on recollection of specific details from the experience or on a sense of familiarity that accompanies re-exposure to information from that episode (Margaret M. Keane, Frances Orlando, Mieke Verfaellie 2006). This is just a small step in the understanding of amnesia in general and the day will definitely come in future whereby different treatment options for source amnesia become available.

There are no specific preventive measures for source amnesia but the general guidelines outlined below can be followed.

1. Keep brain cells active by reading, doing puzzles, singing, exercising, conversing and eating a balanced diet to stimulate blood flow, improve activity and preserve them from degeneration

2. Antioxidants like Vitamin E and Selegiline can be taken when appropriate

3. Avoid head injuries. Wear seat belt and helmet at all times during driving. Children, adolescents and adults should also be taught sports safety guidelines.

4. Strokes, which can cause amnesia, should be prevented by treating predisposing factors like hypertension, obesity, diabetes mellitus, dyslipidemia, smoking, alcoholism, stress and hyperhomocystinemia.

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