Case Reports

Loss of Recent Memory as a Sign of Focal Temporal Lobe Disorder

Report of a Case

RICHARD A. SMITH, M.D.,* AND WILLIAM A. SMITH, M.D.
Atlanta, Georgia

Loss of recent memory is well known as a symptom of diffuse cerebral disease such as senile and presenile dementia, toxic and deficiency states, trauma, and encephalitis. In these cases, memory loss may be only a part of a general disturbance of the intellect. Recently it has become recognized that this symptom may also be a more specific sign of a focal disorder involving the vicinity of either the 3rd ventricle or of the hippocampus.

The patient in the following case had a striking loss of recent memory as the predominant symptom, with the intellect otherwise quite well preserved. This deficit proved to be due to a tumor largely confined to the deep left temporal lobe and hippocampus. We are unaware of similar examples of memory loss associated with such a discrete neoplasm in this location, and wish to add this case of medial temporal lobe tumor to the other reported cases of focal lesions producing this syndrome.

Case Report

A 62-year-old nurse was admitted to the Piedmont Hospital on March 6, 1963, complaining of loss of memory of about 2 weeks duration. She said that she frequently forgot what she had done or read in the preceding few minutes, and had difficulty finding things at home and at work. Friends had told her that she repeated statements and questions over and over. For the past week she had been aware of lethargy and loss of initiative. She found herself “daydreaming,” and had once just watched the food she was cooking burn. She felt “too bored” to eat, and neglected writing letters to her son and other duties. She developed a tendency to cry easily, and wondered if she were in a state of depression. There had been one brief episode of disturbed vision in which objects seemed distorted in size and shape, but no other illusions, hallucinations, or seizures.

Examination. The general physical and neurological findings were normal. There was no papilledema or visual field defect. Motor, sensory, and reflex testing were all normal. The outstanding abnormality was a severe disturbance in memory for recent events, without any corresponding loss for events prior to her illness. She could not recall what she had been told or what she had read even a few minutes earlier, and would quickly forget instructions. She was especially embarrassed by her inability to recall names of close friends who visited her. There was a mild nominal aphasia and some dyslexia. Some disorientation in space was apparent, causing confusion as to which door of her room led to the bathroom, and failure fully to recognize her own hospital room after a short absence. She could perform calculations readily, could distinguish her right side from her left, and had no finger agnosia. She was able to draw objects and copy figures without evidence of constructive apraxia.

Two weeks after admission she had 2 generalized convulsions, beginning with deviation of the head and eyes to the right. Her memory loss then became even more striking. She could not recall her age, and was confused as to time. She recognized her physicians, but would forget their visits a short while later. If procedures such as arteriography were explained to her in detail, she would soon have no memory of the conversation. Her son, a physician in the Navy, had returned from the Philippines to see her. She knew this when he was with her, and would talk to him in an intelligent, appropriate, seemingly normal manner. However, if asked 15 minutes later if she had seen him, she might say, “Oh no, he is in the Philippines.” She was fully aware of her memory disorder and said she felt as if “half my brain is gone.”

Finally, she developed a complete homonymous hemianopsia. She became somewhat lethargic and indifferent. There was still no papilledema or other evidence of increased intracranial pressure, and no abnormality on sensory, motor, or reflex examination.

Roentgenograms of the skull, electroencephalogram, and the usual laboratory tests of blood and urine were all normal. The cerebrospinal fluid contained no cells and was under a pressure of 75 mm. of water; it had a protein content of 10 mg. per cent. Bilateral carotid arteriography revealed no abnormality of the intracranial vessels. A radioactive mercury brain scan demonstrated a 4.5 cm. area of hyperconcentration of the isotope deep in the left mid-temporoparietal area, almost adjacent to the midline (Fig. 1).

Operation. A left temporoparietal craniotomy was carried out on April 8, 1963, 14 months after the onset of symptoms. The brain was not under increased pressure, and no surface abnormalities were seen. A core of brain 2 cm. in diameter was resected from the low mid-temporal region, corresponding to the area of abnormality seen on the brain scan. Tumor tissue was encountered, medial to the temporal horn of the ventricle. A biopsy was obtained. This was interpreted by the pathologist, Dr. R. B. Vincenz, as a gemistocytic astrocytoma. There were areas of hemorrhage and necrosis, with perivascular and endothelial proliferation of blood vessels, indicating malignancy.
Her condition showed little change immediately following operation. Gradually she became less responsive, lapsed into a state of akinetic mutism, and died on April 27, 1963.

**Autopsy.** Postmortem examination was limited to the brain. There was no evidence of cerebral swelling. There was a surgical wound in the midportion of the left middle and inferior temporal gyri, 2 cm. in diameter, but no other surface distortion. Vessels of the circle of Willis showed only minimal atherosclerosis at their bifurcations. The lateral and 3rd ventricles were not enlarged or displaced in coronal sections. The mammillary bodies, thalamus, and body of the corpus callosum were normal. Beginning at the level of the splenium of the corpus callosum (Fig. 2), a firm, greyish-red tumor was found, which extended across the splenium and included the hippocampal commissure. There was complete infiltration of the crus of the left fornix, but the right was not invaded. The bulk of the tumor lay medial to the collateral trigone of the left lateral ventricle and posterior temporal horn, occupying the posterior portion of the left hippocampus and hippocampal gyrus. It extended posteriorly, medial to the occipital horn, to within 5 cm. of the occipital pole. The amygdala and approximately the anterior half of the left hippocampus were not involved. Except for invasion of the psalterium and splenium of the corpus callosum, the tumor was unilateral. The brain stem and cerebellum were normal. Microscopic examination of the tumor was similar to that of the biopsy.

**Discussion**

Memory is a complex, integrated function. Besides the initial perception of events, it involves the process which ultimately results in retention, and permits subsequent recall. Only a start has been made in understanding these processes. Much of the knowledge has necessarily come from clinicopathological studies of memory impairment in man, which cannot be exactly duplicated in experimental animals.

In 1887, Korsakow described a peculiar mental syndrome associated with polyneuritis, occurring mostly in alcoholic patients. This syndrome of Korsakow’s psychosis is characterized particularly by the inability to record new memories, old memories remaining relatively intact. Immediate repetition of words and numbers may not be affected as long as attention is not distracted, but memorizing for retention and recall beyond a few minutes is poor or does not occur. There is often disorientation in time, so that supposed dates are erroneous and time intervals are conceived as much shortened, and in space, with difficulty in finding one's way even in familiar surroundings. There may also be a tendency, known as confabulation, to fabricate false accounts as if to conceal these defects of memory. For some time it has been thought that the mammillary bodies are specifically related to the process of memorizing, because of their consistent involvement in cases of Korsakow’s alcoholic psychosis. More recently it has been indicated that lesions in the medial dorsal nucleus of the thalamus may also be of special significance with respect to memory loss in this disease.

In many diseases, such as encephalitis and senility, the diffuse distribution of the lesion prevents meaningful correlation with the symptoms of memory loss. Intraeranial tumors may cause impairment of memory, secondary to the indifference or inattention accompanying frontal lobe destruction, the aphasic disorders with involvement of the dominant cerebral
Fig. 3. Coronal section of brain through splenium of corpus callosum and hippocampal commissure. A. Photograph of gross specimen. B. Schematic representation. Tumor cross-hatched.
hemisphere, or the generalized depression of thought processes attributable to increased intracranial pressure as in posterior fossa tumors.\textsuperscript{5,24} However, a more specific and severe loss of recent memory, with retention of normal personality and intellect, has been reported from tumors such as the craniopharyngioma in the region of the floor and walls of the 3rd ventricle.\textsuperscript{24} Here the mammillary bodies or their connections could be affected.

Scoville\textsuperscript{17,18} has shown that the hippocampi are also important in the memory process. In his patients, marked memory loss for recent events followed bilateral resection of the medial temporal lobes, including the hippocampus and hippocampal gyrus. Older memories remained intact. Removal of the uncus and amygdala, sparing the hippocampus, did not cause memory loss. There was no additional postoperative deterioration in intellect or personality, and no disturbance of perception. In addition to these operative cases, softening of the hippocampi from vascular lesions has also been reported to cause loss of recent memory.\textsuperscript{5,25}

Although Scoville and Milner\textsuperscript{24} found no lasting loss of memory from a radical unilateral right inferior temporal lobectomy extending 9 cm. from the temporal tip, and although memory loss does not occur in most cases following the usual, more limited temporal lobectomy for epilepsy,\textsuperscript{14} there is some evidence that a unilateral temporal lesion on the dominant side may cause such a loss. Thus Penfield and Milner\textsuperscript{14} reported 2 cases in which unilateral partial temporal lobectomy, including a portion of the hippocampus and hippocampal gyrus, did result in severe impairment of recent memory. They suggested that a destructive lesion such as incisural sclerosis must have existed on the opposite side. Similarly, Walker\textsuperscript{27,28} has reported loss of recent memory in 2 cases resulting from unilateral left temporal lobectomy for seizures; and also in a patient after left temporal lobectomy for exposure of an aneurysm; in another patient following left pedunculotomy beneath the temporal lobe for parkinsonism; and in one patient with an astrocytoma of the left temporal lobe. He feels that these cases are evidence that failing recent memory may be a sign of a focal temporal lobe disorder. Furthermore, the dominant importance of the left hippocampus in memory has been indicated from a study of brain injuries by Russell and Espir.\textsuperscript{16}

The fact that similar impairment of memory may occur with lesions involving either the hippocampus or the mammillary bodies and the thalamus suggests the physiological importance of the fornix which connects these areas. Curiously, bilateral section of the fornix has repeatedly failed to produce memory loss,\textsuperscript{1,5,13,14} with the single exception of the case of Sweet.\textsuperscript{19} This apparent inconsistency has not been fully explained. However, rich, efferent, hippocampal-temporal paths discharging from the hippocampus to hippocampal and other temporal lobe gyri, as described by Votaw,\textsuperscript{21} could serve as alternate, indirect connections of the hippocampus to the thalamus and related structures, through the lateral temporal lobe.

A relationship between the lateral temporal cortex and memory has become evident through the work of Penfield.\textsuperscript{15} He has repeatedly obtained subjective responses consisting of the vivid recall of past auditory and visual experiences in epileptic patients by stimulation of the superior temporal convolution, between the primary auditory and visual receptive areas, as well as from the fusiform and hippocampal gyri. However, excision of these same areas of lateral temporal cortex does not abolish either stored memories or the ability to record new memories. Though the lateral temporal lobe is apparently not essential to memory, Penfield views it as important in the subconscious recall of past experiences for comparison in the interpretation of present experience, particularly with regard to visual and auditory impressions.

How the hippocampus, mammillary bodies, thalamus, lateral temporal lobe, and other structures correlate in the physiology of memory is largely unknown. Herrick\textsuperscript{11} has suggested that memory traces persist as tuned resonating circuits in a more or less stable, dynamic pattern, widely embodied in the neuropile of the cortex and brain stem. These memories may then be reactivated by incoming signals through complex processes involving space, time, and possibly other dimensions. Penfield\textsuperscript{14,15} also indicates that the neuronal record of memory is not localized to the temporal lobes, but rather lies in other neurone circuits at a distance, including the central integrating circuits of the brain stem.

Gerard\textsuperscript{7} found that memories require a certain time to become fixed in the nervous system, the repeated passing of impulses over reverberating circuits adding to their reversible effects until some irreversible state is passed. This phenomenon may be the basis for retrograde amnesia in concussion, which occurs when the memorizing process is interrupted before the memory record is firmly established.

Present evidence suggests that the hippocampus, mammillary bodies, and thalamus play a critical role in this process of memory fixation, whereas the actual storing of the memory record is more widespread in the cerebral cortex, diencephalon, and brain stem. Since the hippocampus is anatomically and physiologically related to all these structures,\textsuperscript{4,9,20} Herrick's\textsuperscript{10} early view of the hippocampus as an integrator of cortical and di-
encephalic areas may be significant with respect to its function in the process of memory recording.

In our case, extremely severe loss of memory for recent events was the presenting symptom. In contrast, memory for events prior to the patient's illness remained intact. There was spatial and temporal disorientation typical of Korsakow's syndrome, a mild nominal aphasia with some dyslexia, and, later, right homonymous hemianopsia. The principal structures involved by the tumor were the medial left posterior temporal and occipital lobes, including the posterior left hippocampus and hippocampal gyrus, crus of the left fornix, hippocampal commissure, and splenium of the corpus callosum. Although the tumor obviously involved more than just the hippocampus and crus, this case does support other evidence that the hippocampus and related pathways are essential to memory recording, and that severe impairment of memory for recent events may result from a predominantly unilateral lesion.

**Summary**

We have reported a case in which the patient had severe loss of memory for recent experience, associated with a malignant gemistocytic astrocytoma involving the medial left temporal lobe, including the posterior left hippocampus and crus of the left fornix. We have concluded that this area plays a role of critical importance in the recording of new memories, and that loss of recent memory may point to focal disease such as infarction or neoplasm of the temporal lobe. Related reports on this subject have been reviewed.

**References**


