and posterior temporal seizures can give rise to, among other things, formed visual hallucinations.

Memory tends to be shot through with emotion, and odors — like the aroma of Proust’s “petites madeleines,” which instantly summoned up the world of his childhood — are notoriously evocative: “the smell and taste of things remain poised a long time, like souls, ready to remind us, waiting and hoping for their moment . . . and bear unaltering, in the tiny and almost impalpable drop of their essence, the vast structure of recollection.” It is no surprise, therefore, that the medial temporal lobes contain two key nodes in the neural networks for emotion and smell — the amygdala and the piriform cortex. Their presence here helps to explain the experience of panic and hallucinations of smell in temporal-lobe seizures. The “epigastric aura,” a rising sensation from the stomach, is another common result of activity within this limbic circuitry.

Although the temporal lobes are much involved with the complex integration of experience, they are also home, in their uppermost gyrus, to a primary sensory area, Heschl’s gyrus — the auditory cortex. In the left hemisphere, this region abuts Wernicke’s area, where meaning is extracted from the sounds of speech. Thus, epilepsy arising close to the left superior temporal gyrus can manifest itself in auditory hallucinations, speech arrest, or dysphasia. Epilepsy arising at the adjacent junction of the temporal, parietal, and occipital lobes, close to the cortical representation of information about balance, has been linked in recent work to out-of-body experiences, during which one sees oneself from an external perspective, and to “autoscopy,” the hallucinatory vision of one’s own body seen from one’s current perspective. 5

Déjà vu, short-lived amnesia, the epigastric aura, hallucinations of smell, incongruous emotions, disorders of language, and out-of-body experiences can all be pointers to a disturbance of function in the temporal lobes. The elegance of clinical neurology lies in the possibility of localizing pathologic lesions in the brain on the basis of clinical features such as these, which may consist of a single minor perturbation of experience. But location does not imply process. Don’t rush to diagnose epilepsy the next time you experience déjà vu. The range of possible explanations bridges the divide between neurology and psychiatry: anxiety, depression, and psychosis are important causes, as is the likeliest candidate of all — that your perfectly normal but hard-pressed temporal lobes are temporarily overstretched.

Surgical Repair of Aneurysm Causing Subarachnoid Hemorrhage.

The anatomy of the subarachnoid space and the circle of Willis is shown in Panels A and B. A major artery (the internal carotid artery) enters the skull from below and then follows a course through the subarachnoid space, giving off penetrating branches that supply the parenchyma. High pulsatile pressure on branching points of the proximal artery (arrow, Panel B) just after the arterial wall sheds much of its supporting adventitia can promote the formation of saccular aneurysms in susceptible persons. In such cases (Panel C), an aneurysm forms at the branch point of an artery, where the arterial pulsation stress is maximal. Most lesions remain silent until rupture occurs, at which time blood is rapidly released into the subarachnoid space, leading to early effects such as parenchymal irritation, edema, and hydrocephalus and delayed effects such as vasospasm. During surgical repair of such an aneurysm (Panel D), temporary clips can be placed on the proximal feeding artery alone, or they can be placed on both the proximal and distal arteries in order to “trap” the segment harboring the aneurysm. Both methods reduce flow within the regional segment. Trapping, however, provides complete cessation of flow, and any tissue supplied by an end artery in the trapped segment is particularly susceptible to ischemic consequences. Lowering the brain’s metabolic demands can extend the interval of tolerance of the flow interruption, providing more time for the surgeon to accurately secure the aneurysm. Removal of the skull base provides improved access and operative exposure for the surgeon without the need for substantial brain retraction. Once the exposure is complete (Panel E), a permanent clip is placed on the neck of the aneurysm, effectively excluding it from arterial circulation. The aneurysm is then collapsed, and the field inspected to make sure no branches are compromised by the clip placement. The inner wall of the aneurysm base is approximated by the clip, generally providing a lifelong cure of the lesion.
A ruptured cerebral aneurysm is an intracranial catastrophe, associated with very high morbidity and mortality. When an aneurysm ruptures, blood spurts into the subarachnoid space under arterial pressure, continuing until increased local or generalized intracranial pressure stops the bleeding. Acute hydrocephalus may develop as the blood fills the subarachnoid space and impedes the normal flow and absorption of cerebrospinal fluid.

Focal clot formation or parenchymal edema and irritation can disturb the regulation of cardiac or respiratory function or further increase the intracranial pressure, culminating in death. Aneurysmal subarachnoid hemorrhage is associated with mortality rates between 25 and 50 percent from the consequences of the initial bleeding. Half of untreated survivors have an additional bleeding episode at least once within the next six months, and among such patients, morbidity and mortality are even higher. Even with aggressive modern treatment, good neurologic function is restored in less than one third of all affected patients.

If the patient survives the immediate effects of the bleeding episode and reaches a medical facility alive, the initial management must be directed toward stabilizing or reversing acute life-threatening conditions, including tissue hypoxia from seizures or respiratory depression, cardiovascular dysfunction, hydrocephalus, and focal intracranial clots. Particularly in obtunded patients, the establishment of an airway and urgent ventriculostomy with drainage of cerebrospinal fluid can be lifesaving, since these procedures reduce the effects of brain hypoxia, acute hydrocephalus, and increased intracranial pressure.

Once the patient’s condition has stabilized, the primary focus of treatment becomes the prevention of rebleeding. The cause and site of the subarachnoid hemorrhage are determined by means of some form of arteriography. The best method of obliterating the aneurysm is then selected and implemented, usually within 24 hours after presentation, unless a life-threatening clot necessitates emergency surgical evacuation. The selection of the appropriate treatment — either open surgery (clipping) or an endovascular approach (coiling) — is based primarily on the age and clinical status of the patient and the size, shape, and location of the aneurysm; the decision is best made by a team that is proficient in both methods.

Operative clipping is a definitive technique for securing most ruptured aneurysms. During surgery, an opening in the skull is created (craniotomy), the dura mater is opened, and the subarachnoid space is dissected to separate the lobes of the brain and also to take advantage of naturally occurring corridors in order to reach an aneurysm arising near the base of the skull. In the process, the brain must be manipulated in a gentle way so as not to create additional risks for the already irritable or injured organ. Methods that reduce the brain’s volume (the use of osmotic agents and the drainage of cerebrospinal fluid) and techniques that minimize brain retraction (the release of arachnoid membranes and the removal of bone from the skull base) facilitate the exposure of a broad area of the brain while minimizing trauma.

Once exposure is complete, the aneurysm is dissected from adjacent branches and obliterated with the placement of a titanium clip across the origin (or neck) of the aneurysm. The operative manipulation of a recently ruptured aneurysm, however, is not without substantial hazards and technical obstacles. The ability to visualize and control blood flow within the entering and exiting branches is crucial, especially in the event of premature intraoperative rupture of the aneurysm before dissection and anatomical clarification have been completed. Temporary clips may be placed on the proximal feeding vessel alone or also on the exiting branches (a technique called trapping), providing focal circulatory arrest in the vessels adjacent to the aneurysm; this vital adjunct technique is often used in the final stages of the dissection and clipping. Temporary clipping reduces the flow of blood into the aneurysm, makes it softer and less pulsatile, allows for easier and safer manipulation of the aneurysm during permanent clip placement, and controls bleeding in the event of premature rupture. During temporary clipping, however, the regions supplied by the clipped vessels are susceptible to ischemic injury, especially if the clip remains in place for a prolonged period. Four basic methods are used in efforts to expand the safe clipping interval in order to give the surgeon more time to reconstruct the artery without incurring hypoperfusion injury. First, a proximal clip may be placed on the feeding artery earlier in the process; this technique...
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Reduces the tension on the aneurysm (hence reducing the incidence of premature rupture during the dissection and clipping) without completely arresting flow within the two exiting vessels or their branches. Second, the systemic blood pressure may be raised slightly above normal levels in order to enhance perfusion through collateral channels. Third, the surgeon may avoid including a vessel that feeds a perforating end artery in the trapped segment. And fourth, the brain’s energy requirements and metabolic activity may be suppressed, either with medications (i.e., barbiturates) or through the induction of mild hypothermia.³,⁴

After the permanent clip has been placed, the aneurysm is collapsed, and the temporary clips are removed, restoring normal blood flow to the region. The base of the aneurysm is then carefully inspected to make sure that complete obliteration has been achieved without compromise of perforators or exiting trunks. Intraoperative angiography is used routinely at many centers to confirm the obliteration of the aneurysm and the patency of distal vasculature.

Even after successful aneurysm obliteration, the patient remains at risk for later problems related to the subarachnoid hemorrhage. Vasospasm is a delayed and often severe vasoconstriction that reaches a peak intensity and incidence around the seventh day after subarachnoid hemorrhage; it is caused by a combination of clot retraction, mechanical deformation, and the release of vasoactive substances on the regional arterial system adjacent to the bleeding site. Early and complete obliteration of the aneurysm allows for more aggressive treatment of this condition with the use of combinations of angioplasty and hypervolemic, hypertensive hemodilutional therapies. If problems with absorption of cerebrospinal fluid persist after vasospasm has passed, a permanent ventricular shunt may be required.

Outcomes after aneurysmal subarachnoid hemorrhage have substantially improved over the past 30 years, particularly in highly specialized neurosurgical centers with high-volume practices.⁵ Early intervention, aggressive treatment of hydrocephalus and vasospasm, emerging endovascular techniques, and refined surgical techniques such as approaches through the skull base and temporary clipping have contributed greatly to this trend. The induction of mild hypothermia during the interval of temporary clipping, as discussed by Todd et al. in this issue of the Journal (pages 135–145), represents an attempt to prolong the safe interval of focal circulatory arrest. Unfortunately, the methods used in the study by Todd et al. failed to produce significant benefits in this group of patients.