

need for loans and to adjust financial policies so as to reduce tuition. Schools that can increase grants and scholarships and develop subsidized loan programs will be able to ameliorate their students' debt — a solution, however, that depends on each school's ability to raise funds and increase its endowment. Most important, medical schools must take another look at the costs of education and determine how to halt the rising cost of tuition. This is a difficult task, since only a third of medical-school administrations have the authority to set their own tuition; in about half of U.S. medical schools, tuition is set by a board of trustees, and in others, it is set by the state legislature or other state authorities. A few medical schools have recently taken the bold step of capping their tuition; it is time for all medical schools to consider this option.

As more and more medical students accumulate more debt than many Americans amass in their lifetime, medical schools must take the lead in decreasing this financial burden. The future of our health care workforce depends on it.

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NEUROLOGY

Tales from the Temporal Lobes

Adam Zeman, D.M.

Ten years ago, the story I was hearing did not ring any bells. It went like this. During recent months, an engineer in his 40s had been having episodes of amnesia. Every so often, his mind would scramble: "Where am I? How did I get here? What am I meant to be doing?" This sense of bewilderment lasted for just a few minutes, during which time he failed to take in the answers offered to his insistent questioning. Some of the attacks occurred on awakening. A curious story: recurrent transient amnesia, affecting both retrograde memory, for the recent past, and anterograde memory, for ongoing events. I arranged some standard tests and wondered whether his symptoms might turn out to be psychogenic (a cop-out carrying the implication that I would soon be able to direct the problem elsewhere).

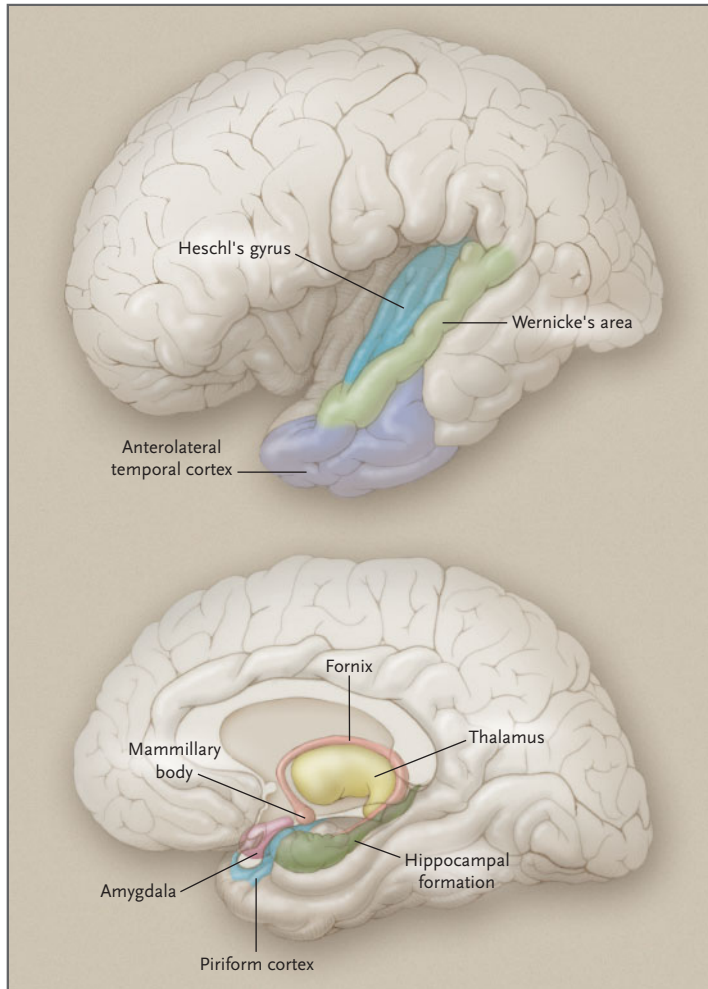
Charles Dickens wrote eloquently of the converse experience, a disconcerting excess of recollection — the sense "of what we are saying and doing having been said and done before, in a remote time —

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of our having been surrounded, dim ages ago, by the same faces, objects and circumstances — of our knowing perfectly well what will be said next, as if we suddenly remembered it." We call this feeling, which often carries with it the knowledge that our recollection is fictitious, "déjà vu." Most of us have it once in a while. When déjà vu can be tracked to its neurologic source, it turns out to originate in the temporal lobes, where epilepsy, among other things, can provoke this illusory sense of familiarity.¹ Temporal-lobe epilepsy was also the cause, as I later discovered, of my patient's transient amnesia.²

A woman is awakened by a premonition of sickening panic for the third night in a row. As she lies anxiously in bed, now wide awake and panting, a procession of disturbingly violent images passes through her mind. She feels she needs to piece these images together with the thoughts that travel with them, but she can't find a pattern — and then they are gone, as abruptly as they came.

A middle-aged man has grown accustomed to a curious state of mind that comes over him without warning. It combines, pleasantly enough, an indefinable smell or taste, the sense of a memory hovering at the fringe of consciousness, and a slight but



The Lateral Surface (Top) and Medial Surface (Bottom) of the Cerebral Hemispheres.

In the top illustration, part of the cortex has been omitted to expose the primary auditory cortex in Heschl's gyrus, and the rough position of Wernicke's area is indicated (authorities differ on its precise extent). The anterolateral temporal cortex is a region of the temporal neocortex that becomes atrophic in semantic dementia. The hippocampus, fornix, mammillary bodies, and parts of the thalamus contribute to the Papez circuit; bilateral disruption of the circuit causes severe amnesia.

unmistakable tug of sexual arousal. One morning, this familiar and mysterious experience is followed by a graceful slide to the floor and a few moments of unconsciousness.

Both of these patients also proved to have epilepsy arising from the temporal lobes. How can the electrical clamor of a seizure give rise to such well-developed experiences? What links distortions of memory, hallucinations of smell, emotions of panic, and sexual arousal to a single lobe of the brain?

The stereotype of epilepsy in the public mind is the grand mal (or tonic-clonic) seizure, characterized by loss of consciousness and tonic stiffening of the body, followed by the vigorous jerking of all four limbs, which lasts a minute or so. The attack results from an electrical rebellion: during the seizure, the highly differentiated electrical signaling that normally sustains our mental lives is replaced by monotonous, high-amplitude discharges, firing in synchrony across the brain. But electrical rebellion can be a local affair: the symptoms of a "focal" seizure illuminate the normal functions of the affected region of the brain. John Hughlings Jackson, the 19th-century father of British neurology, described the "double consciousness" that results, when the patient has a simultaneous awareness of the everyday world and the experiences — often bizarre and sometimes indescribable — arising from the seizure focus.³

Focal seizures are the most common variety of adult-onset epilepsy, and of the brain's four lobes, the temporal is most commonly the source. What do our temporal lobes do for us in health? The brain's massive interconnectedness implies that this question has no simple answer. Yet although almost every psychological act involves multiple areas of the brain, there is no doubt that specific regions are critical for specific functions. Allow me to use a broad brush: if the occipital lobe is an observatory scrutinizing the visual world, the parietal lobe an architect's office where space is mapped, and the frontal lobe home to a cabinet of war plotting our actions, then the temporal lobe is a monumental library equipped to catalogue, store, and retrieve the experiences of a lifetime.

This metaphor is an extravagant simplification, but the key role of the temporal lobes in memory is undoubted. The medial temporal cortex — including the hippocampus, a region with a relatively primitive "archicortical" microanatomy, named after its resemblance, when sectioned, to a seahorse — is required for the normal acquisition of conscious memories. Bilateral damage to this region causes lasting amnesia; epilepsy arising from it can cause both amnestic seizures and the sense of *déjà vu*. The lateral temporal cortex — the temporal "neocortex" — is associated particularly with the storage of "semantic memories," the database we constantly deploy in the recognition, naming, and use of our environment. Progressive atrophy of the temporal neocortex causes "semantic dementia,"⁴ a gradual depletion of our conceptual resources,

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 unfaltering, 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.

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Ruptured Cerebral Aneurysms

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Related articles, pages 135 and 146

The brain derives its blood supply from two internal carotid arteries, which supply most of the cerebrum, and two vertebral arteries, which merge to form the basilar artery and supply the brain stem,

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the cerebellum, and the visual cortex of the cerebrum. These vessels shed most of their external supporting layers as they enter the skull and are therefore considerably thinner and more fragile than vessels elsewhere in the body. On penetrating the dura mater, each vessel traverses the subarachnoid space at the base of the skull, where communications are established between the major trunks to form the circle of Willis (see diagram). The hemodynamic stresses (high pressure and pulsations)