NEUROSCIENCE

Widespread Cortical Networks Underlie Memory and Attention

David Gaffan

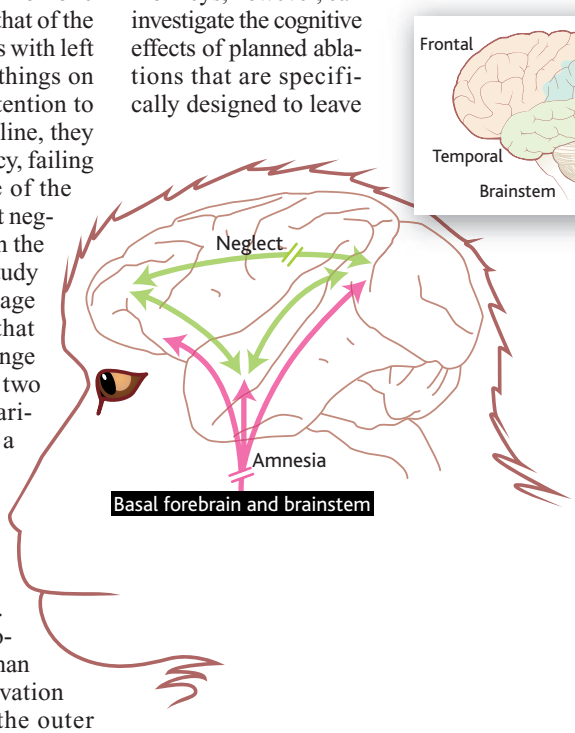
Circumscribed brain lesions can cause some profound but highly specific cognitive losses. One important example is anterograde amnesia, the inability to acquire new memories. This condition has been attributed to lesions in the temporal lobe, one of the major regions of the brain (see the figure). Another example is called neglect, the inability to sense, comprehend, and/or respond to stimuli on one side of space (the side opposite to that of the brain lesion). For instance, patients with left unilateral neglect will ignore all things on the left side of space and shift attention to the right side. If asked to bisect a line, they will do so with a rightward tendency, failing to even notice the left-hand side of the image. It has long been known that neglect is often produced by lesions in the parietal lobe of the brain. Now a study by Thiebaut de Schotten *et al*. on page 2226 of this issue (*1*) indicates that the phenomenon involves long-range functional connections between two brain regions—the frontal and parietal cortices—perhaps resolving a long-standing debate over the anatomical localization of the damage that produces this behavior. It also raises an interesting similarity with a recent proposed explanation for amnesia.

Thiebaut de Schotten *et al*. produced "temporary" neglect in human patients by brief electrical inactivation of the region that lies beneath the outer layer, or cortex, of the parietal lobe. Neuronal cell bodies constitute the cortical gray matter of the brain, whereas neuronal axons constitute the underlying white matter. This finding gives strong support to the idea that neglect results from the disruption of a widespread cortical network that involves neuronal cell bodies in large areas of cortex, including the frontal lobe, parietal lobe, and possibly the occipital lobe. This explanation of neglect, in terms of the white matter beneath the gray matter of the parietal cortex, stands in contrast to the traditional account of neglect that holds destruction of a specific set of neuronal cell bodies in the area of the lesion (the parietal cortex) accountable.

Naturally occurring lesions in the human brain usually involve both neuronal cell bodies in the cortex and subcortical axons. Experiments in monkeys, however, can investigate the cognitive effects of planned ablations that are specifically designed to leave white matter or gray matter intact. Ablation of large areas of the gray matter of the parietal cortex in the monkey, leaving the underlying white matter intact, does not result in neglect. However, neglect can be produced by cutting through the white matter, with only minimal damage to the gray matter (*2*). Evidence from the human brain reported by Thiebaut de Schotten *et al*. confirms that the cause of neglect is the same in the monkey and human brain.

Similarly, evidence for a white matter explanation of temporal lobe amnesia comes both from the human brain and from experiments with monkeys. Transection of the fornix, a subcortical white matter tract carrying axons to and from the medial temporal lobe, has quantitatively similar effects on memory in monkeys and in humans (*3*). A much more severe and dense amnesia than that produced by fornix transection is seen when other subcortical pathways, in addition to the fornix, are cut (*4*). These other pathways are damaged by naturally occurring lesions in the human brain only in conjunction with damage to medial temporal cortex itself. However, in the monkey, dense amnesia can be produced experimentally by a surgical procedure that sections the subcortical white matter pathways while leaving intact the medial temporal cortical areas [namely the hippocampus, the entorhinal and perirhinal cortex, and the parahippocampal gyrus (*4*)]. The crucial pathways that are interrupted in dense amnesia are thought to be the ascending axon projections from the basal forebrain and brainstem that pass through the anterior medial temporal lobe en route to widespread cortical targets (see the figure) (*5*, *6*).

The cognitive features of neglect and amnesia seem to be quite different from each other. However, recent evidence shows that neglect can be considered a failure to construct a representation of hemispace that is contralateral to the lesion, a representation that in normal function is based on memory retrieval just as much as on perception. Bisiach *et al*. (*7*) asked a left unilateral neglect patient to describe a familiar scene from memory, on two occasions from two opposite imagined viewpoints. The memory description omitted the left side of the scene as would be seen from whatever the current imagined viewpoint was. This is neglect in memory, with no input from current perception. Similarly, Hornak (*8*)



Long-range communications pathways in the brain facilitate attention and memory. Some long-range subcortical axonal pathways are illustrated on a lateral view of the left hemisphere of the macaque monkey cortex. Many ascending axonal pathways from the basal forebrain and brainstem (red) pass through a bottleneck in the anterior medial temporal lobe en route to their cortical targets. Cortical axonal pathways (green) pass through subcortical white matter to connect widespread areas of temporal, frontal, and parietal cortex. Interruptions to these long-range pathways (indicated by slashed lines) may underlie amnesia and neglect. (**Inset**) Lobes of the human cerebral cortex and cerebellum, as seen from the left side. The front of the brain is to the left.

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showed that a neglect patient's failure to explore the side of space contralateral to the lesion could be attributed to a failure to form and retrieve a representation of that side of space, rather than to any perceptual failure. Experiments with monkeys, in which one hemisphere of the brain was deprived of the visual information (although cortically completely intact) that would enable that hemisphere to form a representation of the contralateral side of space, gave further support to this representational account of neglect (*2*).

Neglect and amnesia are radically different clinical syndromes, and the point of this comparison is not to blur the distinction between them. Rather, the point is to suggest that widespread cortical networks spanning temporal, frontal, and parietal lobes subserve both memory and attention. The different clinical syndromes arise from different kinds of disruption to the long-range axonal communication among parts of the brain. This view contrasts with the traditional view of cortical localization of function, in which cognitive functions such as attention and memory are supposed to be subserved by spatially segregated areas of cortex. Understanding subcortical control of cortical plasticity in terms of widespread cortical networks, rather than assigning discrete parcels of cognitive function to discrete cortical areas, will enhance our current understanding of memory, learning, and other cognitive functions.

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PHYSICS

Double Quantum Dot as a Quantum Bit

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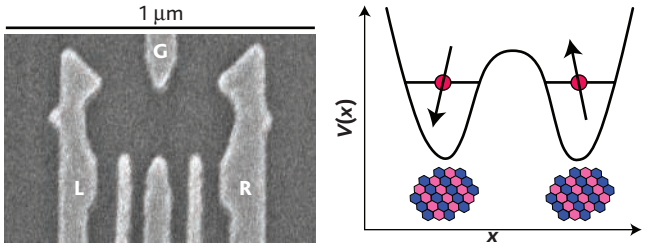
Quantum dots, solid-state structures that are capable of confining a very small number of electrons, have long been thought of as artificial atoms. With the help of these dots, the tools of device engineering can be used to dissect new atomic physics phenomena. Important advances in recent years have made it routine in several labs to construct the smallest possible dots, each holding exactly one electron. One might expect this artificial "hydrogen" to have extremely simple electronic properties. In fact, because the host crystal is the semiconductor gallium arsenide, the quantum properties of this artificial atom are different from those of its natural analog in one striking respect: The single electron spin, rather than being coupled to the spin of one nuclear proton, is coupled to about a million spins carried by the gallium and arsenic nuclei. This bath of spins has previously been a nuisance, in the sense that it has obscured the quantum coherence of the bare electronic spin. On page 2180, Petta *et al.* (*1*) report that they have used a double quantum dot—in essence, an artificial H_2 molecule—to tame the effect of the nuclear spins. The results suggest novel ways in which the physics of these nuclear spins may be put to use in the search for a viable quantum computer.

As a result of years of steady improvement, the double-dot device (see the figure) of Petta *et al.* is a superb system for precise control of this artificial H_2 molecule. This is accomplished via the electric potentials of the six electrical leads shown. Overall variation of their potentials (with respect to a ground) sets the number of electrons in the two dots. The low-lying electronic states of the two-electron system, as with natural H_2, consist of a spin singlet (S) and three spin triplets (T), in which the two spin $^1/_2$ electrons combine to form either a state of spin quantum number 0 (S) or 1 (the Ts). The energies of these states are tuned in a variety of ways: There is an externally applied magnetic field that splits the triplets. The gate potential (G) controls the tunneling barrier between the two dots. Increasing tunneling increases the energy splitting between S and T, because of the Pauli principle—a singlet can lower its energy by (virtual) tunneling of one of the electrons to the other dot, forming a temporary polarized state; but this state is disallowed if the spin configuration is a triplet. One can also vary the degree of virtual tunneling in an unsymmetrical way, by applying a voltage between electrodes L and R. The virtual tunneling then is only in one direction, but the result is the same: control (in fact, much more reliable control) of the singlet-triplet splitting.



Dot SWAP. Double quantum dot device used by Petta *et al.* (*1*) to coherently manipulate electron spins. G is the gate electrode that controls the barrier between the dots. Voltages on the L and R electrodes control number of electrons in the left and right dots, respectively. Pulsing the potentials on these electrodes causes a SWAP of the spin states of the two dots. [Adapted from (*1*)]

This splitting arises from an effective spin-spin coupling that is very aptly named the exchange interaction in physics, because it does really correspond to an interchange of spin states: As a function of time, $|$up-down$\rangle$ is converted to $|$down-up$\rangle$, and back again. The computer science terminology for this operation is SWAP. SWAP is a very useful primitive for quantum computing (*2*), because it can be done partially, in superposition. In fact, the exchange interaction permits all transformations of the form (*3*) $|a,b\rangle \rightarrow \cos(\theta)\,|a,b\rangle + i\sin(\theta)\,|b,a\rangle$ to be done, for any value of θ, where θ is proportional to the interaction time. (This equation emphasizes that any pair of spin states a and b, pointing in any direction, get SWAPPED, not just the states $|$up$\rangle$ and $|$down$\rangle$.)

If this were the end of the story, the engineering of the quantum computer could be initiated immediately: It is well known how to use "fractional SWAP," either alone or in conjunction with other simple primitives, to implement a quantum algorithm. But nuclear spins, the state of which is not under external control in the device shown in the figure, make the story more complicated, and interesting.

Because each atomic nucleus in the GaAs crystal carries a nuclear spin (with angular momentum quantum number equal to $3/\hbar 2$), a simple calculation shows that the wave function of a single electron in one quantum dot has appreciable overlap with

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