## Cognition without a Neural Code: How a Folded Cortex Might Think by Harmonizing Its Own Electromagnetic Fields

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## ABSTRACT

Extensive investigation of the brain's synaptic connectivity, the presumed material basis of cognition, has failed to explain how the brain thinks. Further, the neural code that purportedly allows the brain to coordinate synaptic modulation over wide areas of cortex has yet to be found and may not exist. An alternative approach, focusing on the possibility that the brain's internally generated electromagnetic fields might be biologically effective, leads to a model that solves this "binding problem." The model of cognition proposed here permits mind and consciousness to arise naturally from the brain as trains of signifying states, or stationarities. Neuronal circuits in suitably constructed hierarchies produce thought by reconciling themselves with each other through the forward- and back-broadcast of specific electromagnetic fields, executing a natural algorithm as a harmonized set is selected. Beyond the postulation that information is encoded in specifically organized electromagnetic fields, the only other "code" necessary is topographic, one that is already known. That the brain might use its own fields to think is supported by the literature on the widespread sensitivity of biological organisms to small, windowed fields. This model may help explain the coherence of the brain's fields, the conservation of the folded cortex, and, in its emphasis on a self-harmonizing process, the universality of the esthetic impulse as a projection of the brain's basic mechanism of thought.

# COGNITION BASED BOTH ON FIELDS AND ON SYNAPTIC CONNECTIVITY

ny credible model of thought must embody in a natural way the known physical features of the human brain. Such a model will encode a myriad of memory traces, and it will assemble them rapidly and reliably, excluding extraneous matter without having to consult any list of rules, for which there is no time or place. The model will automatically focus attention where attention is needed, and it will spontaneously apply an internal logic that leads to survivable behavior. At the same time, it will demonstrate the human capacity for loose associations, self-contradictions, and even wild delusions. The model must be self-reading (no homunculi allowed), self-validating, and self-conscious. A model of human cognition should perform as the brain does; it should be at once reliable and suspect, esthetic and erratic.

Most such models rest on synaptic connectivity. Disappointingly, the study of neurons and their connections in the cortex has produced much detailed physiology and many wiring diagrams but no convincing model of how the cortex makes an image, let alone a thought (Felleman and Van Essen 1991; Van Essen 1997).

Neuroscientists have demonstrated that the brain parses its tasks into multiple subunits, all of which involve toand-fro axonal conduction followed by modification of synaptic interconnectivity, and they argue that after abundant "parallel processing" the brain somehow integrates what it has previously divided. But it is not clear how interlocking webs of neurons can represent one discrete "subunit" in an instant while suppressing all other representations. An even deeper mystery is how modification of synapses while thought is in progress can turn a mass of electrical activity into thought. Synaptic change does not proceed at the rate humans think, but over many seconds and even over days and months (Donoghue 1995). While synaptic modulation is indispensible for learning, how does this learning actually help make thought?

Even if brisk synaptic modulation were possible, say by rapid functional changes rather than by structural remodeling (which requires time-consuming protein synthesis), we are still left with the "binding problem," the chief failure of cognitive neuroscience (Treisman 1996). The idea of synaptic modulation simply does not possess the power to explain how the brain synthesizes its "massively parallel processing." Consider, for example, the work of Gerald Edelman (1987, 1992). He has built an admirable model of how different domains of representation within the brain "map" themselves onto each other. The various domains stimulate each other and thereby mutually adjust, modifying their synapses accordingly. Edelman calls his model the "theory of neuronal group selection," or TNGS, drawing on Darwinian ideas of mutual accommodation. Through reciprocal

signaling along axons, synapses are fine-tuned, and a group of firing units is selected. This selection provides the material basis of thought.

Because TNGS requires a plethora of axonal signaling from one unit to another it requires time. We can get a rough idea of the time pressure on TNGS by considering a batter trying to hit a baseball. How much time does he have for central processing? A fastball pitched at 100 mph arrives at home plate in about 410 ms. Time is needed for visual input: at a minimum the 33 ms required to glimpse and distinguish one numeral from another (Saarinen and Julesz 1991), if not the approximately 100 ms needed to register a visually evoked potential. On the output side, an expert needs about 160 ms to swing a bat. This leaves at most 200 ms for central processing (Endo et al. 1999; Regan 1997). With practice, a batter can train his motor cortex, refining synaptic structures over years, and he can guess pitches in real time, but he cannot know a pitch's type or location. He has 200 ms to

decide to swing or not, to craft and launch a swing, an impressive feat, given that the "what" (curve?) and the "where" (over the plate?) of vision are processed separately (Ungerleider and Haxby 1994).

There is simply no known mechanism by which axonal messaging and synaptic modulation can go that fast, even if we allow for functional rather than structural changes. The nervous system contains both electrical and chemical synapses: the former devoted to rapid reflexes, the latter to slower, more modulated responses (Kandel et al. 1991). While electrical transmission across a synapse is almost instantaneous, the gap junctions on which this kind of transmission relies are not readily modified. In contrast, the chemical synapse, which can be modified and thus serve as a basis for memory and learning, requires a minimum of .3 ms of synaptic time, often up to 5 ms. TNGS, which rests on sets of discrete modulations of synaptic connectivity, inevitably requires much synaptic time, say 20 ms for five instances of mod-

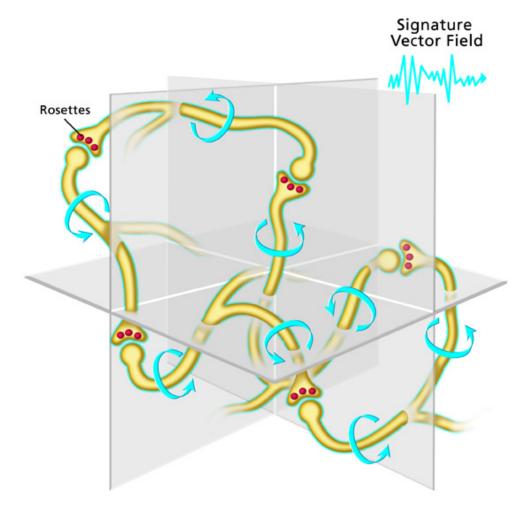


FIGURE 1: A three-dimensional neuronal circuit anatomically fixed by rosettes. According to the right-hand rule, this circuit will automatically broadcast a signature electromagnetic field, determined by the course of its current.

ulation after round-trips between two sets of coordinated neurons (a small number of round-trips, since any one center is already modulated by third parties before it receives news back from a center it just signaled). In addition, there is the time spent conducting along axons. Even if we assume optimal compaction (Cherniak 1994), the magnetoencephalographic evidence cited in support of TNGS shows coordination between areas separated by 10 cm or more (Gaetz et al. 1998; Srinivasan et al. 1999). With a maximum conduction velocity in lightly myelinated neurons of 8 m/s, five round-trips along 10 cm of axon take 125 ms. Next, there is the membrane constant, the time a neuron spends summing its multiple synaptic inputs, another 8 to 20 ms (Kim and Connors 1993; Shadlen and Newsome 1994) for each summation, say 100 ms for just five round-trips between two interconnected neurons with other inputs. The time requirements of axonal conduction, synaptic transmission, and membrane constants exceed what is available to hit a baseball, without any time spent on the synaptic modulation that would be necessary to create the cortical representation of a discrete swing. TNGS works too slowly to explain how the cortex assembles its output, and so do other models based on synaptic modulation and axonal conduction. In contrast, a model partially based on fields allows much of the brain's computation to proceed instantly.

Further, the schema of integration in TNGS, abstracted from its physical basis, is not sufficient to serve as a model of thought. Edelman rightly insists that the world is unlabeled and therefore not categorized a priori, that the brain "synthesizes" patterns and does not take them from the world; but he deprives his model of power when he insists that it run on "value" and "not logic," that "the cognitive science view of the mind based on computation or algorithmic representations is illfounded" (Edelman 1992). But thought is roughly logical. What is needed is a model possessing a natural logic, not the absence of logic. Yet no logic is currently available to explain how neurons come to a signifying consensus (Shadlen and Newsome 1994). A model using fields can provide the logic and obviate the elusive "neural code."

That the rapidly calculating brain might use a process different from synaptic modulation to achieve a running series of coherent states is strongly suggested by studies of other human activities severely constrained by time. The processing of spoken language at the approximate rate of one word every 250 ms requires the coordination of widely dispersed areas of the brain from both hemispheres (Damasio et al. 2004), and there is no explanation of how axonal messaging and consequent synaptic modulation might accomplish this daunting task in the time available. But there is evidence that trains of coherent electromagnetic states, in rolling windows of about 200 ms, can help constitute the material basis for understanding speech (Luo and Poeppel 2007). The model herein rests on the very fields recorded by electro- and magnetoencephalograpy, with the novel hypothesis that complex brains not only broadcast these fields but read them as part of the process that produces thought. Because the brain's patterned electromagnetic fields depend on the connected neurons that generate them, the synapse also plays a role in the model. No theory of the brain can be complete if it does not account for the brain's connectivity. But, according to the view taken here, no theory of higher brain function can be complete if it does not explain why the brain broadcasts coherent fields that correlate with cognition. Lashley, never persuaded that patterns of synaptic connectivity alone could explain thought, failed to confirm any theory of cognitive function based on field theories, including those of Kohler (Lashley et al. 1951). Lashley's failure convinced many that field theory is a dead end. But experiments continue to demonstrate a relationship between the brain's thoughts and its fields (Freeman 2004a, 2004b, 2005; Freeman et al. 2003).

### **BIOLOGICALLY EFFECTIVE ELECTROMAGNETIC FIELDS**

Azanza and del Moral (1994), who review the large subject of the biological effects of magnetic fields, conclude, "The paramount result, in our opinion, is the ubiquitous response of biological systems, of any complexity, to magnetic fields, which shows that magnetosensitivity is, indeed, a general phenomenon in living systems." Since the beginning of biological time on earth, some primitive bacteria have used magnetosomes to align themselves in the earth's magnetic fields (Bazylinski et al. 1994). Bacteriorhodopsin evolved more than three billion years ago as a hydrogen-ion channel responsive to electromagnetic fields in the visible range, a cousin of rhodopsin (Zuber 1986). Ciliates change their swimming patterns when exposed to a magnetic field, an effect almost certainly due to the opening of calcium channels (Hemmersbach et al. 1997).

Sharks navigate and hunt with ampullae of Lorenzini, a sensing organ responsive to tiny changes in the earth's magnetic field and to nanovolt fields produced by prey (Paulin 1995), thus demonstrating the capacity of highly evolved nervous systems to respond to the tiniest of fields. Birds, bees, and rats sense the earth's tiny magnetic field (Able and Able 1995; Burda et al. 1990; Walker and Bitterman 1989; Wiltschko and Wiltschko 1988). Exposure to extremely low-frequency electromagnetic fields improves social recognition in rats (Vazquez-Garcia et al. 2004). Human beings placed in certain tiny magnetic fields show measurable changes in heart rate and encephalographic patterning (Graham et al. 1994).

For eons, biological organisms have responded to external electromagnetic fields. I suggest that human brains, as well as brains of other species, respond to *internally generated* fields. The model I construct implies that

# Compound Signature Field from Domain 1 Chemical signal from Rosettes Lateral Geniculate Nucleus R Neuronal circuit emitting unique Domain 1 electrical signal Domain 2 Domain 3

#### How a Folded Cortex Might Think by Harmonizing Its Own Electromagnetic Fields

FIGIRE 2A: schematic view of effective broadcast and back-broadcast. Each domain possesses its own class of rosettes and antennas, so that domain 1 cannot pick up broadcast intended for domain 2. This is indicated by distinct class markings for the circuits of each domain. Figs. 2a–2e illustrate an evolving stationarity among three hypothetical domains, which are for clarity teased apart, though in the brain they would likely be interlaced, thus optimizing local field strength. See text.

the evolutionary adaptation that permits the brains of higher species to achieve cognition and consciousness is the ability of the cortex to read its own fields.

### FIELDS IN THE BRAIN

Magnetoencephalography (MEG) records rhythmic, coherent magnetic fields, generated mainly by pyramidal cells, which constitute roughly two-thirds of cortical neurons (Gallen et al. 1995). The recorded fields are the result of the coordinated firing of many thousands of these cells, mostly located in layers III and IV, 2 or 3 mm below the cortical surface. In general, the underlying rhythms of MEG reflect the synchronous activity of these neurons (Hamalainen et al. 1993; Lopes da Silva 1991; Nunez 1986).

This synchronicity is important to the model, which depends on the replicable superposition of fields from distinct sources. All circuits relevant to a thought must keep a beat. Recordings of electromagnetic fields indeed demonstrate this property (Gray et al. 1989), with the 10-beats-per-second rhythm commonly seen over occipital regions on EEG also seen on MEG (Cuffin and Cohen 1979). Similarly, the 14-beat-per-second rhythm seen on EEG during sleep ("sleep spindles") has an MEG analog (Nakasato et al. 1990). Various components of the cortical representation of the motor program are each associated with a distinct frequency, mainly in the 10-, 20-, and 40 Hz ranges (Hari and Salenius 1999). These rhythms, generated by the thalamus, also correlate with consciousness and cognition both in normal and in pathological states (Llinas et al. 1998, 1999). The model proposed here depends on, and assigns specific function

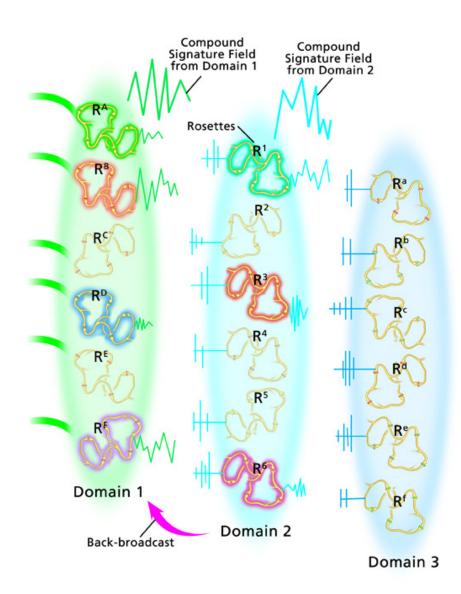


FIGURE: 2B

to, the brain's production and conservation of *rhythmic* activity.

The second critical property of recorded electromagnetic fields is their tiny amplitudes. Is there enough energy in measured fields to be biologically effective? The magnetic fields, recorded from 3 cm above the surface of the scalp, are in the picoTesla (pT) range. The cortex is another 1.5 cm still deeper, and the fields that can be picked up are only those that are perpendicular to the scalp. This means that only neurons oriented parallel to the scalp generate the recorded fields, and these neurons tend to be in the walls of cortical sulci, still farther from sensors above the scalp. A field of 2 to 6 pT, 4 cm from its source, will be 1,600 times stronger at 1 mm, in

the range of 25 to 100 nT, within the known range of biologically effective fields, as we shall see.

The third important feature of recorded electromagnetic fields is their tendency to fall into a continuous series of brief coherent states, each one different from the next. This quality can best be illustrated with data from EEG studies of "microstates" (Koenig et al. 1998; Lehmann et al. 1987; Strik and Lehmann 1993). Microstates are patterns of coherence in electromagnetic fields that last from 50 to 250 ms and correspond with cognitive states. This kind of time-dependent coherence is now demonstrated on MEG as well, with perceptions correlated to patterns of magnetic fields (Srinivasan et al. 1999). Nouns and verbs elicit specific and distinguishable microstates

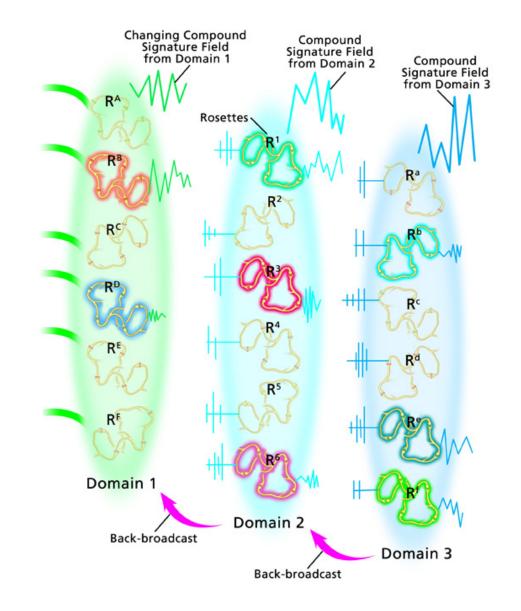


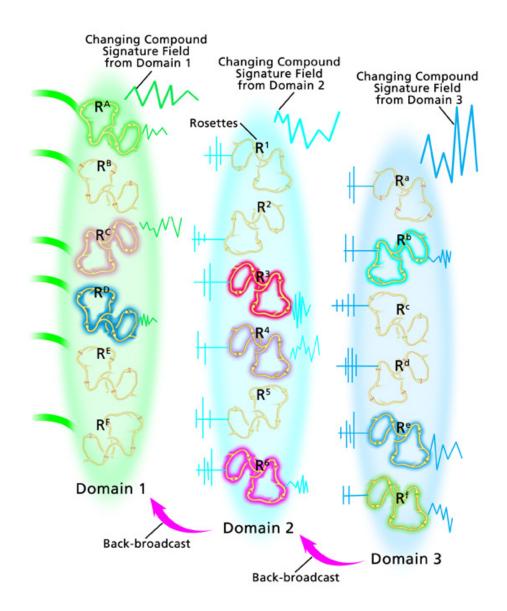
FIGURE: 2C

(Koenig and Lehmann 1996), and so do visual images as compared to abstract thoughts (Lehmann et al. 1993); even the perception of different odors falls into distinguishable microstates (Harada et al. 1996). The model presented here assumes that these microstates constitute part of the physical basis of thought, not merely the by-products of thought.

### THE PARTS OF A HYPOTHETICAL THINKING MACHINE

The model is built from four parts. The first is composed of three-dimensional neuronal circuits, neurons joined together in the cortex according to the usual understanding of synaptic connectivity, with the provision that the neurons circle back and form a closed loop. For the purpose of modeling, these circuits are treated as a unit made up of a single necklace of neurons, though in fact they are ropes.

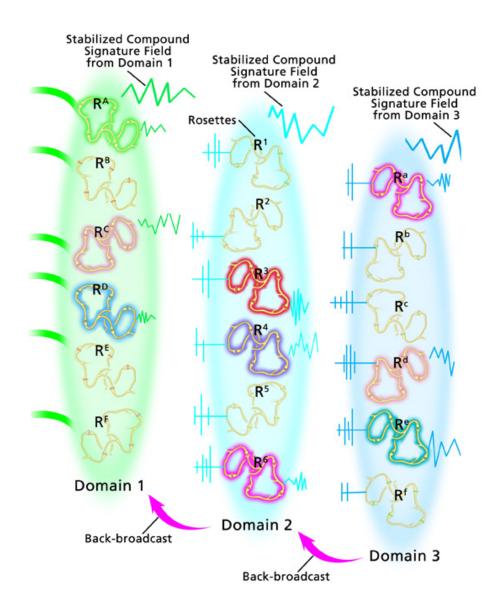
These circuits are fixed in highly specific and permanent anatomical arrangements by the second component of the model, the secretory rosette, an organelle made up of intramembranous particles. The rosette takes up its position in the presynaptic membrane and does not move because it is anchored into the cell's submembranous scaffolding (Heuser et al. 1979, 1974). These secretory rosettes are found throughout all phyla. They are particularly well studied in the paramecium *Tetrahymena* (Satir 1980), in the frog neuromuscular junction (Ceccarelli et al. 1979a, 1979b), and in the rat sensorimotor cortex (Bozhilova-Pastirova 1998), and they



#### FIGURE: 2D

are also found in the human brain (Heuser and Reese 1977). Once these secretory rosettes are deployed in a specific pattern, their positioning and their number are carefully preserved. Since rosettes determine where synaptic vesicles may dock and discharge neurotransmitters, they can fix a pattern of discharge that will repeat itself, a microfragment of memory (fig. 1).

These anatomically fixed circuits inevitably broadcast electromagnetic fields. I hypothesize that they are biologically effective—the third component of my model. Each fixed circuit beams its own signature of electromagnetic fields. These fields obey Maxwellian principles of superposition. If brain-generated fields have effects, they must have receptors. A class of receptors sensitive to the brain's signature magnetic fields constitutes the fourth component of the model. These receptors, postulated to come in a vast variety like immunoglobulins, would attach to ion channels. Each receptor would reverberate to a specific pattern of broadcast, just as each immunoglobulin molecule responds to a particular antigen. When activated, these antennas would open the ion channels to which they are attached and fire the associated neuronal circuits. This system of ion channels, which can be called *vector-gated*, would be distinct from ligand-operated, voltage-gated, and tension-gated ion channels, but similar to the others in the capacity to initiate an action potential.



#### FIGURE: 2E

There is now good evidence that an immunoglobulinlike class of receptors does exist at mammalian cerebral synapses. These are protocadherins, members of a super family of molecules that bind one cell to another (including neurons) and facilitate communication, both during embryogenesis and afterward. In the mouse brain, one type of protocadherin, called the cadherin-related neuronal receptor (CNR), comes in a "striking" variety, with variable and constant regions, just like immunoglobulins (Wu and Maniatis 1999). The CNR even demonstrates "a folding topology that is remarkably similar to that of immunoglobulin-like molecules," distinct in evolutionary origins but "convergent on a common folding pattern" (Fannon and Colman 1996). Fannon and Colman emphasize the cadherins' selective guidance of one cell's adherence to another as an early step of locking in a functional synapse. Kohmura and colleagues, noting the diversity of these receptors, as well as their spanning of the postsynaptic membrane, conclude that CNR family members might "enhance highly differentiated neural networks and behaviors" (1998). I propose molecules like CNRs, as receptors for signature magnetic fields, capable of opening ion channels and firing neurons.

Additionally, the model requires that the secretory rosettes themselves be stabilized by the back-broadcast of vector fields, as described below. This stabilization would be similar to the chemical stabilization of the secretory rosettes by antidiuretic hormone in the kidney's collecting tubules (Stern et al. 1982). The capacity

of magnetic fields to rearrange rosettes has actually been documented (Bersani et al. 1997) and, since rosettes are almost certainly calcium channels or calcium-associated potassium channels (Pawson et al. 1998; Pumplin et al. 1981; Roberts et al. 1990; Robitaille et al. 1990; Robitaille and Charlton 1992), they can probably be stabilized in the way that tiny magnetic fields, when applied to ciliates with mutant calcium channels, stabilize these channels and improve swimming (Hemmersbach et al. 1997).

To summarize, the four components of the proposed apparatus are: neuronal circuits; secretory rosettes; vector-gated ion channels; and effective broadcast and back-broadcast of electromagnetic fields. An intelligent apparatus requires not only components but an intelligent design. Thus the proposed apparatus possesses functional domains, arranged hierarchically, so that lower domains broadcast to higher domains. Each domain possesses its own subclass of rosettes and its own subclass of vector-gated ion channels. The rosettes and the antennas of each subclass come in a large variety, like immunoglobulins. Neuronal circuits in each domain broadcast a superposition of signature fields to the domain above it in the hierarchy, thereby opening selected vector-gated ion channels and not others. In addition, neuronal circuits in each domain are stabilized by back-broadcast from the domain above. This permits the execution of a natural algorithm implicit in Darwin's theory of natural selection.

## THE NATURAL ALGORITHM

Darwin answered his critics who claimed that a natural system, operating by chance, would fail to exhibit the logic needed to produce the "beautiful ramifications" visible in the world. He described the required natural algorithm and natural logic, the critical feature of which is to allow all creatures to interact with each other and with their environment, so that each being shapes the destiny of others while submitting itself to the influence of others. What survives is what falls into mutual adaptation. The present model requires an immense speed-up in the Darwinian algorithm in the brain itself, so that it works at the pace of thought. Each thought emerges as a pattern in an apparatus whose state can and does "evolve" rapidly. Just as in a Darwinian ecosystem, the current model features a brain that produces results that are coherent and workable, sometimes even beautiful, but not necessarily logically consistent. The model features a "fuzzy," nonmonotonic logic (Marek et al. 1990, 1992). Whatever falls into a salient pattern is "true," automatically validated, just as in the evolution of humans, in whom the senseless male nipple is "validated" as part of a design with overall good sense.

The model that follows is analogous to TNGS. But since it requires no time-consuming synaptic modulation during the making of thought, and only minimal axonal signal-

ing, it can act with the necessary speed. Each "thought" produced by the model must emerge from the teeming neurochemistry of the brain as a coherent pattern with stability and a specific identity, like the microstates measured by MEG. A discrete identity achieved by a vibrant system bereft of equilibrium is called a *stationarity*. The classical example is a vibrating piano string. Another example, on a longer time scale, is the state of the ecosystem at any time. Each of Darwin's generations is a stationarity, a coherent pause in a system that will move on to new stationarities. Similarly, in the model at hand, a thought emerges as a stationarity, one esthetically sculpted from its background by a natural algorithm, only to give way to another.

## THE MODEL AND COGNITION

Let us present a Necker cube to the apparatus sketched above (Necker 1832). The Necker cube, a stick figure that can be perceived in either of two configurations, is useful precisely because it demonstrates that the brain assembles images from fragments, rather than retrieving them as whole files. Observers of the Necker cube, when formally studied, first make one version of the cube, then the other, with rates of assembly increasing with observation time (Brown 1955). From the study of dreams, false memories, and illusory conjunctions (Treisman and Schmidt 1982), it also seems clear that the brain indeed works by assembling fragments. In any case, the amount of storage in the brain is enormously enhanced if it does assemble images rather than retrieve them whole, as we shall see.

Let us begin with the Necker cube mapped onto the retina with throughput from there to our apparatus's version of the visual cortex, along the usual pathways. Simple information, topographically coded, passes to the lateral geniculate nucleus and from there to the calcarine cortex. This information is assumed to be of the type described by Hubel and Wiesel in cats (Hubel and Wiesel 1962), mapping edges, corners, verticals, and horizontals to specific circuits in domain 1 of the model's version of the visual cortex (see fig. 2). The information enters there by stimulating the usual ligand-operated receptors, which are attached to an initiating neuron in a three-dimensional circuit like that in figure 1, represented in small in figures 2a-e. The symbol R-- indicates the various subclasses of rosettes, with each domain possessing its own subclass and each rosette subtly different from the next. The situation at the beginning of the operation that will build an internal representation of the Necker cube is represented in figure 2a. Some of the neuronal circuits are stimulated by the specific blend of incoming axons, while others are not, though they would respond to other stimuli. Circuits that respond to a topographic pattern now firing in response to the Necker cube are highlighted in figure 2a.

The active circuits in domain 1 automatically broadcast their signature fields, and these fields automatically superpose, forming a specific composite of all the fields currently active in domain 1. The superposed fields broadcast to domain 2 where, again, there are many circuits, all fitted with varying antennas on vector-gated ion channels. These antennas, given the symbol -l-l---, are specific to this domain only. Because they vary, they respond differentially (or not at all) to composites of vector fields coming from domain 1. (The variety of antennas is indicated by slight variations in the symbol.) The responding receptors fire certain circuits in domain 2, specific to the composite signal coming from domain 1, as in figure 2b. These active circuits (highlighted) in domain 2 automatically broadcast to domain 3 and back-broadcast to domain 1. The forward-broadcast stimulates certain circuits in domain 3, and the backbroadcast stabilizes certain, but not all, of the active circuits in domain 1, as in figure 2c. The stabilization is accomplished by maintaining the viability of some rosettes but not others, depending on their subtle differences. In the figures, back-broadcast is indicated by curved arrows. Note that back-broadcast from domain 2 to domain 1 "focuses" domain 1's representation of external reality. This feature is built into the model and is a requirement of any model that hopes to capture the human capacity to zero in on "relevant" features of the environment, as in a listener's (still mysterious) capacity to isolate a single speaker amidst the din of a party.

The stabilization of some but not all of the circuits in domain 1 alters the composite firing from there, and this in turn alters the firing pattern of domains 2 and 3, as indicated in figure 2d. Forward- and back-broadcast lead to further adjustments, as in figure 2e, and, eventually, this to-and-fro broadcast comes to rest as a cycling stationarity. That is, since there is a finite number of possible states, and since the system is adapted to fall into order quickly, it soon cycles in a stationary pattern. In this model, the "two" Necker cubes are represented by two distinguishable stationarities in the apparatus. The apparatus "knows" that a stationarity has been achieved presumably when the persistence over sufficient time of a coherent set of fields triggers validation and consciousness.

The huge capacity of such a system is readily apparent. If we assume a mere hundred circuits in each domain, and if just three of 100 circuits are active in domain 1 after stationarity is achieved, then the number of possible patterns in domain 1 is 100 x 99 x 98, approximately 10^6. If the other two domains are similar, the apparatus possesses a representational capacity of 10^18 patterns, enormously robust. A multitude of "thoughts" can thus arise in a relatively constant apparatus, though experience will, through synaptic modulation, change the apparatus over time periods much longer than the instant it takes to generate a single stationarity.

Suggestive support for the view that coherent fields are integral to the perception of the Necker cube comes from Gaetz et al., who have demonstrated specific and "coherent MEG activity . . . over occipital, parietal and temporal regions . . . related to the perception of Necker cube reversals" (1998). The two percepts of the Necker cube arise as distinct patterns of firing in synaptically fixed neuronal circuits, but because each pattern forms via broadcast of effective fields, not via synaptic modification, the model explains how the percepts can interchange so quickly. Only minimal axonal activity is necessary. The only neural code needed here is the known topographic code from the retina. The "two" Necker cubes are the two "best fits," given the topographic pattern of inputs to the field-generating and field-reading apparatus. Circuits that don't fit together are automatically silenced.

The pattern of the whole stationarity, not just the pattern in the highest domain, constitutes the internal representation of the Necker cube in one of its orientations. Thus, there are no "grandmother" cells in this model, no higher-order cells whose firing might somehow represent an integration of lower-order firing. The Necker cube is represented in its vivid instantiation in domain 1, while its associated qualities are simultaneously embodied in domains 2 and 3. In this view, areas distant from each other would not be recruited by neural code but would be set in motion by secondary topographic projections, similar to those coming from the lateral geniculate nucleus to the calcarine cortex.

If effective biological broadcast is a basic mechanism of thought, the brain's well-documented habit of building coherent states of rhythmic electromagnetic fields takes on a more precise physiologic function. The cerebral rhythms that we find on EEG and MEG are present and conserved in several species in order to facilitate reproducible Maxwellian superposition, and the brain's coherent fields exist because they are essential components of thought, not mere by-products of thought. Similarly, the conservation of the folded cortex takes on a precise anatomic purpose. Vicq d'Azir's observation two hundred years ago that the pattern of folding in the human cortex is conserved (Schiller 1965) has never been well explained. Van Essen (1997) suggests that the conserved folding of the visual cortex allows two functionally related areas to communicate through a minimal length of white matter, with V1 and V2 backto-back on walls of the same gyrus. However, this view cannot be generalized to the entire cortex, where all is connected to all. But if one allows effective fields, then a folded cortex, with a conserved pattern, would be evolutionarily useful in that it would enlarge any area's range of broadcast. One region of cortex could integrate with another across sulci, thus explaining why, for example, the "what" of vision must reside across the superior temporal sulcus from the "where" of vision.

## SOME OBJECTIONS

The model requires an array of molecules responsive to specific signatures of superposed magnetic fields. Two problems immediately arise. First, can the postulated receptors pick up the tiny electromagnetic fields that the brain generates? The earth's (static) magnetic field is about 50  $\mu$ T, and the human brain does not seem to register it. Magnetic fields in the brain are in the nT range. Can the brain detect fields three orders of magnitude smaller than the earth's puny ambient field?

The second difficulty arises from the opposite end of the scale: the saturation problem. If the brain reads its own tiny fields, why is it not swamped by large fields, such as those generated by magnetic resonance imaging, up to and beyond 3 T?

The answer to both objections lies in the experimental data, which show that modulated (nonstatic) fields of certain frequencies and certain tiny amplitudes can have effects when frequencies and amplitudes outside the effective window have none, even when the effective fields are seemingly below expected thresholds. On pure biophysical grounds, the minimum biologically effective magnetic field should be in the range of 6 mT, four to six orders of magnitude too high for magnetic fields in the brain to have any effect, "unless large, organized, and electrically amplifying multi-cellular systems such as the ampullae of Lorenzini . . . are involved" (Weaver et al. 1998). But there is every reason to suspect that the brain is as finely organized as the shark's organ of navigation. Indeed, we know that biological tissue is far more sensitive to electromagnetic fields than would be predicted by Weaver's threshold. Bees can be trained to detect the source of a weak magnetic field down to a threshold of 25nT (Walker and Bitterman 1989), and the severed trigeminal nerve of the bobolink can respond to fields down to 200 nT (Semm and Beason 1990). Kobayashi and Kirschvink (1995) sketch possible mechanisms of signal amplification that would allow organisms to surpass Weaver's limit.

Azanza and del Moral favor the molecular solitons of Davydov (Davydov 1979) as the "most powerful mechanism" of nonlinear signal amplification. A soliton is a highly localized wave that draws and condenses energy from surrounding sources. Davydov postulated a very weak, "magnetically correlated" field that would perturb the polar heads of lipid molecules in a cell membrane. The energy would funnel into a single protein protruding through the membrane, rendering it capable of resonating in just the right way to modulate a calcium channel and thereby allow a triggering influx of ions. Davydov's model has the advantage of resembling the chlorophyll and rhodopsin arrays of antennas, which condense extremely low energies into a resonant chain of proteins. Like rhodopsin and chlorophyll, and like the model described herein, Davydov's model responds only

Since the review by Azanza and del Moral, at least three more mechanisms for amplifying weak magnetic signals have appeared. One draws on stochastic resonance (Galvanovskis and Sandblom 1997). A randomly opening and closing calcium channel is stabilized in an open position by a sinusoidal magnetic wave of low amplitude. A more robust amplification might be achieved by a system in which oscillating calcium channels are resistant to incoherent noise but are "extremely sensitive to coherent and periodic perturbations" (Eichwald and Kaiser 1995). A third mechanism invokes the Hall effect, the nonlinear bending of a current by a tiny magnetic field placed across it (Balcavage et al. 1996). Thus physics does not forbid *in principle* the hypothesis that the brain registers its tiny fields.

But what about the saturation problem, those huge fields that have no biological effects? The answer lies in the same windowing phenomenon demonstrated in experiments summarized by Azanza and del Moral. For example, studies of chicken embryogenesis (Delgado et al. 1982) showed a "'window' effect for both frequency and intensity variables-the intermediate values of 100 Hz/1.2  $\mu$ T being more effective than any other values used (10, 100, and 1000 Hz frequencies were applied with intensities of 0.12, 1.2, and 12 µT)." Bawin and colleagues (Bawin et al. 1978) and Adey (1988) add other examples of magnetic fields operating within narrow windows. Similarly, Graham and colleagues (Graham et al. 1994) exposed three groups of normal humans to various combinations of low electric and magnetic fields. In the group exposed to 9kV/m and 20  $\mu$ T there was a significant alteration in an evoked EEG response to a socalled oddball task. These effects were not observed in those exposed to lower fields of 6kV/m and 10 µT, nor in those exposed to higher fields of 12 kV/m and 30 µT. Thus, high-Tesla MRI machines may lie outside biologically effective windows.

## CONCLUSION

This model of thought uses the biologically effective broadcast of electromagnetic fields to bring into coherence a set of cortical circuits that previous learning has facilitated. The circuits constitute reproducible analogues of fragments of thought, fixed in specific three-dimensional pathways by synaptic rosettes and hierarchically arranged in domains. These circuits generate fields with distinct signatures in narrow windows, which the apparatus spontaneously registers, just as many organisms register tiny, externally generated fields. These signature fields obviate the problematic search for a neural code and for a mechanism of rapid synaptic modulation. The only *neural* code necessary for the model is topographic, which is the only code known to exist in the brain. The model draws on Darwin's natu-

ral algorithm to shape a thought and distinguish it from others that might have been assembled by the same apparatus. Through an unconscious process, what fits together emerges as a conscious "thought." The model is rapid, robust, generally reliable but sometimes erratic, just as human beings are—because whatever evolves into a stationarity is automatically validated, "rational" or not.

The model's potential for malfunction of its parts, not yet explored, may shed light on unexplained thought disorders. It offers an explanation for the existence and conservation of the brain's coherent rhythms and for the conservation of the folded cortex's pattern. And its mechanism, requiring a harmony of parts, may help explain the universality of the human esthetic impulse as an externalization of the brain's basic procedure.

The model can be tested. One can test whether or not neurons have field effects on other neurons to which they are not connected. One can test Wu and Maniatis's cadherin-like receptors, currently without any known physiologic function, for sensitivity to electromagnetic fields. Synaptic rosettes can be further subjected to such tests. One might search for externally applied fields with specific, windowed signatures that can evoke or disrupt thought. One can extend the experiments that already purport to show that small, externally applied electromagnetic fields have effects on sleep (Graham and Cook 1999) and cognition (Crasson et al. 1999; Graham et al. 1994; Sandyk 1999).

#### SYMBOLS

## <i> <i> = italics

- $10^{6} = 10$  to the sixth power
  - T = Tesla
  - $\mu$  = micro, as in  $\mu$ T = microTesla
- -I-I-- = vector-gated ion channel
- **R**-- = rosette-fixed synapse

#### REFERENCES

Able, K. P., and M. A. Able. 1995. Interactions in the flexible orientation system of a migratory bird. *Nature* **375**:230–32.

Adey, W. R. 1988. The cellular microenvironment and signaling through cell membranes. *Prog. Clin. Biol. Res.* 257:81–106.

Azanza, M. J., and A. del Moral. 1994. Cell membrane biochemistry and neurobiological approach to biomagnetism. *Prog. Neurobiol.* **44**: 517–601.

Balcavage, W. X., T. Alvager, et al. 1996. A mechanism for action of extremely low frequency electromagnetic fields on biological systems. *Biochem. Biophys. Res. Commun.* 222: 374–78.

Bawin, S., A. Shephard, et al. 1978. Possible mechanism of weak electromagnetic field coupling in brain tissue. *Bioelectrochem. Bioenergetics* 5:67–76.

Bazylinski, D. A., A. J. Garratt-Reed, et al. 1994. Electron microscopic studies of magnetosomes in magnetotactic bacteria. *Microsc. Res. Tech.* **27**:389–401.

Bersani, F., F. Marinelli, et al. 1997. Intramembrane protein distribution in cell cultures is affected by 50 Hz pulsed magnetic fields. *Bioelectromagnetics* **18**:463–69.

Bozhilova-Pastirova, A. 1998. Freeze-etching study of the axosomatic synapses in the rat sensorimotor cortex. *Eur. J. Morphol.* **36**:189–200.

Brown, K. T. 1955. Rate of apparent change in a dynamic ambiguous figure as a function of observation time. *Am. J. Psychology* **68**:358–71.

Burda, H., S. Marhold, et al. 1990. Magnetic compass orientation in the subterranean rodent Cryptomys hottentotus (Bathyergidae). *Experientia* **46**:528–30.

Ceccarelli, B., F. Grohovaz, et al. 1979a. Freeze-fracture studies of frog neuromuscular junctions during intense release of neurotransmitter. I. Effects of black widow spider venom and Ca2+-free solutions on the structure of the active zone. J. Cell Biol. 81:163–77.

Ceccarelli, B., F. Grohovaz, et al. 1979b. Freeze-fracture studies of frog neuromuscular junctions during intense release of neurotransmitter. II. Effects of electrical stimulation and high potassium. *J. Cell Biol.* **81**:178–92.

Cherniak, C. 1994. Component placement optimization in the brain. J. Neurosci. 14:2418–27.

Crasson, M., J. J. Legros, et al. 1999. 50 Hz magnetic field exposure influence on human performance and psychophysiological parameters: Two doubleblind experimental studies. *Bioelectromagnetics* **20**:474–86.

Cuffin, B. N., and D. Cohen. 1979. Comparison of the magnetoencephalogram and electroencephalogram. *Electroencephalogr. Clin. Neurophysiol.* **47**:132–46.

Damasio, A. R. 1989a. The brain binds entities and events by multiregional activation from convergence zones. *Neural Composition* 1:123–32.

Damasio, A. R. 1989b. Time-locked multiregional retroactivation: A systemslevel proposal for the neural substrates of recall and recognition. *Cognition* **33** (1–2): 25–62.

Damasio, H., D. Tranel, T. Grabowski, R. Adolphs, and A. Damasio. 2004. Neural systems behind word and concept retrieval. *Cognition*. **92**:179-229.

Darwin, C. 1993. On the origin of species by means of natural selection or the preservation of favoured races in the struggle for life. New York: Random House.

Davydov, A. S. 1979. Solitons in molecular systems. Physica Scripta 20:387-94.

Delgado, J. M., J. Leal, et al. 1982. Embryological changes induced by weak, extremely low frequency electromagnetic fields. *J. Anat.* 134 (3): 533–51.

Donoghue, J. P. 1995. Plasticity of adult sensorimotor representations. *Curr. Opin. Neurobiol.* **5**:749–54.

Edelman, G. M. 1987. Neural Darwinism: The theory of neuronal group selection. New York: Basic Books.

Edelman, G. M. 1992. Bright air, brilliant fire: On the matter of the mind. New York: Basic Books.

Eichwald, C., and F. Kaiser. 1995. Model for external influences on cellular signal transduction pathways including cytosolic calcium oscillations. *Bioelectromagnetics* **16**:75–85.

Endo, H., T. Kizuka, et al. 1999. Automatic activation in the human primary motor cortex synchronized with movement preparation. *Brain Res. Cogn. Brain Res.* 8:229–39.

Fannon, A. M., and D. R. Colman. 1996. A model for central synaptic junctional complex formation based on the differential adhesive specificities of the cadherins. *Neuron* **17**:423–34.

Felleman, D. J., and D. C. Van Essen. 1991. Distributed hierarchical processing in the primate cerebral cortex. *Cereb. Cortex* 1:1–47.

Freeman, W. J. 2004a. Origin, structure, and role of background EEG activity. Part 1. Analytic amplitude. *Clin. Neurophysiol.* **115**:2077–88.

Freeman, W. J. 2004b. Origin, structure, and role of background EEG activity. Part 2. Analytic phase. *Clin. Neurophysiol.* **115**:2089–107.

Freeman, W. J. 2005. Origin, structure, and role of background EEG activity. Part 3. Neural frame classification. *Clin. Neurophysiol.* **116**:1118–29.

Freeman, W. J., B. C. Burke, et al. 2003. Aperiodic phase re-setting in scalp EEG of beta-gamma oscillations by state transitions at alpha-theta rates. *Hum. Brain Mapp.* **19**:248–72.

Gaetz, M., H. Weinberg, et al. 1998. Neural network classifications and correlation analysis of EEG and MEG activity accompanying spontaneous reversals of the Necker cube. *Brain Res. Cogn. Brain Res.* 6:335–46.

Gallen, C. C., E. C. Hirschkoff, et al. 1995. Magnetoencephalography and magnetic source imaging: Capabilities and limitations. *Neuroimaging Clin. N. Am.* 5:227–49.

Galvanovskis, J., and J. Sandblom. 1997. Amplification of electromagnetic signals by ion channels. *Biophys. J.* **73**:3056–65.

Graham, C., and M. R. Cook. 1999. Human sleep in 60 Hz magnetic fields. *Bio-electromagnetics* 20:277-83.

Graham, C., M. R. Cook, et al. 1994. Dose response study of human exposure to 60 Hz electric and magnetic fields. *Bioelectromagnetics* **15**:447–63.

Gray, C. M., P. Konig, et al. 1989. Oscillatory responses in cat visual cortex exhibit inter-columnar synchronization which reflects global stimulus properties. *Nature* **338**:334–37.

Hamalainen, M., R. Hari, et al. 1993. Magnetoencephalography: Theory, instrumentation, and applications to noninvasive studies of the working human brain. *Rev. Mod. Phys.* **65**:413–97.

Harada, H., K. Shiraishi, et al. 1996. Coherence analysis of EEG changes during odour stimulation in humans. *J. Laryng. Otol.* **110**:652–56.

Hari, R., and S. Salenius. 1999. Rhythmical corticomotor communication. *NeuroReport* 10:R1–R10.

Hemmersbach, R., E. Becker, et al. 1997. Influence of extremely low frequency electromagnetic fields on the swimming behavior of ciliates. *Bioelectromagnetics* **18**:491–98.

Heuser, J., and T. S. Reese. 1977. Structure of the synapse. In *Handbook of physiology: The nervous system,* edited by J. M. Brookhart and V. B. Mount-castle, 1:261–94.. Bethesda: American Physiological Society.

Heuser, J., T. S. Reese, et al. 1974. Functional changes in the frog neuromuscular junctions studied in freeze fracture. J. Neurocytol. 3:109–31.

Heuser, J., T. S. Reese, et al. 1979. Synaptic vesicle exocytosis captured by quick freezing and correlated with quantal transmitter release. *J. Cell Biol.* **81**:275–300.

Hubel, D. H., and T. N. Wiesel. 1962. Receptive fields, binocular interaction, and functional architecture in the cat's visual cortex. *J. Physiol.* (London) 160:106–54.

Kandel, E. R., J. H. Schwartz, et al., eds. 1991. *Principles of neural science*. New York: Elsevier.

Kim, H. G., and B. W. Connors. 1993. Apical dendrites of the neocortex: Correlation between sodium- and calcium-dependent spiking and pyramidal cell morphology. *J. Neurosci.* **13**:5301–11.

Kobayashi, A., and J. L. Kirschvink. 1995. Magnetoreception and electromagnetic field effects: Sensory perception of the geomagnetic field in animals and humans. In *Electromagnetic fields: Biological interactions and mechanisms*, edited by M. Blank, **250**:367–94. Washington, DC: American Chemical Society.

Koenig, T., K. Kochi, et al. 1998. Event-related electric microstates of the brain differ between words with visual and abstract meaning. *Electroencephalogr. Clin. Neurophysiol.* **106**:535–46.

Koenig, T., and D. Lehmann. 1996. Microstates in language-related brain potential maps show noun-verb differences. *Brain Lang.* **53**:169–82.

Kohmura, N., K. Senzaki, et al. 1998. Diversity revealed by a novel family of cadherins expressed in neurons at a synaptic complex. *Neuron* 20 (6): 1137–51.

Lashley, K. S., K. L. Chow, et al. 1951. An examination of the electrical field theory of cerebral integration. *Psychol. Rev.* 58:123–36.

Lehmann, D., B. Henggeler, et al. 1993. Source localization of brain electric field frequency bands during conscious, spontaneous, visual imagery and abstract thought. *Brain Res. Cogn. Brain Res.* 1:203–10.

Lehmann, D., H. Ozaki, et al. 1987. EEG alpha map series: Brain micro-states by space-oriented adaptive segmentation. *Electroencephalogr. Clin. Neurophysiol.* **67**:271–88.

Llinas, R., U. Ribary, et al. 1998. The neuronal basis for consciousness. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.* 353:1841-49.

Llinas, R. R., U. Ribary, et al. 1999. Thalamocortical dysrhythmia: A neurological and neuropsychiatric syndrome characterized by magnetoencephalography. *Proc. Nat. Acad. Sci. USA* **96**:15222–27.

Lopes da Silva, F. 1991. Neural mechanisms underlying brain waves: From neural membranes to networks. *Electroencephalogr. Clin. Neurophysiol.* **79**:81–93.

Luo, H., and D. Poeppel. 2007. Phase patterns of neuronal responses reliably discriminate speech in human auditory cortex. *Neuron* **54**:1001–10.

Marek, W., A. Nerode, et al. 1990. A theory of nonmonotonic rule systems I. Ann. Math. Artif. Intell. 1:105–35.

Marek, W., A. Nerode, et al. 1992. A theory of nonmonotonic rule systems II. Ann. Math. Artif. Intell. 5:7229-64.

Merrin, E. L., P. Meek, et al. 1990. Topographic segmentation of waking EEG in medication-free schizophrenic patients. *Int. J. Psychophysiol.* **9**:231–36.

Nakasato, N., H. Kado, et al. 1990. Magnetic detection of sleep spindles in normal subjects. *Electroencephalogr. Clin. Neurophysiol.* **76**:123–30.

Necker, L. A. 1832. Observations on some remarkable optical phaenomena seen in Switzerland; and on an optical phaenomenon which occurs on viewing a figure of a crystal or geometrical solid. *London and Edinburgh Philosoph. Mag. J. Sci.* 3:329–37.

Nunez, P. L. 1986. The brain's magnetic field: Some effects of multiple sources on localization methods. *Electroencephalogr. Clin. Neurophysiol.* 63:75–82.

Paulin, M. G. 1995. Electroreception and the compass sense of sharks. J. Theor. Biol. 174:325–39.

Pawson, P. A., A. D. Grinnell, et al. 1998. Quantitative freeze-fracture analysis of the frog neuromuscular junction synapse. I. Naturally occurring variability in active zone structure. *J. Neurocytol.* **27**:361–77.

Pumplin, D. W., T. S. Reese, et al. 1981. Are the presynaptic membrane particles the calcium channels? *Proc. Nat. Acad. Sci. USA* 78:7210–13.

Regan, D. 1997. Visual factors in hitting and catching. J. Sports Sci. 15:533-58.

Robitaille, R., E. M. Adler, et al. 1990. Strategic location of calcium channels at transmitter release sites of frog neuromuscular synapses. *Neuron* **5**:773–79.

Robitaille, R., and M. P. Charlton. 1992. Presynaptic calcium signals and transmitter release are modulated by calcium-activated potassium channels. *J. Neurosci.* **12**:297–305.

Roberts, W.M., R.A. Jacobs, and A.J. Hudspeth. 1990. Colocalization of ion channels involved in frequency selectivity and synaptic transmission at presynaptic active zones of hair cells. *J Neurosci.* **10**: 3664-84.

Saarinen, J., and B. Julesz. 1991. The speed of attentional shifts in the visual field. *Proc. Nat. Acad. Sci. USA* 88:1812–14.

Sandyk, R. 1999. Impairment of depth perception in multiple sclerosis is improved by treatment with AC pulsed electromagnetic fields. *Int. J. Neurosci.* **98**:83–94.

Satir, B. H. 1980. The role of local design in membranes. In *Membrane-membrane interactions*, edited by N. B. Gilula, 45–58. New York: Raven Press.

Schiller, F. 1965. The rise of the "enteroid processes" in the nineteenth century: Some landmarks in cerebral nomenclature. *Bull. Hist. Med.* **39**:326–38.

Semm, P., and R. C. Beason. 1990. Responses to small magnetic variations by the trigeminal system of the bobolink. *Brain Res. Bull.* 25:735–40.

Shadlen, M. N., and W. T. Newsome. 1994. Noise, neural codes, and cortical organization. *Curr. Opin. Neurobiol.* **4**:569–79.

Srinivasan, R., D. P. Russell, et al. 1999. Increased synchronization of neuromagnetic responses during conscious perception. J. Neurosci. 19:5435–48.

Stern, P., M. C. Harmanci, et al. 1982. Vasopressin and intramembranous particle clusters in collecting duct cells of Brattleboro and Long-Evans rats. *Ann. N.Y. Acad. Sci.* **394**:518–23.

Strik, W., and D. Lehmann. 1993. Segments of the brain's electrical fields as functional microstates: Their role in psychiatry. *Pharmacopsychiatry* 26:205.

Treisman, A. 1996. The binding problem. Curr. Opin. Neurobiol. 6:171-78.

Treisman, A., and H. Schmidt. 1982. Illusory conjunctions in the perception of objects. *Cognit. Psychol.* 14:107–41.

Ungerleider, L. G., and J. V. Haxby. 1994. "What" and "where" in the human brain. *Curr. Opin. Neurobiol.* **4**:157–65.

Van Essen, D. C. 1997. A tension-based theory of morphogenesis and compact wiring in the central nervous system. *Nature* **385**:313–18.

Vazquez-Garcia, M., D. Elias-Vinas, et al. 2004. Exposure to extremely low-frequency electromagnetic fields improves social recognition in male rats. *Physiol. Behav.* 82:685–90.

Walker, M. M., and M. E. Bitterman. 1989. Honeybees can be trained to respond to very small changes in geomagnetic field intensity. *J. Exp. Biol.* **145**:489–94.

Weaver, J. C., T. E. Vaughan, et al. 1998. Theoretical limits on the threshold for the response of long cells to weak extremely low frequency electric fields due to ionic and molecular flux rectification. *Biophys. J.* **75**:2251–54.

Wiltschko, W., and R. Wiltschko. 1988. Magnetic orientation in birds. Curr. Ornithol. 5:67-121.

Wu, Q., and T. Maniatis. 1999. A striking organization of a large family of human neural cadherin-like cell adhesion genes. *Cell* **97**:779–90.

Zuber, H. 1986. Structure of light-harvesting antenna complexes of photosynthetic bacteria, cyanobacteria, and red algae. *Trends Biochem. Sci.* **11**:414–19.