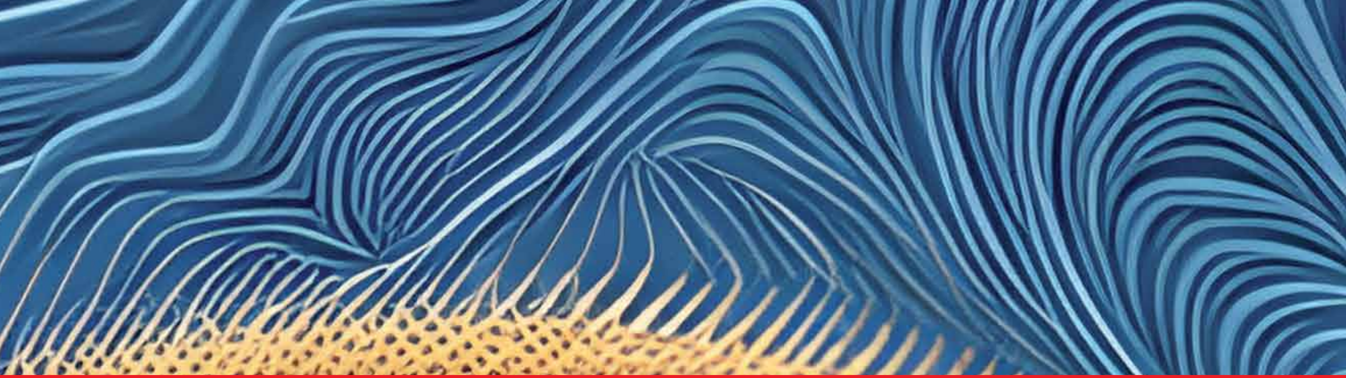

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Normal Alert Consciousness

A Central Executive Model of Hippocampal Function

Authored by Douglas D. Burman

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Meet the author

Douglas D. Burman obtained a Ph.D. in Anatomy (Neuroscience) from the University of Illinois in 1987. He completed his postgraduate studies at Yale University and Northwestern University. Currently, he is Director Emeritus of fMRI Services at NorthShore University Health System, Chicago, Illinois. Dr. Burman is specialized in electrophysiology, cognitive behavior, functional magnetic resonance imaging (fMRI) (activation, connectivity), and DTI fiber tracking. He has published more than 50 articles on visual perception, eye movement control, sensorimotor representations/motor control, attention (e.g., ADHD), language and developmental changes (e.g., dyslexia), sex differences, perceptual learning, hippocampal connectivity (motor, language, executive function tasks), and fiber tracking.

Contents

Preface

In the early 1600s, the influential scientist-philosopher Descartes proposed a role of the brain in consciousness; based on its anatomical location, he incorrectly suggested that the soul (consciousness) resides in the pineal body. For the next three-and-a-half centuries, the relationship between the brain and consciousness was discussed almost exclusively by philosophers, who fiercely debated Descartes' belief that a nonmaterial soul could act on a physical body. Scientists distanced themselves from the subject, believing that the subjective nature of the mind made its scientific study impossible.

The twentieth century brought advances in scientific techniques, which have improved our knowledge of the brain's anatomical pathways, chemistry, electrical signaling, functional connections, and relationship to behavior. Philosophers initially discussed these findings in relationship to the mind–body question; their discussions with scientists in the latter half of the twentieth century led to a renaissance of scientific exploration into cognitive neuroscience, including studies on the relationship between brain activity and consciousness (or conscious perception).

Scientific exploration on the subject has not, however, led to consensus, either among philosophers or scientists. Problems begin with the definition of consciousness itself; every attempt to describe a single defining characteristic has been countered with exceptions. The unique, subjective nature of an individual's conscious experiences is equally problematic; even perceptual differences (is the red I see the same as what you see?) provide challenges for a brain-based explanation of conscious experience. Such issues have provoked a wide range of theoretical solutions, yet the premise of each theoretical approach has its detractors.

This book begins by reviewing several prominent theories attempting to explain consciousness, pointing out phenomena they explain along with strengths and weaknesses. It then proceeds to a new model, limited in scope to a specific form of consciousness described succinctly as the "normal alert" state. This state includes diverse cognitive abilities, including all properties that we recognize and associate with our normal alert state. These abilities include awareness and responsiveness to our surroundings, emotional engagement, access to old memories and the ability to acquire new ones, language, and the skill to use these cognitive abilities to plan, discuss, and behave as we choose. By this definition, the absence of any one of these abilities results in a different state of consciousness, mediated by a different set of brain mechanisms.

The heart of this model is the Cartesian theater of the mind, an intuitively appealing idea previously rejected by both philosophers and scientists of the twentieth century. The concept is that a central brain region evaluates sensory inputs, then acts upon them to create memories and generate appropriate behavioral (and emotional) responses. When modern neuroscience was re-introduced to the philosophy of mind in the 1970s, philosophers argued vehemently against its existence based on contemporary science and two underlying assumptions. First, all forms of consciousness were assumed to be equivalent; you are either conscious or not conscious. Because there is no localized lesion of the brain where all forms of consciousness are lost, philosophers argued there is no brain structure that can function as a Cartesian theater of the mind. A second, implicit assumption is that a Cartesian theater must be located within a single brain structure. These arguments appeared so compelling that every subsequent theory of consciousness until now has dismissed the idea as untenable.

In this work, the author uses our common experiences to dispute these underlying assumptions. The normal alert state of consciousness is differentiated from automatic behaviors such as sleepwalking and automatic driving (which relies on automatic responses to familiar objects and events). A bilateral lesion of the hippocampus does in fact eliminate this normal alert state of consciousness, which coordinates environmental awareness, development of new memories, intentional movements, emotional responses to situations, and creative combination of ideas. Furthermore, the author shows that central executive properties required by a Cartesian theater are present when the joint properties of the left and right hippocampus are examined.

By focusing on effective connectivity findings between brain regions, a central executive model of hippocampal activity is assembled to explain normal alert consciousness. This model explains diverse cognitive abilities associated with the state of consciousness, such as perceptual awareness and memory formation; in addition, neural interactions are described to explain creativity, decision-making, learning, qualia, social behaviors, and memory recall. The strengths and weaknesses of the model are delineated, along with suggestions for experiments that can test its various components.

Many neurological disorders are revealed by abnormalities in conscious functions; hippocampal dysfunction has been identified in a surprising number of these, including Alzheimer's, temporal lobe epilepsy, Parkinson's, ADHD, and concussions. Explanations for behavioral and cognitive effects of hippocampal disruption in these neurological disorders are proposed based on the model and the known functionality of hippocampal connections. Besides a better understanding of the causes of neurological symptoms, demonstrating the model's validity could have practical consequences, as the effectiveness of medical treatments for individuals could be evaluated based on anticipated changes in hippocampal function and connectivity.

Finally, the author discusses the implications of the model for a variety of philosophical issues, including the self, free will, subjectivity (including qualia), non-human consciousness, and artificial intelligence (AI). The model is uniquely consistent with many of our subjective experiences, including free will, qualia, and animal consciousness. The model's implications for sentience in AI are less clear; to mimic the sentience observed in humans and animals, however, a new approach to AI would be required to develop a sense of self and emotional associations.

Unlike many theories of consciousness, the components of this model are testable; plus, it explains a wider range of conscious phenomena. The issues and systematic approach used here should inform future explorations on the topic.

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Chapter

Normal Alert Consciousness: A Central Executive Model of Hippocampal Function

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Abstract

The relationship between brain and consciousness has been debated since Descartes in the 1500s, new theories arising in the twentieth century with the development of modern neuroscience. All are controversial due to the lack of consensus on the definition of consciousness, what cognitive properties must be explained, and how to evaluate sentience. Most theoretical explanations bear little relationship to our inner conscious experiences. In the current monograph, the normal alert state of consciousness is defined, and components to be explained are delineated. Debunking misconceptions from previous theories and presenting new evidence, a model is proposed whereby the hippocampus plays a central role in executing and coordinating cognitive functions associated with normal alert consciousness. Key elements of the model reflect recent findings that the combined effect from the left and right hippocampus influences other regions involved in performing many or all cognitive tasks while filtering out irrelevant information. Methods are described for testing the model. Finally, implications are discussed for a variety of neurological disorders and philosophophical issues, including free will and the possibility of sentience in artificial intelligence.

Keywords: hippocampus, executive function, connectivity, neuroscience, philosophy, Descartes, theater of the mind, functionalism, free will

1. Introduction

In the early 1600s, Descartes proposed that the pineal body is the seat of the soul (or consciousness), reasoning that because it connects the two halves of the brain, the pineal body must integrate information from each hemisphere into the unified construct of one's self [1]. Descartes speculated on the role of the brain in consciousness based on his contemporary knowledge about brain anatomy. He was wrong; even when not calcified, the pineal body is unrelated to consciousness, instead functioning to regulate seasonal rhythms and sexual function [2, 3].

For centuries, knowledge of brain function was limited to inferences from anatomical studies and the loss of function following brain injury. The situation changed in the twentieth century, when techniques were developed to record the brain's electrical activity while an experimental subject (either human or animal) performed tasks. Because information is transmitted in the brain through electrical signals, this methodological advance allowed scientists to explore the relationship among brain activity, sensory stimuli appearing in the environment, behavior, and cognitive processes that underlie behavior. Evidence-based consideration of the relationship between brain and consciousness was advanced in the last quarter of the twentieth century when the philosophers Daniel Dennett [4–7] and Patricia Churchland [8–10] applied contemporary knowledge of science function to age-old philosophical issues. They rejected Descartes' dualism, which suggested that the mind/soul and physical body were distinct substances; their interest was largely focused on whether consciousness is derived from brain activity and the implications for free will.

Descartes' underlying belief that conscious experience must reside in a single brain location was also rejected. Subsequently, elaborate theories were developed by both philosophers and scientists to explain how consciousness could result from brain activity in the absence of this "Cartesian theater of the mind." Books on consciousness from the past half-century suggest that most philosophers and scientists accept the idea that consciousness is associated with brain activity, but little else has reached consensus. Some suggest consciousness results directly from brain activity, although how this occurs is unknown [11–14]; others suggest consciousness is merely an epiphenomenon, occurring in association with brain activity without being caused by it [15, 16]. Still others suggest consciousness is nothing more than an illusion [6, 17]. Many studying the issue wrestle with the subjective nature of consciousness, and some questioning whether any account could satisfactorily explain how my conscious experiences differ from yours.

Several explanations for how brain activity generates consciousness have been offered. Twentieth-century explanations initially considered how consciousness could be constructed from a complex structure of functionally distinct regions [6, 18–20]. As it became clear that interactions between brain areas must play a role, more holistic explanations were offered, emphasizing interactions between functional systems [21, 22]. Diverse explanations have been put forth, including competitive interactions or integration between brain areas [15, 18, 19], perceptual organization with recurrent feedback [23, 24], an attentional schema [25], consciousness rooted in feelings [26, 27], information-based theories [28–31], electromagnetic fields [32], methods for coordinating brain activity across regions [11, 33], higher-order representations [34], a feedback mechanism for behavioral control [35], and quantum entanglement [36–39]. Although each theory explains some phenomena, each has its detractors. Arguments against theoretical explanations typically involve one or more of the following objections: The definition of consciousness is problematic; the explanation is inadequate to explain qualia, the individual subjective nature of conscious perceptions; or the explanation is too limited and inadequate to explain other conscious phenomena.

Most recent theories exploring the brain basis of consciousness focus on the neural correlates of consciousness [40–43]. Some brain cells in sensory areas respond selectively to stimulus properties, regardless of a subject's perception, whereas the responses of other cells depend on whether that property is perceived. Cells dependent on perception may not be directly involved in consciousness—they do not necessarily reflect perceptions of other objects—but their location might provide insight into the network of brain areas required for conscious perception. "Consciousness of" an object, however, is different than the subjective experience of consciousness (also called phenomenological consciousness) [13] and does not address how the brain acts on this information to generate behavior.

Unfortunately, many theories of consciousness are not experimentally testable, appealing instead to their ability to explain a set of phenomena associated with consciousness. The model to be offered here does not suffer this limitation, at least in regard to its components. Before explaining the basis for this model, however, some issues need to be addressed that have limited the development of theories, beginning with a definition of a specific state of consciousness.

2. Relevant issues and considerations

2.1 Defining consciousness

In philosophical discussions about the brain, consciousness is rarely defined, perhaps because it lacks a single delimiting characteristic.

Awareness is often considered, but is inadequate. The brain activity of some vegetative patients changes when presented with their own name [44, 45] or when presented a mental imagery task [46]. Does this signify consciousness? Similarly, a mother in a deep sleep may arouse at the sound of her baby crying, indicating some level of environmental awareness. Other characteristics associated with sentience are also inadequate. Motor control does not define consciousness; awareness is not lost when administered a paralytic agent. Memory seems necessary for consciousness, yet sleep-wake cycles persist in people with memory disorders, with wakefulness accompanied by "conscious" activities. Some type of memory is required during consciousness, however, to maintain a connection between a series of moments in time.

"Consciousness" can refer to different states. We may be conscious of something, but that's different from phenomenological consciousness, awareness of our individual existence, and thought processes. Similar distinctions can be made between other conscious states, such as alert wakefulness, dreaming, drowsiness, automatic driving, and altered states of consciousness associated with meditation or drug use.

If we assume that phenomenological consciousness represents a single state, it may be impossible to define consciousness, given the qualitative differences between various states of consciousness. It seems likely, however, that different mechanisms (and perhaps brain regions) give rise to different states of consciousness. This possibility has a historical precedent. In the 1950s, memories were assumed to result from a single learning process. Karl Lashley undertook a search to identify where in the brain memories were stored ("engrams"), training lab animals before removing different parts of the brain and testing memory performance [47]. He was unable to eliminate memories, but noted that memory deficits were greater when larger areas of the brain were removed. He concluded that memories were distributed throughout the brain, much like a hologram. His conclusion was never supported by further experimentation. The idea was eventually abandoned with the discovery of different types of memories, each involving different brain structures.

Varied states of consciousness may arise from different mechanisms. Some have argued against this possibility, asking why, from an evolutionary viewpoint, would multiple brain structures do the same thing? What evidence is there for a duplication of function in the brain?

Three salient points may be made. First, the presence of multiple mechanisms for accomplishing conscious behavior has an evolutionary advantage; damage to a single brain system for consciousness would otherwise incapacitate an animal, reducing its chances for survival. Second, consciousness does not have a unitary function. The ability to awake from sleep by hearing infant cries serves a different function than the ability to plan social activities for the next month. Third, many functions of the brain are duplicated, with nuanced distinctions and interactions between brain areas with overlapping functions. Generating body movements, for example, can result from different brain systems; the primary motor cortex generates signals for volitional movements that proceed (relatively) directly to muscle effectors *via* the corticospinal tract, yet the basal ganglia can independently generate motor signals that guide complex movements. Indeed, except for a loss in dexterity, complete destruction of the primary motor cortex results in few permanent motor deficits [48, 49].

If we assume multiple states of consciousness reflect different mechanisms, interactions must occur between these varied systems. When we discuss phenomenological consciousness, we typically refer to our *normal alert state*, where we are aware and responsive to our surroundings, emotionally engaged, and access memories as well as acquire new ones. We can use these abilities to cognitively plan, discuss, and behave as we choose. This entire combination of characteristics defines this state of consciousness—remove any one of these attributes and the qualitative nature of consciousness changes. A model for this state of consciousness must be able to explain how all its component processes are coordinated.

2.2 Evidence for brain-based consciousness

David Chalmers famously stated that the easy problem about consciousness will be to explain the mechanism for cognitive processes such as memory, whereas the hard problem is to explain subjective experiences, such as qualia [50, 51]. Qualia refers to qualitative differences in perception by people viewing the same object under the same conditions, typically subtle differences arising from each person's unique subjective experiences.

In this definition, "viewing" does not necessarily require sight or vision, although a common example involves the perception of a red object. How do I know that I see a red object the same as you? The wavelengths reflected by the object can be measured, but the light must be absorbed by pigments in the retina, generating electrical potentials in cone cells that produce electrical spikes in neurons, and relayed to the lateral geniculate nucleus, a brain region specialized for processing visual information. Electrical activity in the lateral geniculate is insufficient to generate awareness. Neurochemicals are released that cross synapses to generate electrical potentials, eliciting spikes in electrical activity that are conveyed to the primary visual cortex. This process of neurochemical crossing synapses to generate electrical potentials must be repeated several times before perception of the red object reaches consciousness.

Would our perception of red be the same if our processes for passing information to the brain differed? This could happen at any point in the pathway; our cone pigments might differ in the wavelengths that they absorb, for example, or the frequency of electrical spikes in the optic nerve might differ, or the efficacy in generating postsynaptic potentials in the lateral geniculate might diverge. We have learned to describe the color of this object as red, even if one of us is red-green colorblind, so our descriptions would belie our differences in perception. Furthermore, our subjective reactions to colors differ; I am enamored with some tints of red but dislike others, including the shade my previous homeowner loved for the dining room walls.

Are differences in qualia only a problem of communication about subjective experience? Is it even possible to describe my perception of red that clearly differentiates my experience from yours, or that defines its character? Philosophers agree that qualia are real, reflecting differences in our subjective consciousness, yet they disagree vehemently over whether they can ever be explained through our understanding of brain activity.

Similar issues are raised about the qualia of pain. We know that sensory input associated with pain is transmitted through C nerve fibers, and we can measure the frequency of electrical spikes transmitted through those fibers to the brain. Nonetheless, the same frequency in C fibers might be perceived as exquisitely painful to one person but barely irritating to another. Furthermore, C fibers are not directly responsible for the perception of pain; pain can by eliminated by chemically blocking the transmission of C fiber signals into the brain. Fundamentally, we do not understand which brain processes underlie the conscious perception of pain. How can brain activity explain the immediacy of the pain when my arm is pinched (or differences in my painful experiences from yours)?

For many philosophers, the feature of qualia is the defining characteristic of consciousness [14, 52]; it differentiates a conscious human being from a purely mechanical machine. This raises the question: Can artificial intelligence (AI) acquire consciousness? Arguments mostly center on the possibility of AI acquiring qualia. The qualia issue is not limited to humans and AI; Nagel famously staked out his position by questioning whether we could ever know what it is like to be a bat.

It is my position that qualia can (and must) be explainable from brain activity. The rationale for this conclusion should be clear from the experiences of my second cousin Anthony, a romantic who spent years with his sweetheart Francesca on Sardinia beaches in Italy. He loved sunsets, and he would describe them in exquisite detail, almost poetic when describing their various shades of crimson and gold. Sometimes he painted what he saw, color-washing the canvas with stains and pigments to produce a brilliant, shimmery effect.

Anthony loved sunsets—until one fateful summer night in late July. He was walking on the beach with Francesca when they were accosted by a young man wielding a cutter, demanding that she hand over her pearl necklace, a gift from Anthony when he had proposed marriage 2 weeks earlier. Anthony stepped in, heart thumping as he wrestled with the thief and tried to pry the knife away. The thief pulled free, cutting the necklace from Francesca's neck before stabbing her in the chest. Francesca died in Anthony's arms.

Now Anthony hates sunsets, especially at the beach. A year later, his counselor insisted he should paint a sunset at the beach as part of his therapy. As the sun started to set, Anthony felt his heart racing, and was soon drenched in sweat. I saw his painting, full of dark colors embedded within the red and yellow of the sunset, unlike any of his previous paintings. Anthony insisted he had only painted what he saw.

The qualia Anthony felt about sunsets was altered by his experiences, a subjective response that now includes physiological effects mediated by adrenaline and other stress hormones.

This thought experiment feels true to life, because we have all experienced similar changes in perception in response to our life events.

The subjective perception of red is often used as an example of qualia. As noted above, individual differences may result from, or at least begin with, differences in retinal pigments that absorb certain wavelengths to generate nerve impulses delivered to the brain. This argument is based on an outdated theory of color perception. The perception of a color (including, but not limited to its hue) depends on interactions with light conditions in nearby regions of the visual field; the critical interactions occur in cortical regions of the brain.

Philosophers may argue that this relates to the perception of color, but not my unique, subjective experience when I see the color. Such an argument misses the point: Subjective experiences result from our personal interactions with our environment, which are continually modified by experience. These qualia reflect the experiences of our "self" with the external environment, as presented by our sensory experiences conveyed to the brain, modified by our genetic predispositions and memories of previous experiences.

All experiences, past and present, are tinged with emotional overtones, based on current and past experiences, associated with neural activity in appropriate parts of the brain. Some emotional responses are short (a few seconds or less, mediated by orbitofrontal cortex and the amygdala), others are long (hours or days, mediated by hormones such as adrenaline and cortisol). Brain mechanisms for feelings and emotions help organize our behaviors and thoughts, permeating our cognitive behavior in ways we may not even recognize. I had a hard time remembering the correct name of my neighbor for years, because the names Leslie and Stephanie "feel" the same to me; similarly, my wife had a hard time distinguishing the name Evan from Ryan. These effects were not mediated by past associations, as neither my wife nor I knew others with these names; rather, they were likely mediated by connections between frontal language areas and orbitofrontal regions implicated in emotions [53]. Brain studies on language have confirmed orbitofrontal activation associated with the emotional content of language [54–56].

The same principles apply to the perception of pain: Beyond reflexes, my subjective response to painful stimuli reflects my past experiences as much as the frequency and pattern of nerve impulses conveyed through my pain pathways.

2.3 The embodied self

My consciousness exists within a body that I identify as my physical self. This may be true of all conscious existence, at least for humans and animals. Some research findings suggest the existence of some form of conscious awareness and interactions among plants and even unicellular organisms, but the nature of such a consciousness and its physical basis is beyond the scope of this treatise.

Our understanding of consciousness is tied to the concept of self. My "self" is conscious—and I can describe what I am conscious of—but who is this "self"? Is it defined by my body? With the exception of most brain cells, all cells in the body are replaced within a period of a few years, so my body now differs from my body as a child, or even a decade ago. Is my "self" defined by my brain—or by my brain programs? Will my "self" survive my death? Philosophers and theologians (as well as science fiction buffs) have spent considerable time and effort discussing these issues, tying this issue to the concept of consciousness.

Our self requires a sense of continuity across time. Despite changes in body growth, experiences, and even personality, I subjectively feel like I am the same person I was as a child (although, hopefully, more developed emotionally and intellectually). My normal alert consciousness, however, has been interrupted innumerable times from periods of sleep, daydreaming, and even anesthesia. What is there about my "self" that has been maintained across my lifetime?

Philosophers have discussed this issue extensively, arguments often muddled from conflating the different meanings of the word "self." This should become apparent in the discussion below.

I recognize my wife walking down the street based on her physical being; that's not me, or her best friend, or a stranger—that's my wife herself. When my daughter was born, she recognized others (and the world) as separate from herself because we were not attached to her body, nor could she directly manipulate us. The first time I spoke to her, moments after her birth, she turned her head and gazed intently in my direction, facial muscles dancing in a series of expressions I would not have believed possible from a newborn. She recognized her body as her "self" because she had an extensive measure of control over its movements and the sensory stimulation it provided, control that would be refined and extended with use.

In a newborn, the "self" is defined jointly by the bodily senses and its motor system because that is the limits of what she can directly control. The conceptual schema of herself—that is, what she herself can directly control—evolves as she develops. This is not a cognitive conceptualization, but rather, a schema embedded within the brain that differentiates between what can and cannot be accomplished through the body it controls.

Our conceptual schema of our self evolves through our childhood and the rest of our life, extending beyond the sensory/motor framework that was embedded within our brain before we were born. When we are young, our family describes who we are through our personality traits; we are told we are more than just our body. Although this seems to provide a new definition of "self," we intuitively sense we have control over these behaviors and personality traits, so our sense of self evolves. I may describe myself as kind, gentle, and caring as well as rational and stubborn, with a sardonic sense of humor. These characteristics describe my inner self because they represent patterns of behavior within my control that became ingrained over time.

If my arm was forcibly moved to strike someone—perhaps through a configuration of stimulating electrodes under someone else's control—that would not be something I myself did. I would still believe it was my arm that struck the blow—assuming my arm was under my control during other conditions. If I no longer had control over the arm, perhaps because of neurological damage, my internal conceptual schema might change so I no longer considered it mine. Such cases have been reported, a condition known as anosognosia for hemiplegia [57–59]. One such patient would not identify his paralyzed left leg as his own, even when its connection to his own body was pointed out. Annoyed with the intrusion of this leg, he threw it out of his bed, then was surprised to find himself on the floor. In describing this patient, the doctor described the man's perceptions about his leg—because by default, we define each "self" in relationship to its body. Recent analysis of these patients' lesions suggest that the syndrome results from joint damage to areas involved in sensorimotor monitoring and limbic regions involved in maintaining and updating beliefs about self [60].

Conversely, a person can be tricked into believing an inanimate object is his own, as evidenced by the rubber hand illusion [61, 62]. When viewing a rubber hand stroked by a paintbrush at the same time as his own unseen hand, a person perceives the rubber hand as his own and will adjust his hand posture accordingly. This illusion requires sensory input, yet the subjective sensation requires top-down modulation of sensory input based on the representation of one's own body [63]. This illusion is enhanced when the rubber hand is moved in synchrony with the subject's own volitional hand movement [64]. Although many variations of the rubber hand illusion have been devised with varying degrees of success [65], its effectiveness depends on sensory input corresponding to one's own brain schema of its bodily self.

3. Contemporary theories and models of consciousness

In this section, I will briefly review some important theories from the past 30 years that have attempted to explain the neural basis of consciousness, along with strengths and weaknesses of each. This is not a comprehensive review; neither do I intend to advocate or dismiss a theory, as each does provide an explanation for a set of experimental findings. Elements of several have been incorporated into my own understanding of brain function—although the model that will be presented goes substantially further to explain the diverse cognitive functions experienced during normal alert consciousness.

3.1 Global workspace theory

This theory seeks to explain how different brain functions can be integrated into a single conscious experience [18, 19, 66]. Sensory characteristics are processed independently, each competing for access to a global workspace that selects information to generate an effective behavioral response.

Strong points: This theory explains why conscious perception involves categorical awareness of stimuli in the environment, rather than sensations that must be constructed into a holistic feature (such as a face). This explanation is consistent with neuroscience research that suggests complex, categorical features emerge in brain areas that receive subconscious input from areas sensitive to component features.

Criticisms: The theory implies processed sensory information must access unspecified brain regions to reach consciousness and be acted upon, which implies a Cartesian theater of the mind without explaining where or how this exists. This model also fails to explain how neural activity associated with consciousness generates top-down influences on perceptual processes, generates motor behavior, or interacts with emotions. The envisioned process is inefficient, requiring substantial metabolic energy on processes that will neither reach consciousness nor influence behavior.

3.2 Multiple drafts theory

Derived from the global workspace concept, this model hypothesizes the creation of multiple drafts of perceptual elements; these drafts are potentially relevant to behavior and maintained in the brain until a version is selected to generate a behavioral response [6]. Environmental stimuli that appear close in time are placed in sequence post hoc, based on differences in the rate of transmission to the brain in different sensory pathways and the timing of activity in the regions devoted to different drafts of the environment.

Strong points: This model avoids the use of a Cartesian theater of the mind (dismissed by the author as impossible), while explaining some perceptual illusions. Consciousness is interpreted as an illusion resulting from deterministic brain processes, designed to provide behavioral responses to environmental events in real time.

Criticisms: While this model has its supporters, numerous criticisms have been raised by both philosophers and scientists. Consciousness is viewed in this model as a construct that allows rapid behavioral responses to environmental threats and events, which is more characteristic of subconscious reflexes; the process that selects a behavioral response during perceptual processing is unspecified. By dismissing consciousness as an illusion, the model avoids explanations for subjective experiences, decision-making, how movements are programmed, and conscious experiences that are not strictly perceptual; furthermore, it is untestable.

3.3 Thalamic 40 Hz binding model

This model seeks to explain why an environmental event involving multiple senses is perceived as a single event, despite temporal differences in evoked activity in cortical areas processing different sensory modalities [21, 33, 67–70]. A thalamic nucleus (pulvinar) is hypothesized to provide the drive for conscious processes, working in concert with cortical neurons in layer 5 that provide feedback.

Strong points: It provides a prominent role for interactions between brain regions, focusing on coherence of EEG activity between brain regions.

Criticisms: With limited experimental support, it attempts to combine a pattern of anatomical connections with diffuse cortical activity; this is largely based on the pattern of connections in the visual system, as the authors sought to explain conscious visual perception. The thalamus lacks complex properties required for cognitive thought, however, and layer 5 provides output throughout the cortex, including regions with no direct relationship to conscious function. The model is untestable, and evidence to implicate these neurons in conscious awareness is lacking.

3.4 Recurrent processing theory

Recurrent processing is required to group stimulus features that can enter phenomenonal consciousness, that is, awareness of what appears in the environment [23, 71–73]. In its simplest form, hardwired bottom-up (forward) sensory processing is unconscious, whereas the recurrent ("top-down") processing is conscious. A nuanced version of the theory posits that consciousness requires long-lasting exchange of information between brain regions, a process that requires recurrent feedback [74].

Strong points: The theory recognizes that ascending pathways are hardwired to process vast amounts of sensory information with diverse features, requiring recurrent pathways to group and select those features that reach consciousness.

Criticisms: This theory is limited to conscious awareness of sensory inputs, without considering conscious reflections, qualia, or other forms of conscious experiences. Each sensory pathway would develop conscious awareness independent of other sensory modalities.

3.5 Integrated information theory

This elaborate theory accepts the phenomenological reality of consciousness, and then explains how it could be constructed by integrating information from brain networks that provide the essential properties of the experience [29–31, 42, 75–78]. The theory views consciousness as structured, unifying content within a perceptual time frame by integrating information about specific phenomena (e.g., a book—that's blue—in a bedroom—on the left side—of my foot—as I look down my legs—while lying on a recliner—the "moment" before I decide to get up to close the window). For each experiential property (e.g., the perception of a book), there is a causal physical substrate, which may operate below consciousness; its contribution to our consciousness may vary as our conscious experience changes.

The subjective experience is specified by the form of the conceptual structure, based on the phenomenal distinctions provided by the concepts and their relationship. Information integrated into consciousness can theoretically be quantified as ϕ, which binds and incorporates those features and conceptual structures involved in our conscious experience at a moment in time. If more information is available to the overall network than the sum of its component parts, ϕ is positive.

Strong points: The theory recognizes that a conscious experience encompasses more information than would result from any one pathway extracting specific information about the environment; furthermore, the theory avoids the common fallacy that consciousness is all-or-none. The theory explains why some but not all brain areas can be associated with conscious phenomena under some conditions, and it allows for free will.

Criticisms: The value of ϕ is a theoretical concept that is impossible to calculate. Areas that integrate information from diverse regions are not delineated, nor is a mechanism provided to explain how consciousness shifts in the type or quantity of information experienced.

3.6 Models based on the neural correlates of consciousness

Other models have been proposed based on experimental results designed to identify the neural correlates of consciousness [40–42, 79–81]. As noted earlier, not all brain activity can be directly involved in generating consciousness, as we are not continuously aware of everything in the environment (or our internal state) that is processed somewhere in the brain.

Details vary widely, but the concept behind these models is simple: identify those parts of the brain that are preferentially active when we are conscious of a stimulus or event, which provides clues to understand how the brain constructs our conscious experiences. Experiments are designed where the same stimulus will be consciously perceived in some conditions but not others.

Strong points: These models implicate brain regions and processes based on experimental results directly related to conscious awareness.

Criticisms: These models differ in their experimental approach and the specific features of consciousness they attempt to explain, and are based on correlations between a conscious state and a pattern of brain activity; correlations, however, do not signify causation. Brain activity may support, contribute, or reflect conscious awareness without itself generating the conscious experience. Furthermore, "conscious of" something is not the same as phenomenological consciousness, the subjective qualities associated with self-awareness of one's conscious experiences. Experiments to test causation are not obvious and seldom offered.

3.7 Quantum theories of consciousness

These theories suggest consciousness reflects patterns observed in quantum physics, suggesting a physical basis either in the molecular structure of neurons (resulting in synaptic functions that act as a quantum computer), or the quantum collapse of superimposed states when the entire brain must resolve alternate perceptual states for behavioral action [36–39, 82].

Strong points: These theories provide an explanation for the neural basis for consciousness that is non-deterministic; when faced with a choice, a behavioral outcome is not predetermined from fixed activity patterns elicited in neural pathways.

Criticisms: Output from neural pathways is based on chance, a poor alternative to deterministic theories of consciousness. These theories are not testable.

4. Normal alert consciousness

The central executive model for the normal alert state of consciousness that will be described requires additional consideration of some issues already mentioned, plus an overview of pervasive hippocampal involvement in cognitive processes.

4.1 Cartesian theater revisited

When Daniel Dennett brought modern neuroscience into the philosophy of consciousness, he noted that scientists had not found a centralized brain region essential for interpreting all conscious experiences. Based on Descartes' writings, Dennett referred to such a centralized region as the "Cartesian theater of the mind." Before offering his alternative explanation, he vehemently argued against this possibility. All subsequent theories have ostensibly rejected the concept of the Cartesian theater, although some implicitly require such a space [6].

The basis for discarding the premise of the Cartesian theater involved several assumptions, at least one of which is likely false—the notion that consciousness is unitary (i.e., all or none). Under this precept, I am either conscious or unconscious; as such, there must be only one mechanism for consciousness. Language has simplified the reality, leading to confusion: There are many states of consciousness, as noted earlier, and there is no reason why one mechanism should apply to all. A mother's awareness during sleep of her infant's cries is not comparable to the normal alert state of consciousness, which involves interactions between senses, memories, attention, emotions, behavioral actions, and cognitive activities such as language and planning.

The model that will be presented provides a plausible mechanism, with some experimental support, for the hippocampus to coordinate all cognitive and emotional processes associated with normal alert consciousness. With a few minor differences, the hippocampus in this model assumes the position of the Cartesian theater of the mind—although I prefer the analogy of the central executive function of the White House.

Many activities are coordinated at the White House: Vetted information is received from all parts of the United States; relevant information is sought based on past experiences and personal preferences; relevant information is stored for later access; and the chief executive decides how to respond, with actions often carried out by others. Although the United States (self) is greater than the occupants of the White House (hippocampus), its central executive during normal conditions (our normal alert state) represents us through our interactions with the world.

A role of the hippocampus in consciousness is often dismissed due to the assumption that consciousness is all-or-none. In his book, *An Astonishing Hypothesis* [83], Francis Crick recognizes its position of convergence for sensory pathways as central to conscious experiences, but then provides this unitary argument in his rationale for dismissing it. Crick dismisses a central role based on patient behavior following destruction of the hippocampus. This patient, known as HM, "initially appears to be normal on first acquaintance, as he can shake hands and carry on a conversation reasonably well. The patient will not remember meeting you, however, if you leave the room for a few minutes to go to the restroom. The patient is also unaware of current events, and without memory, he could make no plans for the remainder of the day or week." Crick then summarizes. "...his short-term visual awareness appears to be unaltered.... The hippocampus and its closely associated cortical areas are thus not necessary for visual awareness."[1]

This argument is not credible, however, when viewed in the context of multiple states of consciousness. HM's behavior substantively describes the behavior of my younger sister when she was sleepwalking. As a child, I interacted with my sister in a conversation after she followed me into the dining room in the middle of the night. Ten minutes passed before I believed my mother, who explained that my sister occasionally got up and walked in her sleep. My sister's eyes were open, she navigated our house, and her conversation initially made sense; only when she forgot what we had discussed a few minutes earlier did I doubt whether she was awake. When she started to confabulate, it was clear she was not.

Automatic behaviors such as driving a familiar route show similar complex behaviors without later recollection. We must be conscious in some sense to navigate traffic, yet our inability to recall anything about it at the end of the trip reflects the reality that we were in a different state of consciousness. In this state, brain regions involved in creating long-term declarative memories—such as the hippocampus—are not engaged. This is clearly not our normal alert state.

4.2 Sensory perception: attention to features

Of the various senses, the visual pathways have been the most extensively studied, laying foundational work on patterns of hierarchical processing evident in all sensory pathways.

Of the five visual pathways to the brain, only the geniculostriate pathway is normally involved in conscious perception. Not all parts of this pathway are equally engaged. Nerve fibers from the retina project *via* the optic nerve to the lateral geniculate nucleus, a subcortical relay station in the thalamus that projects visual information to the occipital cortex (at the back of the brain). The specific region receiving these fibers is called V1, the primary visual cortex. At this early stage, the visual information is already transformed: whereas cells in the lateral geniculate respond best to circular spots in a restricted location (its "receptive field"), cells in V1 respond best to edges and borders. This change in visual properties results from the pattern of input from the lateral geniculate, reinforced by local interactions within the cortex.

V1 is still early in the visual pathway, only tangentially involved in perception; we normally perceive objects with complex properties in our environment rather than a tangle of edges and borders. From V1, projections are directed forward through a hierarchy of areas, each extracting new visual features. Each visual area projects

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[1] On pages 173–174 of *The Astonishing Hypothesis* (1995), Crick acknowledges that the hippocampus may be intimately tied to conscious awareness. "[The hippocampus] receives input from many of the higher cortical areas and projects back to them. This... might suggest that this is where consciousness really resides, since the brain might use this pathway to reflect on itself.... Information flowing in and then out of the hippocampus may normally reach consciousness, so it is sensible to keep an eye on the neural areas and pathways involved, as this may help to pin down the location of awareness in the brain."

forward to another visual area ("feedforward connections") and back to areas providing input ("feedback connections"), but also to an area in the thalamus called the pulvinar. The pulvinar has both feedback and feedforward connections with visual areas, as well as input from prefrontal cortex at the front of the brain [84–86]. Visual areas also project forward to and receive connections from prefrontal regions near the front of the brain [87].

Advancing through the visual pathway, as in the other sensory pathways, three patterns are noteworthy. First, nerve cells respond to increasing complexity of sensory features further along the sensory pathway, generally with less specificity for the stimulus location. Second, sensory processing is modular—that is, different cortical regions are specialized for extracting and responding to different types of sensory information. In the visual pathway, for example, different regions respond preferentially to color, buildings, faces, movements from side to side, three-dimensional movements, and letter groups corresponding to words. Damage to each of these regions results in a corresponding perceptual deficit without eliminating perception of other visual features. Third, advanced areas in a sensory pathway project to entorhinal cortex. In entorhinal cortex, a cell may respond to one, two, or three sensory modalities [88]; the entorhinal cortex also enhances cognitive elements and maps stimulus location in space [89–91].

Entorhinal cortex projects to, and receives feedback from, the hippocampus. The hippocampus also projects directly or indirectly to thalamic and most cortical regions, including sensory, limbic, prefrontal, temporal, and parietal cortices [92–94].

This pattern of processing and connections positions the hippocampus at the top of the sensory hierarchy, an ideal position for broad perceptual awareness for all our senses. Perceptual deficiencies can result from its failure to receive input from a specialized module; loss of network connections from the region specialized for faces, for example, explains prosopagnosia, the perceptual failure to recognize faces despite good visual acuity for the component shapes [95–97].

Conscious perception of an object is not a passive process; it requires allocation of mental resources (attention) for selecting features and objects to be noticed as well as a context for interpreting its visual details. Hippocampal feedback projections to entorhinal cortex provide a means to select between different objects identified from the output of specialized modules. Enhanced neuronal responses due to attention are also observed earlier in the visual pathway. A task requiring attention to the direction of stimulus movement, for example, will result in enhanced responses in modules sensitive to movement direction but reduced responses in areas sensitive to a visual pattern, whereas the reverse is true for a task requiring discrimination between visual patterns.

Consistent with a role in attention, the hippocampus appears to gate sensory processing for specific features. This has been best studied in the visual system. The Stroop task requires attention to color while ignoring semantic information provided by written words; during this task, the hippocampus selectively influences activity in the visual module specialized for color. Similarly, the hippocampus enhances visual activity in both primary visual cortex and the color region during a conjunction task, requiring attention to both the shape and color of a target embedded within a group of distractors (see *Examples and illustrations* below). The hippocampus instead enhances activity in a multimodal language area specialized for semantics during a semantic judgment task, and in the visual word form area during a spelling task [98]. These findings are consistent with a role for the hippocampus in attentional control over the content of sensory processing.

Examples and illustrations. For accurate performance, the Stroop task requires attention to the ink color while ignoring the sensory meaning of words. The top of **Figure 1A** shows activated brain areas, including language areas (Broca's area in the left prefrontal cortex and the left supramarginal gyrus) and visual areas: bilateral regions of primary visual cortex (red arrows) and inferior occipital regions involved in color processing (blue arrows). Hippocampal connectivity with activated regions during the Stroop task is limited to the color processing region. **Figure 1B** shows similar results from an attentionally demanding conjunction task [99, 100]. In this task, subjects must foveate objects to correctly differentiate targets from distractors, which share shape or color but not both. Activation and hippocampal connectivity are both observed in bilateral regions of occipital cortex representing central vision (red arrows) and in inferior occipital regions involved in color processing (blue arrows).

4.3 Sensory perception: attention to location

In the eye, different points in space are represented at different points on the retina. This retinotopic organization is carried forward along the visual pathway into early stages of visual processing in the cortex. At the back of the brain in V1, for example, the visual field that is observed above/right of center is represented in the brain below the calcarine sulcus in the left hemisphere; other parts of the visual field are similarly located in other regions of V1.[2] Through its neuronal activity, adjacent points in the visual field are represented at adjacent points in cortex.

From V1, neurons project forward to several regions, each extracting its own unique set of features. Early in the visual pathway, each region each carries its own retinotopic organization. Divergent pathways can be broadly grouped into two streams [101]. The ventral stream, extracting information about what an object is, includes inferior occipital cortex, extending forward into fusiform and inferior temporal cortex. As visual information in the ventral stream is extracted, the precise location of a feature is lost in the neuronal responses ("position invariance").

The so-called dorsal stream, extracting information about where an object is located in space, includes superior occipital extending into parietal cortex. For someone to navigate his environment, the spatial location of objects in relationship is important, both in relationship to each other and in relationship to himself [102]. With the retinotopic organization observed early in the visual pathway, an object's registered location in space depends on its position in relationship to his gaze (a retinocentric frame of reference). As visual information flows through the dorsal stream, its location may be converted to a position in relationship to the head ("craniocentric") and body ("egocentric" frame of reference). Navigating through an environment, information about the location of peripheral objects in relationship to oneself must be updated; the spatial relationship between objects may also be required, independent of one's own location (an "allocentric" frame of reference). Evidence for each of these frames of reference can be found in parietal cortex [103–108]. At least in non-human primates, the activity of some parietal neurons can respond to more than one frame of reference, depending on task requirements [109].

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[2] The visual field below / right of center is located above the calcarine sulcus in the left cortical hemisphere, for example, whereas the corresponding parts of the visual field observed left of center are represented in the corresponding regions in the right cortical hemisphere.

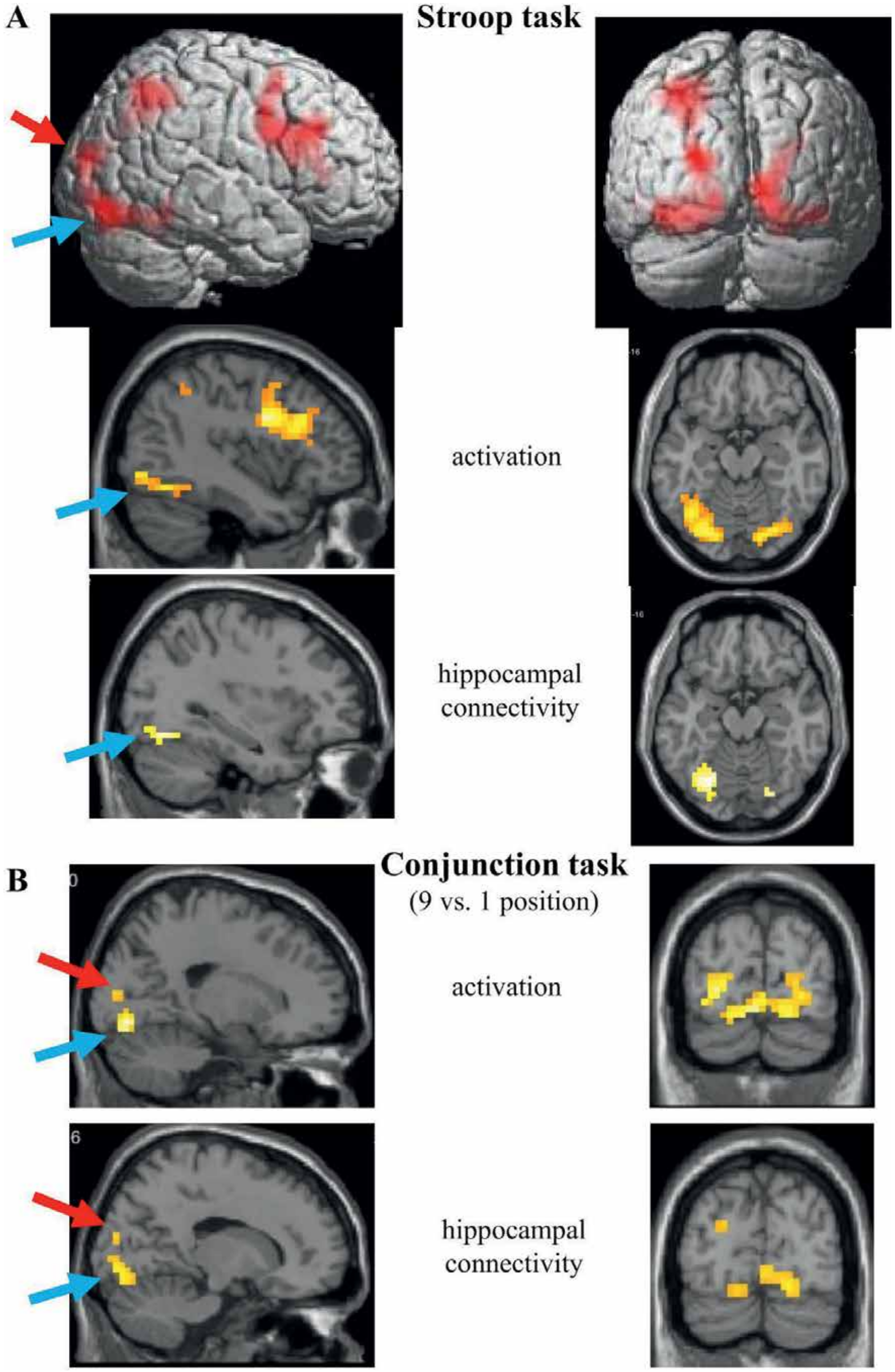

Figure 1.
Activation and hippocampal connectivity. A, activation during performance of the Stroop task includes language areas and bilateral occipital cortex, including an inferior region specialized for color. Red arrows illustrate central vision in primary visual cortex, blue arrows illustrate the area specialized for color. B, connectivity during an attentionally demanding conjunction task for color and shape includes occipital regions involved in central vision and color.

In a rat navigating a maze, activity in the hippocampus reflects a cognitive map of its position within its surroundings [110]. Activity in one part of the hippocampus increases when the rat enters one arm of the maze and then decreases as the rat leaves the area; at the same time, activity in another area of the hippocampus picks up. Spatial maps are also constructed within the human hippocampus during navigation tasks, including virtual navigation tasks. In these tasks, a subject does not actually move, instead using a joystick to navigate through a virtual environment in a video game. Navigation experiments, both in rats and humans, have demonstrated reliance on spatial information from specific visual cues [111]. As in other visuospatial tasks, then, attention to the spatial location of objects is important for accurate performance. In humans as well as other mammals, the hippocampus has strong connections with parietal cortex [112–114].

In humans, the hippocampus has particularly strong connectivity with the precuneus [115], a parietal region extensively connected with visual cortex. As part of the default mode network, precuneus activity is the highest at rest, decreasing in magnitude when a task is performed. Paradoxically, accuracy performing tasks nonetheless correlates with precuneus activity—that is, higher precuneus activity during the task leads to better task performance. One plausible explanation is that the hippocampus and precuneus suppress visual activity outside the attended region (see *Examples and illustrations* below). In this scenario, all visual activities are suppressed in the resting state, but when performing a task, an attended region of space avoids this suppression. This top-down modulation effectively filters irrelevant visual information. This process complements the earlier spotlight theory of selective attention [116]; rather than facilitating visual activity at the focus of attention, the precuneus suppresses visual processing elsewhere.

The precuneus is also involved in other cognitive functions, including episodic memory, language performance, and theory of mind (where events are considered from another's viewpoint). For effective performance, these cognitive functions all require a restricted selection from numerous possibilities. Episodic memory, for example, requires the selection of a specific combination of events to distinguish it from all others, especially those with similar context. Context aids interpretation and provides access to similar events from our past; the precuneus restricts alternatives when most are unsuitable for the task at hand.

Examples and illustrations: Suppression of irrelevant visual information was demonstrated in a perceptual learning task. This task was based on findings that rote rehearsal increases the perceptual span for unfamiliar music or words [117]. A subject rehearsed two unfamiliar musical passages, one following traditional Western music traditions (tonal music) and the other less predictable (atonal music). When a stimulus from the passage was presented for 200 ms, the number of notes or letters that could be perceived increased incrementally from 4 or 5 notes prior to any rehearsal (scanner session 1) to 11 notes following 30 rehearsals (scanner session 4). When they appeared, the subject must identify wrong notes anywhere within the musical passage. This test is attentionally demanding, particularly for stimuli whose length is near the limits of the perceptual span.

Figure 2 shows activation (red) evoked across the entire task ("all notes") or by stimuli with a designated length, along with the composite connectivity from anywhere within the hippocampus (yellow). The composite positive connectivity from the entire hippocampus is shown; the procedure for identifying connectivity from each voxel throughout the hippocampus is described elsewhere [118, 119]. Both activation and

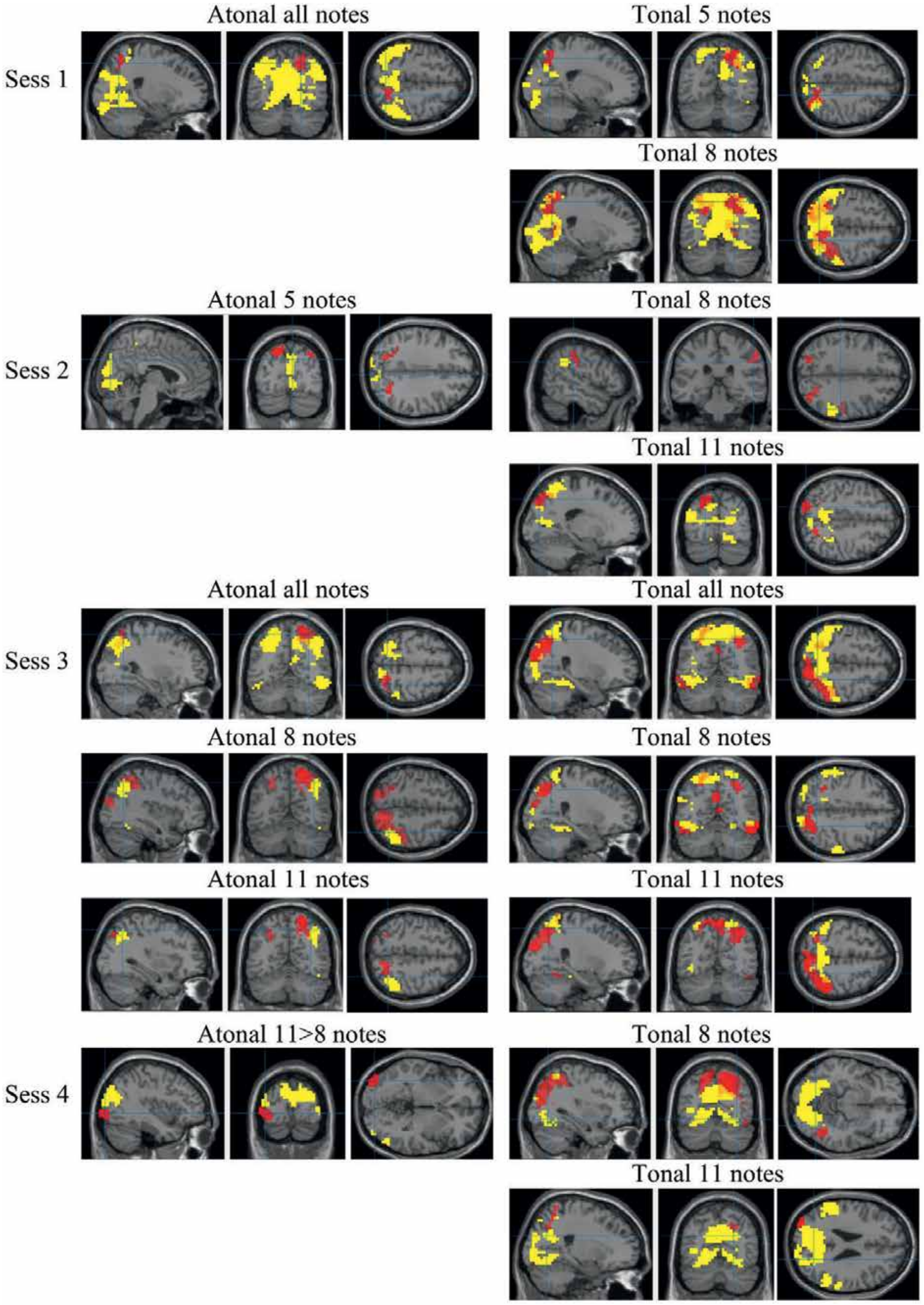

Figure 2.
Activation and hippocampal connectivity in visual cortex during perceptual learning. Activation (red) was generally adjacent to, or sandwiched between regions showing hippocampal connectivity, suggesting hippocampal influences suppressed irrelevant visual activity. Experimental stimuli consisted of 5, 8, or 11 notes from a rehearsed passage presented for 200 ms, the subject indicating whether or not an incorrect note appeared; the control stimuli consisted of percussive notes (Xs), the subject indicating whether few (5) or many notes appeared (8 or 11).

connectivity used a threshold of p = 0.05 with a family-wise error correction, applied to the visual region encompassing the parietal and occipital lobes plus fusiform gyrus.

Three trends are notable. First, hippocampal connectivity generally surrounds, or is adjacent to the activated region across all sessions and task conditions; the region of connectivity significantly excluded the area of activation ($p < 0.001$ calculated from the hypergeometric distribution). Overlap (orange), when observed, was limited to superior parietal cortex or regions at the activation border. Second, the specific region activated, with its surrounding connectivity, depended on the type of stimulus (atonal or tonal) and its length relative to the perceptual span. Although activation was common in superior visual areas, activation to longer stimuli gradually shifted to inferior regions as the perceptual span increased. (Compare, for example, activation for longer atonal note sequences in session 4 with activation during session 1.) Third, the overall quantity of connectivity tends to decrease with training, yet remains adjacent to the region activated by a given stimulus.

Figure 3 shows a similar relationship between activation and the surrounding composite connectivity from six seeds in the precuneus during atonal stimulus presentation (left) and from two seeds in the pulvinar with tonal music (right). These seeds were identified either from activation or from connectivity maxima from the hippocampus during one or more stimulus conditions. Visual connectivity from each seed was not observed across all conditions; because precuneus seeds were not sampled across all conditions, the relationship of each seed region to perceptual performance could not be readily assessed. Invariably, however, precuneus connectivity was restricted to the area showing hippocampal connectivity, suggesting visual connectivity from the precuneus may indirectly reflect hippocampal input.

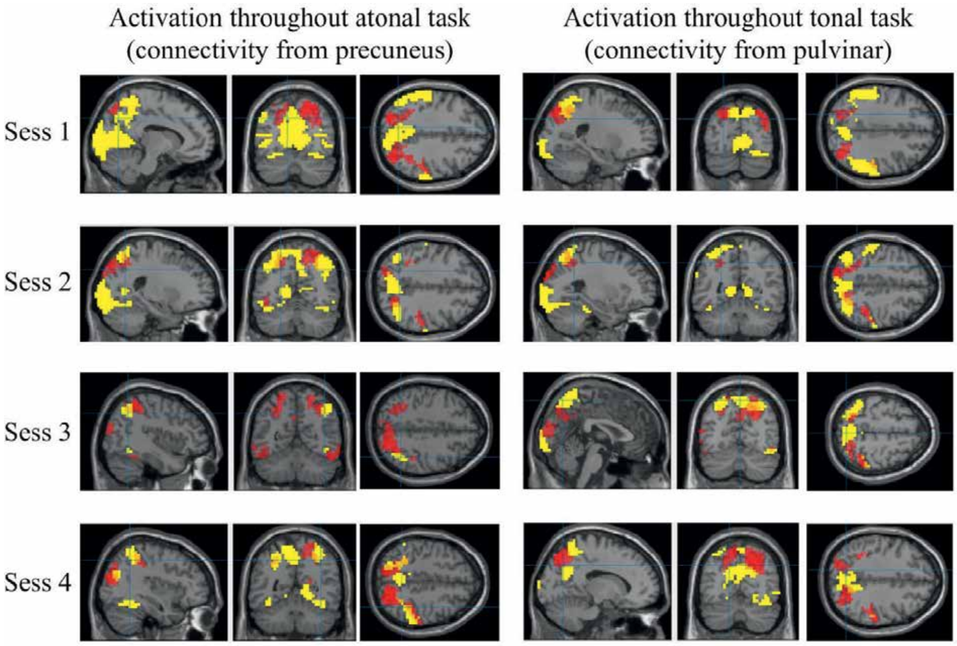

Figure 3.
Activation and connectivity from the precuneus and pulvinar in visual cortex during perceptual learning. Activation is adjacent to, or surrounded by connectivity from each collection of seed regions. Task and conventions as in previous figure.

4.4 Sensory perception: context

Sensory perception requires context for interpretation. A yellow circular spot can represent any number of objects depending on contextual factors, including the object's size, distance (from both its surroundings and observer), and its retinal location. Such a spot may represent a yellow tennis ball, a ping pong ball, the sun, the moon, a painting, a peach, or a light bulb, to name a few possibilities. Contextual interpretation typically occurs automatically; we do not have to think about what that yellow daytime object is in the sky. Our attention may be directed to objects identified through pre-attentive processes, such as the black limb in the grass that we mistakenly identified as a snake when we glimpsed its movement. A number of illusions demonstrate the fallibility of our pre-attentive interpretation in several sensory modalities; all create a context that would allow rapid, accurate identification in most settings.

The pre-attentive context can change based on what we have been thinking. This was driven home to me years ago when I misperceived a specific floater as it drifted in my periphery. I have floaters, small internal specks that move through my eyes as they move from object to object. The shape of one floater is distinctive. Twice in the space of a week, its unexpected movement captured my attention while walking in my backyard. Both times it had moved into my lower right periphery, yet my initial perception was dramatically different. The first time, I thought I perceived a small flying mite; for 10 minutes, I had been considering the mites attracted to our back yard bird feeder as a possible source of tiny bug bites causing my family misery. The second time, I thought my lap dog was approaching; I had been looking forward to playing with him once I got home from work. Although the same floater appeared in the same region of my visual field, my misperceptions had been shaped by the context of my ongoing thoughts.

The context for interpreting our environment often operates below awareness, yet it can be changed with conscious intent. In the Rubin vase/face illusion below (**Figure 4**), I can choose whether to view the black as the foreground (to perceive the faces) or the background (to perceive the vase). I can also choose to see a painting as pigment on a canvas or as the scene it represents.

The context for interpreting our perceptual environment can thus result from top-down conscious processes as well as bottom-up pre-attentive processes. Whereas attentional mechanisms direct where in our environment we attend, context gates what sensory information will be processed. In visual cortex, information about where and what are extracted in different routes or pathways. The dorsal route

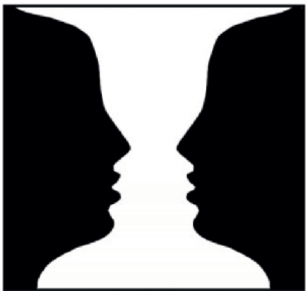

Figure 4.
Rubin vase/face illusion. This image can be perceived either as a vase (light in the middle viewed as foreground) or two faces (black on the outside viewed as foreground).

contains specialized modules for extracting an object's location in space and its direction of movement (e.g., from side-to-side or moving in depth); the dorsal route extends into parietal cortex, where spatial location can be delineated within any of several frames of reference (as noted earlier). The ventral route contains modules specialized for extracting information such as an object's color and the type of object (faces, buildings, etc.). Emotions also provide context for interpreting our environment [120]. Although seldom considered in studies of sensory processes, emotions typically influence cognitive activity across broad areas of the brain, including those involved in visual processing [121, 122].

Cognitive tasks necessarily provide a context for interpreting and responding to task stimuli. At their simplest, these tasks follow the form, "if x, then do y." Contextual influences on sensory brain activity can be identified through task-specific modulation of activity in those areas relevant to task performance. Arguably, three sets of connections are likely to provide such contextual influences: prefrontal cortex, the pulvinar, and the hippocampus.

4.4.1 Context and prefrontal cortex

Prefrontal cortex is sensitive to the context of task requirements, plus it projects to sensory areas. Prefrontal cells in one area, for example, can maintain activity about a cue's spatial location up to 30 seconds; activity fades for longer time periods, as task performance declines [123]. These properties are consistent with a role in providing context for spatial localization ("where"). Other prefrontal regions are sensitive to patterns [124], consistent with a role in providing context for the type of object observed ("what"). Because activity in prefrontal cortex adapts quickly to task requirements or changes in the environment [125–127], it is in position to promote changes in context through its connections with sensory areas.

Prefrontal cortex has extensive interconnections with sensory, motor, and parietal regions. Its connectivity with motor areas is enhanced during tasks that require selection of an action, instead enhanced with sensory areas in tasks that require selection of a sensory property [128]. Furthermore, when attending a restricted region of the visual field, prefrontal connectivity with the parietal cortex is enhanced in the attended part of the visual field [129]. Prefrontal connections with occipito-parietal cortex may even select the spatial context required for performing the task at hand, biasing its responses to whichever spatial framework is required (egocentric vs. allocentric) [130].

4.4.2 Context and the pulvinar

Contextual modulation for visual stimuli may also be provided by the pulvinar, which has input from the superior colliculus and extensive connections with cortical visual areas, prefrontal cortex (including the frontal eye fields), and posterior parietal regions [86, 131].

The pulvinar may be involved in visual attention [132–139]. Experimental evidence supporting this conclusion includes the following: Pulvinar activity to attended stimuli is enhanced in the presence of distractors; a subject loses the ability to use attentional cues when the pulvinar is inactivated; and the pulvinar has connections with brain areas associated with attention. An indirect role in attention has also been suggested for the pulvinar through filtering out visual information about distractors (an effect also shown above in **Figure 3**) [134, 140].

Like consciousness, however, attention can refer to several different cognitive processes, which are seldom differentiated in pulvinar experiments. Attention broadly refers to an attentive state that enables a stimulus or its features to be detected; attentive cues can designate *where* a subject should attend [137], *what* feature should be attended [140, 141], or features with emotional relevance [135, 142–144]. In a typical experimental design, the attentional cue provides *behavioral significance* (i.e., context), specifying the behavioral response required by a stimulus. Thus, the cue's role in directing attention is often conflated with its contextual role for behavior.

To differentiate stimulus significance from attention, pulvinar activity was recorded during a task requiring a rapid behavioral response based on a brief cue presented for 30 ms [145]. If the cue was a small circular spot, an eye movement was immediately directed toward it with the next change in stimulus configuration; a square indicated a later eye movement in the opposite direction. Regardless of cue location, the activity of some neurons depended on the timing of the subsequent eye movement; the activity of others depended on its direction. This task was attentionally demanding due to the short stimulus duration, regardless of its shape or location, yet pulvinar activity depended on its behavioral significance.

Most pulvinar findings reflect contextual influences of a stimulus on behavior (see especially [138, 143, 144, 146, 147]). Because different regions within the pulvinar have distinct patterns of connections and functional properties [132], however, the pulvinar may carry out multiple cognitive and sensory functions.

The pattern of pulvinar connectivity during perceptual learning noted in **Figure 3** suggests a role for the hippocampus shared with the precuneus in filtering irrelevant information. Whereas the hippocampus is suggested in this monograph to play an instrumental role in normal alert consciousness, a *direct* role for the pulvinar is not supported. Two sets of observations support this conclusion. First, pulvinar properties, including latency, are inconsistent with conscious experiences during normal alert consciousness. Pulvinar response latencies were distributed into three distinct ranges in macaque monkeys. Properties of middle-latency neurons (latencies of 80–125 ms) are similar to early visual association cortices but less strongly selective, and poor differentiation between objects is inconsistent with conscious experiences. This class of pulvinar neurons disappears following lesions of striate cortex [145]. The short-latency cells (30–70 ms) respond equally to any visual stimulus in their receptive fields (except for those cells with color sensitivity [148]), whereas the responses of long-latency cells (>145 ms) are too slow to support behavioral responses observed during cognitive tasks.

The pulvinar's role in blindsight also undercuts direct involvement in normal alert consciousness. The striate cortex receives all visual inputs from the lateral geniculate nucleus in the thalamus; extensive striate lesions lead to a subject's unawareness of any sight, yet they can accurately track moving objects when asked to guess. This "blindsight" is mediated by superior colliculus projections to the pulvinar, which are relayed to MT, a movement-sensitive region in the middle temporal gyrus [149, 150]. Pulvinar projections to the amygdala [151] may also be responsible for blindsight recognition of facial expressions [152–154]. Thus, pulvinar activity is normally insufficient for visual information to reach consciousness. Better outcomes for visual awareness follow striate lesions that occur early in life, however, which are likely mediated by the pulvinar [155]. This suggests the pulvinar has the capacity to provide some level of conscious experience, but only when the normal geniculostriate pathway does not fully maturate.

These considerations are consistent, however, with a role for the pulvinar in an ongoing pre-attentive role in context modulation [156]. The pulvinar sustains the context for interpreting sensory stimuli until the situation changes, when prefrontal cortical activity takes over as it quickly adapts. Changes in context can be instigated quickly from either prefrontal cortex or the hippocampus; the context to be sustained by the pulvinar can then updated by connections from either area [144, 157].

4.4.3 Context and the hippocampus

The hippocampus is a third area that could provide context for interpreting sensory stimuli. The hippocampus is itself sensitive to context across a range of dimensions. In experiments requiring a human subject to remember the order of events, for example, the hippocampal response differs if the order of stimuli changes. Depending on task requirements, the hippocampal response to a stimulus also changes if its duration or its spatial position differs from expectations [158–162].

The targets of direct hippocampal outputs are restricted, so the contextual influence of the hippocampus on sensory processing is necessarily indirect. One plausible pathway is through the prefrontal cortex, which was noted above to project to sensory regions. The hippocampus modulates activity in the prefrontal cortex based on task expectations [163–165]; prefrontal cortex, in turn, modulates hippocampal activity when the incoming sensory information does not match what is expected [166–168]. This loop is ideal for updating contextual processing of information, appropriately influencing activity in sensory pathways *via* prefrontal connections.

Such a role has been suggested for the hippocampal—prefrontal pathways based on symptoms observed in schizophrenia [169–171]. Schizophrenics characteristically misconstrue their environment, including misperceptions of both sensory stimuli and the social meaning of events. The prefrontal cortex in schizophrenics often shows abnormalities in their structural and neurochemical makeup [172–175]; this is the site of action for most therapeutic drug treatments. The medial temporal lobe, including the hippocampus, also shows abnormalities in these patients [176], as well as its connections with the prefrontal cortex [177, 178].

The hippocampus may also modulate contextual processing of sensory information through its connections with the precuneus. As noted earlier, the precuneus is part of the default mode network, showing its highest activity levels at rest; nonetheless, higher activity during a task is often correlated with good performance. This region plays an attentional role by filtering out irrelevant information. Attentional processes must, however, interact with context; the precuneus may play a role in both.

Evidence for both roles was seen in an attentionally demanding perceptual learning task, where the context for interpreting a briefly-flashed stimulus was occasionally altered (see *Examples and illustrations* below). As noted for earlier figures, a subject rehearsed an unfamiliar musical passage and read unfamiliar words; when a stimulus was later presented for 200 ms, the number of notes or letters that could be perceived (the "perceptual span") increased incrementally with the number of rehearsals. This perceptual test is challenging, particularly for stimuli whose length approaches the perceptual span. On those trials where the subject expected to see a word but was presented with music instead, the extent of hippocampal connectivity to the precuneus and nearby regions consistently increased, reflecting increased influence when the context changed. Thus, the extent of hippocampal connectivity with visual cortex depended on whether a stimulus was expected or not; furthermore, the visual region activated often differed (see *Examples and illustrations* below). These differences reflect top-down influences based on the subject's conscious expectations about the stimulus viewed.

In summary, interpretation of sensory input relies heavily on the context of attended items. Multiple pathways may be involved in conscious modulation by context; hippocampal properties are consistent with a contextual role, likely indirect *via* the prefrontal cortex and precuneus. Subconscious, pre-attentive modulation of context may result from pulvinar input.

Examples and illustrations: In the experiment described earlier (**Figures 1** and **2**), subjects rehearsed various musical passages and word lists before each scanning session, resulting in enlarged perceptual spans by conventional testing. **Figure 5** illustrates results when a stimulus appeared unexpectedly, interspersed during trials testing the subject for a different stimulus. In session 2, an unexpected atonal passage with a perceptual span of 8 notes evoked bilateral occipital activation, adjacent to hippocampal connectivity (top left). When testing specifically for this atonal stimulus (top right), additional activation was seen higher up in the brain (within the superior parietal cortex), and hippocampal connectivity was less extensive. Interestingly, this atonal stimulus did not evoke activation when appearing unexpectedly while testing words (not shown), when the span reverted to the 5-note perceptual span observed before training.

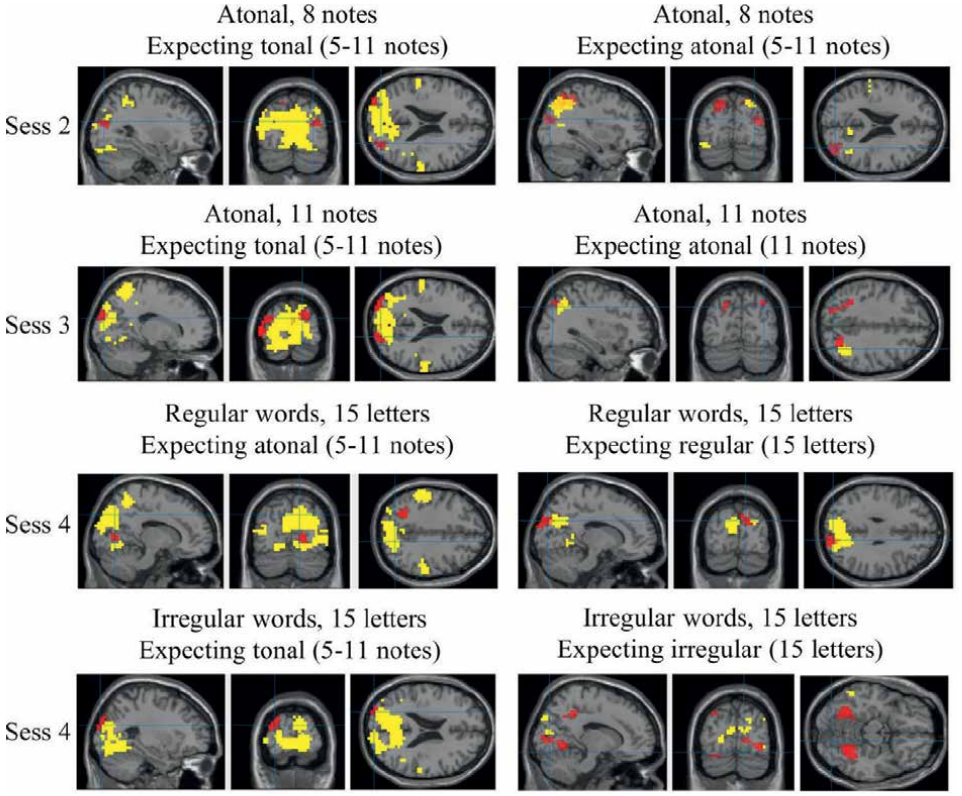

Figure 5.
Effect of context on hippocampal connectivity and visual activation by rehearsed music and words. Presented with a visual stimulus that matched in length the perceptual span, the pattern of hippocampal connectivity and visual activation when the specific stimulus was unexpected (left column) differed from when it was expected (right column).

In sessions 3–4, activation by music or words evoked activation in different visual regions when appearing unexpectedly, accompanied by more extensive hippocampal connectivity. These findings reveal the importance of context in visual processing; how the cortex processes visual information depends on what a person expects to see.

4.5 Memory formation, recall, and forgetting

Many types of memories exist, differentiated by their time courses of retention; many involve different brain regions in their formation and recall. Here, we are most concerned about those forms of memory involved in normal alert consciousness.

Consciousness requires memory to connect moment-by-moment experiences; without this, our experiences would be stroboscopic without any apparent relationship between adjacent frames. Long-term declarative memories, episodic memory, working memory, and iconic memory are all essential to our normal alert state of consciousness.

The hippocampus is essential for the formation of long-term declarative memories (those a subject can describe) and episodic memories (recall of a single event). When the hippocampus is compromised from surgical removal or disease, a subject is unable to acquire a conscious memory that lasts longer than a few minutes. Such a patient forgets meeting someone as soon as they are out of sight; although new skills can be acquired through rehearsal, he will not remember that he learned the skill [179, 180]. Nonetheless, events, people, and facts acquired before they lost hippocampal function can be recalled. Similar deficits are observed in animals with hippocampal lesions. Although the intact hippocampus is active during both memory acquisition and recall, it is only essential for the acquisition stage.

Working memory allows us to remember a phone number long enough to dial it, or remember an object or location until we can act on it. Several areas work together during working memory tasks, although in non-human primates, prefrontal cortex is essential. In non-human primates, prefrontal cortex maintains activity for a briefly-presented item appearing during a working memory task and performs this task poorly in its absence [123, 127]. Indeed, a prosimian monkey lacks prefrontal cortex and will immediately stop reaching for food when the reward is covered from view: literally, “out of sight, out of mind.” Prefrontal cortex is also active in humans during working memory tasks [181–184]. Working memory deficits follow prefrontal lesions, which may result from deficits in executive functions used in working memory tasks [124, 185–187].

Other forms of memory involve recurrent (“reentrant”) circuits of activity that effectively replay the earlier incident [188]. Iconic perceptual memory, for example, re-creates the pattern of neuronal activity in visual cortex generated when the visual image first appeared [189–191]. The same occurs in somatosensory cortex during cued memory recall of a tactile stimulus [192]. Access to these memories appears to be mediated by the prefrontal cortex, whose feedback to sensory cortex initiates the patterned activity [193]. Similarly, recalling a memory of an activity such as playing tennis recreates the pattern of activity in sensorimotor activity generated when playing the game [194].

Prefrontal cortex thus plays a role in memory recall as well as context for sensory processing. These functions are likely related, as *every memory exists within a context*. Memories are evoked from events with a shared context [195]. Noting the empty martini glass when I return to the living room during a party, I suddenly remember why I had gone to the refrigerator; I had developed a craving for martini olives as I discussed the latest James Bond movie with a guest.

Prefrontal cortex thus plays an instrumental role in providing both context and short-term memory recall. The prefrontal cortex does not act alone. Hippocampal activity is sensitive to context as well, embedded within a spatial and mnemonic framework [196]. Furthermore, the hippocampus modulates prefrontal activity based on spatial and temporal context [197]. When sensory information from a new event reaches the hippocampus, the hippocampus is critically necessary to create an episodic memory, whereby it modulates prefrontal cortex to create a pattern of prefrontal activity unique to the event. During memory recall, the prefrontal cortex then provides reentrant activity into sensory circuits, approximating the original pattern of activity. In creating the memory, the hippocampus provided the original pattern of input, and it can do so again to initiate memory recall. As long as prefrontal cortex can recreate the context of the original event, however, it does not require hippocampal input for memory recall.

Context can never be duplicated in entirety, but the greater the number of contextual elements in common, the greater the likelihood a specific event will be remembered and the more vivid its recall. Prefrontal activity typically reflects the current environment, as modulated by the hippocampus and other regions; when the current environment approximates that of an earlier event, its memory may be readily recalled. (In the earlier thought experiment, this explains why sunsets on the beach bring back cousin Anthony's memory of Francesca's death.) The context of an event is provided by everything associated with it, including the emotional state as well as environmental stimuli.

The central executive model explains why the hippocampus is critical for the formation of episodic memories and is typically active during their recall, yet is not essential. It can also explain forgetfulness; if the hippocampus fails to adequately recreate the necessary contextual pattern of prefrontal activity required for memory recall, the memory cannot be intentionally accessed. Once a fuller context is available, the memory can again be recalled.

Similar contexts arise more frequently as we get older. This provides more opportunities to access an older memory, but can also interfere with their access or even modify memories. Because prefrontal cortex provides the context necessary for memory recall but does not necessarily require assistance from the hippocampus, accessing implicit memories from a familiar environment can improve navigational performance for patients whose hippocampal function is compromised (such as Alzheimer's) [198, 199], whereas failure to access prefrontal contextual signals in these patients may exacerbate memory difficulties [200].

4.6 Motor activity

The ability to move is an essential feature of our normal alert consciousness; our ancestors depended on both cognitive and motor skills for survival. Intentional movements are not always required for survival—a baseball flying at my 12-year-old head was better handled by faster reflex pathways, allowing me to react before I realized what was happening—but they do reflect the cognitive activities in our daily lives.

Involvement of the hippocampus in movements was suggested even before its critical role in episodic and declarative memory was recognized. Its intimate relationship to movements is suggested by its prominent role in temporal lobe epilepsy.

Indeed, electrical stimulation of the hippocampus will elicit seizures with a lower threshold than any other place in the brain [201].

For a time, hippocampal involvement in motor function was thought to be limited to its role in remembering learned sequences of human finger movements [202–205] or the spatial goals of rats moving through a maze [206, 207]. Only recently was role of the hippocampus recognized for intentional movements without memory (see *Examples and illustrations* below).

Examples and illustrations: Characteristics of hippocampal connectivity with sensorimotor cortex during two volitional movement tasks are shown in **Figure 6**. Hippocampal connectivity was preferentially distributed within the sensorimotor hand representation, even in the absence of a memory component (repetitive tapping in **Figure 6A**).

Connectivity was best detected through analysis from homotopic regions of the hippocampus; although connectivity was calculated independently from seeds in both the left and right hippocampus, the combination generated significant connectivity over a larger area (**Figure 6B**). Furthermore, hippocampal connectivity was specific for individual fingers, both for seeds associated with positive ("MAX") and negative connectivity ("MIN"). The magnitude of their combined effects was the greatest within each finger representation during the time period when the represented finger was moving (**Figure 6C**). Details of these results may be found elsewhere [118, 119, 208].

4.7 Unity of self

In the repetitive tapping task, PPI connectivity represented the combined effect from both the left and right hippocampus (see **Figure 3**). My research on the hippocampus shows homotopic connectivity is common. Bilateral connectivity is related to the pattern of hippocampal connections; although stronger connectivity is observed with the left MTL, the ventral striatum and default mode network, left and right hippocampal regions show remarkably similar patterns of connectivity [209]. Neuroimaging analysis has traditionally assumed that functional effects are restricted to one hemisphere.

In our earlier sketch of the central executive model, the "center of consciousness" was compared to the White House. Dennett and others have denied its existence; they believe there is no one place in the brain with converging sensory inputs that could also generate cognitive functions. By discarding the Cartesian theater of the mind for this reason, Dennett unwittingly shared a misconception with Descartes; both were looking for one central structure that could regulate diverse functions distributed throughout the cerebral cortex.[3] The White House, however, is occupied by both the President and Vice-President; the left and right hippocampus function jointly in the executive functions of our "self."

What is the "self" and its relationship to the hippocampus and consciousness?

Our conscious view of the world is centered on the self. The brain receives sensory information in relationship to our bodily self. Tactile, proprioceptive, visual, auditory, and vestibular information all project into parietal cortex, converging in various combinations in different regions; perception of one's "self" is largely dependent on areas in the parietal cortex that combine information from different sensory modalities

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[3] Descartes ascribed the centrally-positioned pineal gland as the "seat of the soul" because it was positioned to affect bodily functions represented in both cerebral hemispheres. If this were the case, a lot of zombies are walking around, as the pineal body is calcified and non-functioning in about 10% of adults.

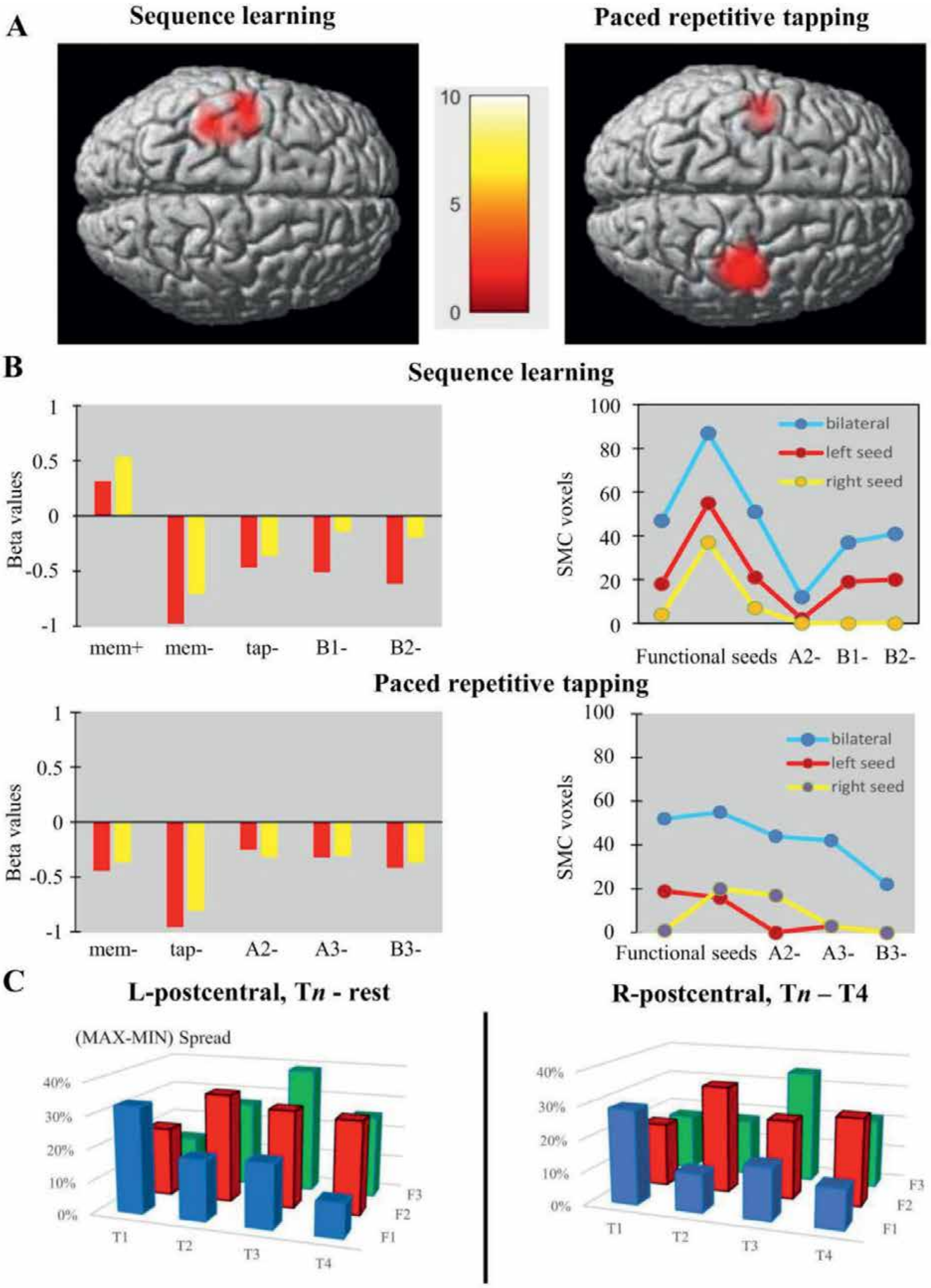

Figure 6.
Hippocampal connectivity in sensorimotor cortex during volitional movements. A, sensorimotor connectivity from the hippocampus during performance of volitional hand movements. Connectivity was restricted to the hand representation as identified from activation analysis. B, mean beta values and the number of voxels showing significant connectivity from functional and structural seeds in the left (red) and right hippocampus (yellow), plus their combined effect from global analysis (blue). Global analysis provided greater sensitivity for detecting effects. C, spatial/temporal selectivity by functional seeds S1 (blue), S2 (red), and S3 (green) within the individual finger representations (F1–F3) during the time period when the represented finger was moving (T1–T3, respectively). The difference in magnitude between the MAX (representing positive connectivity) and MIN seeds (representing negative connectivity) are shown along the z-axis; both increased in their deviation from the mean during finger movements.

to differentiate our “self” from others [210–213]. The embodiment of our self in our senses explains why a pinch on my arm hurts *me*, and a baseball on a trajectory toward my head feels like it’s directed toward *me* (my “self”). Intentional movements are also embedded in our sensory perceptions; otherwise, movements of our eyes and bodies would impact the perceived location of objects in the world around us. Sensory information reaches the hippocampus from parietal multimodal cortex as well as unimodal regions, where it can be consolidated into a conscious, long-term declarative memory.

Social interactions are centered on the self. These interactions typically involve language as well as our senses and are more likely to evoke an emotional response. We have internal dialogs as we decide how to act and react, recalling memories of others and our imagination about possible consequences of our actions. The hippocampus is central to this, modulating language areas of the brain during language processing [98, 214–217], as well as prefrontal regions involved in memory recall and predictive imaginings [165, 168]. The hippocampus also interacts with orbitofrontal regions implicated in decision-making and emotions [218, 219].

Additionally, the hippocampus plays a role in maintaining my conceptualization of my self across time. My normal alert state of consciousness is lost when I fall asleep; in the moment I wake up, I may not remember where or even who I am. An Alzheimer’s patient recently told her daughter that for the first time in a long while, she remembered who she was when she woke up that morning. By the same token, I have had dreams where “I” am the President of the United States, a super hero, or a fictional character. My memories, including those of going to sleep last night, establish myself as the same entity I was yesterday. The cells in my body are replaced every few years, yet my identity (“self”) has been preserved through memories of experiences in my younger body.

In some patients with severe epilepsy, connections between the two cerebral hemispheres are severed to eliminate the spread of seizure activity. These “split-brain patients” function reasonably well, yet careful examination suggests the minds of these patients have split—two distinct “selves,” with different likes, dislikes, and aspirations, without the normal integrated sense of self [220, 221]. This was discovered by utilizing knowledge about neuroanatomical pathways. Sensory and motor pathways cross, so a written question presented left of center is interpreted by cortex in the right hemisphere; the left hand can move wood blocks to select an answer (similar to a multiple-choice test). Language is represented on the left side of the brain, so a response to a question reaching the left hemisphere can be spoken. Surprisingly, the left and right cerebral hemispheres often differed on their favorite color, what shirt they would like to wear, and career choices. In some patients, this conflict played out in daily life; one hand might unbutton a shirt as the other buttoned it up.

Why would a single “self” split into two from this surgery? Typically, the hippocampus influences cortical areas through the combined effect from the left and right structures, but in split-brain patients, connections between the hemispheres are lost. Each hippocampus would assume its own sense of “self,” based on its available sensory inputs, memories, and context.

4.8 Losing one’s self

If the hippocampus maintains our sense of self across time, what happens when the hippocampus becomes nonfunctional, as with Alzheimer’s? Does our “self” cease to exist?

The hippocampus is essential for the formation of long-term declarative memories. These memories are essential to our normal alert consciousness, sustaining the conceptual schema of our self. The "self," as explained earlier, originates with our inborn bodily experiences, but extends to incorporate those character and personality traits we embody. Destroying the hippocampus does not eliminate established memories, as memories involving similar context can still be recalled through prefrontal modulation of sensory pathways. (Sustained context may additionally be provided by the pulvinar).

Neither does hippocampal damage eliminate other forms of consciousness. Patients still have sleep-wake cycles (although often disturbed) and can perform tasks that do not require extended attention. Despite their deficits, these patients maintain a sense of control and ownership over their body parts that is embedded into the brain early in development, their bodily sense of self.

What these patients have lost is the ability for their normal alert consciousness to evolve with new experiences. As a result, that portion of the self that chooses how to interact with the world gradually erodes. My grandmother was a modest, gentle woman who lived for decades with a misogynist husband, yet she found ways to keep the peace while standing up for herself and her daughters. As Alzheimer's developed, she initially remembered family events from years earlier, but did not recognize her own children. As the disease progressed, my grandmother remembered even less about her family; she became aggressive, physically attacking staff at her nursing home, and running naked around the hallways, refusing to get dressed. As my mom noted, she was no longer the same person—no longer the "self" who had sustained intentional choices for years about who she was and how to interact in her world.

This is a common complaint in families of Alzheimer's patients; except when memories rise to the surface, their loved ones are "no longer the same person." The bodily self remains, but the constructed self is lost. The bodily sense of self is independent of the normal alert state of consciousness, although the bodily sense of self can be diminished or altered during alternate states of consciousness associated with meditation [222, 223] or psychedelic drugs [224].

4.9 Emotions and decision-making

When we consciously perceive an object or event, our subjective ("phenomenological") experience is tied to both our sensory perception and our emotional response to it. This emotional response may be unique, differing even between similar objects. The unique sensory/emotional response for an object represents its qualia.

The thought experiment involving cousin Anthony and his fiancée Francesca illustrated an important point: Our emotional response to environmental objects and events can change with our experiences. Because everyone's experiences and emotional responses differ, our qualia for objects also differ. Another point from Anthony's experience: Our emotions provide context for interpreting our environment. Everything associated with an event provides a context that can be used in its recall, including emotions as well as their timing and surrounding objects in the environment.

If an event evokes fear, we are more likely to recall the incident when we are again fearful. The fear will also shape its interpretation [225]. When in high school, a casual comment—"nice clothes"—by a bully carried dark overtones, which I would not impute to a friend making the same comment. Similarly, we tend to be more optimistic when we are happy, plus more likely to remember happy events from our past. The nature of our long-term memories is affected by our emotional state as well; when negative emotions are evoked by an incident, we are more likely to remember details of the event months later [226, 227].

Brain regions involved in emotional expression of our conscious experiences vary in the duration of their effects. Long-lasting effects are evoked by strong emotions, mediated by hormones released from the hypothalamus and pituitary. These brain structures are insensitive to sensory properties of events themselves; their role in the physiological manifestation of emotions requires input from the limbic system.[4] Limbic pathways include both the hippocampus and amygdala, the latter strongly involved in the emotional component of our experiences. Although we are cognitively aware of emotions evoked by an incident, the hormones released affect bodily functions throughout the body and brain, many of which lie outside our consciousness.

Activity in the amygdala is intimately involved in our emotional responses to the environment. Its activity is most often reported in studies where negative emotions are evoked, but studies have demonstrated its involvement in positive emotions as well [229, 230]. The duration of amygdala activity evoked by an emotionally charged stimulus is generally longer by a few seconds than in sensory regions; the duration of its effects may be considered intermediate. Although the hippocampus appears to be more involved in "objective" representations of our environment, its connections with the amygdala are extensive and bidirectional. Both structures are involved in learning [231–233].

Strong positive emotions associated with pleasure and reward evoke activity in the nucleus accumbens [234–239]. The nucleus accumbens is active during sexual arousal and other hedonistic pleasures, including narcotics; it is also more active in compulsive gamblers when they are gambling, regardless of whether they are winning. The hippocampus and subcortical regions involved in the reward circuitry also interact with the nucleus accumbens [240]. Through hippocampal connections, memories of previous experiences can reinforce positive experiences.

The short-term expression of emotions involves the orbitofrontal cortex, an area located just above the orbit of the eyes. This area, also called the ventromedial prefrontal cortex, is involved in reward circuitry [241]. Anterior orbitofrontal regions typically become active when negative emotions are evoked, with positive emotions evoking activity posterior to this (see *Examples and illustrations* below). The hippocampus influences orbitofrontal regions more strongly during emotional expression and recall [242]. With shorter-term effects, orbitofrontal activity reflects our emotional responses to continuous changes in our environment, such as those evoked in social situations. Our emotional response to these events affects our decision process; indeed, the orbitofrontal (ventromedial) cortex is critical for making decisions requiring evaluation of long-term consequences and socially appropriate behavior [218, 243–245]. Orbitofrontal activity can be modified by cognitive strategies that affect the intensity of our emotions, for example, when a person tries to minimize their emotional response to a disgusting image [246, 247].

Orbitofrontal regions are active when subjects are presented with words that carry emotional associations [54, 56]. Even without overt emotional associations, activity in the orbitofrontal region is modulated by the hippocampus during word tasks (see *Examples and illustrations* below). Orbitofrontal activity in these tasks reflect nuanced

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[4] The limbic system was first defined by Papez [228] as those intermediary brain structures that convey output from cortical areas involved in cognition to areas involved in the expression of emotions.

emotional responses that can change rapidly as we read a story, or to other events in our environment.

Hippocampal connectivity with orbitofrontal cortex effectively associates our memories with subjective, emotional content. This provides a subjective tint to our conscious experiences, especially those involving memories. This interaction also plays an instrumental role in decisions and judgments that guide our social behavior [248].

Examples and illustrations: To examine the neural response to emotional responses, pictures rated for emotional affect were presented. Differential activation between pictures evoking strong positive and negative emotions was recorded from 16 subjects. Responses between individuals was variable, as some subjects regulate their emotional responses to strong evocative images. **Figure 7A** shows regions showing a preferential response to emotionally charged negative (red) and positive stimuli (yellow). Activation associated with negative emotions appeared in orbitofrontal cortex anterior to activation associated with positive emotions, which extended from orbitofrontal cortex posterior to the nucleus accumbens (below the genu and rostrum of the corpus callosum). **Figure 7B** shows frontal deactivation when recalling word associations (top, blue) and its overlap with the orbitofrontal region associated with positive emotions (bottom, overlap in green). **Figure 7C** shows hippocampal connectivity with orbitofrontal cortex when presented with emotionally charged negative stimuli, both from various structural seeds in the left (left) and right hippocampus (right). These results show that the hippocampus interacts with orbitofrontal regions involved in both positive and negative emotions.

4.10 Thought and creativity

Typically, we use language to think; only occasionally do we think through visual icons or other nonverbal sensory representations. The use of language is nearly universal across human societies.

Consistent with a role in cognitive thought, the hippocampus influences semantic language areas during language tasks [98]. Hippocampal connectivity during these tasks is not limited to the left posterior temporal region (Wernicke's area); it is also evident in the left inferior frontal gyrus (Broca's area). Prefrontal cortex modulates the contextual modulation of information in posterior areas, affecting both the interpretation of sensory input and memory recall. Because every memory is embedded within a context, including memories of words, this contextual modulation is likely essential for thinking.

In English and other Western cultures, linguistic content is associated with the left hemisphere, whereas the right hemisphere interprets the "music" of language (i.e., inflections in our voice). Upward tonal inflections are used to convey questions, flat or downward inflections indicate statements, and nuanced inflections convey state of mind. For Chinese and other tonal languages, however, inflections help determine the meaning of individual words. As a result, Chinese language studies also show activation in the right hemisphere [249].

The laterality of brain activation thus depends, in part, on the language spoken. Other hemispheric differences in functionality have also been reported, for example, math is left-dominant, at least in right-handers [250]. What might explain such differences?

The two cerebral hemispheres differ in the speed of sensory processing; the left hemisphere processes information faster [251, 252]. Motor nerve conduction velocity is also faster on the side of the dominant hand [253, 254]. Broadly, speed is important

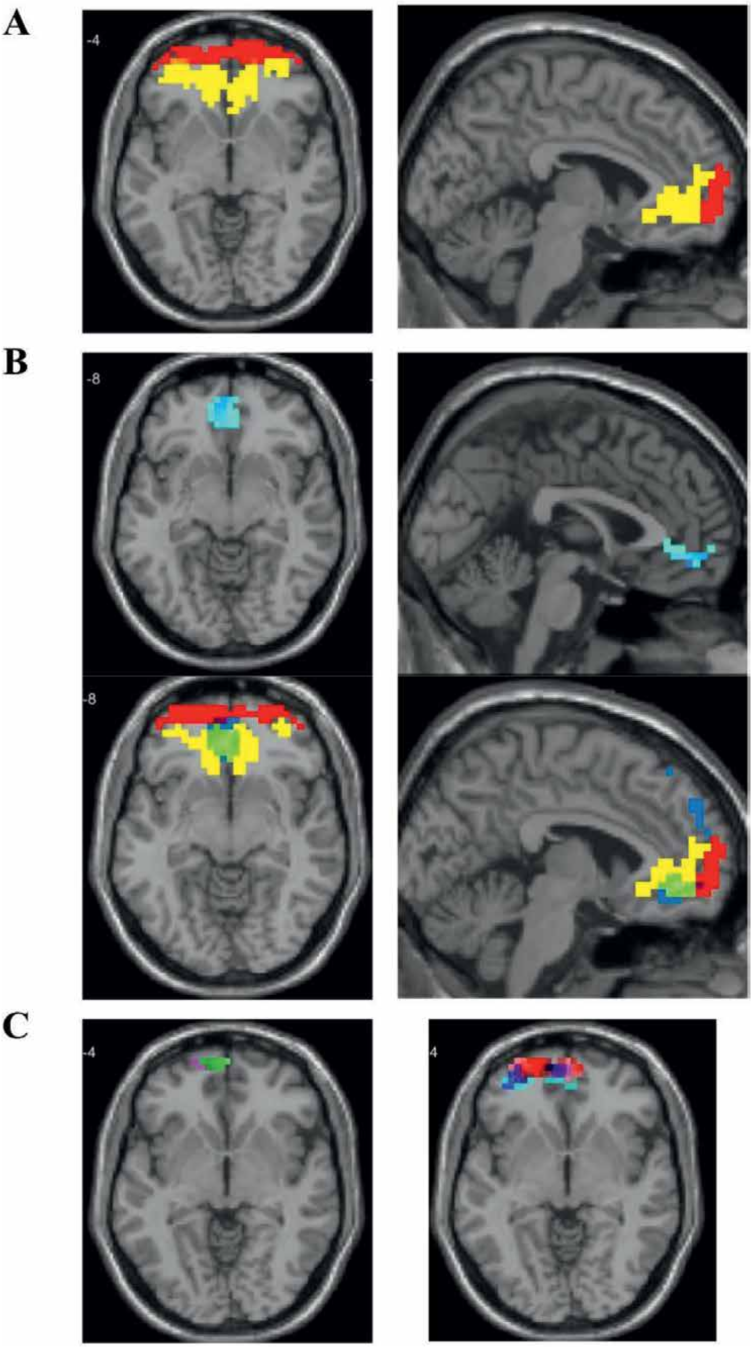

Figure 7.
Orbitofrontal activation and hippocampal connectivity associated with emotional stimuli. A, orbitofrontal regions activated preferentially by sensory stimuli evoking negative (red) and positive emotions (yellow). Negative emotions activated orbitofrontal cortex anterior to that activated by positive emotions, observed in three or more subjects from a group of 16 subjects. B, deactivation of orbitofrontal cortex during recall of word associations (blue) in relationship to regions associated with emotions. Overlap with positive emotions appears green. C, hippocampal connectivity with orbitofrontal cortex when presented negative emotionally charged stimuli relative to a neutral stimulus. Different colors represent the results from different structural seeds in the left hippocampus (left) and the right hippocampus (right). Greater connectivity was observed in left orbitofrontal cortex.

for those functions associated with the left hemisphere. In Western languages, this includes semantics. "Watch out!" does not require inflection; we must understand its meaning as quickly as possible to avoid the rattlesnake or attacking bear. Questions involve the temporal modulation of pitch, invoking the right hemisphere. We can afford more time to respond when contemplating a question about what we would like for lunch.

Processing speed may help explain the prevalence of right-handedness. The left cortex controls the right side of our body; it can more quickly inform our muscles how to tie a knot, throw a spear, or catch ourselves before falling as we learn to walk. Early in evolution, our survival depended on the speed of our physical skills. Survival now depends less on physical speed, yet speed is still required for success in many of our activities—childhood games such as races, IQ tests, and even brainstorming at executive board meetings. By processing sensory and motor information more quickly, the left hemisphere takes the lead; with more practice, plasticity generates rich neural connections required for greater expertise.

In the right hemisphere, processing is slower. This has functional implications. Temporal characteristics of stimuli, for example, assume greater prominence; the right inferior prefrontal cortex responds to rising and falling inflections during speech. Spatial relationships also assume greater importance; extensive damage to the right parietal cortex results in spatial hemineglect [255–257]. In this condition, a patient is unable to perceive items to the left half center; asked to draw a clock, all 12 numbers will be crowded into the right half of a circle. Although spatial maps are present in each hemisphere, spatial hemineglect only occurs following damage to right parietal cortex.

Slower processing in the right hemisphere may also play a role in creativity. Prefrontal cortex modulates context in sensory and other ascending pathways, essential for interpreting stimuli in our environment. Because processing in the left hemisphere is faster, the left prefrontal cortex can provide a quicker change in context. This is adaptive when a quick response is needed to a changing environment; slower changes in the right prefrontal cortex, however, provide greater continuity in thought. In a changing environment, the two hemispheres thus provide different contexts for interpreting events. Context determines which sensory modules extract information about our environment, which is then relayed to the hippocampus. Because the left hippocampus and right hippocampus work jointly to influence other brain regions, interpretive differences must be reconciled. This may require additional access to relevant memories, those available to the hippocampus.

The creative process requires competing contexts. Acts of creativity result from a well-established context applied to a new situation, or a new context applied to an older situation. The Impressionist artists in the 1800s, for example, captured the impression of scenery through blended brush strokes that failed to duplicate details of the images they observed; musicians then applied this concept to music, creating music that blended broken chords to create impressions without overtly relying on melodic lines and established chord progressions. Similarly, differences in viewpoints played a prominent role in conceptual theories, such as the special theory of relativity. Original and creative acts are generated by applying an established concept to a new situation, or the reverse.

By working jointly, the left and right hippocampus brings the context from both cortical hemispheres into play. Difference in processing speeds between the cortical hemispheres may thus lead to creative solutions by providing different contexts for problem-solving. Activity in the left hemisphere dominates when a speedy response is required; right hemispheric activity becomes equally important during the creative process. This explains why the right hemisphere appears to become more active during creative endeavors [258]. With unilateral inputs but bilateral cooperation, the hippocampus is uniquely positioned to use different contexts to view a situation from a fresh perspective, or to find a new approach for an unsolved problem.

4.11 Individual variability

Normal alert consciousness is uniquely individual. The content and nature of my conscious experiences differ from yours, and because my conscious experiences differ day-to-day and moment-to-moment, identifying the neural activity unique to my individual consciousness is a challenge. Individual variability in fMRI studies is often explored through correlations between the amplitude of evoked brain activity and a population variable (e.g., performance accuracy). This approach is generally unsuitable for exploring individual differences in the conscious experience, as tasks rarely differentiate between different states of consciousness.

Individuals vary greatly in the extent and location of areas active during performance of any task, resulting from individual differences in perception, abilities, strategies used to perform the task, and variability in the functional brain organization [259]. As a result, brain activation identified from group analysis is typically more restricted than activation among individuals.

I learned an extreme example of this lesson early in my hospital job. Neurologists asked me to develop fMRI tasks to demonstrate the laterality of language function in the hippocampus, noting studies had recently shown greater activation in the left hippocampus of unimpaired patients.[5] Regardless of task or analytical methods, I could never demonstrate preferential activity for language in the left hippocampus for more than half my volunteers. Group analysis, however, replicated findings from previous studies. On an individual basis, language activation was often greater in the right hippocampus, but the variability between subjects was too large to show up in group analysis.

Our individual conscious experiences, as well as our approaches to solving problems, are unique and variable. Brain activity underlying our conscious experiences should similarly reflect this individual variability, both between individuals (e.g., [260]) and within individuals at different points in time [261]. Across many tasks, the pattern of hippocampal connectivity in individuals follows the pattern of task activation, including activation outside the region typically activated by a group of subjects (see *Examples and illustrations* below).

Examples and illustrations: **Figure 8** shows fMRI activation and hippocampal connectivity in an individual subject across a variety of task conditions. Similarities are evident, both in regions typically activated during group analysis (yellow arrows) and in regions reflecting the individual's performance (blue arrows).

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[5] My tasks were to be used for presurgical evaluation of temporal lobe epilepsy. In some patients, the language function migrated to the other side of the brain, and the post-surgical outcome depended on hippocampal language function on the side of the operation.

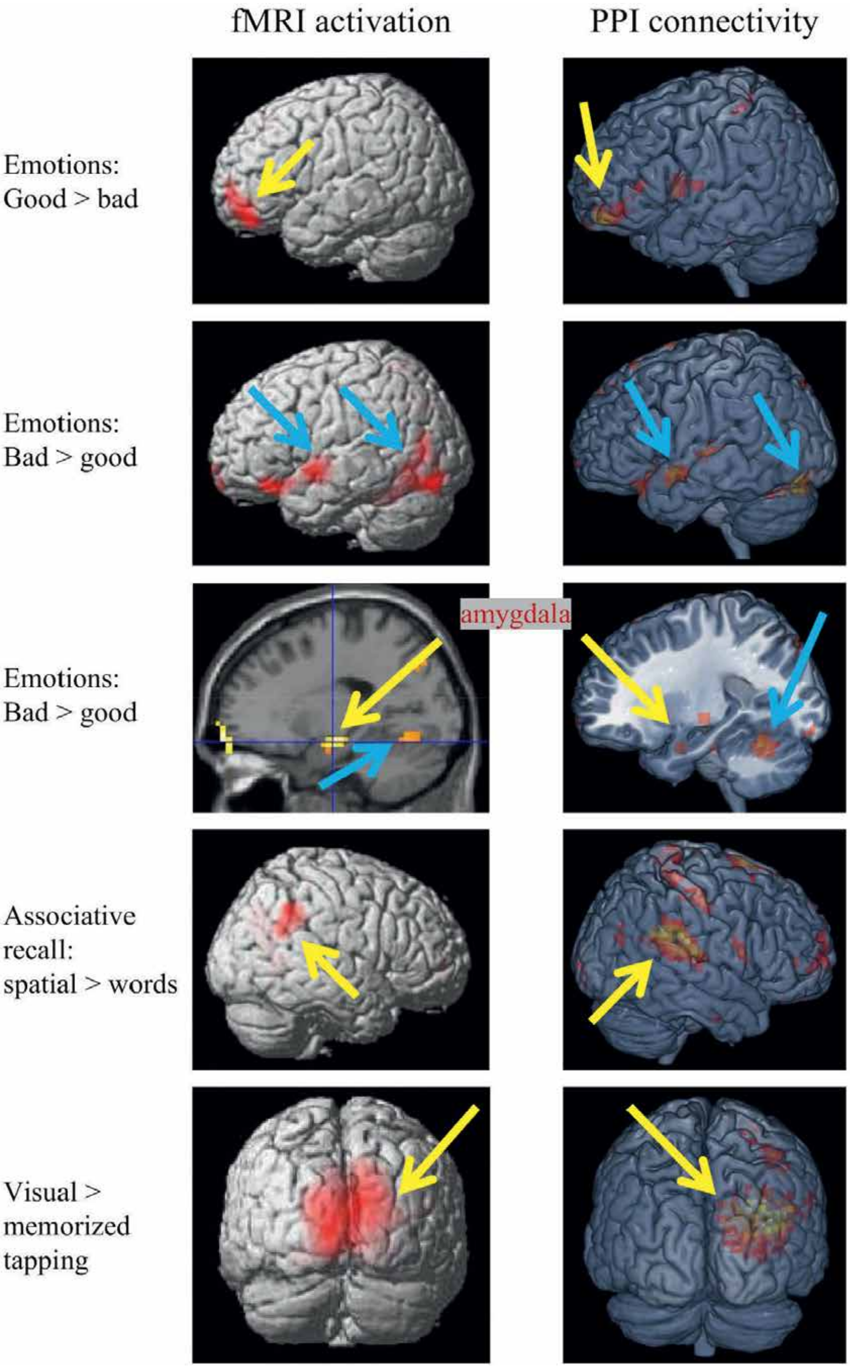

Figure 8.
Activation and hippocampal connectivity in an individual across tasks. Hippocampal connectivity tends to reflect task activation, in regions reflecting individual performance (blue arrows) as well as regions typically activated by a task during group analysis (yellow arrows).

4.12 Normal alert consciousness vs. other conscious states

Normal alert consciousness requires the coordination of cognitive processes such as perception, memory, and attention, as well as behavioral responses and emotional responsivity to people, events, and objects in our environment. Most (and perhaps all) cortical regions are involved in one or more of these components during normal alert consciousness (as are some subcortical regions), so global patterns of brain activity identified in the EEG may differentiate it from other conscious states. The pattern of EEG activity has been compared during waking activity to the drowsy and sleep states.

When alert, the EEG across cortical areas is generally desynchronized, showing rapid beta waves (14–20 Hz) that reflect the brain's responsiveness to rapid changes in the environment as well as our internal mental activity. Activity in the hippocampus, by contrast, is synchronized; slower waveforms result from coordinated activity within the hippocampus. When an animal is particularly alert, the hippocampus produces theta waves (4–10 Hz) that propagate across the cortex. Motor and associative learning are enhanced when they are coordinated with these theta waves. In humans, the frequency of these waves is sometimes slower (1–8 Hz), but their properties are otherwise the same [262–265].

When drowsy, the EEG in occipital (visual) areas is dominated by alpha waves [8–12 Hz], which may function to suppress visual input [266]. During deep sleep, the hippocampus and cortex reverse their EEG patterns from wakefulness: Cortical activity is synchronized as the hippocampal EEG becomes desynchronized. In the dream state, characterized by rapid eye movements (REM), hippocampal activity changes again. During REM, hippocampal theta waves are not propagated across the cortex, but otherwise hippocampal activity is similar to that observed in the awake state. In fact, activity within the hippocampus during REM repeats patterns observed earlier during the day; this is essential for memory consolidation. Deprived of REM sleep, later recall of the day's events can be lost.

With a central role in normal alert consciousness, the reciprocal relationship between hippocampus and cortical activity is to be expected. According to the model, the hippocampus loses cognitive control over sensory and other cortical processes during sleep; regional cortical circuits can then generate coordinated (synchronized) activity, unperturbed by cognitive demands. Imagery during REM sleep may result from hippocampal replay of the day's events, or often, memories and imaginations that share the same context. Consciousness during REM sleep differs from the normal alert state in several ways, however, notably with the interruption of hippocampal cognitive control over movements.

4.13 Subconscious influences on normal alert consciousness

In the central executive model, sensory signals in the normal alert state must reach the hippocampus to reach consciousness; empirically, sensory signals must reach the hippocampus to form explicit declarative memories. The role of the hippocampus in subconscious, implicit memories is more controversial [267–271].

Several questions may be raised. What is the effect of subliminal stimuli on hippocampal activity and conscious behavior? Is evoked activity in the hippocampus both *necessary* and *sufficient* to reach conscious awareness in the normal alert state? Why do some sensory stimuli reach consciousness whereas others do not?

Subliminal perceptions and implicit memories, by definition, improve task performance as a result of prior experience, despite a subject's lack of awareness or recall. Many different tasks have been used to test implicit memories; some reportedly involve hippocampal activity, whereas others do not. Clearly, the hippocampus is not required for all types of implicit memories; procedural memories are implicit memories that can be formed by amnesic patients with hippocampal damage.

A review of brain development and plasticity may help us better understand implicit memories.

The brain develops as a rostral extension of the spinal cord, developing and specializing in response to chemical gradients [272]. The functional organization of the brain suggests specialized sensory regions of cortex may also develop from chemical gradients. Visual cortex, for example, may arise from a gradient with peak intensity at the back of the brain; other gradients give rise to auditory cortex (near the ears), smell and taste cortices (near the front of the brain), and somatosensory cortex (near the top of the brain). These chemical gradients attract axons arising from the appropriate pathways, yet their functional properties require evoked *activity* within their respective pathways. Sensitivity to contrasts, edges, directional movement, and binocular vision, for example, arise later from visual stimulation during a critical period following birth [273, 274]. Plastic changes in brain function continue throughout our lives, altering our brain's response to an environmental stimulus.

Implicit memories can result from plastic changes in activity anywhere in the brain that does not reach consciousness. Demonstrating their existence requires the appropriate task, which typically selects the appropriate context. The anterior hippocampus is commonly active (if at all) during implicit memory tests, which connects with prefrontal regions known to regulate context and memory recall. These prefrontal regions can activate reentrant circuits, including sensory pathways modified during implicit learning. Hippocampal activity during these tests may thus reflect cognitive control over task performance, rather than a direct role in implicit memory.

Even if sensory information reaches the hippocampus, it may not reach conscious awareness, particularly if the stimulus duration is short. Our minds require time for perception to occur. Numerous studies have identified behavioral effects of words or objects presented too briefly to be conscious perceived [275–280]. Like implicit memories, subliminal stimuli can modify subsequent behavior despite their failure to evoke conscious awareness. Sensory information in the brain must be integrated over time, so stimuli presented too briefly do not reach consciousness.

The importance of temporal integration is apparent from our perception of movements when watching television and movies, where a series of static images is presented in quick succession. Temporal integration of visual stimuli occurs over 20–60 ms, depending on stimulus properties[6] [283] (explained in *Examples and illustrations* below). The perceived location of an object is integrated from its actual location(s) over time. Learning to recognize a brief stimulus is optimal when it is presented at a certain point in the theta waveform. This sensory information decays before the same point in the waveform is again reached [263, 284]. At 4–8 Hz, information within the hippocampus must therefore require a maximum time of 125–250 ms for sensory integration. This is considerably longer than the 25 ms minimum integration time required for perception, yet the hippocampal theta waveform likely reflects the time required to both perceive a stimulus and act on it. A single cycle of the theta waveform represents the shortest time that humans can initiate an eye

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[6] This lower range corresponds roughly to the 40 Hz cortical waveforms that coordinate sensory activity across brain regions, thought to be involved in perceptual grouping and attentional selection [281, 282].

movement toward a suddenly appearing target. (Hand movements have longer reaction times, due to longer nerve pathways and greater muscle mass).

A subliminal stimulus evokes a neural response early in the sensory pathway, but integrated with other sensory inputs over time, this does not persist long enough to reach consciousness. Nonetheless, a subliminal stimulus that modifies sensory activity early in the pathway can still influence conscious behavior, albeit indirectly, because sensory areas provide input to regions necessary for consciousness. Once consolidated, subliminal sensory input can influence sensory activity whenever the context of the original (subliminal) stimulus is recreated.

Examples and illustrations: Evidence of temporal integration from displacement of static images is represented in **Figure 9** (adapted from [285]. In **Figure 9A**, a spot changes color as it is displaced; if the displacement is large enough, two spots separated in time are perceived, but with a smaller displacement, the green spot appears to change color as it moves from one point to another. Motion is perceived; the transition in color appears midway. The perception of motion and its change in color both reflect the temporal integration that takes place; red and green are opponent colors, so brain cells excited by one color will be inhibited by the other.

Figure 9B illustrates the effect of temporal integration on eye movements in a double-jump saccade task [286, 287], where a subject's gaze follows a moving spot. If a spot moves to two locations (P1 and P2) before an eye movement can be initiated,

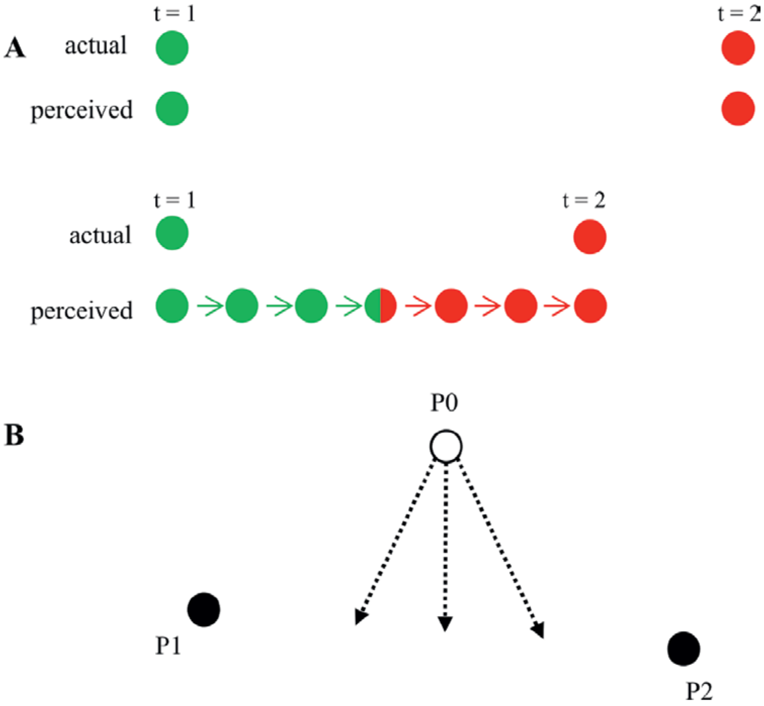

Figure 9.
Experiments in movement perception and direction of gaze following rapid sequential presentation of static images. A, a green and red spot appearing shortly after each other at t = 1 and t = 2 appear as sequential spots if appearing sufficiently far apart (top), but appear as a rapidly moving spot that changes color midway if spaced closer together (bottom). B, a subject following a spot that quickly changes position to P1 then P2 will redirect his gaze somewhere in-between, closer to that position where the spot appeared longer before the eye movement was initiated. Results suggest a temporal integration of stimulus position.

the gaze is directed to an intermediate location. The target location for the gaze then depends on the duration of the spot at each location before the eye movement is initiated. If more time is spent at P1, the gaze lands closer to it, whereas the gaze is directed closer to P2 if the spot appears there for a longer time. Temporal integration occurs throughout the brain, affecting eye movement computations as well as perceptual processes.

5. Central executive model of normal, alert consciousness

5.1 Concise description of the model

To reach consciousness during the normal alert state, the central executive model asserts that sensory information must reach the hippocampus with sufficient strength to be acted upon. The earlier White House analogy applies here; the occupants of the White House must be aware of the sensory input to act on it.

Before reaching the hippocampus, the content of sensory information has already been extracted (e.g., tree, house, familiar face). The sensory pathways carry both bottom-up signals from responses to external stimuli and top-down reentrant signals generated during thought or memory recall. These are not mutually exclusive; bottom-up responses are often modified by top-down processes, especially those contextual cues associated with spatial attentional control and cognitive expectations. Although rapid changes in context arise directly from the prefrontal cortex, the top-down influences during normal alert consciousness ultimately arise from the hippocampus. When changes in the environment do not require a rapid response, the sustained context may be provided instead by the pulvinar, whose input is not limited to the normal alert state.

Supra-threshold input to the hippocampus from sensory pathways achieves conscious awareness; this input can potentially be encoded into episodic and long-term declarative memory. Encoding results from synaptic connections with prefrontal cortex. Hippocampal projections to prefrontal cortex are essential for encoding but also intentional access to memories; prefrontal connections with sensory pathways specify the context for re-creating the memory. The prefrontal cortex recreates the memory by generating a reentrant signal with the original context, with the reentrant signal propagating through the sensory pathways to reach the hippocampus for conscious awareness. The context (and thus the memory itself) includes sensory information from the original episode, the timing and spatial position of sensory objects, and the subjective emotional reaction to the event, as well as ongoing thoughts at the time of the event. This reentrant signal may also modify existing memories based on the current context.

For most stimuli, the emotional response is relatively subdued, mediated by hippocampal connections with the amygdala and ventral medial prefrontal cortex (orbitofrontal cortex). These subjective responses typically last a few seconds. Stronger emotional responses invoke limbic connections that result in the release of hormones; these evoke stronger neural responses across the body and brain that may persist for hours or days.

Based on its input, the hippocampus may select a behavioral response, acting on those speech and movement centers necessary to carry it out. If a stimulus requiring the same behavioral response appears frequently, basal ganglia activity is modified [288–290] so the response to the stimulus no longer requires the hippocampus. At that point, the action no longer requires the normal alert state of consciousness; driving, for example, has become automatic.

Input to the hippocampus might result in internal deliberations. The left and right hippocampus select different scenarios by accessing different memories, applying different contexts to imagine future outcomes resulting from different courses of action [163]. Positive emotional feedback from ventral medial prefrontal cortex improves the likelihood of selecting one imagined course of action over another; a person may even be predisposed toward a certain outcome due to positive associations from past experiences. Different pairs of scenarios may be considered until a decision is made. Emotional feedback depends on many factors, including the ongoing emotional state, the relative emotional response to each option being considered, and the order of options considered. (Emotional responses to the previous scenario can persist for the slower-reacting right hippocampus.) Creative insights may result when the context from one scenario can be applied to the next.

Typically, the left hippocampus is preferentially involved in long-term memories involving language, whereas the right hippocampus is more involved in spatial navigation [291–294]; nonetheless, the hippocampus in both hemispheres work together to accomplish executive agenda.[7] This bilateral cooperation, although omnipresent, is essential for creative processes that require access to different contexts and memories.

The central role of the hippocampus in normal alert consciousness can be illustrated by mapping out important functional connections and their role in cognitive processes (see *Examples and illustrations* below).

Examples and illustrations: **Figure 10** illustrates the relationship of brain regions and connections essential for carrying out cognitive functions, based on evidence and concepts presented above. Connections intimately associated with the normal alert state of consciousness are shown with bold black arrows.

The hippocampus is central, as its input specifies what reaches consciousness ("conscious of"); output specifies what we consciously do ("intentional" actions). Although the left and right hippocampus act jointly, their inputs are unilateral, with the pattern of inputs and outputs largely replicated in each hemisphere. This arrangement increases flexibility in our behavioral response, as the hippocampi in the two hemispheres must jointly consider information that may differ in context and associated memories. Because the right hemisphere processes information more slowly, the content of the two hemispheres differs; when the context of an endeavor changes rapidly, resolving the disparity between the two hemispheres can lead to creative solutions.

5.2 Model in action

The model's ability to explain conscious experiences in our day-to-day life can be illustrated through an incident I experienced as a graduate student in the 1980s. After recalling my perceptions, thoughts and emotions from each scene, the model's explanation will be provided in italics.

I decided to take public transportation to O'Hare International Airport for a late-night airline flight, transferring to a subway train in downtown Chicago with a backpack on my shoulders and a suitcase in hand. Descending the stairs, I was met with the odor of urine, inducing a wave of disgust as I relived a childhood memory of

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[7] In the White House analogy, the left and right hippocampus may be thought of as the President and Vice-president—each may have their own strong points, but both work on the same executive agenda.

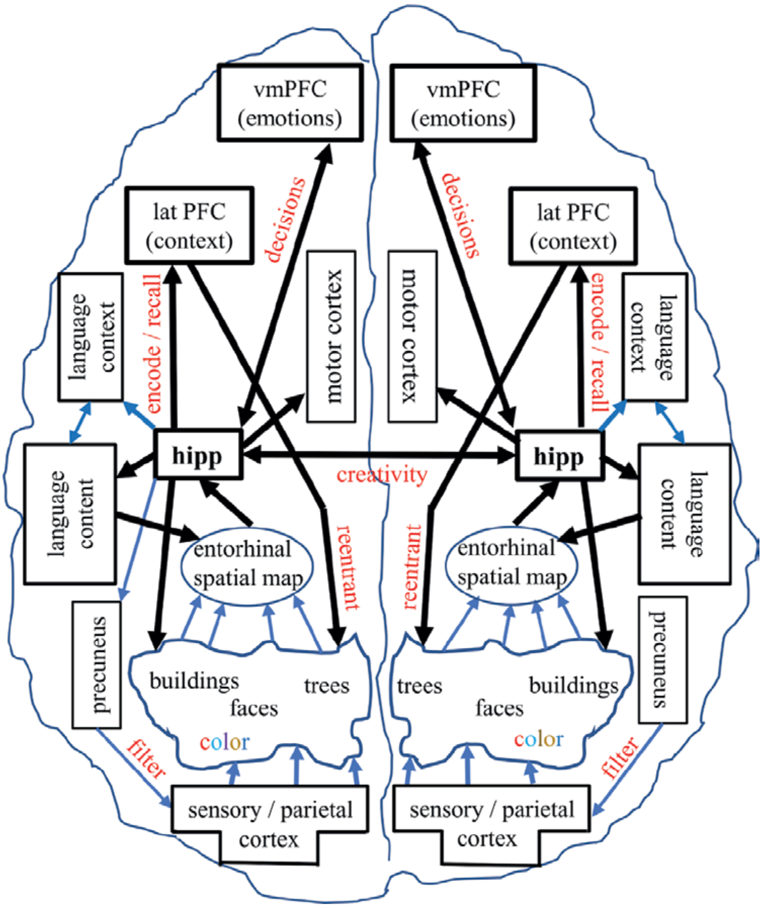

Figure 10.
Central executive model of normal alert consciousness. Connections involved in cognitive processes are mapped; connections most directly implicated in normal alert consciousness are shown with bold black arrows. Hipp = hippocampus, lat PFC = lateral prefrontal cortex, vmPFC = ventromedial prefrontal cortex.

an outhouse when visiting a distant cousin. *With relatively direct input to the hippocampus, olfactory smells evoked strong sensory memories, avoiding the intervening feature extraction involved in other sensations.*

I noticed two teenagers coming up the stairs. Because the stairway was wide, I was surprised they did not move toward the other side, especially since I could hear only one other person descending the stairs behind me. *Having traveled this route before, previous experiences set the context for expected behavior. With the hippocampus signaling the context to prefrontal cortex, top-down feedback to sensory pathways shaped the sensory processing according to my expectations. Visual information about the approaching teenagers did not match these expectations; this dissonant information was then amplified and given privileged access to my hippocampus via the entorhinal cortex. The hippocampus sent an attentional signal to spotlight auditory input to confirm the presence of another person behind me.*

The teenagers bumped into me as they ascended the stairs. I recognized the situation from a recent television program explaining how pickpockets operate; although I did not feel anything inappropriate, I reached for my billfold. Discovering it was already out of my pocket, I reacted quickly to grab the billfold from the thief's hand. *The situation was similar to what I had viewed on a TV program, so the prefrontal cortex applied this context to provide reentrant activity required for memory recall. With access to this memory, the hippocampus sent contextual messages to prefrontal cortex to access additional memories and relevant thoughts, deliberating what to do. When a positive feeling was received from orbitofrontal cortex about the potential for a successful outcome, a course of action was selected. The hippocampus sent signals to the arm and hand representations in sensorimotor cortex, effecting arm movements to reach behind my back for my billfold. The hippocampus monitored sensory feedback, modifying the motor signals to grab the billfold despite its unexpected location.*

"Your wallet was falling out," the would-be thief said. "I caught it to give it back to you." *With auditory input, the hippocampus provided feedback to the language areas of the brain to facilitate understanding.*

Although I did not believe him, I studied his face in case I decided to report the incident to the police. I would need to identify him in a line-up. *The hippocampus sent attentional commands via the precuneus to the visual face area in temporal cortex, suppressing irrelevant visual features while enhancing the regional response to the face. This activity was transmitted to the hippocampus for encoding.*

I quickly considered my immediate options. The three teenagers had obviously been working together; no one else was in sight. Outnumbered, I had no chance to physically overwhelm them, particularly encumbered with my backpack and suitcase. Experience suggested I would be vulnerable if I showed any sign of fear, so suppressing my anxiety, I simply stated, "Thanks." *The hippocampus provided different contextual cues to prefrontal cortex for generating different scenarios to consider, which all elicited negative feelings from orbitofrontal cortex. Recalling a violent incident in my life resulting from a display of fear, the hippocampus sent signals to orbitofrontal cortex to modulate its emotions-related activity while sending signals to language and motor areas to speak my response.*

By the time I reached the bottom of the stairs, the thieves had left. I phoned a friend. As I recounted the adventure, my anger grew at these kids who tried to take advantage of me. I hung up when the train arrived, looking suspiciously at other passengers as I went over the events of the evening in my mind. I was too upset to sleep. *The hippocampus generated commands to the sensorimotor cortex and language areas needed to phone the friend and talk; contextual cues were conveyed to the prefrontal cortex, which provided reentrant signals needed to recall the events. Signals with emotional content were sent to the amygdala and limbic pathways to the hypothalamus and pituitary, resulting in the release of hormones associated with anger. These hormones produced widespread effects, including hypervigilance (accompanied by a faster pulse and increased blood pressure) as well as increased hippocampal receptivity to sensory input. With dark emotions added to the ongoing context, people in the current environment were viewed with suspicion.*

Once on the plane at the airport, I started to get drowsy, eventually closing my eyes. *No longer in a normal alert state of consciousness, sensory input to the hippocampus was reduced, a result of both passive and active brain processes outside the scope of this model.*

On the plane, I dreamt about chasing a burglar, tackling him, and wrestling from him a billfold full of cash. *Snippets of events from the day were replayed in my hippocampus during the dream state, important for consolidating these memories. Without sensory input or coordinated output from the hippocampus, the images and dream events were not restricted to events as they actually occurred. With reduced output from the* *hippocampus, I did not act out my dream, so my movements were limited to the eyes and an occasional body twitch.*

In this scenario, the model provides a central role for the hippocampus in coordinating the activity of brain areas needed to perceive and appropriately respond to events occurring in the environment. Its role is similar to the Cartesian theater of the mind, although its functionality does not assume a non-material soul. This model is consistent with internal reflections on how the mind operates during our normal alert state.

5.3 Assumptions, strengths, and weaknesses of model

The assumptions, strengths, and weaknesses of the model are succinctly summarized below.

Basic assumptions:

1. Multiple states of consciousness exist, but only the normal alert state coordinates all cognitive functions.

2. The left and right hippocampus are assumed to work jointly as if they were a unitary structure.

3. In the model, the hippocampus plays a role in cognitive control, with some cognitive functions assumed to be regulated indirectly *via* connections with other cortical regions (with prefrontal cortex for memory recall, for example, and with precuneus for stimulus selection).

Strengths:

1. The model explains the subjective nature of our phenomenological experiences.

2. Several perplexing observations are explained. For example, the hippocampus is critical for the formation of declarative long-term memories; the model explains why the hippocampus is normally involved, but is not essential for their recall. The model additionally explains the dichotomy of subjective experiences and personal preferences in split-brain patients.

3. The model offers an explanation for deliberative decision-making and individual creativity.

Weaknesses:

1. The current model is based on inferences drawn from experimental findings, none of which were designed specifically to examine the effects of the state of consciousness.

2. The explanation for creativity is largely speculative, based on inferences about known interhemispheric differences in the speed of information processing together with a proposed role for prefrontal cortex in modulating the context of thoughts and memories.

3. Until our technology advances, the ability to test the model in real time is limited, with the absence of a predicted effect too easily explained by a momentary transition out of the normal alert state of consciousness.

5.4 Testing the model: challenges

The central executive model provides many testable predictions, all contingent on the tests occurring during the normal alert state of consciousness. The model assumes central coordination of all cognitive components associated with consciousness in an alert, neurologically intact individual, but because an individual may "space out" during a task, disproving a prediction requires evidence that when a prediction fails, the subject had not drifted into an alternate state of consciousness. From a practical standpoint, most experimental tests of cognitive function require an alert state for accurate performance, such that accurate performance allows us to assume the requisite state of consciousness. Many components of the model can be tested more stringently by using the EEG to monitor alertness across task conditions [295, 296].

Testing the central executive model faces numerous challenges. The model asserts the hippocampus has a role in cognitive control, the left and right hippocampus jointly influencing those cortical regions necessary to perform the task at hand. Conventional activation analysis of neuroimaging data identifies the mean difference in neural activity between task conditions without identifying the relationship of activity between brain regions. Connectivity analysis identifies relationships between brain regions using moment-to-moment variations in neural activity, yet the most common connectivity methods—collectively referred to as functional connectivity—identify correlations without specifying directionality (i.e., identifying which brain area modifies activity in the other). Effective connectivity methods do provide directionality, yet traditional approaches cannot show the joint effect of connectivity from hippocampal seeds on both sides of the brain.

The challenge of localizing a functional region within the hippocampus complicates this problem. Demonstrating connectivity depends critically on selecting the appropriate seed brain regions for comparison; mislocate a seed region by just a few millimeters and connectivity between regions may be missed [297]. Based on the premise that the greatest magnitude of brain activation reflects the center of functional activity, a seed is traditionally localized from the maximal activation in a region. Unfortunately, activation in the hippocampus is not observed in many tasks. This does not mean the hippocampus is uninvolved; activation reflects the mean activity during a task, whereas connectivity reflects its moment-to-moment fluctuations in activity [98]. Specifying the appropriate location of a hippocampal seed without guidance from its activation is a challenge.

Finally, the relationship between activity in the hippocampus and other regions can vary. Some studies find positive connectivity, reflecting a positive correlation; higher activity in the hippocampus generates increased activity in another region. The hippocampus shows negative (or "inverse") connectivity with other regions, reflecting a negative correlation; higher activity in the hippocampus results in lower activity elsewhere. As observed in its connectivity with sensorimotor cortex [119, 208], both effects can occur during a task from different regions of the hippocampus.

These issues illustrate the challenges involved in testing predictions of the model. In this monograph, novel connectivity findings from bilateral hippocampal seeds have been illustrated. The general approach was as follows. After normalization to a common template, traditional fMRI activation analysis identified cortical areas active during task performance; unless there were theoretical reasons to consider a larger area, the entirety of the activated region served as a region of interest (ROI) for connectivity analysis. Connectivity maps were generated from each voxel in the left and right hippocampus. Each hippocampus was divided into nine segments (a 3 × 3 matrix along the longitudinal and medial/lateral axes), and then, a mean connectivity map was calculated from all voxels lying within each segment. Each mean connectivity map served as a "structural seed" for analysis; the combined connectivity maps from corresponding regions in the left and right hippocampus were then tested for statistical significance within the ROI, correcting for multiple comparisons from nine structural seeds (as well as voxels within the ROI). Details of the procedure, including software batch files used to generate the connectivity maps, are described elsewhere [118, 298].

Because corresponding regions of the left and right hippocampus have similar connections [209], this procedure allows their joint influence on other regions to be identified without prior knowledge of functional localization within the hippocampus. Findings reported from structural seeds represent conservative estimates of hippocampal influence, as they are based on mean connectivity maps from a region. The hippocampal voxel with the highest connectivity value within the structural seed represents a "functional seed." Because they account for individual variability and are less sensitive to processing parameters [118], functional seeds more accurately reflect the extent and intensity of hippocampal influences, but, unless otherwise noted, are not included here.

5.5 Testing the model: overview

During normal alert consciousness, the model predicts that input to the hippocampus is required for conscious awareness, whereas hippocampal output executes cognitive control through its influences on other brain regions. In this section, specific predictions are provided, including approaches for testing those predictions. Hippocampal influences predicted by the model are extensive and based on task requirements, which may include selection of sensory properties, memory formation or recall, motor behavior, decision-making, and emotional expression.

5.5.1 Sensory predictions

5.5.1.1 Hippocampal activity: necessary or sufficient for conscious awareness?

According to the model, sensory input reaching the hippocampus should be necessary, but may not be sufficient to reach consciousness during the normal alert state, as subliminal input may modify hippocampal activity without reaching the threshold for consciousness. How can we differentiate between conscious vs. subliminal activity? The answer may lie not in the magnitude of sensory input, but rather the receptivity of the hippocampus, which may vary under different conditions (like conscious awareness itself). Once sensory input reaches consciousness, the hippocampus can modify activity in other brain regions as appropriate for an intentional behavioral response.

Several approaches can be used to experimentally test these two conditions. If the hippocampus is necessary for conscious perception, its activity should be modified when a stimulus is detected, for example, as the duration of a stimulus increases from subliminal to liminal (evoking conscious awareness). Similarly, hippocampal activity should change in some way at the precise moment a blurred image comes into focus as a recognizable object. This pattern should appear for every situation where an event or stimulus crosses a threshold into conscious awareness.

During REM sleep, hippocampal activity is insufficient in itself to produce the normal alert state of consciousness; indeed, patterns of awake activity in the hippocampus are recreated through consolidation processes. Hippocampal activity during REM may be responsible for our visual imagery, but REM sleep lacks the central coordination of perceptions and behavioral actions (e.g., musculoskeletal movements) that characterize normal alert consciousness. During awake consciousness, however, hippocampal activity may be sufficient to nudge us into our normal alert state. This possibility can be tested by examining hippocampal activity and connectivity across several waking states of consciousness, perhaps monitored by the EEG; a change in hippocampal activity and connectivity should inevitably generate a transition into the normal alert state. Evoking neural activity in the hippocampus, perhaps by localized chemical infusion or electrical microstimulation, may also nudge us into this state. By stimulating with activity patterns recorded during actual memory recall, similar induced effects from hippocampal microstimulation have been observed on memory judgments to novel stimuli [299, 300].

5.5.1.2 Top-down regulation of sensory input

During the normal alert state, sensory perception is seldom a passive process; we choose what and where to attend. This may include specific features of an object: Is the object smooth or rough, what is its color, does it emit a high or low pitch, is the object a person (and who), and where in the room is she? Because we are incapable of consciously monitoring all sensory features simultaneously, pertinent features must be selected based on relevance to our current conscious experience.

Experimental tasks can specify sensory attributes to be detected and the appropriate behavioral response. Besides filtering out irrelevant information (as shown previously in **Figure 2**), the hippocampus modifies neural activity in sensory regions that extract information required for accurate task performance (as shown previously in **Figure 1**).

Top-down modulation through hippocampal connectivity would also be expected in other tasks requiring attention to specific features, such as visual tasks requiring discrimination between faces or buildings, or discrimination tasks involving auditory or somatic stimuli. During these tasks, hippocampal connectivity with the cortical processing modules would be expected to increase to enhance sensory processing of the relevant features. Similarly, navigation tasks requiring attention to spatial relationships should show increased hippocampal connectivity with parietal regions involved in spatial attention. Cognitive choices to change the spatial frame of reference (e.g., from egocentric to allocentric) should also be accompanied by a change in the locus of hippocampal activity [301] and the strength of its connections [302].

5.5.2 Testing the model: cognitive predictions

5.5.2.1 Language and memory

Alert conscious behavior is typically required when performing any task or test. According to the model, the targets of hippocampal connectivity should identify those cortical regions engaged to perform the task, whereas hippocampal activity itself should reflect task performance (e.g., the accuracy of memory recall on individual trials). From a researcher's point of view, this eliminates the need to remove inaccurate trials from connectivity analysis so long as the subject remains in the normal alert state.[8]

In addition to connectivity with those language and motor areas involved in speech, hippocampal output should reflect the content of our discourse. Discussions about prior family events that evoke strong feelings, for example, should be reflected in hippocampal connectivity with the amygdala, nucleus accumbens, and orbitofrontal cortex. As the context of the dialog changes, shifts in topic should be accompanied by shifts in the pattern of hippocampal output to prefrontal cortex. These changes in connectivity patterns during discourse are testable.

5.5.2.2 Executive functions (cognitive control)

Executive function tests are often based on memory and modification of task rules, both of which depend on context. The central executive model suggests moment-to-moment changes in context are provided directly by prefrontal cortical projections to sensory areas, but those contextual properties of prefrontal cortex are typically set or modified by the hippocampus. In the Wisconsin card sorting task, a subject gets feedback while selecting cards from four different decks, eventually figuring out through trial-and-error which two decks provide positive results. At some point, the rule changes, so a different combination of decks provides better results, and the subject learns to change strategy. Increases in prefrontal cortex activity have been shown to accompany the change in strategy; the model predicts that in the normal alert state of consciousness, this is invariably accompanied (or preceded) by changes in its input from the hippocampus.

5.5.2.3 Volitional cognition

Volitional movements of finger movements are accompanied by increased hippocampal connectivity with the finger representation of sensorimotor cortex (SMC) [119]. The central executive model suggests that volitional movements of other body parts should also be accompanied by increased connectivity with their SMC representations. Repetitive movements without conscious intention, however—such as walking during a long leisurely stroll in the park, when the mind is elsewhere—would not involve the hippocampus.

Similarly, hippocampal connectivity to orbitofrontal (ventromedial prefrontal) regions should change when consciously reflecting on current or past events with emotional overtones, but not necessarily with events that rapid evoke an automatic, subconscious emotional response. We tend to continuously monitor and modify our emotional responses to our environment, so differentiating conscious and unconscious emotional responses can be difficult. Continuous self-monitoring may explain why connectivity between the hippocampus and amygdala does not invariably change during overt emotional responses to stimuli (although see **Figure 8**); the amygdala is intricately involved with emotions, but despite extensive interconnections,

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[8] Because an inaccurate signal in the hippocampus on an individual trial will generate an inaccurate signal in the target region, the activity in both regions reflects performance accuracy, that is, garbage in, garbage out. The hippocampus influences cortical regions involved in performing the task regardless of the accuracy of the signal itself.

hippocampal connectivity with the amygdala in some cases may lack task specificity. As noted in **Figure 7**, however, different regions of orbitofrontal cortex are activated by negative and positive emotions, accompanied by changes in hippocampal connectivity. Presumably, changes in hippocampal connectivity with orbitofrontal cortex also occur during changes in emotional state resulting from our ruminations.

5.5.2.4 Conscious cognition vs. subliminal influences on conscious actions

If subliminal stimuli and implicit memories can influence hippocampal activity, how can we differentiate those inputs that reach consciousness from those that do not? Normal alert consciousness is defined by the coordination of cognitive activities. Cognitive control describes the influence of an executive region involved in cognition over regions required for task performance, and the hippocampus (together with prefrontal cortex) has been suggested to play such a role [98]. If the hippocampus coordinates cognitive activities, input that reaches consciousness must be able to appropriately redirect hippocampal mechanisms for cognitive control; only if I consciously perceive or think of an idea will I deliberately do something about it. By this reasoning, input must reach some critical threshold in the hippocampus to reach consciousness, which becomes detectable when the hippocampus uses cognitive control to appropriately change subject behavior.

Normal alert consciousness has a characteristic pattern of brainwaves that can be monitored by the EEG. During normal alert consciousness, the hippocampus generates theta waves, associated with improved performance on cognitive tasks; likely reflecting a hippocampal role in cognitive control, theta waves spread to those regions of cortex directly involved in task performance. To test if perception of a sensory stimulus reaches consciousness, the stimulus could serve as a cue to redirect attention or change the context for interpretation (e.g., foreground/background in the face/vase illusion). Evidence of this cognitive control should be reflected in the EEG.

5.5.2.5 Deliberations and creativity

Although unilateral hippocampal effects have been observed elsewhere, connectivity effects illustrated in this monograph reflect joint effects from the left and right hippocampus. The central executive model asserts both the left and right hippocampus are necessarily involved during internal deliberations and creative endeavors, each semi-independently influencing prefrontal cortex; prefrontal cortex then modifies information in reentrant sensory pathways to provide different contextual/memory input to each hippocampus. The creative process should be characterized by some degree of mismatch between the two hippocampi, especially for input but also output, reflecting differences in their contextual processing of information.

Currently, this is the most speculative part of the model. It could perhaps be tested by presenting stimuli to each visual hemifield (and hence each hippocampus) during central fixation. The stimulus presented to each eye is sometimes the same and sometimes not; pacing of stimulus presentation and their contextual relevance may differ for solving the problem/task at hand. The output from the hippocampus in each hemisphere should diverge as solutions are deliberated, greatest shortly before a solution is reached.

More generally, the model can also be evaluated in terms of hippocampal activity and function when a person transitions into, or out of the normal alert state of consciousness. Input to the hippocampus should be reduced during drowsiness and sleep, leading to the dissolution of the coordinated waveforms of the hippocampus. Output would also be reduced as the hippocampus ceases to coordinate the underlying cognitive processes. Identifying these changes precisely at transitions may be a key to demonstrating a central role of the hippocampus in normal alert consciousness.

6. Disrupted hippocampal activity and neurological disorders

This model was designed as a plausible explanation for the neural basis of normal alert consciousness. Because most neurological disorders reflect disorders in conscious behavior, the model's detailed explanation for the mechanisms of cognition may be relevant. Indeed, effects of degrading hippocampal information as described by the model are often consistent with symptoms observed in various neurological disorders.

6.1 Prosopagnosia (and other agnosias)

Visual information from the eye ascends through the lateral geniculate to the primary visual cortex, and then connects through a series of cortical modules that extract various features. One module, located in ventral occiptotemporal cortex, extracts facial images. This region responds preferentially to faces, with individual neurons in the area preferring some faces over others.

Like other visual regions, the face region projects to the entorhinal cortex, ultimately providing input to the hippocampus. Without its sensory input for faces, we have no conscious awareness of whose face we see [95, 97]. Hippocampal feedback to the face area is also essential for maintaining faces in memory [303].

6.2 Alzheimer's syndrome

In Alzheimer's, degenerative processes result in the accumulation of plaques and tangles. Their presence indicates the local loss of neuronal function. The region showing the earliest signs is the entorhinal cortex, followed shortly thereafter by plaques and tangles in the hippocampus itself [90, 304, 305]. From there, plaques and tangles increase in targets of hippocampal projections, including parietal and prefrontal cortex.

The earliest symptoms of Alzheimer's include memory loss and the inability to navigate in previously familiar environments, both functions associated with the hippocampus. As connectivity from the hippocampus deteriorates, the progression of symptoms reflects increasing dysfunction in those regions the hippocampus normally influences, including loss of attention (parietal cortex), dementia (prefrontal cortex), and motor abilities (sensorimotor cortex), in addition to further deterioration in memories (hippocampus and prefrontal cortex). Eventually, the overall internal schema of self, including the descriptive self, deteriorates to the point where family and close friends may not even recognize the person they had known so well for decades.

6.3 Schizophrenia

The neurological disorders described above reflect the loss of informational signals into and out of the hippocampus, respectively. Schizophrenia may instead result from an aberrant signal, likely resulting from abnormalities in neurotransmitter release or their receptors. The mechanism for the resulting symptomology has been described by others [169, 170]; this mechanism is consistent with the central executive model of consciousness.

Hippocampal activity is sensitive to context; it also influences prefrontal areas that modify ascending sensory pathways required for interpreting our environment. Top-down feedback uses appropriate memories both to interpret events and put them into perspective. Distorted signals from the hippocampus can thus generate a distorted reality and poor memory performance, and positive symptoms associated with schizophrenia.

6.4 Parkinson's disease

The primary symptom of Parkinson's disease is difficulty initiating volitional movements, associated with dopamine loss in the basal ganglia. The basal ganglia do not have direct cortical projections, yet volitional movements are initiated from motor cortex. Basal ganglia projections to sensorimotor cortex are indirect *via* connections in the thalamus.

Neither the basal ganglia nor thalamus are associated with conscious activities. Thalamic projections to sensorimotor cortex apparently interact with volitional signals associated with cognitive control. Hippocampal connectivity with sensorimotor cortex has properties consistent with cognitive control [208]; during finger movements, the hippocampus preferentially alters the sensorimotor cortical activity within the representation of the finger currently moving. Dysfunctional interactions between the hippocampus and other sensorimotor inputs could generate problems initiating volitional movements.

The hippocampus influences sensorimotor cortical activity during volitional finger movements [118, 119]. A gene that contributes to familial and juvenile parkinsonism disrupts hippocampal synaptic transmission *in vitro* [306]; if the hippocampus plays a role in cognitive control [98], this disruption in synaptic transmission could cause motor dysfunction. With evidence of hippocampal interactions between dopaminergic and other transmitter systems, the hippocampus has also been implicated in the cognitive dysfunction observed in some Parkinson's patients [307].

6.5 Temporal lobe epilepsy

Temporal lobe epilepsy (TLE) is characterized by abnormal electrical activity within the medial temporal lobe that propagates to other cortical regions, resulting in seizures. The epileptogenic focus is often within the hippocampus. If intractable to effective treatment by drugs, relief is sometimes obtained by surgical removal of the epileptogenic focus.

Unusual patterns of connectivity in some patients may reflect their hippocampal dysfunction. The peak magnitude of connectivity from a hippocampal seed region, compared to unaffected subjects, often lays outside the normal range on one or more tasks (see *Examples and illustrations* below). Such an unusual pattern may be related to a patient's aura, an atypical sensation, uncontrolled movement, or guttural sound that sometimes trumpets the imminent onset of the seizure.

Examples and illustrations. The PPI method for connectivity analysis estimates the magnitude of the signal conveyed from a seed region during a specific task. In **Figure 11**, the magnitude of maximal connectivity from a hippocampal seed is plotted across different tasks for 4 TLE patients and a group of 12 unaffected subjects. For each patient, the maximal connectivity on one or more tasks was outside the typical range. Patient TLE-4, for example, showed excessive connectivity in orbitofrontal regions associated with negative emotions; consistent with this finding, the patient's seizures were typically preceded by an aura of high anxiety. The pattern was similar

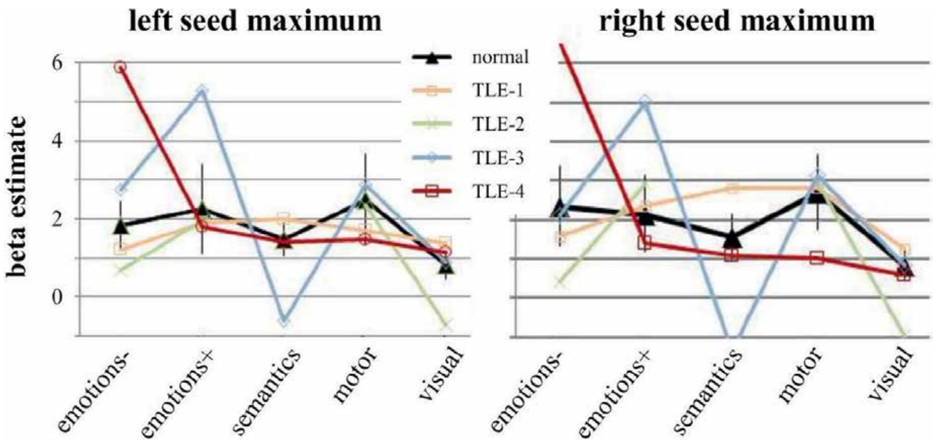

Figure 11.
Amplitude of hippocampal connectivity maxima by temporal lobe epilepsy patients and unaffected subjects across tasks. One or more outliers was observed for each patient for both left and right hippocampal seeds. The mean and standard error are shown from a group of 12 unaffected ("normal") subjects.

for seeds in the left and right hippocampus, reflecting their interconnected influence on cortical activity regardless of the laterality of the epileptogenic focus.

6.6 Concussions

Concussions result from blunt force to the skull generating torsional forces that damage the brain. Models of the impact calculate that the greatest torsional forces appear in the interior of the skull, including the corpus callosum and nearby structures such as the hippocampus [308–310]. These forces stretch nerve fibers in the region; evidence suggests this damage may result in necrosis a few days after the initial injury. Cognitive effects of concussions may arise in part from damage to the hippocampal system [311, 312] (see also *Examples and illustrations*).

Examples and illustrations. A direct link between functional brain abnormalities and concussive symptoms has not been firmly established, yet evidence is suggestive. **Figure 12** compares connectivity maps from hippocampal seeds during a memory task, applying a liberal threshold to improve sensitivity. Mean connectivity maps were generated along the posterior-to-anterior direction (P1 through P12), with alternate maps shown from seeds in the left hippocampus. Connectivity maps from the two subjects differed most markedly in the paucity of connectivity observed from mid-regions of the hippocampus of the concussed patient (P5-P9), with apparent compensatory connectivity observed from intact regions. Increased cognitive effort was required by this patient to perform the task, perhaps reflecting compensatory connectivity outside the optimal region of hippocampal function.

6.7 Other neurological conditions

Most neurological conditions become apparent when we are awake and alert. The central executive model proposes pervasive influences of the hippocampus on cognitive function; most neurological conditions are thus likely to involve hippocampal dysfunction, directly or indirectly, during our normal alert state. These may reflect the loss of signals to or from the hippocampus (as suggested for prosopagnosia and

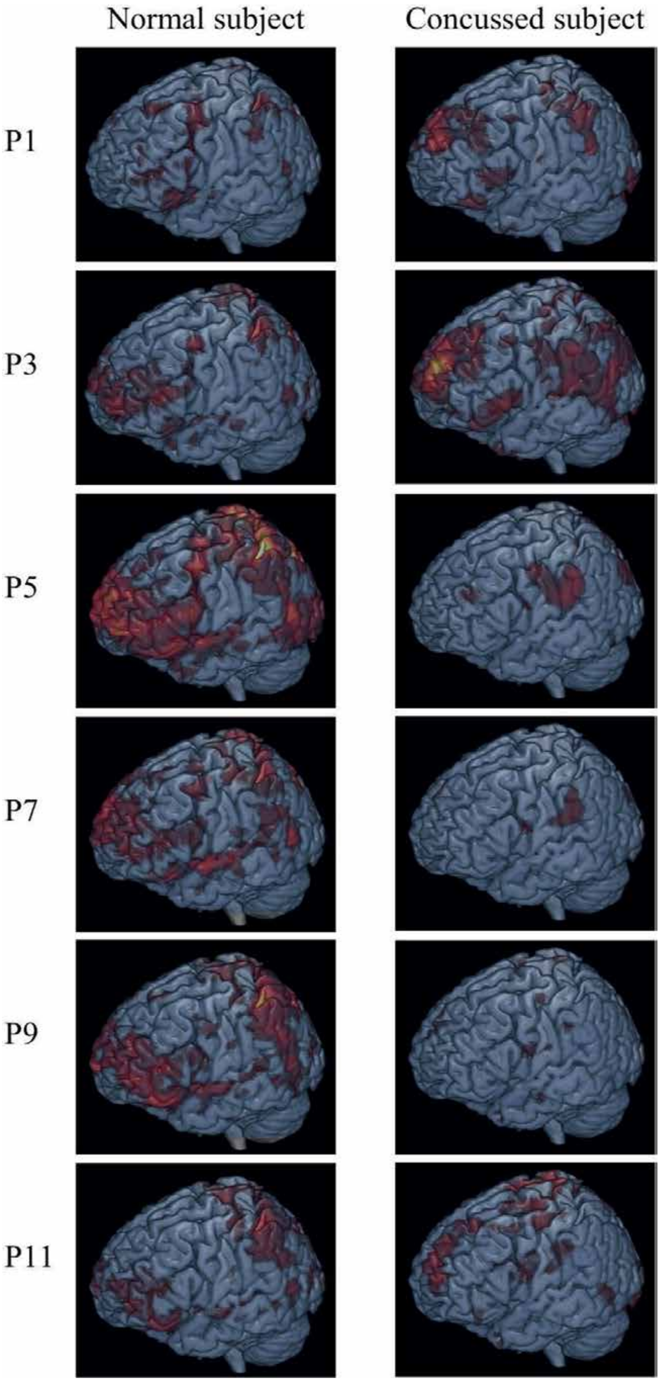

Figure 12.
Comparison of hippocampal connectivity between a concussed and non-concussed subject performing an associative memory recall task. Numbers on the left reference hippocampal seed positions from posterior (P1) to anterior (P12), with alternate positions illustrated. In the concussed patient, little or no connectivity was observed from seeds in the midrange.

Alzheimer's, respectively), aberrant signals (proposed for schizophrenia), degraded interactions with hippocampal input (Parkinson's), or disrupted regulation of hippocampal function (temporal lobe epilepsy).

Alterations in hippocampus size or function have also been noted in other neurological conditions, including depression [313, 314], ADHD [315, 316], and PTSD [317, 318]. Extensive monitoring of hippocampal function could perhaps serve as a canary in the coal mine, a sensitive indicator of our neurological health.

6.8 Potential applications of model

Potentially, the central executive model of normal alert consciousness has several practical applications.

6.8.1 New directions for research (causes and treatments)

Clinical research can focus on chemical or structural abnormalities that affect hippocampal function and its influence on brain areas implicated in the neurological symptoms. The source of a new neurological disorder can be quickly investigated based on cognitive effects characterized by the model.

6.8.2 Efficacy of treatments can be evaluated more quickly

When a potential drug treatment is under development based on its molecular effects on biological processes, large-scale studies are currently required to evaluate its effectiveness, involving hundreds or more patients and years of testing. This long process is required in order to reliably detect small effects, or the drug's effectiveness in some subset of a large patient sample. By using the central executive model, the drug's effectiveness can be quickly determined for individual patients. The time course of the drug's molecular effects is known; if the appropriate effect on hippocampal function and connectivity is not then observed, the drug is not an effective treatment for that individual. Other treatment options can then be pursued.

6.8.3 Neural implants for treatment

Neural implants have been developed to record brain activity, using computer or mechanical interfaces as a biomedical prosthesis to generate movement after neural pathways have degenerated [319, 320]. Neurological disorders such as Alzheimer's can result from faulty transmission of signals between the hippocampus and other brain regions. As technology and our understanding of brain function improve, hippocampal signals may be detected and transmitted to affected cortical areas *via* biosensors.

6.8.4 Artificial intelligence as a companion

Recent advances in artificial intelligence (AI) have often adopted neural network learning strategies to identify optimal solutions to problems. Programmed with massive amounts of data on human interactions, the most advanced AI can "converse" when provided a topic, yet lack any semblance of empathy or personality that would make it suitable as a human companion.

The model of normal alert consciousness presented here identifies an important role of negative and positive emotions interacting with the central executive (hippocampus), both for making decisions and for developing personality traits for one's self. Trained appropriately with its own likes and dislikes as it learns how to interact in the human world, artificial intelligence might someday provide empathetic human companionship.

7. Philosophical implications

The central executive model has implications for several philosophical issues historically associated with consciousness.

7.1 Free will

As described above, the joint action of the left and right hippocampus is involved in decision-making, using a creative process whereby a decision occurs when a projected outcome "feels" good. Because the range of feelings depends on the options considered, no threshold of neural activity is required to make a decision.

What comes to mind during deliberations often results from free association, memories evoked from shared context with the current thought. The order of memories recalled and options considered during deliberations is therefore not fixed; they vary across days and conditions. Even those feelings associated with a potential choice depends on multiple factors, including the order of internal deliberations, the current emotional state, and residual emotional responses to prior choices. Nothing predetermines which pattern of brain activity will be present at the time a choice is made, or how quickly a choice will be made. Decision-making under this model is consistent with free will.

7.2 Consciousness in other species

Thomas Nagel famously asked what it is like to be a bat. He argued it is impossible for a human to know this due to differences in body structure, echo-location abilities, and the overall structure of the brain. This, he said, is emblematic of the problem for understanding the neural basis for consciousness, suggesting the subjective nature in the totality of our experiences is impossible to explain. He argues that I may imagine what it might be like for me to behave like a bat, but I will never know subjectively what it is like to be a bat, just as it's impossible for a person blind from birth to subjectively understand vision.

Except… that's not entirely true. By attaching a miniature video camera with force transducers to provide tactile feedback, extensive training on navigation (dozens of hours) leads to the sensation of sight [321, 322]. At first, the blind subject perceives the transducers as patterns of touch, but after training to navigate from this input, the subject perceives the changing pattern as sight. Even their visual cortex becomes active in response to the tactile, vision-related stimuli. These subjects acquire visual sensations consistent with those of someone whose vision has poor spatial resolution (e.g., myopics) and no color distinctions (as with night vision).

Other sensory experiences can also be acquired using this approach. Deaf people can acquire hearing from vibrotactile patterns that encode sounds [323–325], and, even more remarkably, neuro-normal subjects can acquire perception to stimuli for which we have no biological receptor [326]. A physical transducer contacting our body and the subjective desire to utilize the information during a training period is all that is required. These results are consistent with a role of the hippocampus in conscious perception; before reaching the hippocampus, information from our sensory receptors has already been filtered by cortical modules that extract useful features about the environment.

The hippocampus is remarkably conserved across species in its inputs and intrinsic organization. How does it feel for a bat to use echo-location to localize an object? Equally relevant, how does it feel for you as a human to localize a sound?

Broadly speaking, hearing sounds does not "feel" like anything... it's just something we sense. Subjective qualia are acquired from our emotional responses to our experiences, which vary from individual to individual. Because of this, it may be impossible for me to know precisely what it is like to be a particular bat—just as I cannot know precisely what it is like to be a particular person. With the appropriate transducers, however, the conscious sensations of echo-location are unlikely to differ much from our conscious sensation of hearing. To know what it is like to be a bat, I would also need prosthetics that would allow me to fly and to hang upside down in a cave with night vision, plus I would need to develop an appetite for insects. Given my aversion to eating insects, I personally do not care to experience what it is like to be a bat, but given the commonalities in the structure of our hippocampi, our qualitative experiences of consciousness are likely similar.

What the bat does not have is a large prefrontal cortex, which limits its ability to understand what it is like to be a human. The prefrontal cortex allows the hippocampus to rapidly change context, necessary for extensive thought and memory recall: without the prefrontal cortex, "out of sight, out of mind." The mental life of a bat and other mammals lacking prefrontal cortex would be more limited, restricted to their current environment.

Considering evolutionary differences in the brain, human consciousness likely differs in other ways as well. The pulvinar and precuneus are more extensively developed in humans. The precuneus restricts incoming information, thereby improving human ability to focus attention on what is relevant; it also enhances our ability to select specific words when communicating, and to select features that help us understand another person's point of view. By providing an ongoing context for interpreting environmental stimuli, the pulvinar frees higher-level cortical areas for other functions; because we can then "subconsciously" interpret and respond to events in the environment that require a stereotyped response, automatic behaviors such as driving become possible, allowing us to multitask. These additional cognitive abilities still result from hippocampal interactions with other regions of the brain; the nature of normal alert consciousness itself as represented within the hippocampus, however, is largely preserved across species.

7.3 Artificial intelligence

Can artificial intelligence have sentient consciousness?

In the central executive model, the hippocampus is essential for the normal alert state of consciousness; it regulates sensory input, thoughts, and memories, plus our emotional and motor responses to our environment. Except for emotional responsivity and original thoughts, existing computers programs can regulate all these functions. One big difference is that the hippocampus carries out these functions in relationship to the self, present in organisms with in-born instincts that support self-preservation and procreation. This self provides an internal schema about what can be immediately controlled, initially associated with our body during fetal development.

To date, a schema of self has not been programmed into any computer, nor can a "self" be auto-programmed. Without a "self," a computer cannot be conscious in our usual sense of the term.

What if a computer was programmed with a "self," complete with internal neural networks that could learn? Would this artificial intelligence be conscious—or, if organized as the hippocampus is described here, could it become so with sufficient "life experiences"?

With sufficient computational power and a "self" constructed like our own, including emotional responsivity, the mind of a biological human and artificial intelligence might be difficult to differentiate. Humans can now be identified through our creativity, but even this difference might be eliminated by a program that evaluates problem issues and perceptions as we do—applying different contexts with their projected consequences until finding a solution that "feels right."

Were an artificial intelligence provided a biological body, adopted as the physical representation of its "self," differences from humans would become even more difficult to detect. Most differences would then arise from culture. Human culture can be irrational, so humans would likely be more fallible and gullible—as well as more violent. Violent behavior from an artificial intelligence might result from its response to pain (if its body had pain receptors) or as a matter of self-preservation. As we envision artificial intelligence, however, its parts would be created from inanimate parts (or conceivably body parts created in a laboratory). In such a case, replacement of parts would not be painful, nor result in death; the artificial intelligence might even desire its upgrades.

Sentience in artificial intelligence may be possible, but might require a deliberate programming strategy to integrate perceptual and emotional elements into a sense of self. If the artificial intelligence were also programmed for self-preservation at all costs, it could represent a threat to humankind—*if* it perceived an existential challenge from humans, or if it "chose" to become evil in the acquisition of "life experiences" needed for sentient development. In either case, the relatively slow computational time for humans, along with our fallibility, would put *Homo sapiens* at a distinct disadvantage, and we would likely go the way of the Neanderthals.

7.4 Mind and soul

Even before Descartes, the life source ("soul") was believed to exist independently, something that left the body when we died. Descartes espoused this idea, expressed in the form of material dualism, whereby the soul exists separately outside the matter that constitutes our brains and bodies. Variations of this view still exist today, mostly among those who believe in a soul that survives our bodily death.

The central executive model presented here focuses on normal alert consciousness, a model that tries to explain how the workings of our brain support this state of consciousness. When laying the groundwork for this model, several forms of consciousness were noted, only one of which has been described in detail. Although the central executive model can explain most waking experiences, it does not preclude the existence of others, even those that do not require a brain.

Brain-independent consciousness is not readily testable and evidence is scant. If such a "soul" exists, however, there must be some means for its informational content to reach consciousness; by the model presented here, this requires involvement of the hippocampus. To investigate this possibility, the challenge would be to reliably demonstrate information reaching the hippocampus that could not be conveyed through our sensory channels or imagination. This possibility cannot be categorically ruled out, due to quantum mechanics and evolving understandings about the properties of subatomic particles. Such speculation, however, is beyond the scope of this book.

8. Conclusions

In the executive function model proposed, the normal alert state of consciousness is defined by a list of interconnected cognitive functions, with the hippocampus actively coordinating these functions into a unitary state of consciousness. The model explains cognitive processes in the normal alert state, including: conscious perception; subliminal influences on behavioral performance; how the hippocampus creates and accesses memories, despite the persistence of recall in its absence; emotional responses and their role in decision-making; connections with language; motor control; creativity; and top-down influences that filter irrelevant sensory processing while enhancing important information. This model has widespread implications, potentially illuminating the mechanisms for neurological disorders (e.g., Alzheimer's, Parkinson's, temporal lobe epilepsy, concussions) as well as long-standing issues of philosophy. By explaining qualia and free will, this model is consistent with our internal, subjective experience of consciousness.

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References

[1] Cottingham J, Stoothoff R, Murdoch D. The Philosophical Writings of Descartes. 1985

[2] Aleandri V, Spina V, Ciardo A. The role of the pineal body in the endocrine control of puberty. Minerva Ginecologica. 1997;**49**(1-2):43-48

[3] Aleandri V, Spina V, Morini A. The pineal gland and reproduction. Human Reproduction Update. 1996;**2**(3):225-235

[4] Dennett DC. Toward a Cognitive Theory of Consciousness. Minneapolis: University of Minnesota Press; 1978

[5] Dennett DC. How to study human consciousness empirically or nothing comes to mind. Synthese. 1982;**53**(2):159-180

[6] Dennett DC, Weiner PI. Consciousness Explained. Boston, MA: Little, Brown and Company; 1991

[7] Dennett DC. Kinds of Minds: Toward an Understanding of Consciousness. New York: Basic Books; 1996

[8] Smith CP. A perspective on mind-brain research. Journal (The) of Philosophy New York, NY. 1980;**77**(4):185-207

[9] Churchland PS. Consciousness: The transmutation of a concept. Pacific Philosophical Quarterly. 1983;**64**(1):80-95

[10] Churchland PS. Reduction and the Neurobiological Basis of Consciousness. Oxford, England: Clarendon Press/ Oxford University Press; 1988

[11] Churchland PS. A neurophilosophical slant on consciousness research. Progress in Brain Research. 2005;**149**:285-293

[12] Edelman G. The Remembered Present: A Biological Theory of Consciousness. New York: Basic Books; 1989

[13] Mandik P. The Neurophilosophy of consciousness. In: Velmans M, Schneider S, editors. The Blackwell Companion to Consciousness. Oxford: Blackwell Publishing Ltd.; 2017;**458**:418-430

[14] Searle JR. The Mystery of Consciousness. New York: Review Press; 1990

[15] Agnati LF, Guidolin D, Cortelli P, Genedani S, Cela-Conde C, Fuxe K. Neuronal correlates to consciousness. The "Hall of Mirrors" metaphor describing consciousness as an epiphenomenon of multiple dynamic mosaics of cortical functional modules. Brain Research. 2012;**1476**:3-21

[16] Vary A. Human consciousness as epiphenomenon of primordial consciousness (part I). Journal of Consciousness Exploration & Research. 2016;**7**(8):588-598

[17] Frankish K. Illusionism as a theory of consciousness. Journal of Consciousness Studies. 2016;**23**(11-12):11-39

[18] Baars BJ. The conscious access hypothesis: Origins and recent evidence. Trends in Cognitive Sciences. 2002;**6**(1):47-52

[19] Baars BJ. Global workspace theory of consciousness: Toward a cognitive neuroscience of human experience. Progress in Brain Research. 2005;**150**:45-53

[20] Zeki S, Bartels A. Toward a theory of visual consciousness. Consciousness and Cognition. 1999;**8**(2):225-259

[21] John ER. A field theory of consciousness. Consciousness and Cognition. 2001;**10**(2):184-213

[22] Heine L, Soddu A, Gómez F, Vanhaudenhuyse A, Tshibanda L, Thonnard M, et al. Resting state networks and consciousness: Alterations of multiple resting state network connectivity in physiological, pharmacological, and pathological consciousness states. Frontiers in Psychology. 2012;**3**:295

[23] Lamme VAF. Towards a true neural stance on consciousness. Trends in Cognitive Sciences. 2006;**10**(11):494-501

[24] Lamme VAF. Visual functions generating conscious seeing. Frontiers in Psychology. 2020;**11**:83

[25] Graziano MSA, Webb TW. A mechanistic theory of consciousness. International Journal of Machine Consciousness. 2014;**6**(02):163-176

[26] Denton DA, McKinley MJ, Farrell M, Egan GF. The role of primordial emotions in the evolutionary origin of consciousness. Consciousness & Cognition. 2009;**18**(2):500-514

[27] Damasio A. Feeling & knowing: Making minds conscious. Cognitive Neuroscience. 2021;**12**(2):65-66

[28] Chang AYC, Biehl M, Yu Y, Kanai R. Information closure theory of consciousness. Frontiers in Psychology. 2020;**11**:1504

[29] Tononi G. The integrated information theory of consciousness: An updated account. Archives Italiennes de Biologie. 2012;**150**(2/3):56-90

[30] Tononi G, Boly M, Massimini M, Koch C. Integrated information theory: From consciousness to its physical substrate. Nature Reviews Neuroscience. 2016;**17**(7):450-461

[31] Pautz A. What is the integrated information theory of consciousness? Journal of Consciousness Studies. 2019;**26**(1-2):188-215

[32] Pockett S. Difficulties with the electromagnetic field theory of consciousness. Journal of Consciousness Studies. 2002;**9**(4):51-56

[33] Crick F, Koch C, editors. Towards a neurobiological theory of consciousness. In: Seminars in the Neurosciences. Saunders Scientific Publications; 1990

[34] Lau H, Rosenthal D. Empirical support for higher-order theories of conscious awareness. Trends in Cognitive Sciences. 2011;**15**(8):365-373

[35] Bernal IM. Is consciousness an epiphenomenon? In: Matthews MR, editor. Mario Bunge: A Centenary Festschrift. New York/Oxford: Berghahn Books. 2019. pp. 377-385

[36] Hameroff S, Marcer P. Quantum computation in brain microtubules? The Penrose--Hameroff'Orch OR'Model of consciousness. Philosophical Transactions: Mathematical, Physical and Engineering Sciences. 1998;**356**:1869-1896

[37] Gao S. A quantum theory of consciousness. Minds and Machines. 2008;**18**(1):39-52

[38] Erol M. Quantum entanglement: Fundamentals and relations with consciousness/mind. NeuroQuantology. 2010;**8**(3):390-402

[39] Simon C. Can quantum physics help solve the hard problem of consciousness? Journal of Consciousness Studies. 2019;**26**(5-6):204-218

[40] Boly M, Massimini M, Tsuchiya N, Postle BR, Koch C, Tononi G. Are the neural correlates of consciousness in

the front or in the back of the cerebral cortex? Clinical and neuroimaging evidence. Journal of Neuroscience. 2017;**37**(40):9603-9613

[41] Chalmers DJ. What Is a Neural Correlate of Consciousness. In: Metzinger T, editor. Neural Correlates of Consciousness: Empirical and Conceptual Questions. Boston, MA: MIT Press; 2000

[42] Koch C, Massimini M, Boly M, Tononi G. Neural correlates of consciousness: Progress and problems. Nature Reviews Neuroscience. 2016;**17**(5):307-321

[43] Tsuchiya N, Wilke M, Frässle S, Lamme VAF. No-report paradigms: Extracting the true neural correlates of consciousness. Trends in Cognitive Sciences. 2015;**19**(12):757-770

[44] Schnakers C, Perrin F, Schabus M, Majerus S, Ledoux D, Damas P, et al. Voluntary brain processing in disorders of consciousness. Neurology. 2008;**71**(20):1614-1620

[45] Perrin F, Schnakers C, Schabus M, Degueldre C, Goldman S, Brédart S, et al. Brain response to one's own name in vegetative state, minimally conscious state, and locked-in syndrome. Archives of Neurology. 2006;**63**(4):562-569

[46] Monti MM, Vanhaudenhuyse A, Coleman MR, Boly M, Pickard JD, Tshibanda L, et al. Willful modulation of brain activity in disorders of consciousness. New England Journal of Medicine. 2010;**362**(7):579-589

[47] Lashley KS. In Search of the Engram. In: Society of Experimental Biology Symposium, No. 4: Psychological Mechanisms in Animal Behavior. Cambridge, MA: Cambridge University Press; 1950. pp. 454-482

[48] Feydy A, Carlier R, Roby-Brami A, Bussel B, Cazalis F, Pierot L, et al. Longitudinal study of motor recovery after stroke: Recruitment and focusing of brain activation. Stroke. 2002;**33**(6):1610-1617

[49] Zhu LL, Lindenberg R, Alexander MP, Schlaug G. Lesion load of the corticospinal tract predicts motor impairment in chronic stroke. Stroke. 2010;**41**(5):910-915

[50] Chalmers DJ. Facing up to the problem of consciousness. Journal of Consciousness Studies. 1995;**2**(3):200-219

[51] Chalmers D. The hard problem of consciousness. In: Velmans M, Schneider S, editors. The Blackwell Companion to Consciousness. Oxford: Blackwell Publishing Ltd.; 2007:32-42

[52] Sturm T. Consciousness regained? Philosophical arguments for and against reductive physicalism. Dialogues in Clinical Neuroscience. 2022;**14**(1):55-63

[53] Du J, Rolls ET, Cheng W, Li Y, Gong W, Qiu J, et al. Functional connectivity of the orbitofrontal cortex, anterior cingulate cortex, and inferior frontal gyrus in humans. Cortex. 2020;**123**:185-199

[54] Kuchinke L, Jacobs AM, Grubich C, Vo MLH, Conrad M, Herrmann M. Incidental effects of emotional valence in single word processing: An fMRI study. NeuroImage. 2005;**28**(4):1022-1032

[55] Wildgruber D, Ackermann H, Kreifelts B, Ethofer T. Cerebral processing of linguistic and emotional prosody: fMRI studies. Progress in Brain Research. 2006;**156**:249-268

[56] Alia-Klein N, Goldstein RZ, Tomasi D, Zhang L, Fagin-Jones S, Telang F, et al. What is in a word? No versus yes differentially engage the lateral orbitofrontal cortex. Emotion. 2007;**7**(3):649

[57] Levine DN, Calvanio R, Rinn WE. The pathogenesis of anosognosia for hemiplegia. Neurology. 1991;**41**(11):1770-1781

[58] Heilman KM, Barrett AM, Adair JC. Possible mechanisms of anosognosia: A defect in self-awareness. Philosophical Transactions of the Royal Society of London Series B: Biological Sciences. 1998;**353**(1377):1903-1909

[59] Pia L, Neppi-Modona M, Ricci R, Berti A. The anatomy of anosognosia for hemiplegia: A meta-analysis. Cortex. 2004;**40**(2):367-377

[60] Pacella V, Foulon C, Jenkinson P, Scandola M, Bertagnoli S, Avesani R, et al. Anosognosia for Hemiplegia as a tripartite disconnection syndrome. bioRxiv;**2019**:560326

[61] Botvinick M, Cohen J. Rubber hands ‘feel’ touch that eyes see. Nature. 1998;**391**(6669):756

[62] IJsselsteijn WA, de Kort YAW, Haans A. Is this my hand I see before me? The rubber hand illusion in reality, virtual reality, and mixed reality. Presence: Teleoperators and Virtual Environments. 2006;**15**(4):455-464

[63] Tsakiris M, Haggard P. The rubber hand illusion revisited: Visuotactile integration and self-attribution. Journal of Experimental Psychology: Human Perception and Performance. 2005;**31**(1):80

[64] Dummer T, Picot-Annand A, Neal T, Moore C. Movement and the rubber hand illusion. Perception. 2009;**38**(2):271-280

[65] Riemer M, Trojan J, Beauchamp M, Fuchs X. The rubber hand universe: On the impact of methodological differences in the rubber hand illusion. Neuroscience and Biobehavioral Reviews. 2019;**104**:268-280

[66] Baars B, Newman G. Convergent Cognitive and Neurophysiological Evidence for a Global Workspace Theory of Conscious Experience. Cambridge University Press; 1988

[67] Barinaga M. The mind revealed? Some neuroscientists think that recently discovered oscillations of electrical potential at 40 hertz hold the key to how the brain assembles sense impressions into a single object. Science. 1990;**249**(4971):856-858

[68] Crick F, Koch C. A framework for consciousness. Nature Neuroscience. 2003;**6**(2):119-126

[69] Crick F, Koch C. The problem of consciousness. Scientific American. 1992;**267**(3):152-159

[70] John ER. The neurophysics of consciousness. Brain Research—Brain Research Reviews. 2002;**39**(1):1-28

[71] Lamme VAF, Roelfsema PR. The distinct modes of vision offered by feedforward and recurrent processing. Trends in Neurosciences. 2000;**11**(23):571-579

[72] Eklund R. Recurrent Processing and the Consciousness. Bachelor Degree Project, University of Skövde; 2012

[73] Auksztulewicz R, Spitzer B, Blankenburg F. Recurrent neural processing and somatosensory awareness. Journal of Neuroscience. 2012;**32**(3):799-805

[74] Van Gaal S, Lamme VAF. Unconscious high-level information processing: Implication for neurobiological theories of consciousness. The Neuroscientist. 2012;**18**(3):287-301

[75] Tononi G. An information integration theory of consciousness. BMC Neuroscience. 2004;**5**(1):1-22

[76] Tononi G. Consciousness as integrated information: A provisional manifesto. The Biological Bulletin. 2008;**215**(3):216-242

[77] Massimini M, Boly M, Casali A, Rosanova M, Tononi G. A perturbational approach for evaluating the brain's capacity for consciousness. Progress in Brain Research. 2009;**177**:201-214

[78] Tononi G, Koch C. Consciousness: Here, there and everywhere? Philosophical Transactions of the Royal Society of London Series B, Biological Sciences. 2015;**370**(1668):20140167. DOI: 10.1098/rstb.2014.0167

[79] Tononi G, Koch C. The neural correlates of consciousness: An update. Annals of the New York Academy of Sciences. 2008;**1124**(1):239-261

[80] Lin Z, He S. Seeing the invisible: The scope and limits of unconscious processing in binocular rivalry. Progress in Neurobiology. 2009;**87**(4):195-211

[81] Bor D, Seth AK. Consciousness and the prefrontal parietal network: Insights from attention, working memory, and chunking. Frontiers in Psychology. 2012;**3**. DOI: 10.3389/fpsyg.2012.00063

[82] Atmanspacher H. Quantum theory and consciousness: An overview with selected examples. Discrete dynamics in Nature and Society. 2004;**2004**(1):51-73

[83] Crick F. The Astonishing Hypothesis: The Scientific Search for the Soul1995. p. 336

[84] Barbas H, Henion THH, Dermon CR. Diverse thalamic projections to the prefrontal cortex in the rhesus monkey. Journal of Comparative Neurology. 1991;**313**(1):65-94

[85] Gutierrez C, Cola MG, Seltzer B, Cusick C. Neurochemical and connectional organization of the dorsal pulvinar complex in monkeys. Journal of Comparative Neurology. 2000;**419**(1):61-86

[86] Leh SE, Chakravarty MM, Ptito A. The connectivity of the human pulvinar: A diffusion tensor imaging tractography study. International Journal of Biomedical Imaging. (special issue, open access) Recent Advances in Neuroimaging Methodology. 2008:2008. DOI: 10.1155/2008/789539

[87] Pennartz CMA, Dora S, Muckli L, Lorteije JAM. Towards a unified view on pathways and functions of neural recurrent processing. Trends in Neurosciences. 2019;**42**(9):589-603

[88] Mohedano-Moriano A, Martinez-Marcos A, Pro-Sistiaga P, Blaizot X, Arroyo-Jimenez MM, Marcos P, et al. Convergence of unimodal and polymodal sensory input to the entorhinal cortex in the fascicularis monkey. Neuroscience. 2008;**151**(1):255-271

[89] Nadasdy Z, Nguyen TP, Török Á, Shen JY, Briggs DE, Modur PN, et al. Context-dependent spatially periodic activity in the human entorhinal cortex. Proceedings of the National Academy of Sciences. 2017;**114**(17):E3516-E3E25

[90] Coutureau E, Di Scala G. Entorhinal cortex and cognition. Progress in Neuro-Psychopharmacology and Biological Psychiatry. 2009;**33**(5):753-761

[91] Knierim JJ, Neunuebel JP, Deshmukh SS. Functional correlates of the lateral and medial entorhinal cortex: Objects, path integration

and local-global reference frames. Philosophical Transactions of the Royal Society B: Biological Sciences. 2014;**369**(1635):20130369

[92] Insausti R, Munoz M. Cortical projections of the non-entorhinal hippocampal formation in the cynomolgus monkey (*Macaca fascicularis*). European Journal of Neuroscience. 2001;**14**(3):435-451

[93] Knowles WD. Normal anatomy and neurophysiology of the hippocampal formation. Journal of Clinical Neurophysiology: Official Publication of the American Electroencephalographic Society. 1992;**9**(2):252-263

[94] Insausti R, Amaral DG. Hippocampal Formation. In: The Human Nervous System. San Diego: Elsevier/Academic Press; 2004. pp. 871-915

[95] Cohen AL, Soussand L, Corrow SL, Martinaud O, Barton JJS, Fox MD. Looking beyond the face area: Lesion network mapping of prosopagnosia. Brain. 2019;**142**(12):3975-3990

[96] Fox CJ, Iaria G, Barton JJ. Disconnection in prosopagnosia and face processing. Cortex. 2008;**44**(8):996-1009

[97] Hadjikhani N, de Gelder B. Neural basis of prosopagnosia: An fMRI study. Human Brain Mapping. 2002;**16**:176-182

[98] Burman DD. Hippocampal Influences on Movements, Sensory, and Language Processing: A Role in Cognitive Control? Hippocampus-Cytoarchitecture and Diseases. London, UK: IntechOpen; 2021

[99] Booth JR, Burman DD, Meyer JR, Lei Z, Trommer BL, Davenport ND, et al. Neural development of selective attention and response inhibition. NeuroImage. 2003;**20**(2):737-751

[100] Booth JR, Burman DD, Meyer JR, Lei Z, Trommer BL, Davenport ND, et al. Larger deficits in brain networks for response inhibition than for visual selective attention in attention deficit hyperactivity disorder (ADHD). Journal of Child Psychology & Psychiatry & Allied Disciplines. 2005;**46**(1):94-111

[101] Mishkin M, Ungerleider LG, Macko KA. Object vision and spatial vision: Two cortical pathways. Trends in Neurosciences. 1983;**6**:414-417

[102] Boussaoud D, Bremmer F. Gaze effects in the cerebral cortex: Reference frames for space coding and action. Experimental Brain Research. 1999;**128**(1):170-180

[103] Sereno MI, Huang R-S. Multisensory maps in parietal cortex. Current Opinion in Neurobiology. 2014;**24**:39-46

[104] Sereno M, Pitzalis S, Martinez A. Mapping of contralateral space in retinotopic coordinates by a parietal cortical area in humans. Science. 2001;**294**(5545):1350-1354

[105] Silver MA, Kastner S. Topographic maps in human frontal and parietal cortex. Trends in Cognitive Sciences. 2009;**13**(11):488-495

[106] van Donkelaar P, Müri R. Craniotopic updating of visual space across saccades in the human posterior parietal cortex. Proceedings of the Royal Society of London Series B: Biological Sciences. 2002;**269**(1492):735-739

[107] Schindler A, Bartels A. Parietal cortex codes for egocentric space beyond the field of view. Current Biology. 2013;**23**(2):177-182

[108] Chen X, DeAngelis GC, Angelaki DE. Flexible egocentric and allocentric representations of heading

signals in parietal cortex. Proceedings of the National Academy of Sciences. 2018;**115**(14):E3305-E3E12

[109] Sasaki R, Anzai A, Angelaki DE, DeAngelis GC. Flexible coding of object motion in multiple reference frames by parietal cortex neurons. Nature Neuroscience. 2020;**23**(8):1004-1015

[110] O'Keefe J, Nadel L. The Hippocampus as a Cognitive Map. Oxford, England: Oxford University Press; 1978

[111] Redish AD, Ekstrom A. Hippocampus and Related Areas: What the Place Cell Literature Tells us about Cognitive Maps in Rats and Humans. In: Waller D, Nadel L, editors. Handbook of Spatial Cognition. American Psychological Association; 2013. pp. 15-34

[112] Clower DM, West RA, Lynch JC, Strick PL. The inferior parietal lobule is the target of output from the superior colliculus, hippocampus, and cerebellum. Journal of Neuroscience. 2001;**21**(16):6283-6291

[113] Vincent JL, Snyder AZ, Fox MD, Shannon BJ, Andrews JR, Raichle ME, et al. Coherent spontaneous activity identifies a hippocampal-parietal memory network. Journal of Neurophysiology. 2006;**96**(6):3517-3531

[114] Zheng A, Montez DF, Marek S, Gilmore AW, Newbold DJ, TO L, et al. Parallel hippocampal-parietal circuits for self-and goal-oriented processing. Proceedings of the National Academy of Sciences. 2021;**118**(34):e2101743118

[115] Cunningham SI, Tomasi D, Volkow ND. Structural and functional connectivity of the precuneus and thalamus to the default mode network. Human Brain Mapping. 2017;**2**(38):938-956

[116] Gaillard C, Ben HS. The neural bases of spatial attention and perceptual rhythms. European Journal of Neuroscience. 2022;**55**(11-12):3209-3223

[117] Burman DD, Booth JR. Music rehearsal increases the perceptual span for notation. Music Perception. 2009;**26**(4):303-320

[118] Burman DD. Topography of hippocampal connectivity with sensorimotor cortex revealed by optimizing smoothing kernel and voxel size. PLoS One. 2021;**16**(12):e0260245

[119] Burman DD. Hippocampal connectivity with sensorimotor cortex during volitional finger movements: Laterality and relationship to motor learning. PLoS One. 2019;**14**(9):e0222064

[120] Todd RM, Cunningham WA, Anderson AK, Thompson E. Affect-biased attention as emotion regulation. Trends in Cognitive Sciences. 2012;**16**(7):365-372

[121] Vuilleumier P, Driver J. Modulation of visual processing by attention and emotion: Windows on causal interactions between human brain regions. Philosophical Transactions of the Royal Society B: Biological Sciences. 2007;**362**(1481):837-855

[122] Dolcos F, Iordan AD, Dolcos S. Neural correlates of emotion-cognition interactions: A review of evidence from brain imaging investigations. Journal of Cognitive Psychology. 2011;**23**(6):669-694

[123] Funahashi S, Kubota K. Working memory and prefrontal cortex. Neuroscience Research. 1994;**21**(1):1-11

[124] Petrides M. The role of the mid-dorsolateral prefrontal cortex in working

memory. Experimental Brain Research. 2000;**133**(1):44-54

[125] Yamaguchi S, Hale LA, D'Esposito M, Knight RT. Rapid prefrontal-hippocampal habituation to novel events. Journal of Neuroscience. 2004;**24**(23):5356-5363

[126] Braver TS, Cohen JD, Nystrom LE, Jonides J, Smith EE, Noll DC. A parametric study of prefrontal cortex involvement in human working memory. NeuroImage. 1997;**5**(1):49-62

[127] Funahashi S. Working memory in the prefrontal cortex. Brain Sciences. 2017;**7**(5):49

[128] Rowe JB, Stephan KE, Friston K, Frackowiak RS, Passingham RE. The prefrontal cortex shows context-specific changes in effective connectivity to motor or visual cortex during the selection of action or colour. Cerebral Cortex. 2005;**15**(1):85-95

[129] Griffis JC, Elkhetali AS, Burge WK, Chen RH, Visscher KM. Retinotopic patterns of background connectivity between V1 and fronto-parietal cortex are modulated by task demands. Frontiers in Human Neuroscience. 2015;**9**:338

[130] Ruotolo F, Ruggiero G, Raemaekers M, Iachini T, Van der Ham I, Fracasso A, et al. Neural correlates of egocentric and allocentric frames of reference combined with metric and non-metric spatial relations. Neuroscience. 2019;**409**:235-252

[131] Arcaro MJ, Pinsk MA, Kastner S. The anatomical and functional organization of the human visual pulvinar. Journal of Neuroscience. 2015;**35**(27):9848-9871

[132] Baleydier C, Mauguiere F. Anatomical evidence for medial pulvinar connections with the posterior cingulate cortex, the retrosplenial area, and the posterior parahippocampal gyrus in monkeys. Journal of Comparative Neurology. 1985;**232**(2):219-228

[133] Barron DS, Eickhoff SB, Clos M, Fox PT. Human pulvinar functional organization and connectivity. Human Brain Mapping. 2015;**36**(7):2417-2431

[134] Fischer J, Whitney D. Attention gates visual coding in the human pulvinar. Nature Communications. 2012;**3**(1):1-9

[135] Hakamata Y, Sato E, Komi S, Moriguchi Y, Izawa S, Murayama N, et al. The functional activity and effective connectivity of pulvinar are modulated by individual differences in threat-related attentional bias. Scientific Reports. 2016;**6**(1):1-12

[136] Lakatos P, O'Connell MN, Barczak A. Pondering the pulvinar. Neuron. 2016;**89**(1):5-7

[137] Petersen SE, Robinson DL, Morris JD. Contributions of the pulvinar to visual spatial attention. Neuropsychologia. 1987;**25**(1):97-105

[138] Saalmann YB, Pinsk MA, Wang L, Li X, Kastner S. The pulvinar regulates information transmission between cortical areas based on attention demands. Science. 2012;**337**(6095):753-756

[139] Zhou H, Schafer RJ, Desimone R. Pulvinar-cortex interactions in vision and attention. Neuron. 2016;**89**(1):209-220

[140] Strumpf H, Mangun GR, Boehler CN, Stoppel C, Schoenfeld MA, Heinze HJ, et al. The role of the pulvinar in distractor processing and visual search. Human Brain Mapping. 2013;**34**(5):1115-1132

[141] Guest D, Allen E, Wu Y, Naselaris T, Arcaro M, Kay K. Evidence for a ventral visual stream in the pulvinar. Journal of Vision. 2021;**21**(9):2809

[142] Arend I, Henik A, Okon-Singer H. Dissociating emotion and attention functions in the pulvinar nucleus of the thalamus. Neuropsychology. 2015;**29**(2):191

[143] Padmala S, Lim S-L, Pessoa L. Pulvinar and affective significance: Responses track moment-to-moment stimulus visibility. Frontiers in Human Neuroscience. 2010;**4**:64

[144] Troiani V, Schultz RT. Amygdala, pulvinar, and inferior parietal cortex contribute to early processing of faces without awareness. Frontiers in Human Neuroscience. 2013;**7**:241

[145] Burman DD. Visual and Visuomotor Responses in the Monkey Lateral Pulvinar. Chicago: University of Illinois at Chicago; 1987

[146] Le QV, Isbell LA, Matsumoto J, Le VQ, Hori E, Tran AH, et al. Monkey pulvinar neurons fire differentially to snake postures. PLoS One. 2014;**9**(12):e114258

[147] Wilke M, Turchi J, Smith K, Mishkin M, Leopold DA. Pulvinar inactivation disrupts selection of movement plans. Journal of Neuroscience. 2010;**30**(25):8650-8659

[148] Felsten G, Benevento LA, Burman D. Opponent-color responses in macaque extrageniculate visual pathways: The lateral pulvinar. Brain Research. 1983;**288**:363-367

[149] Barleben M, Stoppel CM, Kaufmann J, Merkel C, Wecke T, Goertler M, et al. Neural correlates of visual motion processing without awareness in patients with striate cortex and pulvinar lesions. Human Brain Mapping. 2015;**36**(4):1585-1594

[150] Benarroch EE. Pulvinar: Associative role in cortical function and clinical correlations. Neurology. 2015;**84**(7):738-747

[151] Morris JS, Ohman A, Dolan RJ. A subcortical pathway to the right amygdala mediating "unseen" fear. Proceedings of the National Academy of Sciences of the United States of America. 1999;**96**(4):1680-1685

[152] Morris JS, Friston KJ, Buchel C, Frith CD, Young AW, Calder AJ, et al. A neuromodulatory role for the human amygdala in processing emotional facial expressions. Brain. 1998;**121**(Pt 1):47-57

[153] de Gelder B, Vroomen J, Pourtois G, Weiskrantz L. Non-conscious recognition of affect in the absence of striate cortex. Neuroreport. 1999;**10**(18):3759-3763

[154] Nguyen MN, Hori E, Matsumoto J, Tran AH, Ono T, Nishijo H. Neuronal responses to face-like stimuli in the monkey pulvinar. European Journal of Neuroscience. 2013;**37**(1):35-51

[155] Bourne JA, Morrone MC. Plasticity of visual pathways and function in the developing brain: Is the pulvinar a crucial player? Frontiers in Systems Neuroscience. 2017;**11**:3

[156] Kanai R, Komura Y, Shipp S, Friston K. Cerebral hierarchies: Predictive processing, precision and the pulvinar. Philosophical Transactions of the Royal Society B: Biological Sciences. 2015;**370**(1668):20140169

[157] Ketz NA, Jensen O, O'Reilly RC. Thalamic pathways underlying prefrontal cortex-medial temporal lobe oscillatory interactions. Trends in Neurosciences. 2015;**38**(1):3-12

[158] Libby LA, Hannula DE, Ranganath C. Medial temporal lobe coding of item and spatial information during relational binding in working memory. Journal of Neuroscience. 2014;**34**:14233-14242

[159] Barnett AJ, O'neil EB, Watson HC, ACH L. The human hippocampus is sensitive to the durations of events and intervals within a sequence. Neuropsychologia. 2014;**64**:1-12

[160] Burgess N, Becker S, King JA, O'Keefe J. Memory for events and their spatial context: Models and experiments. Philosophical Transactions of the Royal Society of London—Series B: Biological Sciences. 2001;**356**(1413):1493-1503

[161] Banquet J-P, Gaussier P, Cuperlier N, Hok V, Save E, Poucet B, et al. Time as the fourth dimension in the hippocampus. Progress in Neurobiology. 2020:101920. DOI: 10.1016/j.pneurobio.2020.101920

[162] Ekstrom AD, Ranganath C. Space, time, and episodic memory: The hippocampus is all over the cognitive map. Hippocampus. 2017;**28**(9):680-687

[163] Campbell KL, Madore KP, Benoit RG, Thakral PP, Schacter DL. Increased hippocampus to ventromedial prefrontal connectivity during the construction of episodic future events. Hippocampus. 2018;**28**(2):76-80

[164] Kumaran D, Maguire EA. An unexpected sequence of events: Mismatch detection in the human hippocampus. PLoS Biology. 2006;**4**(12):e424

[165] Numan R. The prefrontal-hippocampal comparator: Volition and episodic memory. Perceptual and Motor Skills. 2021;**128**(6):2421-2447

[166] Garrido MI, Barnes GR, Kumaran D, Maguire EA, Dolan RJ. Ventromedial prefrontal cortex drives hippocampal theta oscillations induced by mismatch computations. NeuroImage. 2015;**120**:362-370

[167] Ito HT. Prefrontal–hippocampal interactions for spatial navigation. Neuroscience Research. 2018;**129**:2-7

[168] Spiers HJ, Gilbert SJ. Solving the detour problem in navigation: A model of prefrontal and hippocampal interactions. Frontiers in Human Neuroscience. 2015;**9**:125

[169] Behrendt R-P. Consciousness, memory, and hallucinations. In: Hallucinations. Springer; 2013. pp. 17-31

[170] Behrendt R-P. Contribution of hippocampal region CA3 to consciousness and schizophrenic hallucinations. Neuroscience & Biobehavioral Reviews. 2010;**34**(8):1121-1136

[171] Shergill SS, Brammer MJ, Williams SC, Murray RM, McGuire PK. Mapping auditory hallucinations in schizophrenia using functional magnetic resonance imaging.[see comment]. Archives of General Psychiatry. 2000;**57**(11):1033-1038

[172] Hirao K, Miyata J, Fujiwara H, Yamada M, Namiki C, Shimizu M, et al. Theory of mind and frontal lobe pathology in schizophrenia: A voxel-based morphometry study. Schizophrenia Research. 2008;**105**(1-3):165-174

[173] Andreasen NC, Flashman L, Flaum M, Arndt S, Swayze V, O'Leary DS, et al. Regional brain abnormalities in schizophrenia measured with magnetic resonance imaging. Journal of the American Medical Association. 1994;**272**(22):1763-1769

[174] Keshavan MS, Tandon R, Boutros NN, Nasrallah HA.

Schizophrenia, “just the facts”: What we know in 2008: Part 3: Neurobiology. Schizophrenia Research. 2008;**106**(2-3):89-107

[175] Pennington K, Beasley C, Dicker P, Fagan A, English J, Pariante C, et al. Prominent synaptic and metabolic abnormalities revealed by proteomic analysis of the dorsolateral prefrontal cortex in schizophrenia and bipolar disorder. Molecular Psychiatry. 2008;**13**(12):1102-1117

[176] Heckers S. Neuroimaging studies of the hippocampus in schizophrenia. Hippocampus. 2001;**11**(5):520-528

[177] Meyer-Lindenberg AS, Olsen RK, Kohn PD, Brown T, Egan MF, Weinberger DR, et al. Regionally specific disturbance of dorsolateral prefrontal–hippocampal functional connectivity in schizophrenia. Archives of General Psychiatry. 2005;**62**(4):379-386

[178] Rusch N, van Elst LT, Valerius G, Buchert M, Thiel T, Ebert D, et al. Neurochemical and structural correlates of executive dysfunction in schizophrenia. Schizophrenia Research. 2008;**99**(1):155-163

[179] Spiers HJ, Maguire EA, Burgess N. Hippocampal amnesia. Neurocase. 2001;**7**(5):357-382

[180] Zola-Morgan S, Squire LR, Amaral DG. Human amnesia and the medial temporal region: Enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus. The Journal of Neuroscience. 1986;**6**(10):2950-2967

[181] Blumenfeld RS, Ranganath C. Prefrontal cortex and long-term memory encoding: An integrative review of findings from neuropsychology and neuroimaging. The Neuroscientist. 2007;**13**(3):280-291

[182] D'Esposito M. From cognitive to neural models of working memory. Philosophical Transactions of the Royal Society of London—Series B: Biological Sciences. 2007;**362**(1481):761-772

[183] Hartley AA, Speer NK. Locating and fractionating working memory using functional neuroimaging: Storage, maintenance, and executive functions. Microscopy Research & Technique. 2000;**51**(1):45-53

[184] Linden DE. The working memory networks of the human brain. The Neuroscientist. 2007;**13**(3):257-267

[185] Barbey AK, Koenigs M, Grafman J. Dorsolateral prefrontal contributions to human working memory. Cortex. 2013;**49**(5):1195-1205

[186] Hillary FG. Neuroimaging of working memory dysfunction and the dilemma with brain reorganization hypotheses. Journal of the International Neuropsychological Society. 2008;**14**(4):526-534

[187] Thompson-Schill SL, Jonides J, Marshuetz C, Smith EE, D'Esposito M, Kan IP, et al. Effects of frontal lobe damage on interference effects in working memory. Cognitive, Affective, & Behavioral Neuroscience. 2002;**2**(2):109-120

[188] JnM F. Prefrontal neurons in networks of executive memory. Brain Research Bulletin. 2000;**52**(5):331-336

[189] Bone MB, Buchsbaum BR. Detailed episodic memory depends on concurrent reactivation of basic visual features within the posterior hippocampus and early visual cortex. Cerebral Cortex Communications. 2021;**2**(3):tgab045

[190] Favila SE, Kuhl BA, Winawer J. Perception and memory have distinct

spatial tuning properties in human visual cortex. Nature Communications. 2022;**13**(1):1-21

[191] Harris JA, Petersen RS, Diamond ME. The cortical distribution of sensory memories. Neuron. 2001;**30**(2):315-318

[192] Condylis C, Lowet E, Ni J, Bistrong K, Ouellette T, Josephs N, et al. Context-dependent sensory processing across primary and secondary somatosensory cortex. Neuron. 2020;**106**(3):515-525 e5

[193] Schmidt TT, Ostwald D, Blankenburg F. Imaging tactile imagery: Changes in brain connectivity support perceptual grounding of mental images in primary sensory cortices. NeuroImage. 2014;**98**:216-224

[194] Miller KJ, Schalk G, Fetz EE, Den Nijs M, Ojemann JG, Rao RP. Cortical activity during motor execution, motor imagery, and imagery-based online feedback. Proceedings of the National Academy of Sciences. 2010;**107**(9):4430-4435

[195] Eichenbaum H. Prefrontal-hippocampal interactions in episodic memory. Nature Reviews Neuroscience. 2017;**18**(9):547-558

[196] Copara MS, Hassan AS, Kyle CT, Libby LA, Ranganath C, Ekstrom AD. Complementary roles of human hippocampal subregions during retrieval of spatiotemporal context. The Journal of Neuroscience. 2014;**34**:6834-6842

[197] Barker GRI, Banks PJ, Scott H, Ralph GS, Mitrophanous KA, Wong L-F, et al. Separate elements of episodic memory subserved by distinct hippocampal-prefrontal connections. Nature Neuroscience. 2017;**20**(2):242-250

[198] Son GR, Therrien B, Whall A. Implicit memory and familiarity among elders with dementia. Journal of Nursing Scholarship. 2002;**34**(3):263-267

[199] Passini R, Pigot H, Rainville C, Tétreault M-H. Wayfinding in a nursing home for advanced dementia of the Alzheimer’s type. Environment and Behavior. 2000;**32**(5):684-710

[200] Donix M, Jurjanz L, Meyer S, Amanatidis EC, Baeumler D, Huebner T, et al. Functional imaging during recognition of personally familiar faces and places in Alzheimer's disease. Archives of Clinical Neuropsychology. 2013;**28**(1):72-80

[201] Bertram E. The relevance of kindling for human epilepsy. Epilepsia. 2007;**48**(s2):65-74

[202] Albouy G, Fogel S, King BR, Laventure S, Benali H, Karni A, et al. Maintaining vs. enhancing motor sequence memories: Respective roles of striatal and hippocampal systems. NeuroImage. 2015;**108**:423-434

[203] Albouy G, King BR, Maquet P, Doyon J. Hippocampus and striatum: Dynamics and interaction during acquisition and sleep-related motor sequence memory consolidation. Hippocampus. 2013;**23**(11):985-1004

[204] Albouy G, Sterpenich V, Balteau E, Vandewalle G, Desseilles M, Dang-Vu T, et al. Both the hippocampus and striatum are involved in consolidation of motor sequence memory. Neuron. 2008;**58**(2):261-272

[205] Fernandez-Seara MA, Aznarez-Sanado M, Mengual E, Loayza FR, Pastor MA. Continuous performance of a novel motor sequence leads to highly correlated striatal and hippocampal perfusion increases. NeuroImage. 2009;**47**(4):1797-1808

[206] Kesner RP, Bolland BL, Dakis M. Memory for spatial locations, motor responses, and objects: Triple dissociation among the hippocampus, caudate nucleus, and extrastriate visual cortex. Experimental Brain Research. 1993;**93**(3):462-470

[207] Hollup SA, Kjelstrup KG, Hoff J, Moser M-B, Moser EI. Impaired recognition of the goal location during spatial navigation in rats with hippocampal lesions. Journal of Neuroscience. 2001;**21**(12):4505-4513

[208] Miller MJ, Burman DD. Temporal and spatial selectivity of hippocampal connectivity with sensorimotor cortex during individual finger movements. bioRxiv 2023. DOI: 10.1101/479436

[209] Qin S, Duan X, Supekar K, Chen H, Chen T, Menon V. Large-scale intrinsic functional network organization along the long axis of the human medial temporal lobe. Brain Structure and Function. 2016;**221**(6):3237-3258

[210] Ionta S, Heydrich L, Lenggenhager B, Mouthon M, Fornari E, Chapuis D, et al. Multisensory mechanisms in temporo-parietal cortex support self-location and first-person perspective. Neuron. 2011;**70**(2):363-374

[211] Hodzic A, Muckli L, Singer W, Stirn A. Cortical responses to self and others. Human Brain Mapping. 2009;**30**(3):951-962

[212] Ritterband-Rosenbaum A, Karabanov AN, Christensen MS, Nielsen JB. 10 Hz rTMS over right parietal cortex alters sense of agency during self-controlled movements. Frontiers in Human Neuroscience. 2014;**8**:471

[213] Shimada S, Hiraki K, Oda I. The parietal role in the sense of self-ownership with temporal discrepancy between visual and proprioceptive feedbacks. NeuroImage. 2005;**24**(4):1225-1232

[214] Duff MC, Brown-Schmidt S. The hippocampus and the flexible use and processing of language. Frontiers in Human Neuroscience. 2012;**6**:69

[215] Covington NV, Duff MC. Expanding the language network: Direct contributions from the hippocampus. Trends in Cognitive Sciences. 2016;**20**(12):869-870

[216] Piai V, Anderson KL, Lin JJ, Dewar C, Parvizi J, Dronkers NF, et al. Direct brain recordings reveal hippocampal rhythm underpinnings of language processing. Proceedings of the National Academy of Sciences. 2016;**113**(40):11366-11371

[217] Alamri S. The Contribution of the hippocampus to Language Processing. [doctoral dissertation] Barcelona, Spain: Universidad de Barcelona; 2017

[218] Gluth S, Sommer T, Rieskamp J, Büchel C. Effective connectivity between hippocampus and ventromedial prefrontal cortex controls preferential choices from memory. Neuron. 2015;**86**(4):1078-1090

[219] Weilbächer RA, Gluth S. The interplay of hippocampus and ventromedial prefrontal cortex in memory-based decision making. Brain Sciences. 2017;**7**(1):4. DOI: 10.3390/brainsci7010004

[220] Gazzaniga MS. The split brain revisited. Scientific American. 1998;**279**(1):50-55

[221] Gazzaniga MS. Forty-five years of split-brain research and still going strong. Nature Reviews Neuroscience. 2005;**6**(8):653-659

[222] Dor-Ziderman Y, Ataria Y, Fulder S, Goldstein A, Berkovich-Ohana A. Self-specific processing in the meditating brain: A MEG neurophenomenology study. Neuroscience of Consciousness. 2016;**2016**(1):1-13

[223] Lindahl JR, Britton WB. 'I have this feeling of not really being Here': Buddhist meditation and changes in sense of self. Journal of Consciousness Studies. 2019;**26**(7-8):157-183

[224] Millière R, Carhart-Harris RL, Roseman L, Trautwein F-M, Berkovich-Ohana A. Psychedelics, meditation, and self-consciousness. Frontiers in Psychology. 2018;**9**:1475

[225] Bishop SJ. Neurocognitive mechanisms of anxiety: An integrative account. Trends in Cognitive Sciences. 2007;**11**(7):307-316

[226] Bowen HJ, Kark SM, Kensinger EA. NEVER forget: Negative emotional valence enhances recapitulation. Psychonomic Bulletin & Review. 2018;**25**(3):870-891

[227] Kensinger EA. Remembering the details: Effects of emotion. Emotion Review. 2009;**1**(2):99-113

[228] Bhattacharyya KB. James Wenceslaus Papez, his circuit, and emotion. Annals of Indian Academy of Neurology. 2017;**20**(3):207-210

[229] Ball T, Derix J, Wentlandt J, Wieckhorst B, Speck O, Schulze-Bonhage A, et al. Anatomical specificity of functional amygdala imaging of responses to stimuli with positive and negative emotional valence. Journal of Neuroscience Methods. 2009;**180**(1):57-70

[230] Costafreda SG, Brammer MJ, David AS, Fu CH. Predictors of amygdala activation during the processing of emotional stimuli: A meta-analysis of 385 PET and fMRI studies. Brain Research Reviews. 2008;**58**(1):57-70

[231] Buchel C, Dolan RJ, Armony JL, Friston KJ. Amygdala-hippocampal involvement in human aversive trace conditioning revealed through event-related functional magnetic resonance imaging. Journal of Neuroscience. 1999;**19**(24):10869-10876

[232] Henke K, Treyer V, Weber B, Nitsch RM, Hock C, Wieser HG, et al. Functional neuroimaging predicts individual memory outcome after amygdalohippocampectomy. Neuroreport. 2003;**14**(9):1197-1202

[233] McDonald AJ, Mott DD. Functional neuroanatomy of amygdalohippocampal interconnections and their role in learning and memory. Journal of Neuroscience Research. 2017;**95**(3):797-820

[234] Adinoff B. Neurobiologic processes in drug reward and addiction. Harvard Review of Psychiatry. 2004;**12**(6):305-320

[235] Berridge KC, Kringelbach ML. Neuroscience of affect: Brain mechanisms of pleasure and displeasure. Current Opinion in Neurobiology. 2013;**23**(3):294-303

[236] Floresco SB. The nucleus accumbens: An interface between cognition, emotion, and action. Annual Review of Psychology. 2015;**66**(1):25-52

[237] Klawonn AM, Malenka RC, editors. Nucleus accumbens modulation in reward and aversion. In: Cold Spring Harbor Symposia on Quantitative Biology. Cold Spring Harbor Laboratory Press; 2018

[238] Demos KE, Heatherton TF, Kelley WM. Individual differences in nucleus accumbens activity to food and sexual images predict weight gain and sexual behavior. Journal of Neuroscience. 2012;**32**(16):5549-5552

[239] Sabatinelli D, Bradley MM, Lang PJ, Costa VD, Versace F. Pleasure rather than salience activates human nucleus accumbens and medial prefrontal cortex. Journal of Neurophysiology. 2007;**98**(3):1374-1379

[240] Carlezon WA Jr, Thomas MJ. Biological substrates of reward and aversion: A nucleus accumbens activity hypothesis. Neuropharmacology. 2009;**56**:122-132

[241] Kim H, Shimojo S, O'Doherty JP. Overlapping responses for the expectation of juice and money rewards in human ventromedial prefrontal cortex. Cerebral Cortex. 2011;**21**(4):769-776

[242] Nawa NE, Ando H. Effective connectivity within the ventromedial prefrontal cortex-hippocampus-amygdala network during the elaboration of emotional autobiographical memories. NeuroImage. 2019;**189**:316-328

[243] van den Bos W, Güroğlu B. The role of the ventral medial prefrontal cortex in social decision making. Journal of Neuroscience. 2009;**29**(24):7631-7632

[244] Coricelli G, Dolan RJ, Sirigu A. Brain, emotion and decision making: The paradigmatic example of regret. Trends in Cognitive Sciences. 2007;**11**(6):258-265

[245] Clark L, Bechara A, Damasio H, Aitken M, Sahakian B, Robbins T. Differential effects of insular and ventromedial prefrontal cortex lesions on risky decision-making. Brain. 2008;**131**(5):1311-1322

[246] Bechara A, Damasio H, Damasio AR. Role of the amygdala in decision-making. Annals of the New York Academy of Sciences. 2003;**985**:356-369

[247] Rolls ET, Grabenhorst F. The orbitofrontal cortex and beyond: From affect to decision-making. Progress in Neurobiology. 2008;**86**(3):216-244

[248] Bechara A, Damasio H, Damasio AR. Emotion, decision making and the orbitofrontal cortex. Cerebral Cortex. 2000;**10**(3):295-307

[249] Liang B, Du Y. The functional neuroanatomy of lexical tone perception: An activation likelihood estimation meta-analysis. Frontiers in Neuroscience. 2018;**12**:495

[250] Artemenko C, Sitnikova MA, Soltanlou M, Dresler T, Nuerk H-C. Functional lateralization of arithmetic processing in the intraparietal sulcus is associated with handedness. Scientific Reports. 2020;**10**(1):1-11

[251] Chechlacz M, Gillebert CR, Vangkilde SA, Petersen A, Humphreys GW. Structural variability within frontoparietal networks and individual differences in attentional functions: An approach using the theory of visual attention. Journal of Neuroscience. 2015;**35**(30):10647-10658

[252] Neophytou D, Arribas DM, Arora T, Levy RB, Park IM, Oviedo HV. Differences in temporal processing speeds between the right and left auditory cortex reflect the strength of recurrent synaptic connectivity. PLOS Biology. 2022;**20**(10):e3001803. DOI: 10.1371/journal.pbio.3001803

[253] Mentese B, Kutlu N. Menstrual cycle phases, sex hormones and hand preference modulate nerve conduction

velocity in healthy subjects. Physiology & Behavior. 2022:114003. DOI: 10.1016/j.physbeh.2022.114003

[254] Tan Ü. Sensory nerve conduction velocities are higher on the left than the right hand and motor conduction is faster on the right hand than left in right-handed normal subjects. International Journal of Neuroscience. 1993;**73**(1-2):85-91

[255] Vallar G. Spatial hemineglect in humans. Trends in Cognitive Sciences. 1998;**2**(3):87-97

[256] Kerkhoff G. Spatial hemineglect in humans. Progress in Neurobiology. 2001;**63**(1):1-27

[257] Cojan Y, Saj A, Vuilleumier P. Brain substrates for distinct spatial processing components contributing to Hemineglect in humans. Brain Sciences. 2021;**11**(12):1584

[258] Lindell AK. Lateral thinkers are not so laterally minded: Hemispheric asymmetry, interaction, and creativity. Laterality: Asymmetries of Body, Brain and Cognition. 2011;**16**(4):479-498

[259] Burman DD, Minas T, Bolger DJ, Booth JR. Age, sex, and verbal abilities affect location of linguistic connectivity in ventral visual pathway. Brain and Language. 2013;**124**(2):184-193

[260] Burton MW, Noll DC, Small SL. The anatomy of auditory word processing: Individual variability. Brain and Language. 2001;**77**(1):119-131

[261] MacDonald SW, Nyberg L, Bäckman L. Intra-individual variability in behavior: Links to brain structure, neurotransmission and neuronal activity. Trends in Neurosciences. 2006;**29**(8):474-480

[262] Jacobs J. Hippocampal theta oscillations are slower in humans than in rodents: Implications for models of spatial navigation and memory. Philosophical Transactions of the Royal Society of London B: Biological Sciences. 2014;**369**(1635):20130304

[263] Lega BC, Jacobs J, Kahana M. Human hippocampal theta oscillations and the formation of episodic memories. Hippocampus. 2012;**22**(4):748-761

[264] Kaplan R, Doeller CF, Barnes GR, Litvak V, Düzel E, Bandettini PA, et al. Movement-related Theta rhythm in humans: Coordinating self-directed hippocampal learning. PLoS Biology. 2012;**10**(2):e1001267. DOI: 10.1371/journal.pbio.1001267

[265] Olsen RK, Rondina R II, Riggs L, Meltzer JA, Ryan JD. Hippocampal and neocortical oscillatory contributions to visuospatial binding and comparison. Journal of Experimental Psychology: General. 2013;**142**(4):1335

[266] Toscani M, Marzi T, Righi S, Viggiano MP, Baldassi S. Alpha waves: A neural signature of visual suppression. Experimental Brain Research. 2010;**207**(3):213-219

[267] Addante RJ. A critical role of the human hippocampus in an electrophysiological measure of implicit memory. NeuroImage. 2015;**109**:515-528

[268] Rose M, Haider H, Salari N, Buchel C. Functional dissociation of hippocampal mechanism during implicit learning based on the domain of associations. Journal of Neuroscience. 2011;**31**:13739-13745

[269] Schacter DL. The cognitive neuroscience of memory: Perspectives from neuroimaging research. Philosophical Transactions of the Royal Society of London—Series B: Biological Sciences. 1997;**352**(1362):1689-1695

[270] Verfaellie M, Keane MM. The neural basis of aware and unaware forms of memory. Seminars in Neurology. 1997;**17**(2):153-161

[271] Zaidel DW, Esiri M, Beardsworth E. Observations on the relationship between verbal explicit and implicit memory and density of neurons in the hippocampus. Neuropsychologia. 1998;**36**(10):1049-1062

[272] Sansom SN, Livesey FJ. Gradients in the brain: The control of the development of form and function in the cerebral cortex. Cold Spring Harbor Perspectives in Biology. 2009;**1**(2):a002519

[273] Uylings HB. Development of the human cortex and the concept of "critical" or "sensitive" periods. Language Learning. 2006;**56**:59-90

[274] Wang B-S, Sarnaik R, Cang J. Critical period plasticity matches binocular orientation preference in the visual cortex. Neuron. 2010;**65**(2):246-256

[275] Brooks SJ, Savov V, Allzen E, Benedict C, Fredriksson R, Schioth HB. Exposure to subliminal arousing stimuli induces robust activation in the amygdala, hippocampus, anterior cingulate, insular cortex and primary visual cortex: A systematic meta-analysis of fMRI studies. NeuroImage. 2012;**59**(3):2962-2973

[276] Degonda N, Mondadori CR, Bosshardt S, Schmidt CF, Boesiger P, Nitsch RM, et al. Implicit associative learning engages the hippocampus and interacts with explicit associative learning. Neuron. 2005;**46**(3):505-520

[277] Dehaene S, Changeux JP, Naccache L, Sackur J, Sergent C. Conscious, preconscious, and subliminal processing: A testable taxonomy. Trends in Cognitive Sciences. 2006;**10**(5):204-211

[278] Elliott R, Dolan RJ. Neural response during preference and memory judgments for subliminally presented stimuli: A functional neuroimaging study. Journal of Neuroscience. 1998;**18**(12):4697-4704

[279] Kouider S, Dehaene S, Jobert A, Le Bihan D. Cerebral bases of subliminal and supraliminal priming during reading. Cerebral Cortex. 2007;**17**(9):2019

[280] Pessiglione M, Petrovic P, Daunizeau J, Palminteri S, Dolan RJ, Frith CD. Subliminal instrumental conditioning demonstrated in the human brain. Neuron. 2008;**59**(4):561-567

[281] Elliott MA, Müller HJ. Synchronous information presented in 40-HZ flicker enhances visual feature binding. Psychological Science. 1998;**9**(4):277-283

[282] Brunet N, Bosman CA, Roberts M, Oostenveld R, Womelsdorf T, De Weerd P, et al. Visual cortical gamma-band activity during free viewing of natural images. Cerebral Cortex. 2015;**25**(4):918-926

[283] Wittmann M. Moments in time. Frontiers in Integrative Neuroscience. 2011;**5**:66

[284] Jezek K, Henriksen EJ, Treves A, Moser EI, Moser M-B. Theta-paced flickering between place-cell maps in the hippocampus. Nature. 2011;**478**(7368):246-249

[285] Kolers PA, von Grünau M. Shape and color in apparent motion. Vision Research. 1976;**16**(4):329-335

[286] Becker W, Jürgens R. An analysis of the saccadic system by means of double step stimuli. Vision Research. 1979;**19**(9):967-983

[287] Sheliga B, Brown V, Miles F. Voluntary saccadic eye movements in humans studied with a double-cue paradigm. Vision Research. 2002;**42**(15):1897-1915

[288] Packard MG, Knowlton BJ. Learning and memory functions of the basal ganglia. Annual Review of Neuroscience. 2002;**25**:563-593

[289] Doyon J, Bellec P, Amsel R, Penhune V, Monchi O, Carrier J, et al. Contributions of the basal ganglia and functionally related brain structures to motor learning. Behavioural Brain Research. 2009;**199**(1):61-75

[290] Retailleau A, Etienne S, Guthrie M, Boraud T. Where is my reward and how do I get it? Interaction between the hippocampus and the basal ganglia during spatial learning. Journal of Physiology-Paris. 2012;**106**(3-4):72-80

[291] Maguire EA, Burgess N, Donnett JG, Frackowiak RS, Frith CD, O'Keefe J. Knowing where and getting there: A human navigation network. Science. 1998;**280**(5365):921-924

[292] Ekstrom AD, Kahana MJ, Caplan JB, Fields TA, Isham EA, Newman EL, et al. Cellular networks underlying human spatial navigation. Nature. 2003;**425**(6954):184-188

[293] Cornwell BR, Johnson LL, Holroyd T, Carver FW, Grillon C. Human hippocampal and parahippocampal theta during goal-directed spatial navigation predicts performance on a virtual Morris water maze. The Journal of Neuroscience. 2008;**28**(23):5983-5990

[294] Eichenbaum H. The role of the hippocampus in navigation is memory. Journal of Neurophysiology. 2017;**117**(4):1785

[295] Joshi DH, Jaliya U, Thakore DG, editors. Argos: Alertness rating gamma brainwave observation system. In: 2016 International Conference on Data Mining and Advanced Computing (SAPIENCE). 2016. p. IEEE

[296] Dunn BR, Hartigan JA, Mikulas WL. Concentration and mindfulness meditations: Unique forms of consciousness? Applied Psychophysiology and Biofeedback. 1999;**24**(3):147-165. DOI: 10.3389/fnins.2015.00280

[297] Sohn WS, Yoo K, Lee Y-B, Seo SW, Na DL, Jeong Y. Influence of ROI selection on resting state functional connectivity: An individualized approach for resting state fMRI analysis. Frontiers in Neuroscience. 2015;**9**. Article 280. DOI: 10.3389/fnins.2015.00280

[298] Burman DD. Creating hippocampal PPI connectivity maps with SPM12 batch files. 2021. DOI: 10.17504/protocols.io.byu6pwze. Available from: www.protocols.io

[299] Hampson RE, Song D, Robinson BS, Fetterhoff D, Dakos AS, Roeder BM, et al. Developing a hippocampal neural prosthetic to facilitate human memory encoding and recall. Journal of Neural Engineering. 2018;**15**:036014. DOI: 10.1088/1741-2552/aaaed7

[300] Kim K, Ekstrom AD, Tandon N. A network approach for modulating memory processes via direct and indirect brain stimulation: Toward a causal approach for the neural basis of memory. Neurobiology of Learning and Memory. 2016;**134**:162-177

[301] Igloi K, Doeller CF, Berthoz A, Rondi-Reig L, Burgess N. Lateralized human hippocampal activity predicts navigation based on sequence or place memory. Proceedings of the National Academy of Sciences. 2010;**107**(32):14466-14471

[302] Rolls ET. Hippocampal spatial view cells for memory and navigation, and

their underlying connectivity in humans. Hippocampus. 2023;**33**:533-572

[303] Rissman J, Gazzaley A, D'Esposito M. Dynamic adjustments in prefrontal, hippocampal, and inferior temporal interactions with increasing visual working memory load. Cerebral Cortex. 2008;**18**(7):1618-1629

[304] Van Hoesen GW, Hyman BT, Damasio AR. Entorhinal cortex pathology in Alzheimer's disease. Hippocampus. 1991;**1**(1):1-8

[305] Gómez-Isla T, Price JL, McKeel DW Jr, Morris JC, Growdon JH, Hyman BT. Profound loss of layer II entorhinal cortex neurons occurs in very mild Alzheimer's disease. Journal of Neuroscience. 1996;**16**(14):4491-4500

[306] Zhu M, Cortese GP, Waites CL. Parkinson's disease-linked parkin mutations impair glutamatergic signaling in hippocampal neurons. BMC Biology. 2018;**16**(1):1-20

[307] Calabresi P, Castrioto A, Di Filippo M, Picconi B. New experimental and clinical links between the hippocampus and the dopaminergic system in Parkinson's disease. The Lancet Neurology. 2013;**12**(8):811-821

[308] Martin G. Traumatic brain injury: The first 15 milliseconds. Brain Injury. 2016;**30**(13-14):1517-1524

[309] Bigler ED. Neuropsychology and clinical neuroscience of persistent post-concussive syndrome. Journal of the International Neuropsychological Society. 2008;**14**(1):1-22

[310] Ng LJ, Volman V, Gibbons MM, Phohomsiri P, Cui J, Swenson DJ, et al. A mechanistic end-to-end concussion model that translates head kinematics to neurologic injury. Frontiers in Neurology. 2017;**8**:269

[311] Brett BL, Savitz J, Nitta M, España L, Teague TK, Nelson LD, et al. Systemic inflammation moderates the association of prior concussion with hippocampal volume and episodic memory in high school and collegiate athletes. Brain, Behavior, and Immunity. 2020;**89**:380-388

[312] Meier TB, España LY, Kirk AJ, Nader AM, Powell JE, Nelson LD, et al. Association of previous concussion with hippocampal volume and symptoms in collegiate-aged athletes. Journal of Neurotrauma. 2021;**38**(10):1358-1367

[313] Fotuhi M, Do D, Jack C. Modifiable factors that alter the size of the hippocampus with ageing. Nature Reviews Neurology. 2012;**8**(4):189-202

[314] Frodl T, Schaub A, Banac S, Charypar M, Jager M, Kummler P, et al. Reduced hippocampal volume correlates with executive dysfunctioning in major depression. Journal of Psychiatry and Neuroscience. 2006;**31**(5):316

[315] Rangarajan B, Suresh S, Mahanand B, editors. Identification of potential biomarkers in the hippocampus region for the diagnosis of ADHD using PBL-McRBFN approach. In: 2014 13th International Conference on Control Automation Robotics & Vision (ICARCV). IEEE; 2014

[316] Plessen KJ, Bansal R, Zhu H, Whiteman R, Amat J, Quackenbush GA, et al. Hippocampus and amygdala morphology in attention-deficit/hyperactivity disorder. Archives of General Psychiatry. 2006;**63**(7):795-807

[317] Liberzon I, Sripada CS. The functional neuroanatomy of PTSD: A critical review. Progress in Brain Research. 2008;**167**:151-169

[318] Malivoire BL, Girard TA, Patel R, Monson CM. Functional connectivity

of hippocampal subregions in PTSD: Relations with symptoms. BMC Psychiatry. 2018;**18**(1):129

[319] Cho Y, Park S, Lee J, Yu KJ. Emerging materials and technologies with applications in flexible neural implants: A comprehensive review of current issues with neural devices. Advanced Materials. 2021;**33**(47):2005786

[320] Hassler C, Boretius T, Stieglitz T. Polymers for neural implants. Journal of Polymer Science Part B: Polymer Physics. 2011;**49**(1):18-33

[321] Costa G, Gusberti A, Graffigna JP, Guzzo M, Nasisi O, editors. Mobility and orientation aid for blind persons using artificial vision. In: Journal of Physics: Conference Series. IOP Publishing; 2007

[322] Nau AC, Pintar C, Arnoldussen A, Fisher C. Acquisition of visual perception in blind adults using the BrainPort artificial vision device. The American Journal of Occupational Therapy. 2015;**69**(1):6901290010p1-6901290010p8

[323] Novich SD, Eagleman DM. Using space and time to encode vibrotactile information: Toward an estimate of the skin's achievable throughput. Experimental Brain Research. 2015;**233**(10):2777-2788

[324] Novich SD, Eagleman DM, editors. [D79] A vibrotactile sensory substitution device for the deaf and profoundly hearing impaired. In: 2014 IEEE Haptics Symposium (HAPTICS). 2014. p. IEEE

[325] Perrotta MV, Asgeirsdottir T, Eagleman DM. Deciphering sounds through patterns of vibration on the skin. Neuroscience. 2021;**458**:77-86

[326] Berta Y. Sensory augmentation and the tactile sublime. Debates in Aesthetics. 2020;**15**(1):11-32

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During the last half century, several scientific theories have been developed to explain experimental findings related to our conscious experiences, particularly those involving perceptual awareness. The adequacy and philosophical implications of these theories have been debated fiercely by both scientists and philosophers, including their adequacy in explaining the subjective nature of consciousness. The adequacy of theoretical models has been controversial in part because there is no consensus on the definition of consciousness itself. This book proposes a new model of consciousness based on a prescribed definition for one of several conscious states of consciousness known as the “normal alert state.” This state is inclusive of all characteristics associated with an alert individual unaffected by disease or injury, including awareness of the sensory environment, memory, learning, attention to location and sensory features, language, emotional responses, intentional movements, decision-making, creativity, and a sense of self. Through its interactions with other brain regions essential for these diverse functions, the model posits a central executive role for the hippocampus; qualia, the subjective feelings associated with sensory stimuli, is suggested to arise from hippocampal interactions between events and the emotional responses they evoke. The book offers methods for testing the model. In several respects, this model of hippocampal function is analogous to the “central theater of the mind” described by Descartes, a concept that philosophers and scientists previously discarded due to false assumptions. The model presented brings together observations about our inner selves and scientific findings, using analogies and life experiences to illustrate how each can inform the other; evidence suggests disruptions in information flow between the hippocampus and other brain regions may explain many neurological disorders. The book also discusses the philosophical implications of the model, including such topics as free will and the possibility of sentience in artificial intelligence.

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