A Systems-Based Understanding of Visual Processing, Mental Imagery, and Their Absence

by Aramis D. Munoz-Valverde

for Mind & Language Seminar with Ned Block & Erik Mandelbaum

New York University

May 10th, 2025

A Systems-Based Understanding of Visual Processing, Mental Imagery, and Their Absence

by Aramis D. Munoz-Valverde

for Mind & Language Seminar with Ned Block & Erik Mandelbaum

May 10th, 2025

Abstract

The visual system is among the best mapped neural systems in the brain. Nonetheless, there exist a substantial set of questions regarding the general processing structures critical to the operation of this system, and what happens when this system goes awry, as in blindsight and aphantasia. Here we outline a simple anatomic-computational structure for visual processing by combining recent advances in the visual processing literature with a neuroscientifically derived minimal framework for reportable perceptual awareness[1][2]. Throughout we will build up a basic idea of what the perceptual and visual systems we are engaging with are doing computationally. For the constituent neural systems, we will similarly outline their roles, and the evidence supporting their validity. Once established, we utilize this tentative visual processing framework to parse an abstract visual percept, and then examine the specific neuro-computational aberrations which give rise to the phenomena of blindsight and aphantasia.

In doing so we will embrace the idea recently put forward by Ian Phillips that by breaking with assumptions of simplicity and taking on the scope of variability in human cognition we can arrive at more elegant explanations for complex phenomena. We also take to heart Phillips' distinctions in potential variations in kinds of percepts[3] which are directly and functionally distinguishable according to their underlying structures and dynamics. Despite the agreement there, we disagree with the assertion that blindsight and aphantasia are best understood as cases of minimally perceptible stimuli being mistaken for conscious stimuli. We instead propose that cases of function without direct awareness are indeed cases of function without direct awareness. We propose that cases of blindsight are cases of genuinely unconscious ascertainment by the neural system of visual phenomenon, and that cases of aphantasia are indeed cases of an inability to adequately utilize top-down signal flows to manipulate the visual cortex in a utile way. We will go over case studies which will clarify this view, and we will proceed to derive the expected and actual symptom profile of patients based on their particular lesions and accompanying aberrant information flows.

We will conclude with general discussion on what the visual processing system might tell us or not tell us about consciousness and cognition generally, and engage with some disagreements in the literature.

[1] By "reportable awareness", we mean a percept that is accessible for explicit report, verbal or behavioral, and is further accessible in such a manner that the perceiver can confirm the presence of the percept or describe at least one of its attributes replicably and above chance.

[2] In preparation.

[3] What Phillips calls "imagery".

A Systems-Based Understanding of Visual Processing, Mental Imagery, and Their Absence

The mammalian visual cortex is a complex and exceptionally well-studied cognitive system, with the basic computations of the visual cortex and neural architectures they are instantiated through being well documented [1], [2], [3][4], and our understanding advancing to a computationally coherent [5], [6] and predictive, if imperfect and incomplete [7], [8], consensus. The larger perceptual system has been more slowly elucidated, often in conjunction with the visual system and through general anatomical and computational terms [1], [9], [10]. This paper aims to achieve two goals. First, we will outline a basic model of a perceptual system, drawing on recent scientific advancements and integrating the model with our current knowledge of how the visual system functions. Second, we will explain how malfunctions in this combined system can lead to the symptoms observed in clinical and psychological studies.

It is important to note that the paper assumes the not at all uncontroversial idea that computations are the sine-qua-non of cognitive processes, and that the biological structures which instantiate these reflect the fact that they do. Due to the significant and wide-spanning debate around such ideas, we do not engage with potential objections on non-empirical grounds until the discussion section, with the exception of footnotes. Additionally, all terms not within the purview of basic introductory neuroscience will be explained in the footnotes[4].

The Anatomy, Structure, and Function of the Perceptual-Visual System

The general structure of the minimal perceptual system we will be working with is composed of a frontal cortical-basal ganglia-thalamic-cortical circuit. This is the main circuit of interest as it is the circuit generally understood [11] to be necessary for basic human activity, directed cognition and perception[12] [5]. This paper also utilizes a relatively simple criterion for whether or not something is "in" perception or not, namely that the sub-system affect and be affected by the entirety of the perceptual circuit. The reasoning for this is the assumed non-controversial statement that everything in perception can affect and be affected by any other thing in perception. If perception were not able to combine constituent percepts together by this standard, the functions of multi-sensory integration and abstract computation would not be possible by definition, since the percept would be fragmented.

Due to the substantial interconnectivity of cortical systems and the small-world structure of the brain, we will attempt to isolate a few representative cortical areas, and simplify their roles. As such we will simplify the cortex to three cortical areas[6], two of which are the compositional representational systems the Anterior Temporal Lobe and Prefrontal Cortex, and the third of which we will cover first, the Visual Cortex[7]. Because we will be speaking about blindsight later on we will also cover atypical and "redundant" pathways.

The Visual System

The visual system naturally begins at the retina, where the initial propagations and signal processing begin. The retina performs a substantial set of initial computations on the signals, including well understood edge-enhancement and movement detection computations [15]. After these initial computations, the

---

[4] As introductory neuroscience is assumed, knowledge certain facts about the neural system, including well known anatomical and computational dynamics are excluded.

[5] There are substantial variations in human cognitive capacities and cerebral organization. Here we first concern ourselves with a typical case, since any damage or developmental aberration will cause compensation, and induce an area typically associated with some function in most people to instead take over another function. That being said, we will cover edge cases in the case study sections, including cases of plasticity in blindsight.

[6] Until our discussion on mental imagery.

[7] It is generally understood that information about any given neural ensemble corresponding to an idea or representation will have information stored about it *across the brain*, in what can be described as a "hub and spoke" configuration. Here we refer to what can be called the informative "central hub". That the ATL is a centrally important structure is evident from lesion and imaging studies [13][14].

propagations continue down their respective optic nerves, combining at the optic chiasm, and then separating into two pathways, the first evolutionarily older pathway being the tectopulvinar pathway, and the second being the geniculostriate pathway. There exist two other pathways which merit mention, since functions relating to those have been cited as evidence in philosophy of mind and psychology literature [16], [17]. Propagations continue down the optic tract and terminate at the pretectum, to control among other things reflexive control of pupils to light, and separately terminate at the hypothalamus, to help regulate circadian rhythms[18].

The propagations going through the tectopulvinar pathway continue from the optic chiasm towards the superior colliculus (a brainstem area responsible for such things as control of eye movements and general "target" tracking using a map-like structure), and then continue to the lateral posterior nucleus, and neighboring pulvinar. The propagations down the lateral posterior nucleus lead to the precuneus [19], superior parietal lobule [20], the temporal lobe [21], and the visual association area [22]. The propagations down the pulvinar separate into the medial and lateral pulvinar nuclei.

Propagations down the medial pulvinar nucleus pass to the cingulate, posterior parietal, motor, and prefrontal cortical areas. Propagations through the lateral pulvinar nucleus go to the early visual cortical areas [23], and the posterior parietal and dorsal stream cortical areas [24]. These pathways are important in articulating the reasons for visual discernment capacity in blindsight [25], [26]. In particular, the pulvinar nucleus and its efferent endpoints are understood to provide non-visual-cortex-mediated visual information to the middle temporal area sufficient for complex tasks. More impressively, this information stream is sufficient for a reportable perceptual evaluation of complex visual stimuli, granted extensive re-wiring in early childhood [27][8].

The geniculostriate pathway, by far the most extensively studied, has signals go down the optic tract towards the lateral geniculate nucleus, and pass through two different white matter pathways which combine and synapse at V1 [29]. From there, ventrolateral propagations move through short white matter tracts to V2, then V3, then V4, and from V4, continue to the fusiform face area, V8, and the posterior inferotemporal cortex [30], [31]. The ventromedial propagations move from V2 to the prestriate cortex[9] and the dorsal transitional visual area. Both signal the ventromedial visual regions, and from there contribute to various diffuse computations both along the ventromedial path and outside of it [30]. This pathway permits the translation of retinotopic visual information, permitting evaluations of texture, color, depth, form, motion, and object presence. Dorsal propagations continue from V3 and V3a to the intraparietal sulcus[10] and then to the dorsal pre-motor areas[11] [32]. This path is responsible for evaluating spatial characteristics and for the execution and evaluation of movements.

The visual system has direct bidirectional connections to the anterior temporal lobe through the inferior longitudinal fasciculus, which allows the anterior temporal lobe to communicate with the visual cortex, primarily V1[33]. Similarly, the visual cortex has direct reciprocal connections to the frontal lobe, including the prefrontal cortex, through the inferior fronto-occipital fasciculus [34]. In order to understand the peculiar lesion-induced effects on the visual system, it is important to understand that both the fasciculus mediated, and the cortical U tract mediated propagations may inform the same endpoints, but provide different information, which is at times redundant or modulatory.

---

[8] It is important to note that the study focused on the middle temporal area, and, while noting that other areas must also be playing a role in the preserved function sans-V1, they did not investigate the lateral posterior nucleus, which is understood to instantiate a retinotopic map in order to facilitate localization of salience, motion, and fear [28].

[9] Broadman's area 18 & 19.

[10] Broadman's area 5.

[11] Broadman's area 6.

Anterior Temporal Lobe (ATL) & Prefrontal Cortex (PFC)

The ATL retains central object representations and the relational structure between said representations[12] [38]. These are sense invariant data structures of objects, concepts, and relationships, linking various sensory features to an abstract, *modality-independent* identity, permitting what we could identify as abstract computational comparisons. In other words, the ATL distills the varied sights, sounds, and other features of an entity into a stable concept, a unified representation not dependent on sensory modalities [39]. That the ATL is critical for semantic memory, our knowledge of objects, people, words, and facts is evidenced by lesion studies demonstrating a lack of semantic capacity in patients[40], [41], [42] and ECoG demonstrations of the ATL is a "heteromodal" hub [43].

In this perceptual circuit, the ATL provides the general abstract content that is experienced, the "what" that is being represented in mind[13]. It is the repository of the current abstract percepts and thoughts, encoded as interconnected semantic[14] representations. For example, perceiving or recalling[15] a dog would activate an ATL ensemble for "dog,"[47], which then broadcasts this content to the PFC [48]. Top-down signals from PFC [49] and modulatory input from the thalamus [50] can bias ATL activity, strengthening certain features or associations while damping others.

The prefrontal cortex (PFC) acts as the brain's affective evaluator and guidance mechanism within the perceptual circuit. The PFC integrates the information coming from the ATL with context about goals, expectations, preferences, and task-relevance. It performs what is in essence a higher-order appraisal, essentially asking of the current pattern of representations at the ATL, "What does this mean for me? Is this good, bad, or neutral?" Neuronal activity in medial [51] and orbital regions of PFC evaluates[16] the value and emotional significance of stimuli [52], [53], while lateral PFC regions incorporate rule-based and goal-oriented processing [54]. The PFC is richly interconnected with sensory, limbic, and memory systems [55], permitting the PFC to compute an evaluation of the current state of the corresponding system [56], [57], drawing on both the abstract content from ATL [58] and afferent evaluations from limbic systems [59], [60].

In sum, **t**he PFC functionally binds representations and evaluates the pattern of representations in the ATL. The PFC then modulates the activity in the ATL [61], [62] and additionally communicates the determination of the overall value and affective status of the pattern of representations in the ATL to the BG [63], [64], [65].

---

[12] Representations here are of the sort typically described as "engrams", or discrete sense-invariant modular neural ensembles corresponding to a particular object or abstract concept. This description is derived from connectomic analyses of mammalian neo-cortex, which demonstrate that propagations through the neocortex take the form of graph-like structures through what can be adequately characterized as a directed hypergraph neural network [35], [36]. Because of the known overlapping structure of object representations and plentiful neural imaging work, it can now be minimally assumed, regardless of architecture, that the temporal lobe, particularly the anterior temporal lobe, does hold sense-invariant object data [37].

[13] It is important to note that other parts can take part in sense invariant relational representations [44], and that the cortex is plastic enough to generally instantiate the same functions in areas other than the anterior temporal cortex. This is what happens in cases of semantic recovery post-lesion.

[14] Here a semantic representation is a representation which has sensory-feature based capacities attributable to its efferent connections to sensory systems, and category and network-based capacities attributable to computations over the overlapping modular neural architecture. So, a neural ensemble is a semantic representation of a thing of this kind insofar as it elicits or is elicited by the particular pattern of activation corresponding to the pattern elicited by direct ascertainment the thing in the sensory modality under consideration.

[15] Recalling the dog is taken here to constitute the top-down instantiation of the "dog" pattern in the relevant sensory modality, in this case the visual modality. This is consistent with the neuroscientific consensus [45] and the substantial majority of neural imaging evidence, including evidence demonstrating the necessity for primary visual cortex in the creation of mental imagery [46].

[16] Based on prior experience and training.

Basal Ganglia (BG)

The BG, treated as a whole, integrates information sent from the PFC along with inputs from the ATL [66] and many other systems [67] to create a measure of how the system currently is, in other words, the "hedonic value" of the system and its trajectory, to the end of dynamically responding to the current state of the system [57], [68]. The BG communicates that measure to the thalamus [69], [70] which subsequently communicates and modulates systems such as the BG itself, the ATL, and the PFC, thereby completing the loop.

Striatum (Part of Basal Ganglia)

The Striatum is the primary sub-system of interest for this examination[17]. The striatum can be seen as constituting the evaluation of the incoming information from the PFC, and creating a single linear evaluation of the overall incoming information, from which the trajectory of hedonic value can be additionally determined . Valence here is grossly a linear measure, instantiated via population code, of "is this good or bad? And by how much?"

Via extensive reciprocal engagement with the cortex, thalamus and other parts of the BG, the striatum instantiates and heavily influences sensory gating, selective attention, and temporal structure [71]. The striatum serves to guide action, and more importantly serves to change the existing dynamics through the thalamus in order to concur with its evaluation of the overall condition of the system [72], [73]. That the striatum affects the overall circuit dynamics through the thalamus is quite important in understanding why recovery of consciousness and therefore perception in coma patients is substantially determined by the persistence or recovery of striatal function [74]. To reinforce its importance, upon ablation of the striatum, stereotyped repetitive behaviors emerge [75], as does the complete cessation of motivated behavior, including basic survival behavior [76].

Thalamus

The Thalamus modulates the cortex at large, including the ATL and PFC and further modulates a great number of other neural systems, changing the dynamics of the overall system to reflect the BG's evaluation of the current state of events, as well as the concurrent computations of a wide swath of neural structures [77], [78].

The thalamus is often described as the brain's central relay station. Here, the thalamus plays an active role in regulating, directing, coordinating, and synchronizing global neural activity [77], and as such is a necessary prerequisite for the direction of perception towards a given modality [77], [79]. In the general perceptual circuit, the thalamus functions as the global context regulator and broadcaster of the valence evaluation computed by the basal ganglia. Once the BG have evaluated the current condition of the system, the thalamus propagates this evaluative result broadly to the cortex [80], ensuring that the entire network updates according to the new information in a context specific manner[18]. Anatomically, certain thalamic nuclei (like the intralaminar and mediodorsal nuclei) have diffuse projections to widespread cortical areas enabling them to enact modulation across the system. Under this system, the thalamus receives the "verdict" from the basal ganglia, a signal that "this is important" or "this is not rewarding", and then broadcasts that signal to influence processing in sensory, associative, and motor regions accordingly [81]. Essentially, the thalamus helps align the subsequent time-step of the system with the evaluative feedback, so that what the cortex represents and computes in the next moment reflects the outcome of the previous moment's appraisal.

---

[17] In the basal ganglia there are a few structures which can partially perform the role of the striatum,

[18] The context specificity of the regulation by thalamus is due to the modality specific computations undertaken at dedicated modality nodes in thalamus.

Generally, the thalamus is known to help prioritize signals among vast sensory input, and here it is biasing the overall dynamic in accordance with the latest value assessment. Under typical conditions, the thalamus works in tight reciprocal loops with the cortex, where cortical feedback to thalamus helps refine what is broadcast [82], creating a dynamic interplay that sustains and modulates percepts.

For perception, the thalamus's contribution is to maintain a space for the coherent integration of global signals [58]. It ensures that when one part of the brain "decides something is important," the rest of the brain's processing reflects or is able to integrate and interact with that decision in the next moment. We don't experience disjointed bits of processing. Thanks in part to thalamic coordination, we experience an integrated whole, with appropriate weight given to what matters most at that time. If the ATL's role was content and the BG's role was value, the thalamus helps weave those together into a unified perspective, updating the entirety of the perceptual system continuously.

Integration and Significance

Each of the above components performs a distinct computation, and perception arises from their integrated activity. The ATL provides the semantic and relational content of "what is represented," the visual cortex provides the raw information that is processed by several modalities and then passed onto the ATL semantic hubs. PFC attaches interpretive and goal-oriented meaning, or "what it means for me", the BG assigns an affective valence, "good or bad", and the thalamus and hippocampus feed those results back to modulate and contextualize the subsequent timestep of perception.

This looping architecture is self-referential[19] and continuously updating. The brain is, essentially, constantly evaluating its own representations, the changes therein and then modulating itself based on that evaluation. By understanding the roles of each node, ATL (representation), PFC (evaluation), BG (valuation), and Thalamus (global broadcasting), this minimal perceptual system offers a mechanistic account of the functions and interplay which at least partially underlie perception. Each region's contribution is indispensable[20] in this view, as together they form the "core circuit" which is necessary for perceptive processing of the sort that is experientially accessible, as evidenced by conditions where parts of the circuit do not get processed in perception, in which there are invariably lesions or anomalies affecting some part of this circuit.

By simplifying the vast complexity of brain activity into this circuit of "key players", this minimal circuit aims to make perception comprehensible in terms of concrete interactions among well-known brain systems. More importantly, the theoretically delineated circuit maps closely to our current understanding of the necessary clinical structures for experienced perception, especially visual perception. As we will cover in the next section.

Mental Imagery, Aphantasia, & Blindsight: Lesions & Lessons

Aphantasia and blindsight are very dissimilar conditions, which share only the fact that they are conditions affecting the visual system. In this section, we will put forward a set of proposals which will permit us to speak of the visual system in functional terms. The first and most important proposal is that of the existence, nature, and use of neural ensemble binding and the binding of reportable percepts. After going

[19] This simply means that there are computational recursive elements.

[20] It should be evident that in lesion cases, especially cortical lesion cases, plastic processes will recruit other parts of the brain to complete the computations that the lesion has damaged. That the brain can accommodate lesions is assumed, the important thing is that the information is being processed in the characteristic way. So, if you can replace anterior temporal lobe with posterior temporal lobe after lesion, what is important isn't that the brain area labeled "anterior temporal lobe" is no more, but that the function and set of computations which were once instantiated by the brain area labeled "anterior temporal lobe" continue to be computed, albeit by a brain area differently labeled.

over the proposed binding mechanisms, we will provide a general functional account of visual perception, then of mental imagery, and finally of aberrations in said functioning. Throughout we will appeal to lesion studies, anatomical analyses, and arguments in philosophy of mind.

Binding

There are two principal kinds of binding that we are concerned with here, visual feature binding, and binding of percepts. Feature binding refers to the ability to connect information from differing perceptual modalities into a single coherent object. Broadly, the feature binding problem is the question of how brains turn multiple complex sensory inputs into discrete and recognizable objects [83]. The binding of percepts is a greater question, which requires discussion of consciousness and the unity of consciousness. Fundamentally the question is how do the various neural processes construct the "phenomenal object"[84]. Given that there are many neural mechanisms and processes, widely distributed in the brain, how does a unified percept come to be?[85]

Because the debate and history around binding is extensive, here we will attempt to use a minimally controversial account of visual feature binding, and then gradually move to a tentative description of binding at the phenomenal level. There is a relatively simple manner of binding together neural ensembles which will work well for us in describing both local and distal neural ensemble binding, that being obligate reciprocal activation of neural ensembles. What this means is that when one ensemble fires, the other ensemble also fires, and vice versa. In other words, there is *functional co-activation* of the neural ensembles in question. How these ensembles may come to be functionally bound in this manner can be considered an open question, and one which does not necessarily need to be answered here[21].

Still, to develop the intuition for the visual system, an explanation is required. The simplest answer is that of continual reciprocal activation, where a pattern in a given sensory modality elicits the activation of a corresponding neural ensemble. The corresponding representative neural ensemble then sends a propagation down to activate that same pattern, and vice versa. Simulations have demonstrated that a neural system equipped with the machinery for functional co-activation is sufficient to combinatorally compute from multiple modalities [86]. That reciprocal or recurrent pathways and dynamics are important to top-down modulation is of course not a new assertion [87], [88]. The neurophysiological evidence has only recently demonstrated that functional co-activation of this kind, modulated and coordinated by thalamic oscillations, and spread across sensory modalities in the retrieval of memories, exist and are central to function [89].

The question is then asked, what does this do for the neural system? The desirability of such a mechanism is three-fold. First, the relative permanence of the signal will be extended, allowing for further computations to take place using that extended signal [90], [91], [92]. Second, the continual activation in this particular manner ensures that the neural ensemble adequately and accurately corresponds to the pattern it is meant to respond to. If, for instance, the primary visual system sends a signal that is substantially different from the dynamic previously active, then one expects a neural response to ensure that does not happen continuously. Such a disparity would further induce the activation of one of the numerous error-detecting signals in the brain, in this case a visual mismatch negativity signal [93], [94]. Third, and closely related to the second point, recurrent processing could enable the neural system to anticipate or predict stereotyped inputs, by instantiating a learned procession or biasing against a non-learned procession, through the continued temporal evolution of the dynamic. As one might expect, this particular mechanism and processing dynamic has been validated in mice [95], and has demonstrable further utility in delineating stimulus features in mammalian brains [96].

[21] For what we require, that things are bound and that communication between the bound ensembles requires communication, even if indirect, is sufficient for our purposes.

Returning to the question of binding across scales, this local or even distal [97] mechanism may not be sufficient to explain the overall unity of perception, which we may[22] wish to mechanistically explain if we would like to examine what does or does not make a given percept amenable to perceptual report. In order to answer that question, we must reconsider what we are talking about when we talk about a percept. One can contend that a percept is not a state or property of a given neural system, but is instead a process that is instantiated through a neural system.

The explananda of the brain we have recapitulated thus far has focused primarily on mechanisms and propagations through networks, and as such has contained within it an undercurrent of process ontology[23]. If we are to take seriously the idea that perception, much like life [98], [99], could be most coherently and elegantly understood as a process that something performs or *is*, and not a property that something has, then we can begin to delineate precisely what the perceptual process is, and how it can go awry. That is what we are going to do in the following section. As for how binding occurs at the scale of the entire percept; we contend that, given perception is a process, the entirety of the constituent parts of any given process, in order to be a part of said process, must affect the dynamics of the entire process under consideration and must be similarly affected by the entirety of the system [100].

Mental Imagery and Visual Perception

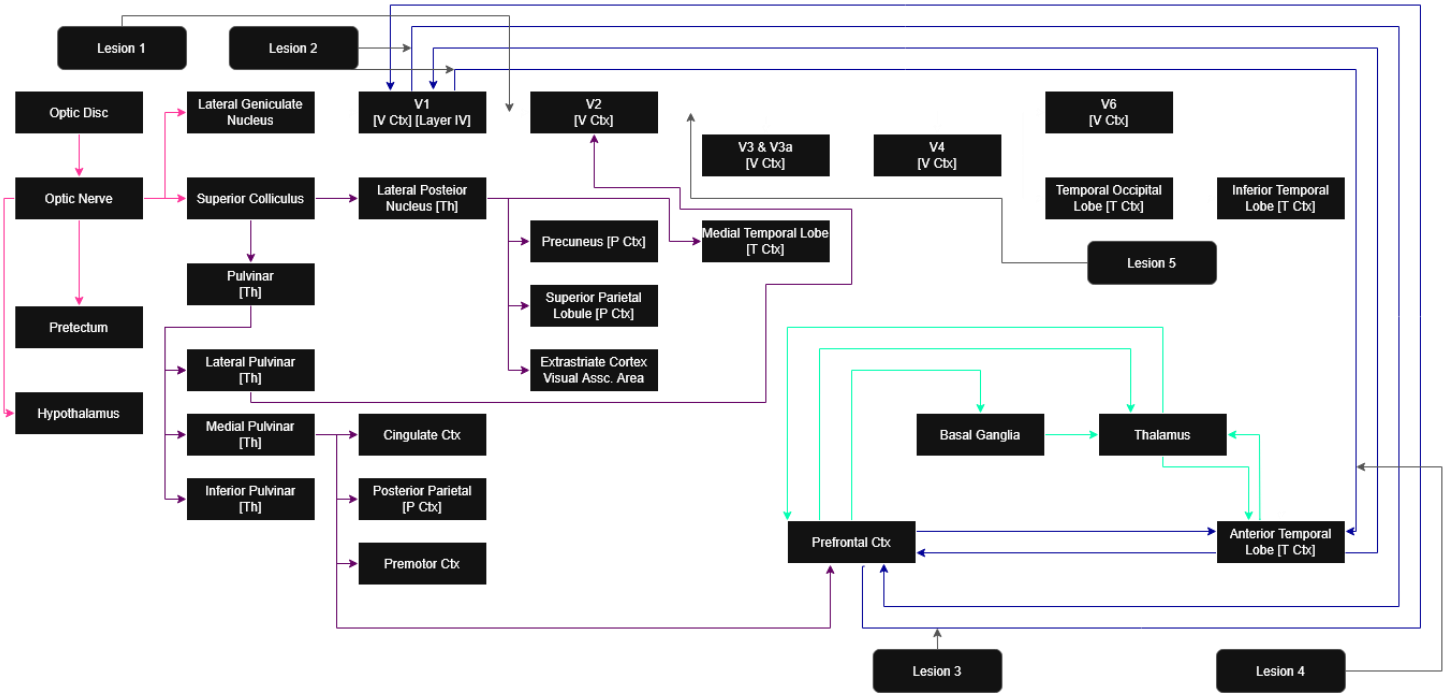

Figure 1: A partial diagram of the perceptual system as relevant to visual perception. In light green is the central circuit, consisting of the prefrontal cortex, anterior temporal lobe, basal ganglia, thalamus, and connections between them. In blue are the bidirectional fasciculi. In purple are the constituent parts of the tectopulvinar pathway. In pink are the direct projections of the optic disc and nerves. In white are the ventral and dorsal visual pathways. In grey are the lesions we will be examining. Abbreviations are as follows; V Ctx: Visual Cortex, Th: Thalamus, P Ctx: Parietal Cortex, T Ctx: Temporal Cortex, Ctx: Cortex.

[22] Once again, that we can say things are bound in some manner across modalities is sufficient for our purposes, since binding of any given set of neural ensembles would permit us to speak about whether or not something can be bound according to lesion studies.

[23] The author confesses that they are sympathetic to dynamical systems theory, which can be described quite adequately as process ontology with computers. That being said, this is not evangelization for Whitehead.

We will begin by examining Mental Imagery, synthesizing all we have covered in order to come to an understanding of this system, assuming a neurotypical adult. A propagation begins at the dorsolateral prefrontal cortex (dl-PFC). The dl-PFC will maintain the "goal" of the mental object to be imagined, and will help maintain the particular desired dynamic coherent and "active" until the task ends [101][102][24]. The dl-PFC signal passes through the superior longitudinal fasciculus III (SLFIII) and is evaluated at Inferior Frontal Gyrus [103]. The Left Inferior Frontal Gyrus (LIFG) acts as a kind of "switch"[25] which, once modulated by the dl-PFC signal, leads to a shift in global dynamics from domain general (non-specific) executive processing, to domain-specific processing, and from perceptually driven processes to higher-order semantic driven processing.

This shift permits the propagation from the dl-PFC to make its way the IFG , and subsequently into the anterior temporal lobe (ATL) through a ventral fronto-temporal white matter tract [26]. This representation then sends a propagation through the visual areas, through the Inferior Longitudinal Fasciculus, terminating at the extrastriate cortex. At this point, feed-back projections translate the representation into a pattern of activation "projected" onto V1. The resulting feedback into the dl-PFC and PFC from the ATL via ventral and dorsal pathways permits information to have efferent effects on the rest of the system, to the degree that it affects the rest of the system and so is computed across the system and therefore reportable. As time passes, direct communication through the ventral fronto-temporal white matter tract permits the desired configuration at PFC to continually guide the configuration at the ATL to the configuration that is required. The projection at visual cortex and the concurrent feedback as mediated by the inferior fronto-occipital fasciculus permits rapid communication and additional evaluation of the success or failure of the endeavor.

The propagation has now resolved into a pattern in visual cortex, and is communicated and kept active through rapid, bidirectional feedback carried along the inferior fronto-occipital fasciculus (IFOF). This IFOF mediated flow permits the frontal cortex to monitor and refine the image in real time and to evaluate whether the imagery goal has been met.

Case Studies

To briefly recapitulate, we now have a tentative system by which the frontal cortex sets a goal, for example, the creation of a mental image of an apple. This apple goal is communicated across cortex and leads to a shift in global processing, which then enables the ATL to project to the extrastriate visual systems a general apple pattern, which, through reciprocal and feed-back processing, resolves into an image which is then evaluated by the ATL and PFC. If all has gone well, we now have a continuing visually ascertained apple percept in our neural system, and insofar as that dynamic holds, so will the imagined apple.

What makes the apple percept an apple percept and not just apple-flavored information is that the apple, which our neural system is still imagining, is having a continual effect on the rest of the neural system. Your visual field now has an apple in it, with texture and maybe even feeling. If so inclined, you will be faster to think apple-y thoughts, and to ascertain an apple in a crowd of things that would occlude an apple [105], [106], [107]. Mental imagery does have a functional role to play in human cognition. As such we will continue

[24] The continual maintenance of the dynamic between the relevant parts is one instantiation of working memory.
[25] There is a dual gradient. The first is the gradient from anterior to posterior, where rostral LIFG supports abstract contextually-modulated processing, and caudal LIFG which supports linguistic and salience driven processing. The second is dorsal to ventral, where the superior LIFG supports broad frontal dynamics, and the inferior LIFG supports semantic and episodic processing [104], [105].
[26] This tract is either the uncinate fasciculus or the extreme capsule. There is contemporary debate on which particular tract actually propagates the signal [61], [104]. That there exists a ventral fronto-temporal white matter tract, and that the IFG and ATL communicate through it is undisputed. Given that not a terrible amount of computation happens in white matter, we find the particular modality to be, for our present purposes, non-consequential.

to treat visual perception as a process that accomplishes tasks as we examine our cases of aberrant visual perception[27].

Blindsight

Blindsight is present when a person reports a lack of visual perception, but is nonetheless capable of exhibiting sighted behavior under certain circumstances. Typically, this condition arises from a lesion or set of lesions leading to a lack of visual processing at V1, the striate cortex. Function in blindsight is dependent on structures such as the hypothalamus, pretectum, and the tectopulvinar visual pathway. The reason that people with blindsight retain the ability to act on visual information is entirely due to the fact that parts of their visual system remain [110].

A person with blindsight is capable of navigating spaces due to the retention of a spatial location containing targeting system localized in the superior colliculus [110]. Pulvinar tracts through the thalamus permit signaling of visual information into a number of cortical areas, including but not limited to the extrastriate cortex, the premotor cortex, the cingulate cortex, and the posterior parietal cortex [24]. Because of the lack of extensive integration of these visual systems into the larger cortical system, these remaining neural structures can only really nudge things in one way or another, given that there is no modality for greater interaction.

The tectopulvinar pathway, and certainly the pulvinar structures themselves demonstrate rigid feed-forward behavior [111], which makes full functional integration unfeasible. Returning to our framework and criteria for integration into perception, that this pathway generally feeds information forward indicates that it is not liable to take part in perception, as it cannot be reasonably affected by the rest of the dynamic. The remaining tectopulvinar visual system simply sends signals, and biases outcomes at the cortical efferents.

Moving on to the explication of obstacle avoidance capacity, the fact that two maps are going to be present in blindsight patients is going to permit a lot of function, especially if one informs the other. The first of these two maps is the aforementioned tectopulvinar target tracking map. The second is the entorhinal cortical map [112]. That a blindsight patient is able to navigate a room is not a great surprise considering that the reciprocal connections between the entorhinal cortex, which mediates spatial navigation, and the medial pulvinar, which integrates information from the remaining visual circuit into the entorhinal cortex [113]. These reciprocal connections permit the translation of the aforementioned visual tract into spatial circuits, which then drive the motor effects, biasing the blindsight patient to move one way or the other as one normally would if those were visually ascertained.[28]

Turning to the forced-choice experiments. The existence of bias is common to human experience. We see the afferent information from the tectopulvinar pathway to be a biasing factor in judgements and actions. Some philosophers of mind have taken blindsight to be a case of "perception without

[27] There does exist evidence of "mental imagery" in a person who is described as being incapable of ascertaining visually the identity of an object, but did report the capacity to imagine objects, and was able to draw the same. This has been taken to mean there is a double dissociation between perception and imagery [108]. This is not the case. Those kinds of cases are examples of highly localized deficits in a particular pathway, here the one leading from V1 to V4 to Anterior temporal lobe, while the direct pathway, the inferior longitudinal fasciculus, remained unharmed. Damage is outlined here [109], the location of the lesion is evidenced by lack of color perception in the patient. She had focal symmetrical lesions at areas 18 and 19. As such, she was able to recruit the rest of the imagery system and utilize V1 through the direct line, and the other areas through the standard mental imagery pathway, in such a manner as to functionally complete and integrate the circuit. When she opened her eyes, the top-down induction of color and shape was no longer available, since she wasn't purposefully adding it in. This example demonstrates one of the benefits of the systems approach to cognition, tying form (anatomy) to function to experience.

[28] We recognize that some people may have to consciously evaluate where things are. Nonetheless, we assume for the most part that the experience of not having to think about avoiding obstacles and nonetheless avoiding them is typical.

consciousness", or constitutive of "degraded vision" [16], [114]. Let us address those cases here. There are very evidently blindsight cases in which visual functions are retained, and there are cases where optic-nerve mediated information is sufficiently integrated into another modality as to permit the affected person to *feel* that information. They do not report this to be *visual* information, on account of having lost said modality. What is required for a visual percept to take place is for the systemic computational and functional roles to be fulfilled. What you need is a grid structure and a mapping of visual information onto that grid in a retinotopic manner.

Retinotopic or simply regular grid structure is not restricted to V1 [9], so one may ask *why it is the case that those maps don't become integrated into vision,* since, in blindsight cases, there remains visual information, and there are multiple map-like configurations in the brain as previously mentioned [29]. The answer is that they do.

B.I. lost his occipital lobes and parts of the parietal lobes at two weeks old, due to complications of a genetic issue. At age six, B.I. was able to adequately report his surroundings, identify colors, expressions, and generally navigate the world. Patient B.I.'s lesions can be characterized as a functional equivalent to the combination of lesions 1 and 5 in figure 1.

It should be apparent that there are a lot of projections from the optic system into cortex. The question is not one of having visual information, it is of integrating it properly. Most blindsight patients become blindsight patients in their later years, and as such have not tended to recover. The difference between B.I. and other blindsight patients is evident. The same pathways exist, but B.I.'s brain, due to the exceptional plasticity available in infancy, was able to accommodate the visual information. In order to make this information workable the brain had to change drastically, as is evidenced by the substantial growth in B.I.'s medial temporal volume [27].

B.I. is an interesting case because, despite his vision, he still exhibits some blindsight behavior which may be instructive to us. B.I. cannot see how thick something is, but when he reaches for a given thing, he always reaches in such a manner as to accommodate the object. He does this unconsciously. The tectopulvinar pathway in typical circumstances is responsible for reaching behavior. And in B.I., it continues that role, while taking on a few afferent roles as well. That part of the circuit functions on its own, in complex but regimented ways. It is in essence a kind of reflex or control system that can be employed but cannot be ascertained directly.

Because the processing is automatic, feedforward, and independent of cognition, blindsight appears to be more akin such to an involuntary reflex than to "unconscious perception", or visual perception too subtle to note. It is important to note that people can walk purely on reflexes adequately trained, it is a manner of maneuvering and fitting to task those reflexes. If blindsight is akin to a reflex, we must also note that there is no belief that one instantiates when a doctor hits one's knee and one's leg moves a bit, it is simply a thing that happens in a certain circumstance.

Aphantasia

A person with aphantasia is incapable of creating mental imagery. While the cutoffs are variable between studies, here we will primarily concern ourselves with cases of aphantasia resulting in absolutely no mental imagery, and will further consider a case of acquired aphantasia. There has been a growth of interest in

[29] The map structure is important, since a map structure would minimally retain the computational structure needed for visual processing.

aphantasia by the philosophical community, as question which has persisted in philosophy of mind [115], [116], [117], the perennial argument over images and semantics[30].

Aphantasia cannot be said to be a distinction without a difference, and does induce quantifiable cognitive differences. People with aphantasia are not able to better find an apple in an apple-occluded search task, and respond differentially to fear-inducing verbal reports[119]. Aphantasia can arise from a multitude of causes. However, in general, what has occurred is some variation of Lesion 3 in figure 1. The top-down mechanisms that we outlined prior are not capable of eliciting the desired pattern through that, or any other, pathway.

This does not have to be caused by an actual lesion, all that has to occur is that the function does not run. Put another way, in people with aphantasia, "feedback originating from frontal brain regions fails to excite activity in earlier brain structures that generate imagined sensory experiences."[120] In people with aphantasia, this can occur because of higher baseline activity in visual cortex [121], lower binding or entraining of dynamics [122], or reduced binding between the visual and frontal cortices [123]. The common pathway is simply the inability to elicit a pattern in the visual cortex. That does not mean however, that people with aphantasia cannot modulate *any* neural patterns, but it means that they cannot modulate the *visual* modality.

In describing what aphantasia is, one must also describe what it is not. A recent study proposed that a subset of aphantasic individuals may lack the metacognitive awareness of mental imagery, but may still be utilizing mental imagery itself. This assertion was based on the fact that, as a whole, people with aphantasia in this study performed on par with their control counterparts in a 3-d computer game where one had to familiarize oneself with a set of objects their position, then solve a distractor and reproduce the configuration. The idea being that in order to integrate the various perspectives into a unified whole, mental imagery could be necessary.

The authors do state that the people with aphantasia could have been using another method, and it does seem very plausible that one could keep either a verbal conceptualization at hand of the configuration, or could have utilized sensorimotor modalities to accomplish the same. If that is the case, then we have succeeded in demonstrating that aphantasics can use other modalities, on par with the controls, and not much else.

Now we move to patient PL518, or the architect. The architect lost the ability to perform mental imagery after a bilateral posterior cerebral artery stroke. The architect lost significant portions of the occipital pole, lingual gyrus, the entire fusiform gyrus, with additional damage extending anteriorly to the parahippocampal region, on his right. On his left, he saw damage to the medial fusiform gyrus (and underlying white matter) and lingual gyrus. The damage on his left, although seemingly less severe, caused his aphantasia.

The extensive damage to grey matter in the brain areas which were affected were not the issue[31], it was the white matter damage under the left medial fusiform gyrus that prevented the process from happening. Fibers from the Inferior Longitudinal Fasciculus pass through the fusiform gyrus [124]. If we return to the basic pathway above, the propagational path from the ATL, the part of the brain with the object representations, to the extrastriate or visual cortex passes through the Inferior Longitudinal Fasciculus. The damage took out the tract enabling the ATL to communicate with the visual cortex, and so the ATL didn't pass along the representation to be projected onto V1, and so the architect couldn't imagine.

---

[30] There are also epistemological issues which have to do with what we are justified in believing, and if percepts are veridical, but those concerns are better addressed elsewhere [118].

[31] The grey matter damage outlined would have had no effect on the particular dynamic we are interested in, mental imagery, since the lesions simply didn't affect the visual circuit we have been outlining directly.

Discussion

We have outlined an empirically based model of visual perception[32] and mental imagery, and have further examined how the localized disruption of this system can lead to blindsight and aphantasia. With this framework in hand, we now turn to some conceptual and empirical issues, including alternative interpretations of these phenomena and their broader implications.

A familiar debate is whether the deficits in blindsight and aphantasia involve genuinely unconscious processing or instead reflect a degraded form of conscious perception. In particular, Phillips has argued that blindsight may constitute a qualitatively diminished kind of visual consciousness. We embrace Phillips' call to consider the full complexity and variability of human cognition, recognizing that perceptual experiences can differ substantially depending on the underlying system. However, we contend that these cases of blindsight and aphantasia are best understood as instances of function separate from cognition in the blindsight case, and a mechanism disabled for the aphantasia case. This is more parsimonious with the empirical narrative. To layer an additional minimally perceived thing onto what seem to be clear cut cases of brain disorders seems unnecessary[33].

In blindsight, a person with V1 damage reports no visual experience yet can respond to visual stimuli above chance. Some philosophers would similarly interpret this as "perception without consciousness" or a severely degraded form of vision. The alternative view posits that the person might have faint visual qualia that they fail to recognize as sight. If that were true, blindsight would not be a purely unconscious process but a subtle form of conscious vision. Our analysis and the evidence weigh against this interpretation.

Blindsight related behavior appears to be driven by residual visual pathways which, independent of any substantial interaction, appear more like reflexes or biasing signals than conscious perception. The person's accurate "guesses" on forced choice tests are made without any accompanying visual feeling, often even without an intuition that they are receiving information. That is Type 1 blindsight. In Type 2 blindsight, people sometimes report a vague feeling or guess that *something* has happened, but crucially they do not describe any visual qualia. These reports suggest that whatever information is guiding behavior is not entering the conscious visual experience, and that it is not necessary for it to enter consciousness to have an effect. It is not immediately apparent why we would expect it to be a conscious influence.[34]

Having addressed some alternative interpretations and touched upon our philosophical framing, we can now highlight points of consensus, limitations of our account, and promising directions for future inquiry. First, there are important areas of agreement between our view and other perspectives. We concur with Phillips and colleagues on the importance of rejecting overly simplistic models of mind. Blindsight and aphantasia indeed demonstrate that human cognition is variable and that simple one size fits all theories of vision or imagery are insufficient. We have heeded Phillips' insight that different kinds of perceptual states exist, distinguishable by their neural underpinnings. Our analysis explicitly distinguishes normal seeing, blindsight "seeing," and imagined seeing as three distinct kinds dynamics and processes that should not be conflated. We also agree with the long-standing neuroscientific consensus that visual processing is distributed across multiple pathways.

---

[32] The process for visual perception and ascertainment of a scene or object is exactly the same, down to the reciprocal dynamics, but without the LIGF switch and ensuing cascade.

[33] That there are people who qualify for aphantasia who are able to use faint visual imagery to complete tasks does not seem at all unlikely. That aphantasia in general utilizes unconscious or minimally conscious faint imagery however does not arise from that.

[34] That being said, there are likely people who maintain parts of the visual cortex such that they are capable of perceiving some imagery. These are cases of partial blindsight, and do not imply people with blindsight are using some sort of unconscious or very faint consciously perceived (but not reported) visual processes.

With these agreements noted, we must also confront the limitations of our approach. Our proposed model of the visual system and perceptual awareness is deliberately simplified, omitting many details of real consequence. The human brain's visual pathways are enormously complex, involving endless feedback loops and interactions with other systems. While we identified key players (V1, thalamus, PFC, ATL, BG) and pathways (like the ILF and pulvinar routes), this is not an exhaustive map of visual consciousness, and should not be seen as such. For instance, we have not discussed in depth the role of the hippocampus in eliciting information from memory to select ATL ensembles.

Moreover, blindsight and aphantasia themselves vary between individuals. There are degrees of blindsight, and this account did not engage with that complexity. Our account focused on broad commonalities and "clean" interpretations, but that does not adequately speak to the experimental realities. Similarly, aphantasia exists on a spectrum, and we focused on an extreme, zero imagery. Many people have weak imagery rather than none, and in those people it is very possible that they utilize partial maps in order to complete rotation tasks. Our model doesn't explicitly address those intermediate cases.

Another limitation is that we primarily considered steady-state lesions or lifelong conditions. We have not delved into dynamic fluctuations, such as transitions in excitability or hallucinations[35]. These related phenomena could further test our model's generality. On the philosophical side, we have explicitly set aside the key question, why do certain neural integrations produce an experience in the first place? We have described mechanistic correlations and necessities but we have not provided a theory of why those neural activities feel like anything. Lastly, the explanatory framework we propose will need to be continually tested and refined. As new science emerges some details of our model will require adjustment. We acknowledge that our current explanation is not definitive and alternative models exist. In summary, while we are confident our systems-based account captures the essence of these phenomena, it is by no means the final word on the subject.

Looking ahead, there are several directions for future research that emerge from this work or in conjunction with it. One line of inquiry is empirical. Hypoxic reperfusion shock is a substantial cause of permanent brain damage, and the mechanisms for it are not entirely understood, and would assist in understanding lesion patterns in patients with disordered perception. On the theoretical front, further integration with parallel bodies of work, including upcoming conference projects will be undertaken. Finally, a substantial omission from this work was the use of the visual system in the blind, in particular, the existence of visual imagery within the congenitally blind.

In conclusion, by adopting a systems-level framework grounded in anatomical and computational neuroscience, we have shown how seemingly disparate phenomena blindsight and aphantasia can be understood as deficits in integration within a core perceptual loop. This account demonstrates how perception depends on reciprocal dynamics among key cortical and subcortical systems, and offers the beginning of a framework for further research into perceptual cognition.

[35] Paper is in the works on LSD.

Works Cited

[1] D. J. Heeger, E. P. Simoncelli, and J. A. Movshon, "Computational models of cortical visual processing," *Proc. Natl. Acad. Sci. U. S. A.*, vol. 93, no. 2, pp. 623–627, Jan. 1996, doi: 10.1073/pnas.93.2.623.

[2] J. A. Bae *et al.*, "Functional connectomics spanning multiple areas of mouse visual cortex," *Nature*, vol. 640, no. 8058, pp. 435–447, Apr. 2025, doi: 10.1038/s41586-025-08790-w.

[3] Y. N. Billeh *et al.*, "Systematic Integration of Structural and Functional Data into Multi-scale Models of Mouse Primary Visual Cortex," *Neuron*, vol. 106, no. 3, pp. 388-403.e18, May 2020, doi: 10.1016/j.neuron.2020.01.040.

[4] G. K. H. Zupanc, "Mapping brain structure and function: cellular resolution, global perspective," *J. Comp. Physiol. A*, vol. 203, no. 4, pp. 245–264, Apr. 2017, doi: 10.1007/s00359-017-1163-y.

[5] J. H. Siegle *et al.*, "Survey of spiking in the mouse visual system reveals functional hierarchy," *Nature*, vol. 592, no. 7852, pp. 86–92, Apr. 2021, doi: 10.1038/s41586-020-03171-x.

[6] R. D. D'Souza *et al.*, "Hierarchical and nonhierarchical features of the mouse visual cortical network," *Nat. Commun.*, vol. 13, no. 1, p. 503, Jan. 2022, doi: 10.1038/s41467-022-28035-y.

[7] D. Deitch, A. Rubin, and Y. Ziv, "Representational drift in the mouse visual cortex," *Curr. Biol.*, vol. 31, no. 19, pp. 4327-4339.e6, Oct. 2021, doi: 10.1016/j.cub.2021.07.062.

[8] K. J. Jeffery, "The mosaic structure of the mammalian cognitive map," *Learn. Behav.*, vol. 52, no. 1, pp. 19–34, Mar. 2024, doi: 10.3758/s13420-023-00618-9.

[9] R. A. Epstein, E. Z. Patai, J. B. Julian, and H. J. Spiers, "The cognitive map in humans: spatial navigation and beyond," *Nat. Neurosci.*, vol. 20, no. 11, pp. 1504–1513, Nov. 2017, doi: 10.1038/nn.4656.

[10] E. T. Rolls, G. Deco, C.-C. Huang, and J. Feng, "Multiple cortical visual streams in humans," *Cereb. Cortex*, vol. 33, no. 7, pp. 3319–3349, Apr. 2023, doi: 10.1093/cercor/bhac276.

[11] L. Weng *et al.*, "Abnormal structural connectivity between the basal ganglia, thalamus, and frontal cortex in patients with disorders of consciousness," *Cortex*, vol. 90, pp. 71–87, May 2017, doi: 10.1016/j.cortex.2017.02.011.

[12] S. N. Haber and R. Calzavara, "The cortico-basal ganglia integrative network: the role of the thalamus," *Brain Res. Bull.*, vol. 78, no. 0, pp. 69–74, Feb. 2009, doi: 10.1016/j.brainresbull.2008.09.013.

[13] G. Elumalai *et al.*, *Detecting Diffusion Imaging constructive connectivity analysis for What stream Visual Pathways with correlations to Visual Agnosia*. 2018. doi: 10.1101/486639.

[14] F. M. Richardson, M. L. Seghier, A. P. Leff, M. S. C. Thomas, and C. J. Price, "Multiple Routes from Occipital to Temporal Cortices during Reading," *J. Neurosci.*, vol. 31, no. 22, pp. 8239–8247, Jun. 2011, doi: 10.1523/JNEUROSCI.6519-10.2011.

[15] T. Gollisch and M. Meister, "Eye smarter than scientists believed: Neural computations in circuits of the retina," *Neuron*, vol. 65, no. 2, pp. 150–164, Jan. 2010, doi: 10.1016/j.neuron.2009.12.009.

[16] I. Phillips, "Blindsight is qualitatively degraded conscious vision," *Psychol. Rev.*, vol. 128, no. 3, pp. 558–584, 2021, doi: 10.1037/rev0000254.

[17] A. Sahraie, L. Weiskrantz, C. T. Trevethan, R. Cruce, and A. D. Murray, "Psychophysical and pupillometric study of spatial channels of visual processing in blindsight," *Exp. Brain Res.*, vol. 143, no. 2, pp. 249–256, Mar. 2002, doi: 10.1007/s00221-001-0989-1.

[18] "Visual Processing: Cortical Pathways (Section 2, Chapter 15) Neuroscience Online: An Electronic Textbook for the Neurosciences | Department of Neurobiology and Anatomy - The University of Texas Medical School at Houston." Accessed: May 12, 2025. [Online]. Available: https://nba.uth.tmc.edu/neuroscience/m/s2/chapter15.html

[19] M. Gamberini *et al.*, "Thalamic afferents emphasize the different functions of macaque precuneate areas," *Brain Struct. Funct.*, vol. 225, no. 2, pp. 853–870, Mar. 2020, doi: 10.1007/s00429-020-02045-2.

[20] L. Passarelli, M. Gamberini, and P. Fattori, "The superior parietal lobule of primates: a sensory-motor hub for interaction with the environment," *J. Integr. Neurosci.*, vol. 20, no. 1, p. 157, Mar. 2021, doi: 10.31083/j.jin.2021.01.334.

[21] M. Catani, "Chapter 1 - The connectional anatomy of the temporal lobe," in *Handbook of Clinical Neurology*, vol. 187, G. Miceli, P. Bartolomeo, and V. Navarro, Eds., in The Temporal Lobe, vol. 187. , Elsevier, 2022, pp. 3–16. doi: 10.1016/B978-0-12-823493-8.00001-8.
[22] R. N. Ramesh, C. R. Burgess, A. U. Sugden, M. Gyetvan, and M. L. Andermann, "Intermingled Ensembles in Visual Association Cortex Encode Stimulus Identity or Predicted Outcome," *Neuron*, vol. 100, no. 4, pp. 900-915.e9, Nov. 2018, doi: 10.1016/j.neuron.2018.09.024.
[23] R. A. Berman and R. H. Wurtz, "Signals Conveyed in the Pulvinar Pathway from Superior Colliculus to Cortical Area MT," *J. Neurosci.*, vol. 31, no. 2, pp. 373–384, Jan. 2011, doi: 10.1523/JNEUROSCI.4738-10.2011.
[24] N. Cortes, H. J. Ladret, R. Abbas-Farishta, and C. Casanova, "The pulvinar as a hub of visual processing and cortical integration," *Trends Neurosci.*, vol. 47, no. 2, pp. 120–134, Feb. 2024, doi: 10.1016/j.tins.2023.11.008.
[25] R. T. Born and D. C. Bradley, "STRUCTURE AND FUNCTION OF VISUAL AREA MT," *Annu. Rev. Neurosci.*, vol. 28, no. 1, pp. 157–189, Jul. 2005, doi: 10.1146/annurev.neuro.26.041002.131052.
[26] I.-C. Mundinano *et al.*, "More than blindsight: Case report of a child with extraordinary visual capacity following perinatal bilateral occipital lobe injury," *Neuropsychologia*, vol. 128, pp. 178–186, May 2019, doi: 10.1016/j.neuropsychologia.2017.11.017.
[27] I.-C. Mundinano *et al.*, "More than blindsight: Case report of a child with extraordinary visual capacity following perinatal bilateral occipital lobe injury," *Neuropsychologia*, vol. 128, pp. 178–186, May 2019, doi: 10.1016/j.neuropsychologia.2017.11.017.
[28] A. E. Allen, C. A. Procyk, M. Howarth, L. Walmsley, and T. M. Brown, "Visual input to the mouse lateral posterior and posterior thalamic nuclei: photoreceptive origins and retinotopic order," *J. Physiol.*, vol. 594, no. 7, pp. 1911–1929, Apr. 2016, doi: 10.1113/JP271707.
[29] F. Briggs and W. M. Usrey, "A Fast, Reciprocal Pathway between the Lateral Geniculate Nucleus and Visual Cortex in the Macaque Monkey," *J. Neurosci.*, vol. 27, no. 20, pp. 5431–5436, May 2007, doi: 10.1523/JNEUROSCI.1035-07.2007.
[30] D. J. Kravitz, K. S. Saleem, C. I. Baker, L. G. Ungerleider, and M. Mishkin, "The ventral visual pathway: An expanded neural framework for the processing of object quality," *Trends Cogn. Sci.*, vol. 17, no. 1, pp. 26–49, Jan. 2013, doi: 10.1016/j.tics.2012.10.011.
[31] E. T. Rolls, "Two what, two where, visual cortical streams in humans," *Neurosci. Biobehav. Rev.*, vol. 160, p. 105650, May 2024, doi: 10.1016/j.neubiorev.2024.105650.
[32] H. Takemura, F. Pestilli, and K. S. Weiner, "Comparative neuroanatomy: Integrating classic and modern methods to understand association fibers connecting dorsal and ventral visual cortex," *Neurosci. Res.*, vol. 146, pp. 1–12, Sep. 2019, doi: 10.1016/j.neures.2018.10.011.
[33] G. Herbet, I. Zemmoura, and H. Duffau, "Functional Anatomy of the Inferior Longitudinal Fasciculus: From Historical Reports to Current Hypotheses," *Front. Neuroanat.*, vol. 12, Sep. 2018, doi: 10.3389/fnana.2018.00077.
[34] A. K. Conner *et al.*, "A Connectomic Atlas of the Human Cerebrum—Chapter 13: Tractographic Description of the Inferior Fronto-Occipital Fasciculus," *Oper. Neurosurg.*, vol. 15, no. Suppl 1, pp. S436–S443, Dec. 2018, doi: 10.1093/ons/opy267.
[35] M. W. Reimann *et al.*, "Cliques of Neurons Bound into Cavities Provide a Missing Link between Structure and Function," *Front. Comput. Neurosci.*, vol. 11, p. 266051, Jun. 2017, doi: 10.3389/fncom.2017.00048.
[36] A. D. M. Valverde, "From Connectome to Cognition: Proposing a Connectome-Based Hypernetwork Model to Interpret the Conjunction Fallacy & Possible Representational Structure of Qualia," presented at the ASSC 27, University of Tokyo, Jul. 04, 2024. doi: 10.13140/RG.2.2.16658.06086.
[37] A. M. Morgan, O. Devinsky, W. K. Doyle, P. Dugan, D. Friedman, and A. Flinker, "Decoding words during sentence production: Syntactic role encoding and structure-dependent dynamics revealed by ECoG," Jan. 24, 2025, *bioRxiv*. doi: 10.1101/2024.10.30.621177.
[38] T. R. del J. Gonzalez Alam, T. Karapanagiotidis, J. Smallwood, and E. Jefferies, "Degrees of lateralisation in semantic cognition: Evidence from intrinsic connectivity," *NeuroImage*, vol. 202, p. 116089, Nov. 2019, doi: 10.1016/j.neuroimage.2019.116089.

[39] Y. Chen *et al.*, "The 'when' and 'where' of semantic coding in the anterior temporal lobe: Temporal representational similarity analysis of electrocorticogram data," *Cortex*, vol. 79, pp. 1–13, Jun. 2016, doi: 10.1016/j.cortex.2016.02.015.
[40] F. Binkofski and L. J. Buxbaum, "Two action systems in the human brain," *Brain Lang.*, vol. 127, no. 2, pp. 222–229, Nov. 2013, doi: 10.1016/j.bandl.2012.07.007.
[41] Y. Chen *et al.*, "White matter basis for the hub-and-spoke semantic representation: evidence from semantic dementia," *Brain*, vol. 143, no. 4, pp. 1206–1219, Apr. 2020, doi: 10.1093/brain/awaa057.
[42] W. K. Simmons and A. Martin, "The anterior temporal lobes and the functional architecture of semantic memory," *J. Int. Neuropsychol. Soc. JINS*, vol. 15, no. 5, pp. 645–649, Sep. 2009, doi: 10.1017/S1355617709990348.
[43] T. J. Abel *et al.*, "Direct Physiologic Evidence of a Heteromodal Convergence Region for Proper Naming in Human Left Anterior Temporal Lobe," *J. Neurosci.*, vol. 35, no. 4, pp. 1513–1520, Jan. 2015, doi: 10.1523/JNEUROSCI.3387-14.2015.
[44] A. Y. Li *et al.*, "Experience transforms crossmodal object representations in the anterior temporal lobes," *eLife*, vol. 13, p. e83382, Apr. 2024, doi: 10.7554/eLife.83382.
[45] J. Pearson, "The human imagination: the cognitive neuroscience of visual mental imagery," *Nat. Rev. Neurosci.*, vol. 20, no. 10, pp. 624–634, Oct. 2019, doi: 10.1038/s41583-019-0202-9.
[46] S. Chang, X. Zhang, Y. Cao, J. Pearson, and M. Meng, "Imageless imagery in aphantasia revealed by early visual cortex decoding," *Curr. Biol.*, vol. 35, no. 3, pp. 591-599.e4, Feb. 2025, doi: 10.1016/j.cub.2024.12.012.
[47] C. R. Cox *et al.*, "Representational similarity learning reveals a graded multidimensional semantic space in the human anterior temporal cortex," *Imaging Neurosci.*, vol. 2, pp. 1–22, Feb. 2024, doi: 10.1162/imag_a_00093.
[48] M. Girn, R. Setton, G. R. Turner, and R. N. Spreng, "The 'limbic network,' comprising orbitofrontal and anterior temporal cortex, is part of an extended default network: Evidence from multi-echo fMRI," *Netw. Neurosci.*, vol. 8, no. 3, pp. 860–882, Oct. 2024, doi: 10.1162/netn_a_00385.
[49] G. Yvert, M. Perrone-Bertolotti, M. Baciu, and O. David, "Dynamic Causal Modeling of Spatiotemporal Integration of Phonological and Semantic Processes: An Electroencephalographic Study," *J. Neurosci.*, vol. 32, no. 12, pp. 4297–4306, Mar. 2012, doi: 10.1523/JNEUROSCI.6434-11.2012.
[50] J. M. Shine, L. D. Lewis, D. D. Garrett, and K. Hwang, "The impact of the human thalamus on brain-wide information processing," *Nat. Rev. Neurosci.*, vol. 24, no. 7, pp. 416–430, Jul. 2023, doi: 10.1038/s41583-023-00701-0.
[51] M. W. Schlund, A. T. Brewer, D. M. Richman, S. K. Magee, and S. Dymond, "Not so bad: avoidance and aversive discounting modulate threat appraisal in anterior cingulate and medial prefrontal cortex," *Front. Behav. Neurosci.*, vol. 9, p. 148777, Jun. 2015, doi: 10.3389/fnbeh.2015.00142.
[52] E. T. Rolls, G. Deco, C.-C. Huang, and J. Feng, "The human orbitofrontal cortex, vmPFC, and anterior cingulate cortex effective connectome: emotion, memory, and action," *Cereb. Cortex N. Y. N 1991*, vol. 33, no. 2, pp. 330–356, Dec. 2022, doi: 10.1093/cercor/bhac070.
[53] P. H. Rudebeck and E. A. Murray, "The orbitofrontal oracle: cortical mechanisms for the prediction and evaluation of specific behavioral outcomes," *Neuron*, vol. 84, no. 6, pp. 1143–1156, Dec. 2014, doi: 10.1016/j.neuron.2014.10.049.
[54] J. Jiang, A. D. Wagner, and T. Egner, "Integrated externally and internally generated task predictions jointly guide cognitive control in prefrontal cortex," *eLife*, vol. 7, p. e39497, Aug. 2018, doi: 10.7554/eLife.39497.
[55] H. Barbas, "General cortical and special prefrontal connections: principles from structure to function," *Annu. Rev. Neurosci.*, vol. 38, pp. 269–289, Jul. 2015, doi: 10.1146/annurev-neuro-071714-033936.
[56] E. T. Rolls, W. Cheng, and J. Feng, "The orbitofrontal cortex: reward, emotion and depression," *Brain Commun.*, vol. 2, no. 2, p. fcaa196, Nov. 2020, doi: 10.1093/braincomms/fcaa196.
[57] M. L. Kringelbach, "The human orbitofrontal cortex: linking reward to hedonic experience," *Nat. Rev. Neurosci.*, vol. 6, no. 9, pp. 691–702, Sep. 2005, doi: 10.1038/nrn1747.

[58] H. T. Ghashghaei and H. Barbas, "Pathways for emotion: interactions of prefrontal and anterior temporal pathways in the amygdala of the rhesus monkey," *Neuroscience*, vol. 115, no. 4, pp. 1261–1279, Dec. 2002, doi: 10.1016/S0306-4522(02)00446-3.

[59] S. J. Banks, K. T. Eddy, M. Angstadt, P. J. Nathan, and K. L. Phan, "Amygdala–frontal connectivity during emotion regulation," *Soc. Cogn. Affect. Neurosci.*, vol. 2, no. 4, pp. 303–312, Dec. 2007, doi: 10.1093/scan/nsm029.

[60] S. J. Granger *et al.*, "Integrity of the Uncinate Fasciculus is Associated with Emotional Pattern Separation-Related fMRI Signals in the Hippocampal Dentate and CA3.," *Neurobiol. Learn. Mem.*, vol. 177, p. 107359, Jan. 2021, doi: 10.1016/j.nlm.2020.107359.

[61] R. J. Von Der Heide, L. M. Skipper, E. Klobusicky, and I. R. Olson, "Dissecting the uncinate fasciculus: disorders, controversies and a hypothesis," *Brain*, vol. 136, no. 6, pp. 1692–1707, Jun. 2013, doi: 10.1093/brain/awt094.

[62] R. Chiou, G. F. Humphreys, J. Jung, and M. A. Lambon Ralph, "Controlled semantic cognition relies upon dynamic and flexible interactions between the executive 'semantic control' and hub-and-spoke 'semantic representation' systems," *Cortex J. Devoted Study Nerv. Syst. Behav.*, vol. 103, pp. 100–116, Jun. 2018, doi: 10.1016/j.cortex.2018.02.018.

[63] H. T. Ghashghaei, C. C. Hilgetag, and H. Barbas, "Sequence of information processing for emotions based on the anatomic dialogue between prefrontal cortex and amygdala," *NeuroImage*, vol. 34, no. 3, pp. 905–923, Feb. 2007, doi: 10.1016/j.neuroimage.2006.09.046.

[64] P. Fettes, L. Schulze, and J. Downar, "Cortico-Striatal-Thalamic Loop Circuits of the Orbitofrontal Cortex: Promising Therapeutic Targets in Psychiatric Illness," *Front. Syst. Neurosci.*, vol. 11, p. 245573, Apr. 2017, doi: 10.3389/fnsys.2017.00025.

[65] H. Yan *et al.*, "Nucleus accumbens: a systematic review of neural circuitry and clinical studies in healthy and pathological states," *J. Neurosurg.*, vol. 138, no. 2, pp. 337–346, Feb. 2023, doi: 10.3171/2022.5.JNS212548.

[66] M. Catani, F. Dell'Acqua, and M. Thiebaut de Schotten, "A revised limbic system model for memory, emotion and behaviour," *Neurosci. Biobehav. Rev.*, vol. 37, no. 8, pp. 1724–1737, Sep. 2013, doi: 10.1016/j.neubiorev.2013.07.001.

[67] E. Y. Choi, S.-L. Ding, and S. N. Haber, "Combinatorial Inputs to the Ventral Striatum from the Temporal Cortex, Frontal Cortex, and Amygdala: Implications for Segmenting the Striatum," *eNeuro*, vol. 4, no. 6, Nov. 2017, doi: 10.1523/ENEURO.0392-17.2017.

[68] M. Yun *et al.*, "Distinct roles of the orbitofrontal cortex, ventral striatum, and dopamine neurons in counterfactual thinking of decision outcomes," *Sci. Adv.*, vol. 9, no. 32, p. eadh2831, Aug. 2023, doi: 10.1126/sciadv.adh2831.

[69] J. L. Lanciego, N. Luquin, and J. A. Obeso, "Functional Neuroanatomy of the Basal Ganglia," *Cold Spring Harb. Perspect. Med.*, vol. 2, no. 12, p. a009621, Dec. 2012, doi: 10.1101/cshperspect.a009621.

[70] E. Mengual, S. de las Heras, E. Erro, J. L. Lanciego, and J. M. Giménez-Amaya, "Thalamic interaction between the input and the output systems of the basal ganglia," *J. Chem. Neuroanat.*, vol. 16, no. 3, pp. 187–200, May 1999, doi: 10.1016/S0891-0618(99)00010-1.

[71] "Contributions of Basal Ganglia Circuits to Perception, Attention, and Consciousness | Journal of Cognitive Neuroscience | MIT Press." Accessed: May 15, 2025. [Online]. Available: https://direct.mit.edu/jocn/article-abstract/36/8/1620/120892/Contributions-of-Basal-Ganglia-Circuits-to?redirectedFrom=fulltext

[72] L. A. Bradfield, J. Bertran-Gonzalez, B. Chieng, and B. W. Balleine, "The Thalamostriatal Pathway and Cholinergic Control of Goal-Directed Action: Interlacing New with Existing Learning in the Striatum," *Neuron*, vol. 79, no. 1, pp. 153–166, Jul. 2013, doi: 10.1016/j.neuron.2013.04.039.

[73] A. J. Weitz, H. J. Lee, M. Choy, and J. H. Lee, "Thalamic input to orbitofrontal cortex drives brain-wide, frequency-dependent inhibition mediated by GABA and zona incerta," *Neuron*, vol. 104, no. 6, pp. 1153-1167.e4, Dec. 2019, doi: 10.1016/j.neuron.2019.09.023.

[74] D. Tonduti *et al.*, "Neurological Disorders Associated with Striatal Lesions: Classification and Diagnostic Approach," *Curr. Neurol. Neurosci. Rep.*, vol. 16, no. 6, p. 54, Jun. 2016, doi: 10.1007/s11910-016-0656-3.

[75] H. Arnts, W. S. van Erp, J. C. M. Lavrijsen, S. van Gaal, H. J. Groenewegen, and P. van den Munckhof, "On the pathophysiology and treatment of akinetic mutism," *Neurosci. Biobehav. Rev.*, vol. 112, pp. 270–278, May 2020, doi: 10.1016/j.neubiorev.2020.02.006.
[76] C. L. Burton, A. Longaretti, A. Zlatanovic, G. M. Gomes, and R. Tonini, "Striatal insights: a cellular and molecular perspective on repetitive behaviors in pathology," *Front. Cell. Neurosci.*, vol. 18, p. 1386715, Mar. 2024, doi: 10.3389/fncel.2024.1386715.
[77] Y. B. Saalmann, "Intralaminar and medial thalamic influence on cortical synchrony, information transmission and cognition," *Front. Syst. Neurosci.*, vol. 8, p. 78285, May 2014, doi: 10.3389/fnsys.2014.00083.
[78] B. J. Hunnicutt, B. R. Long, D. Kusefoglu, K. J. Gertz, H. Zhong, and T. Mao, "A comprehensive thalamocortical projection map at the mesoscopic level," *Nat. Neurosci.*, vol. 17, no. 9, pp. 1276–1285, Sep. 2014, doi: 10.1038/nn.3780.
[79] J. M. Shine, "The thalamus integrates the macrosystems of the brain to facilitate complex, adaptive brain network dynamics," *Prog. Neurobiol.*, vol. 199, p. 101951, Apr. 2021, doi: 10.1016/j.pneurobio.2020.101951.
[80] S. M. Sherman and W. M. Usrey, "A Reconsideration of the Core and Matrix Classification of Thalamocortical Projections," *J. Neurosci. Off. J. Soc. Neurosci.*, vol. 44, no. 24, p. e0163242024, Jun. 2024, doi: 10.1523/JNEUROSCI.0163-24.2024.
[81] E. Yoshida *et al.*, "Whether or not to act is determined by distinct signals from motor thalamus and orbitofrontal cortex to secondary motor cortex," *Nat. Commun.*, vol. 16, no. 1, p. 3106, Apr. 2025, doi: 10.1038/s41467-025-58272-w.
[82] K. Kawabata *et al.*, "Bridging large-scale cortical networks: Integrative and function-specific hubs in the thalamus," *iScience*, vol. 24, no. 10, p. 103106, Oct. 2021, doi: 10.1016/j.isci.2021.103106.
[83] A. Revonsuo and J. Newman, "Binding and consciousness," *Conscious. Cogn.*, vol. 8, no. 2, pp. 123–127, Jun. 1999, doi: 10.1006/ccog.1999.0393.
[84] "Space, Time and Consciousness, by John Smythies." Accessed: May 15, 2025. [Online]. Available: https://www.newdualism.org/papers/J.Smythies/Smythies-JCS2003.htm
[85] A. Revonsuo, "Binding and the Phenomenal Unity of Consciousness," *Conscious. Cogn.*, vol. 8, no. 2, pp. 173–185, Jun. 1999, doi: 10.1006/ccog.1999.0384.
[86] A. K. Seth, J. L. McKinstry, G. M. Edelman, and J. L. Krichmar, "Visual Binding Through Reentrant Connectivity and Dynamic Synchronization in a Brain-based Device," *Cereb. Cortex*, vol. 14, no. 11, pp. 1185–1199, Nov. 2004, doi: 10.1093/cercor/bhh079.
[87] C. D. Gilbert and W. Li, "Top-down influences on visual processing," *Nat. Rev. Neurosci.*, vol. 14, no. 5, p. 10.1038/nrn3476, May 2013, doi: 10.1038/nrn3476.
[88] J. J. Hopfield, "Neural networks and physical systems with emergent collective computational abilities.," *Proc. Natl. Acad. Sci. U. S. A.*, vol. 79, no. 8, pp. 2554–2558, Apr. 1982, doi: 10.1073/pnas.79.8.2554.
[89] H. Miyawaki and K. Mizuseki, "De novo inter-regional coactivations of preconfigured local ensembles support memory," *Nat. Commun.*, vol. 13, no. 1, p. 1272, Mar. 2022, doi: 10.1038/s41467-022-28929-x.
[90] X.-J. Wang, "Synaptic reverberation underlying mnemonic persistent activity," *Trends Neurosci.*, vol. 24, no. 8, pp. 455–463, Aug. 2001, doi: 10.1016/S0166-2236(00)01868-3.
[91] J. Zylberberg and B. W. Strowbridge, "Mechanisms of Persistent Activity in Cortical Circuits: Possible Neural Substrates for Working Memory," *Annu. Rev. Neurosci.*, vol. 40, pp. 603–627, Jul. 2017, doi: 10.1146/annurev-neuro-070815-014006.
[92] C. Liu *et al.*, "Recurrent neural networks with transient trajectory explain working memory encoding mechanisms," *Commun. Biol.*, vol. 8, no. 1, pp. 1–13, Jan. 2025, doi: 10.1038/s42003-024-07282-3.
[93] G. Stefanics, J. Kremláček, and I. Czigler, "Visual mismatch negativity: a predictive coding view," *Front. Hum. Neurosci.*, vol. 8, p. 666, Sep. 2014, doi: 10.3389/fnhum.2014.00666.
[94] G. Stefanics, J. Heinzle, A. A. Horváth, and K. E. Stephan, "Visual Mismatch and Predictive Coding: A Computational Single-Trial ERP Study," *J. Neurosci.*, vol. 38, no. 16, pp. 4020–4030, Apr. 2018, doi: 10.1523/JNEUROSCI.3365-17.2018.
[95] C. E. Deveau *et al.*, "Recurrent cortical networks encode natural sensory statistics via sequence filtering," *bioRxiv*, p. 2024.02.24.581890, Sep. 2024, doi: 10.1101/2024.02.24.581890.

[96] I. A. Oldenburg *et al.*, "The logic of recurrent circuits in the primary visual cortex," *Nat. Neurosci.*, vol. 27, no. 1, pp. 137–147, Jan. 2024, doi: 10.1038/s41593-023-01510-5.
[97] S. Clavagnier, A. Falchier, and H. Kennedy, "Long-distance feedback projections to area V1: Implications for multisensory integration, spatial awareness, and visual consciousness," *Cogn. Affect. Behav. Neurosci.*, vol. 4, no. 2, pp. 117–126, Jun. 2004, doi: 10.3758/CABN.4.2.117.
[98] K. R. Skene, "Systems theory, thermodynamics and life: Integrated thinking across ecology, organization and biological evolution.," *Biosystems*, vol. 236, p. 105123, Feb. 2024, doi: 10.1016/j.biosystems.2024.105123.
[99] A. Goldbeter, "Dissipative structures in biological systems: bistability, oscillations, spatial patterns and waves," *Philos. Transact. A Math. Phys. Eng. Sci.*, vol. 376, no. 2124, p. 20170376, Jul. 2018, doi: 10.1098/rsta.2017.0376.
[100] W. Hofkirchner and M. Schafranek, "General System Theory," in *Philosophy of Complex Systems*, vol. 10, C. Hooker, Ed., in Handbook of the Philosophy of Science, vol. 10. , Amsterdam: North-Holland, 2011, pp. 177–194. doi: 10.1016/B978-0-444-52076-0.50006-7.
[101] Y. Shi, L. Yang, J. Lu, T. Yan, Y. Ding, and B. Wang, "The dynamic reconfiguration of the functional network during episodic memory task predicts the memory performance," *Sci. Rep.*, vol. 14, no. 1, p. 20527, Sep. 2024, doi: 10.1038/s41598-024-71295-5.
[102] J. K. Degutis *et al.*, "Dynamic layer-specific processing in the prefrontal cortex during working memory," *Commun. Biol.*, vol. 7, no. 1, pp. 1–11, Sep. 2024, doi: 10.1038/s42003-024-06780-8.
[103] E. B. Barbeau, M. Descoteaux, and M. Petrides, "Dissociating the white matter tracts connecting the temporo-parietal cortical region with frontal cortex using diffusion tractography," *Sci. Rep.*, vol. 10, no. 1, p. 8186, May 2020, doi: 10.1038/s41598-020-64124-y.
[104] E. Shekari and N. Nozari, "A narrative review of the anatomy and function of the white matter tracts in language production and comprehension," *Front. Hum. Neurosci.*, vol. 17, p. 1139292, Mar. 2023, doi: 10.3389/fnhum.2023.1139292.
[105] J. Moriya, "Visual mental imagery influences attentional guidance in a visual-search task," *Atten. Percept. Psychophys.*, vol. 80, no. 5, pp. 1127–1142, Jul. 2018, doi: 10.3758/s13414-018-1520-0.
[106] M. Monzel, K. Keidel, and M. Reuter, "Imagine, and you will find – Lack of attentional guidance through visual imagery in aphantasics," *Atten. Percept. Psychophys.*, vol. 83, no. 6, pp. 2486–2497, Aug. 2021, doi: 10.3758/s13414-021-02307-z.
[107] M. Monzel and M. Reuter, "Where's Wanda? The influence of visual imagery vividness on visual search speed measured by means of hidden object pictures," *Atten. Percept. Psychophys.*, vol. 86, no. 1, pp. 22–27, Jan. 2024, doi: 10.3758/s13414-022-02645-6.
[108] P. Bartolomeo *et al.*, "Multiple-domain dissociation between impaired visual perception and preserved mental imagery in a patient with bilateral extrastriate lesions," *Neuropsychologia*, vol. 36, no. 3, pp. 239–249, Mar. 1998, doi: 10.1016/S0028-3932(97)00103-6.
[109] B. J. White, S. E. Boehnke, R. A. Marino, L. Itti, and D. P. Munoz, "Color-Related Signals in the Primate Superior Colliculus," *J. Neurosci.*, vol. 29, no. 39, pp. 12159–12166, Sep. 2009, doi: 10.1523/JNEUROSCI.1986-09.2009.
[110] N. Takakuwa, K. Isa, H. Onoe, J. Takahashi, and T. Isa, "Contribution of the Pulvinar and Lateral Geniculate Nucleus to the Control of Visually Guided Saccades in Blindsight Monkeys," *J. Neurosci.*, vol. 41, no. 8, pp. 1755–1768, Feb. 2021, doi: 10.1523/JNEUROSCI.2293-20.2020.
[111] R. M. Cassidy, A. V. Macias, W. N. Lagos, C. Ugorji, and E. M. Callaway, "Complementary Organization of Mouse Driver and Modulator Cortico-thalamo-cortical Circuits," *J. Neurosci.*, vol. 45, no. 5, p. e1167242024, Jan. 2025, doi: 10.1523/JNEUROSCI.1167-24.2024.
[112] H. Sanders, C. Rennó-Costa, M. Idiart, and J. Lisman, "Grid Cells and Place Cells: An Integrated View of their Navigational and Memory Function," *Trends Neurosci.*, vol. 38, no. 12, pp. 763–775, Dec. 2015, doi: 10.1016/j.tins.2015.10.004.
[113] R. C. Saunders, M. Mishkin, and J. P. Aggleton, "Projections from the entorhinal cortex, perirhinal cortex, presubiculum, and parasubiculum to the medial thalamus in macaque monkeys: identifying different pathways using disconnection techniques," *Exp. Brain Res.*, vol. 167, no. 1, pp. 1–16, Nov. 2005, doi: 10.1007/s00221-005-2361-3.

[114] D. Derrien, C. Garric, C. Sergent, and S. Chokron, "The nature of blindsight: implications for current theories of consciousness," *Neurosci. Conscious.*, vol. 2022, no. 1, p. niab043, Jan. 2022, doi: 10.1093/nc/niab043.
[115] J. Pearson and S. M. Kosslyn, "The heterogeneity of mental representation: Ending the imagery debate," *Proc. Natl. Acad. Sci. U. S. A.*, vol. 112, no. 33, pp. 10089–10092, Aug. 2015, doi: 10.1073/pnas.1504933112.
[116] B. Nanay, "Mental Imagery," in *The Stanford Encyclopedia of Philosophy*, Winter 2021., E. N. Zalta, Ed., Metaphysics Research Lab, Stanford University, 2021. Accessed: May 16, 2025. [Online]. Available: https://plato.stanford.edu/archives/win2021/entries/mental-imagery/
[117] V. S. Wadia *et al.*, "A shared code for perceiving and imagining objects in human ventral temporal cortex," Oct. 06, 2024, *bio*R*xiv*. doi: 10.1101/2024.10.05.616828.
[118] M. Ayers, *Locke: Epistemology and Ontology*. Psychology Press, 1993.
[119] M. Wicken, R. Keogh, and J. Pearson, "The critical role of mental imagery in human emotion: insights from fear-based imagery and aphantasia," *Proc. R. Soc. B Biol. Sci.*, vol. 288, no. 1946, p. 20210267, Mar. 2021, doi: 10.1098/rspb.2021.0267.
[120] L. N. Bouyer and D. H. Arnold, "Deep Aphantasia: a visual brain with minimal influence from priors or inhibitory feedback?," *Front. Psychol.*, vol. 15, p. 1374349, Apr. 2024, doi: 10.3389/fpsyg.2024.1374349.
[121] R. Keogh, J. Bergmann, and J. Pearson, "Cortical excitability controls the strength of mental imagery," *eLife*, vol. 9, p. e50232, May 2020, doi: 10.7554/eLife.50232.
[122] E. Megla, D. Prasad, and W. A. Bainbridge, "The Neural Underpinnings of Aphantasia: A Case Study of Identical Twins," *bio*R*xiv*, p. 2024.09.23.614521, Sep. 2024, doi: 10.1101/2024.09.23.614521.
[123] A. Zeman, "Aphantasia and hyperphantasia: exploring imagery vividness extremes," *Trends Cogn. Sci.*, vol. 28, no. 5, pp. 467–480, May 2024, doi: 10.1016/j.tics.2024.02.007.
[124] A. H. Palejwala *et al.*, "Anatomy and white matter connections of the fusiform gyrus," *Sci. Rep.*, vol. 10, no. 1, p. 13489, Aug. 2020, doi: 10.1038/s41598-020-70410-6.