

# **An Integration Model of Mental Imagery and Aphantasia: Conceptual Framework, Neuromechanistic Pathways, and Clinical Implications**

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## **Abstract**

Aphantasia, the strong diminution or complete absence of mental imagery, challenges long-standing views of imagery as central to cognition. Competing accounts variously explain the phenomenon as a failure of sensory reactivation or as unconscious mental imagery. Here, we propose a new framework, the integration model of aphantasia, which argues that reactivated sensory information must undergo multi-stage integration to yield imagery experience. Against unconscious imagery accounts, we argue that the neural activations observed in aphantasics are not imagery but sensory precursors: rudimentary sensory codes that lack perceptual status. Only when sensory precursors are locally integrated do they become perceptual representations, and only when these are further integrated with interoceptive signals do they give rise to conscious imagery experience. We present the integration model as a dual-stream framework that unifies recent attention- and interoception-based accounts, situate the model within existing theories of mental imagery and aphantasia, and highlight its clinical relevance. In doing so, we reframe the debate on unconscious imagery and draw attention to the role of multi-stage integration as a key mechanism underlying mental imagery and its absence across different subtypes of aphantasia.

*Keywords:* aphantasia, mental imagery, unconscious imagery, sensory precursors, integration

# 1. Introduction

Aphantasia, the severe reduction or complete absence of mental imagery<sup>1</sup>, most often involving but not necessarily limited to the visual modality (Zeman et al., 2015, 2025), attracted growing scholarly and public attention since the term was coined in 2015 (see Zeman, 2024, for a recent review). A key driver of this attention is mounting evidence that individuals with aphantasia (henceforth ‘aphantasics’) can perform a wide range of tasks once thought to depend on imagery, including mental rotation and visual memory (Bainbridge et al., 2021; Kay et al., 2024; Liu & Bartolomeo, 2023; Milton et al., 2021; Pounder et al., 2022; Siena & Simons, 2024). These findings, together with arguments against classifying aphantasia as a disorder (Blomkvist & Marks, 2023; Monzel, Vetterlein, & Reuter, 2023) and reports showing that many aphantasics remain unaware of their condition for decades (Scholz, 2023, 2025a; Zeman et al., 2020), directly challenge longstanding philosophical and psychological traditions that assign mental imagery a central role in human cognition (see MacKisack et al., 2016, for a review).

Although aphantasia is often operationalized as a self-reported lack of imagery experience, as assessed via the *Vividness of Visual Imagery Questionnaire* (VVIQ; Marks, 1973), converging physiological (Kay et al., 2022; Monzel, Karnebo, et al., 2024; Wicken et al., 2021), behavioral (Keogh & Pearson, 2024; Monzel, Keidel, et al., 2023; Monzel, Vetterlein, Hogeterp, et al., 2023; Monzel, Handlogten, et al., 2024; Purkart et al., 2025) and neurological (Chang, Zhang, et al.,

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<sup>1</sup> We are here following Nanay’s (2023) definition of mental imagery as perceptual representation that is not directly triggered by sensory input. Furthermore, here and throughout the paper ‘perceptual representation’ will, also following Nanay, be understood as a type of representation that underlies both sensory stimulation-driven perception (what is usually referred to as perception) and mental imagery. We are using Nanay’s definition here since it is the current definition of mental imagery in the Stanford Encyclopedia of Philosophy (Nanay, 2021a), largely aligns with other influential definitions of mental imagery (e.g., Kosslyn et al., 2006; Pearson et al., 2015), and lies at the centre of the unconscious imagery debate (Nanay, 2021b; Scholz, 2025b; see Section 2.1 below).

2025; Liu, Cohen, et al., 2025; Liu, Zhan, et al., 2025; Monzel, Leelaarporn, et al., 2024), evidence demonstrates that the phenomenon cannot be reduced to differences in language use or interpretation (Lorenzatti, 2025; Scholz, 2024). The core question, then, is how to understand the absence of imagery phenomenology in aphantasia.

Existing theories offer sharply diverging answers. One class of accounts holds that aphantasics fail to generate the perceptual representations required for imagery. Pearson (2019), invoking the ventral-dorsal pathway distinction (Ungerleider et al., 1998), suggests that imagery deficits stem from impairments in the ventral pathway (representing colors, textures, and objects), while the dorsal pathway (supporting spatial representation) remains intact. Similarly, Balaban and Ullman (2025) suggest that aphantasia may involve preserved physical simulation in dorsal networks but broken graphical rendering in the visual cortex. Consistent with this perspective, Phillips (2025; see also Teng, 2025) attributes the competence of aphantasics to preserved spatial imagery, and Blomkvist (2023) proposes that aphantasia reflects an episodic system condition, rooted in problems of memory retrieval or indexing.

A second class of theories argues instead that aphantasia is primarily a deficit of metacognition, where aphantasics do generate perceptual representations, but lack the metacognitive awareness related to introspecting them. On this view, aphantasia involves unconscious mental imagery, meaning that aphantasics still activate the neural representations associated with imagery experience, but without conscious experience (Michel et al., 2025; Myers, 2025; Nanay, 2021b, 2023). This account has intuitive appeal, since it explains how aphantasics can succeed on imagery-related tasks without accompanying phenomenology. However, it has also sparked an active debate about the status of the neural representations observed in aphantasics during imagery tasks (Chang, Jiang, et al., 2025; Scholz et al., 2025).

More recently, a third class of theories has shifted the focus from generation to integration. According to the attention model (Liu, 2025), aphantasics can reactivate weak sensory codes at subliminal or preconscious levels, but fail to integrate and amplify them due to deficits in top-down processes associated with frontoparietal attention networks. The interoception model (Silvanto & Nagai, 2025) instead locates the deficit in the integration of sensory representations with interoceptive bodily signals at the level of the insula. These accounts highlight important mechanisms beyond mere sensory reactivation but have so far been developed independently.

In this paper, we build directly on this third approach by proposing a dual-stream integration model of aphantasia that synergizes the attention and interoception models into a unified framework. Furthermore, we argue that integration is the key process in understanding aphantasia, by outlining multiple steps at which integration processes in aphantasia may fail.

Central to our proposal is the notion of *sensory precursors*, that is, rudimentary, preconscious sensory codes (e.g., for shape, color, or orientation) that can feed into conceptual cognition but, in most aphantasics, fail to be integrated into perceptual representations. Building on the distinction between perceptual representation and sensory precursors, we then develop a neuromechanistic model, the *integration model*, that explains both the persistence of task competence and the absence of imagery phenomenology by mapping imagery generation as a multistage integration process. In doing so, our framework draws further inspiration from broader theories of consciousness, including Global Neuronal Workspace Theory (Dehaene & Changeux, 2011; Mashour et al., 2020) and Integrated Information Theory (Tononi et al., 2016), both of which emphasize the role of integration in enabling conscious experience. By clarifying the representational nature of neural activations in aphantasia, and highlighting the importance of subsequent multistage integration processes, we therefore not only aim to reconcile different views

in the unconscious imagery debate but also provide a novel conceptual scaffold that can unify existing models under a common testable integration framework.

We will proceed as follows: Section 2 argues that the representations observed in aphantasics during imagery tasks should not be understood as perceptual representations and therefore do not support unconscious imagery accounts. Instead, we introduce the concept of sensory precursors to account for the representations in aphantasia. Section 3 then presents the integration model as a dual-stream framework synthesizing the attention and interoception models, and Section 4 argues that integration is the key process underlying imagery experience. In Section 5, we then situate the integration model within the broader landscape of theories of mental imagery and aphantasia. Finally, Section 6 explores the implications of viewing aphantasia as a failure of integration, with a specific focus on clinical links to related neurodevelopmental and psychiatric conditions.

## **2. The nature of representation in aphantasia**

A central debate in the aphantasia literature is whether aphantasics have unconscious imagery (Chang, Zhang, et al., 2025; Mckilliam & Kirberg, 2025; Michel et al., 2025; Nanay, 2021b; Scholz, 2025b; Scholz et al., 2025; Zeman, 2025). On this view, although aphantasics lack imagery phenomenology, they still activate the perceptual representations typically associated with imagery (Nanay, 2021b, 2023). Here, we argue that the evidence does not support this interpretation (Section 2.1). Instead, we propose that the activations in aphantasics are better understood as sensory precursors, that is, non-perceptual representations that, under appropriate integration, can be transformed into perceptual representations (Section 2.2).

## 2.1 Evidence against unconscious imagery in aphantasia

Two lines of evidence are typically presented in favor of unconscious imagery. First, aphantasics perform similarly to typical visualizers on tasks that supposedly test for mental imagery (e.g., Liu & Bartolomeo, 2023; Pounder et al., 2022; Siena & Simons, 2024). Second, aphantasics activate the same sensory brain regions as typical visualizers (Chang, Zhang, et al., 2025; Liu, Cohen, et al., 2025; Liu, Zhan, et al., 2025; Weber et al., 2024). For example, Liu, Zhan, et al. (2025) instructed participants to visualize two items (e.g., beaver, fox) and compare them with respect to a shared attribute (e.g., length). Aphantasics performed as accurately as typical visualizers, and brain imaging showed activation in the same domain-specific visual areas (e.g., V4 for color comparison), with stimulus-specific representations (e.g., visual colors, face shapes, and identities) encoded in these regions during imagery (Liu, Zhan, et al., 2025). Similarly, Chang, Zhang, et al. (2025) instructed participants to visualize colored Gabor patches and found that stimulus-specific representations were decodable from aphantasics' early visual cortices.

However, as Scholz et al. (2025) point out, in both studies the representations in aphantasics during the imagery condition lacked perceptual representational geometry, as shown by the absence of cross-decodability and representational overlap with representations during analogous perception conditions (i.e., seeing the items or Gabor patches). This shared representational geometry between imagery and perception is a hallmark of mental imagery (Naselaris et al., 2015; Pearson & Kosslyn, 2015), and was found, in both studies, in the respective typically visualizing control groups (Chang, Zhang, et al., 2025; Liu, Cohen, et al., 2025; Liu, Zhan, et al., 2025).

While Chang, Jiang, et al. (2025) argue that these findings may merely indicate distorted perceptual representations in aphantasia, evidence from behavioral studies suggests that the

representations in aphantasics lack a distinctly perceptual functional profile. For example, while visualization is known to prime behavior on binocular rivalry tasks (Pearson et al., 2008), multiple studies from different groups (Keogh & Pearson, 2018, 2024; Monzel, Agren, et al., 2025; Monzel, Scholz, et al., 2025) found that aphantasics do not exhibit the expected priming effect. This is especially relevant because in another binocular rivalry study (Kwok et al., 2019), where typically visualizing participants were told to suppress visualization following a semantic cue (e.g., ‘red apple’), they still showed the expected priming effect, despite reporting no imagery phenomenology. This shows that imagery representations, despite absent accompanying phenomenology, can still, in principle, prime rivalry. The absence of priming in aphantasics therefore provides strong evidence against unconscious imagery (Nanay, 2021b).<sup>2</sup>

Importantly, the absence of binocular rivalry priming in aphantasia has recently also been shown in an involuntary imagery paradigm (Purkart et al., 2025), thus indicating, pace Nanay (2021b), that unconscious imagery is neither triggered voluntarily nor involuntarily in aphantasics. This finding is corroborated by another study (Monzel, Rademacher, et al., 2025) that found significantly reduced priming in aphantasics in a Stroop task paradigm, which Nanay (2024) hypothesized may trigger (unconscious) mental imagery (see Scholz, 2025b, for discussion; see also Krempel & Monzel, 2024) for a general discussion of involuntary imagery in aphantasia). The same was true for missing priming effects in visual search tasks in which the target was supposed to be primed by visual imagery (Monzel et al., 2021; Monzel & Reuter, 2024).

Physiological studies (Kay et al., 2022; Wicken et al., 2021) showing that aphantasics lack the physiological correlate typically associated with imagery representations add further weight.

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<sup>2</sup> Furthermore, Kwok et al. (2019) showed that the priming effect in typical visualizers could be disrupted by exposing them to a highly luminous stimulus, thus indicating that the effect that took place in the absence of imagery phenomenology was driven by perceptual representations.

For example, while imagining a bright stimulus (e.g., the sun) leads to pupillary constriction in typical visualizers (Laeng & Sultvedt, 2014), no such effect was observed in aphantasics (Kay et al., 2022). And while listening to frightening stories evoked an increased galvanic skin response in typical visualizers (presumably due to the generation of corresponding [unconscious] imagery), aphantasics showed a significantly smaller response, despite showing no difference with visualizers when looking at frightening pictures (Wicken et al., 2021).

Taken together, the evidence thus suggests that the representations observed in aphantasics, despite occurring in the same sensory neural regions as in typical visualizers and supporting competencies typically associated with imagery, differ in important ways from imagery representations. They lack a perceptual representational geometry (Chang, Zhang, et al., 2025; Liu, Cohen, et al., 2025; Liu, Zhan, et al., 2025), fail to produce the behavioral priming effects that (unconscious) imagery can elicit in typical visualizers (Keogh & Pearson, 2024; Purkart et al., 2025), and do not generate the physiological markers associated with imagery (Kay et al., 2022; Wicken et al., 2021). These converging findings indicate that the neural representations in aphantasics should not be understood as unconscious imagery and thus call for a reconceptualization.

## **2.2 Sensory precursors predate perceptual representation**

How then should the activations in aphantasics during imagery tasks be classified? We propose that the representations in aphantasia are best understood as what we call *sensory precursors*, that is, preconscious, domain-specific sensory codes (e.g., for shapes, colors) that can, in principle, be transformed into perceptual representations if subjected to further top-down processing, but in most aphantasics do not reach this state. Only when representations achieve



perceptual status, as indicated by appropriate neurogeometrical, behavioral, and physiological profiles, can they be described as (potentially unconscious) imagery.

The integration model we outline in Section 3 aims to capture the full progression from the (re)activation of sensory precursors, to their integration into perceptual representations, and ultimately to imagery phenomenology. Importantly, this framework does not conceptually exclude the possibility of unconscious imagery in some aphantasics. On the contrary, it allows for heterogeneity of aphantasia subtypes (Nanay, 2025), by specifying multiple states at which integration may fail. Thus, while some aphantasics may indeed reach unconscious perceptual representations (constituting a genuine unconscious imagery subtype), current evidence suggests that most remain at the precursor stage and should be classified as belonging to a *no-imagery subtype* (Table 1).

**Table 1.** Comparison of unconscious imagery and no-imagery subtypes of aphantasia

	<b>Unconscious Imagery Subtype</b>	<b>No-imagery Subtype</b>
Awareness	Absence of imagery experience	Absence of imagery experience
Representation type	Perceptual representation (with distinct perceptual geometry)	Sensory precursors (non-perceptual geometry)
Functional profile	Priming effects (e.g., binocular rivalry, Stroop task)	No imagery-related priming; may contribute to conceptual representations
Physiological profile	Imagery-related effects (pupil constriction, galvanic skin response)	No imagery-related physiological profile

While sensory precursors do not function like mental imagery, they may still causally feature in aphantasics' performance of tasks that are classically associated with imagery. For example, in the visual imagery task by Liu and Bartolomeo (2023), activation of domain-specific precursors may supply information that is further processed in conceptual representations without becoming perceptual codes. A (re)activated color precursor in V4, for instance, may be integrated into the conceptual representation 'strawberry'. In more complex tasks, aphantasics may integrate sensory precursors from multiple domains directly into conceptual representations, bypassing perceptual integration altogether. This idea is compatible with Arcangeli and Bartolomeo's (2025) recent suggestion that aphantasics may access perceptual information via non-perceptual routes (see also Arcangeli, 2023).

We posit that the important distinguishing factor between sensory precursors and perceptual representations, next to their differing neuronal coding and associated behavioral and physiological profiles, is that while perceptual representations can be conscious or unconscious, sensory precursors are necessarily at an unintegrated, subliminal level. However, they may be integrated into cognitively accessible perceptual or conceptual representations in a preconscious stage (Liu, 2025). The underlying process that transforms sensory precursors into perceptual representations is, as we will discuss in detail next, a multi-stage integration process that integrates isolated sensory precursors (e.g., a circular shape code, a red color code, etc.) first into modality-specific but locally integrated object/scene representations (e.g., a cherry), and later into embodied, self-referential, multimodal representations (e.g., eating cherries at the beach). These integrated perceptual representations can then, via further long-range amplification processes, become conscious, leading to imagery phenomenology (e.g., the quasi-sensory experience of seeing cherries or eating cherries at a beach, etc.).

### **3. The integration model of aphantasia**

The integration model builds on and unifies two recent stand-alone accounts of aphantasia, namely the attention model (Liu, 2025) and the interoception model (Silvanto & Nagai, 2025). Although being developed independently, both models go beyond sensory reactivation and stress the importance of integration processes. After briefly introducing each of the two individual models (Sections 3.1, 3.2), we present the integration model as incorporating attention–interoception dual streams, and demonstrate how this framework can explain the absence of imagery experience in aphantasia (Section 3.3). We argue that these accounts are not mutually exclusive but rather highlight two complementary streams of imagery processing, and that their unification in the integration model thus offers a more comprehensive explanation of subjective imagery and its absence in aphantasia than each of the models do on their own.

#### **3.1 The attention model**

The attention model considers aphantasia as a deficit in top-down modulation by frontoparietal networks. It offers a testable neural framework for characterizing unconscious internal representations and their transformation into conscious imagery. According to the model, imagery emerges through three successive stages: generation, integration, and amplification, modulated by top-down attention. In this process, subliminal fragmented visual information is first integrated locally within the visual cortex into perceptual-like preconscious representations, and then globally amplified and broadcast through frontoparietal networks, enabling conscious experience (Liu, 2025).

Subjective imagery emerges from coordinated activity of a fronto-parieto-fusiform network orchestrated by dorsal and ventral attention systems (Liu et al., 2023; Bartolomeo et al.,

2025), potentially coordinated by the anterior insula cortex (AIC). The dorsal attention network (DAN) supports hierarchical processing across three successive stages: (i) generation, where top-down signals initiate weak reactivations in visual areas; (ii) integration, where the visual cortex binds conceptual knowledge with visual features to assemble coherent perceptual-like content; and (iii) amplification, where conscious access arises as dorsal prefrontal cortex strengthens these representations for global broadcasting (Mashour et al., 2020). The fusiform imagery node (FIN) may act as a key interface between frontoparietal networks and the visual cortex, supporting both sensory integration and conscious access. Individual vividness depends largely on the role of integration of the FIN with its more perceptual-like representations (Dijkstra, Bosch, et al., 2017; Liu, Zhan, et al., 2025) and enhanced local efficiency of integration (Kvamme et al., 2025) associated with stronger vividness. Notably, weaker or less perceptual integration in the FIN produces less vivid imagery in typical visualizers; amplification, by contrast, enables conscious access and operates as a thresholded, binary process, determining the presence or absence of imagery. In parallel, the ventral attention network (VAN) mediates inward shifts of attention, disengaging from the external environment while suppressing distractors (Corbetta et al., 2008).

In this view, most individuals with aphantasia may still generate and integrate visual features, to some degree, towards preconscious representations, allowing access to conceptual and visual knowledge at these stages. However, they may show deficits in amplification, preventing internal representations from reaching conscious awareness. Consistent with this, both congenital aphantasia exhibits reduced functional connectivity between the FIN and frontoparietal networks (Liu & Bartolomeo, 2025), and neurogenic aphantasia shows reduced connectivity between the FIN and domain-preferring circuits (Kutsche et al. 2025).

### 3.2 The interoception model

The interoception model reconceptualizes aphantasia not as a purely sensory deficit but as a disruption in the integration of bodily signals with higher-order sensory and cognitive processes. In this framework, mental imagery is not merely viewed as the reactivation of perceptual representations but as an embodied simulation, dependent on interoceptive input to confer affective tone, sense of agency, and self-referential processing (Silvanto, 2025; Silvanto & Nagai, 2025).

At the core of this account is the insula, a principal hub of the brain's interoceptive system. By integrating visceral signals with multisensory input, the AIC provides the foundation for conscious emotion and higher-order representations of the self (Critchley & Harrison, 2013). It thereby anchors mental content in a sense of ownership and agency. Structural and functional connectivity between the insula, somatosensory cortices, and occipito-temporal regions has been associated with enhanced embodiment and self-initiated action (Harduf et al., 2023), underscoring its role in binding bodily and sensory information. As a key node in the salience network, the insula further supports cross-modal integration and the detection of behaviorally relevant signals (Butti & Hof, 2010; Calvert, 2001). Aphantasia may therefore arise when this integrative function is compromised, either due to altered insular processing or dysregulated autonomic input. From a predictive coding perspective, mental imagery arises when top-down prefrontal predictions activate sensory cortices; in the interoceptive model, the insula regulates the precision of these predictions by modulating fronto-parietal signals (Silvanto, 2025). According to this account, aphantasia may result from a failure of interoceptive–sensory integration, due either to suboptimal processing within the insula or impaired afferent input from the body. Consistent with this view, both congenital and psychogenic aphantasia are linked to altered self-reported interoceptive awareness and/or autonomic reactivity (Gao et al., 2025; Monzel, Nagai, et al., 2025). Moreover,

both objective measures of cardiac interoceptive sensitivity and self-reported subjective interoceptive awareness are found to predict imagery vividness (Nagai et al., 2025).

### **3.3 Integration of the two models: toward a dual-stream account of subjective imagery**

Both the attention and interoception models converge on several key principles while differing in their mechanisms. First, both models view imagery experience as an active, constructive process rather than a mere reactivation of sensory cortex activity. In the attention model, imagery arises from hierarchical stages after shifting attention inward: generation, integration, and amplification, whereas in the interoception model, imagery arises from the integration of interoceptive and sensory signals into embodied simulations. Second, both accounts underscore the role of higher-order control networks. The attention model highlights frontoparietal attention networks in shaping imagery experience, while the interoception model emphasizes self-referential integration and sense of agency regulated by the AIC. Third, both frameworks assign a central coordinating role to the insula: it orchestrates attentional shifts and amplification in the attention model and integrates bodily signals to confer affective tone, sense of agency, and self-reference in the interoception model. Finally, both models agree that aphantasia arises not from a purely sensory deficit, but from impaired top-down or integrative processes in large-scale networks associated with subjective experience, with the attention model stressing failures of binding within visual and fronto-parietal systems, while the interoceptive model points to disrupted coupling of sensory representation and interoceptive signals.

Therefore, both models identify long-range network integration as the decisive factor determining whether an internal representation becomes conscious imagery. This convergence

provides the foundation for an integrative dual-stream model, with a dorsal attention stream and a ventral interoception stream (Figure 1).

By combining the two streams, the emergence of subjective imagery may unfold in a sequence of stages (Figure 2):

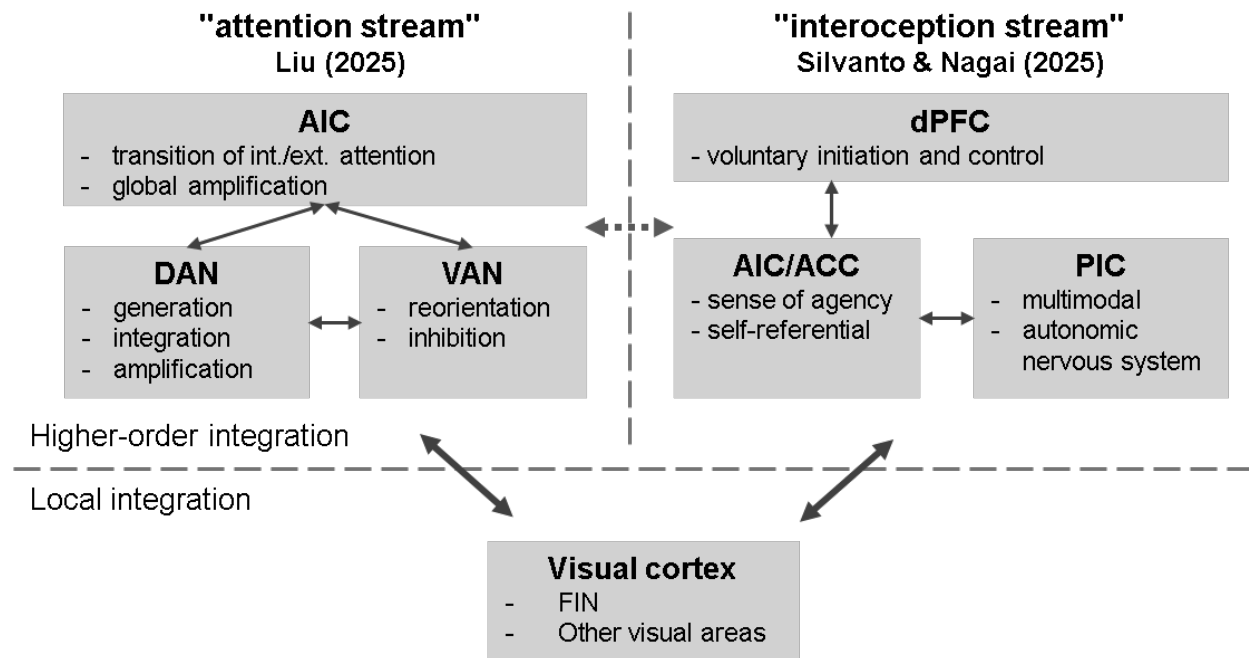
1) the right AIC and VAN first shift attention inward, which is necessary to decouple from external sensory input and prioritise internal representations. Studies on task switching and network-connectivity have shown that the right AIC within the salience network functions as a gatekeeper between the default mode network (supporting internal thought) and the central executive network (enabling attentional control) (Goulden et al., 2014; Huang et al., 2021), allowing flexible attentional orienting or reorienting for conscious processing (Liu et al., 2023). The right AIC thus regulates and prioritizes access to internal representations through salience-based control, such as emotionally charged content (e.g., fear-conditioned stimuli), during imagery (Greening et al., 2022).

2) the inferior frontal gyrus (IFG) initiates semantic and episodic retrieval, leading to reactivation of the visual (or other sensory modalities) cortex, which is essential for re-instantiating sensory precursors (colour, shape, spatial fragments) that form the raw material of imagery, if they are successfully transformed into perceptual codes. However, these precursors alone remain unconscious;

3) the sensory precursors are subsequently integrated through three levels. (i) they undergo *local- or within-modality integration*, e.g., in visual cortex, mediated by the FIN, which integrates modality-specific precursors into perceptual representations; (ii) the locally integrated perceptual representations undergo *multimodal integration*, where modality specific representations (e.g., visual representations) are integrated with representations from other modalities (e.g., auditory

representations)<sup>3</sup> and, crucially, with interoceptive signals from posterior insular cortex (PIC), enabling physiological profiles of imagery; (iii) they undergo *self-referential integration*, supported by the coupling between the dPFC and AIC, which extends beyond sensory integration by rendering the image self-referential and embodiedly- and affectively- grounded;

4) Finally, the AIC and the dorsal fronto-parietal system amplify and stabilize activity, enabling integrated, though, weak perceptual representations to become consciously accessible (Liu, 2025). This attentional amplification is critical for raising integrated perceptual representations above threshold, allowing them to be experienced as quasi-sensory imagery.



**Figure 1. A dual-stream model of subjective imagery: attention (dorsal) and interoception (ventral).** Within the attention stream, images are processed through three successive stages: generation, integration, and amplification, by a fronto-parieto-fusiform network orchestrated by attention networks, coordinated by the AIC. The progress in the attention stream modulates task-related reports associated with internal visual representation. The interoception stream further

<sup>3</sup> We do not take the integration of different sensory modalities to be necessary for imagery experience. For example, one could also simply generate visual imagery experiences of an apple. However, we take the integration of perceptual representations with interoceptive and bodily signals to be necessary for imagery experience.



frames imagery as an embodied simulation, drawing on interoceptive input and multimodal integration by PIC, to confer affective tone, agency, and self-referential grounding through the AIC and its associated circuits.<sup>4</sup> Through this process, interoceptive processing endows imagery with a sense of ownership and self, a critical feature underlying the subjective feeling of imagery. FIN, fusiform imagery node; DAN, dorsal attention network; VAN, ventral attention network; PIC, posterior insular cortex; AIC, anterior insular cortex; ACC, anterior cingulate cortex; dPFC, dorsal prefrontal cortex.

## **4. Integration as the key process in subjective imagery and aphantasia**

The integration model is consistent with established theories of consciousness, such as the Global Neuronal Workspace Theory (Dehaene & Changeux, 2011; Mashour et al., 2020) and the Integrated Information Theory (Tononi et al., 2016), both of which emphasize the integration of sensory information in larger networks as central to the emergence of conscious experience. In line with these theories, we propose that integration constitutes the key process through which mental imagery becomes consciously experienced, and, conversely, that its disruption may underlie aphantasia. In this section, we specify three types of integration processes: local, multimodal and self-referential integration, and demonstrate how different integration steps would fail in aphantasia.

Imagery requires integration at multiple hierarchical levels, from the reconstruction of basic visual features to the binding of multimodal content. Importantly, attention plays a key role

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<sup>4</sup> Importantly, while the current literature on aphantasia focuses mostly on visual cases, aphantasia can occur also in non-visual modalities (Zeman et al., 2025). To account for this heterogeneity, our integration model is aimed to outline a process that occurs in any sensory modality. Thus, we assume that there are sensory precursors and hubs of local integration analogous to the FIN in other sensory modalities, and that locally integrated perceptual representations are undergoing multimodal and self-referential integration processes at the insular level, regardless of the sensory modality (e.g., audition, smell, etc.) in which the precursors were generated. Thus, while we will focus here mainly on visual cases, ‘visual’ can be read as ‘visual (or other sensory modality)’.

in selecting and binding visual features into coherent representations (Treisman and Gelade, 1980). Further, conscious experience depends not simply on the registration of elementary sensory features, but on their binding into a unified whole, either objects, spaces and scenes (Mashour et al., 2020; Robertson, 2003; Tononi and Koch, 2015; Overgaard et al., 2024), often through long-range integration across multiple sensory and bodily systems. This requirement is especially critical for mental imagery, which involves resource-demanding, internally generated processes. Unlike perception, which is continuously stabilized by bottom-up input, imagery is internally generated and therefore disproportionately dependent on integrative mechanisms and anchoring to perceptual or bodily signals to achieve conscious form. When binding fails, individual features or conceptual knowledge may still be accessible, but no vivid, embodied image is experienced. As such, the integration mechanisms co-select distributed feature representations that are part of a single object and explain why the object representations that become part of conscious experience are usually coherent and integrated.

The integration model of aphantasia offers important insights into understanding aphantasia and its potential subtypes, by distinguishing between different processing stages at which imagery generation and integration may fail in aphantasia (Table 2).

At the earliest level, due to altered top–down attention feedback within the attention stream, sensory precursors (i.e., pre-integrated features of colors, shapes, etc.), may not be accurately retrieved.

At the within-modality level, *local (i.e., within modality) integration*: distinct visual components (shapes, colours, spatial layouts) must be combined into perceptual representations that form a coherent scene. Consider the example of imagining a strawberry. One may retrieve schematic features represented as semantic information or sensory precursors—its red colour,

rounded shape, small seeds—but conscious imagery arises only when these features are integrated into a unified perceptual-like content. During imagery, this process is strongly left-lateralized (Liu et al., 2022), particularly for generating multipart images (Kosslyn et al., 1985), with the FIN selectively connecting with areas for specific visual features, such as colors, faces, shapes (Liu, Zhan, et al., 2025), and more intriguingly, enabling the synthesis of two different objects into a unified mental image (Yomogida et al, 2004; e.g. imagine a watermelon-TV), thus supporting the unification of disparate visual elements. Disconnection or inefficiency in these pathways, as observed in lesion-induced aphantasia, may be associated with loss of visual features in specific domains, therefore, failing to coalesce into a coherent internal perceptual representation and preventing both task-related report and imagery experience. In congenital aphantasia, even if sensory precursors are often successfully (re)activated, they may fail to elevate these features to a perceptual status by integrating them into a coherent visual object or scene.

At a higher level, *multimodal integration*: multisensory integration enriches unimodal perceptual imagery representations (from any sensory modality) by incorporating representations from other modalities, (e.g., auditory) and interoceptive states, leading to multimodal perceptual representation. Within this hierarchy, the posterior insula cortex (PIC) acts as a binding hub that links these bodily states with exteroceptive and contextual signals (e.g., visual or somatosensory features processed via parietal relays). A vivid beach scene, for instance, is not merely the reconstruction of sand and sea in the visual cortex, but also mental simulation of sounds, bodily states, and affective resonance that render the image subjectively “real,” for a holistic, embodied re-experiencing.

Furthermore, *self-referential integration*: the AIC transforms these integrated signals into self-referential (recognizing the image as belonging to oneself) and agentic (experiencing

voluntary control over its generation) experiences (see e.g. Uddin, 2007; Critchley & Patchitt, 2024), transforming imagery into personally owned experience. Supporting this view, large-scale connectivity studies have demonstrated that greater global efficiency in the right AIC situated as a key node in salience network predicts higher imagery vividness (Kvamme et al., 2025). Thus, even basic imagery experience (such as imagining a simple stimulus) requires ownership and agency. Loss of self-referential experience, as observed in depersonalisation disorder, a condition characterised by detachment from both the self and the environment, is related to a disrupted connectivity between the AIC and left IFG (Jia et al., 2025); historical clinical reports similarly noted that dissociative disorders can give rise to what is nowadays understood as psychogenic aphantasia (see e.g. de Vito & Bartolomeo, 2016, for review). Therefore, when this self-referential integration process is disrupted, even well-integrated multimodal content remains detached, yielding schematic or depersonalised imagery rather than vivid phenomenology.

In summary, in aphantasia, the process often stalls at the pre-perceptual level: semantic templates are accessed (“strawberries are red, beaches have sand and sea”) and conceptual representations may be further embellished by information derived from (re)activated sensory precursors, but they fail to be integrated into perceptual imagery representations and experiences. In typical imagery this yields a consciously experienced hybrid, but in aphantasia the result remains schematic and pre-perceptual, potentially expressed as a list of attributes as part of a conceptual representation. Therefore, as claimed in Liu (2025), preconscious internal representations exist in aphantasia, but without top-down global integration and amplification they fail to cross the threshold into imagery. This explains why aphantasics can access semantic knowledge yet lack the subjective experience of “seeing with the mind’s eye.”

Here, the model helps clarify the status of activity in the visual cortex in apantasia, which is critical under the current debate related to unconscious imagery. If aphantasics would achieve local integration, manifested as recurrent processing and feedback loops confined to the visual cortex, this activity would be expected to drive unconscious priming effects, including binocular rivalry. However, since they consistently lack the relevant behavioural and physiological signatures of imagery, this suggests that even local integration fails in most aphantasics. Instead, their visual activations remain as fragmented sensory precursors that hardly engage the recurrent processing loops necessary to create coherent representations capable of unconscious influence. As argued above, such activity should thus not be viewed as unconscious imagery and is better described as sensory precursors or preconscious visual representation (Liu, 2025), where feature-specific populations (e.g., colour, shape, spatial fragments) may show local synchrony, but without top-down feedback from frontoparietal attention networks, fail to achieve perceptual status and thus cannot enter into subsequent integration and amplification steps that are necessary to achieve imagery experience.

Notably, the integration model also explains why suboptimal integration manifests so clearly in imagery but not in perception. Perception is anchored by continuous bottom-up input: external signals constrain the scene across multiple levels of processing, providing redundancy and stability. Imagery, by contrast, is “floating”: internally generated without external anchors, and therefore disproportionately dependent on top-down integration of (re)activated sensory precursors, their integration into perceptual representations, attentional amplification, and cross-network coordination. Failures in local visual binding (FIN), long-range attentional control (dorsal PFC), or integration with interoceptive signals (AIC) thus have disproportionately large effects on

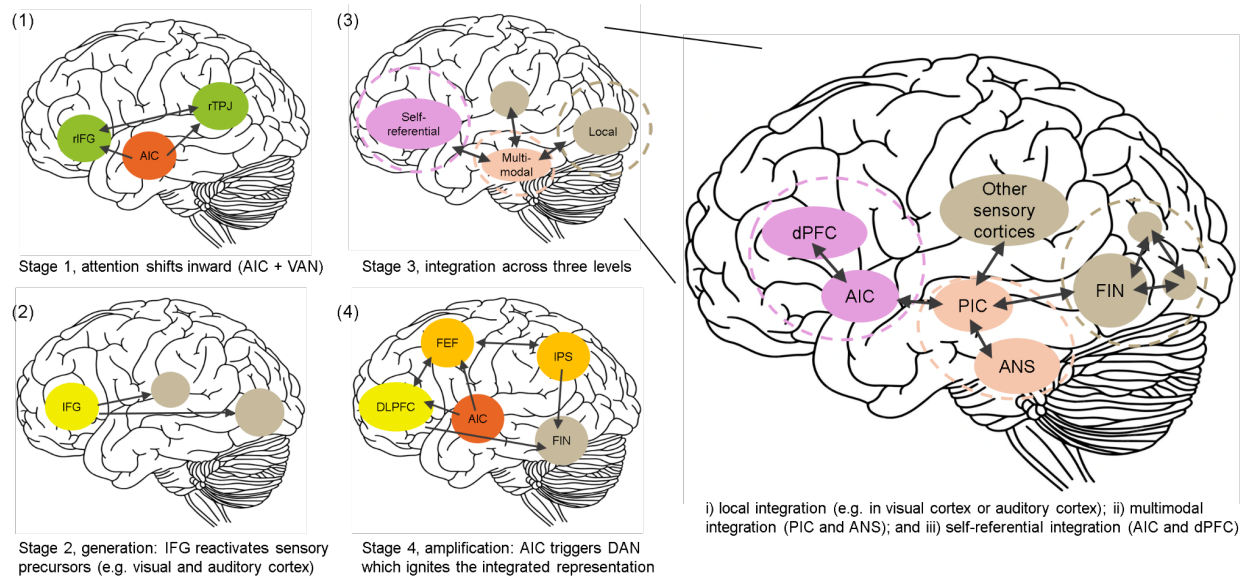
imagery. In short, imagery is uniquely fragile because it is integration-heavy yet unmoored from external stabilisation.

Together, in the integration model, each stage plays a distinct role: orienting attention inward (Stage 1), (re)activating sensory precursors (Stage 2), integrating features across sensory modalities and linking to the sense of self (Stage 3), and amplifying activity into consciously accessible imagery (Stage 4). Disruption at any of these stages—whether attentional, sensory, integrative, or amplificatory—prevents imagery from achieving vivid phenomenology. This, we propose, is the core mechanism underlying aphantasia.

**Table 2.** Comparison of local, multimodal, and self-referential integration

<b>Integration levels</b>	<b>Neural substrates</b>	<b>Function in imagery</b>	<b>Result of failure</b>
Local (within-modality)	Sensory cortex, recurrent feedback loops	Combines sensory precursors into coherent perceptual objects or scenes; elevates sensory precursors to perceptual representations	Activations remain fragmented sensory precursors; lack of perceptual geometry; absence of unconscious priming effects (e.g., binocular rivalry)
Multimodal	Anterior/posterior insula, parietal hubs	Combines separate representations from different modalities and interoceptive signals into multimodal perceptual representations	Representations remain unimodal, schematic or conceptual

Self-referential	Anterior insula, left IFG, salience network	Confers ownership and agency; transforms multimodal content into subjectively “owned” experience	Imagery remains detached, depersonalized, and not affectively grounded
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**Figure 2. Stages of processing for subjective imagery through the integration of attention and interoceptive streams.** The model outlines four stages through which internal visual representation is integrated and crosses the threshold into conscious imagery. Stage 1: The anterior insula cortex (AIC) and ventral attention network including right temporoparietal junction (rTPJ) and right inferior frontal gyrus (rIFG), shift attention inward. Stage 2: The inferior frontal gyrus (IFG) retrieves conceptual and episodic information from medial temporal areas and reactivates sensory precursors, that is, stored fragmented representations in the sensory cortex, establishing pre-integrated visual fragments. Stage 3: Integration occurs at three levels: (i) local- or within-modality binding of visual features in the visual cortex into perceptual representations; (ii) multimodal integration of sensory modalities (e.g. visual and auditory) and interoceptive signals through posterior insular cortex (PIC) and autonomic nervous system (ANS); and (iii) self-referential integration linking internal visual representation to the sense of self via the AIC and the dorsal prefrontal cortex (dPFC). Stage 4: Amplification, in which the AIC engages the dorsal attention network including frontal eye field (FEF), intraparietal cortex (IPS) and dorsolateral prefrontal cortex (DLPFC), igniting the integrated representation to elevate preconscious representations into conscious, embodied imagery. Disruptions at any stage can lead to schematic or propositional recall without phenomenological imagery, as in aphantasia.

## **5. The integration model in the context of existing theories of mental imagery and aphantasia**

In this Section, we will consider the integration model in the context of existing theories of mental imagery and aphantasia. First, we will argue that our model extends theories of mental imagery by highlighting a multi-stage integration process that goes beyond reactivation (Section 5.1). Then, we will critically discuss two theories of aphantasia, namely the spatial imagery theory and the episodic system theory, and locate them within our framework (Section 5.2).

### **5.1 The integration model and other mental imagery theories**

The current models of visual mental imagery either propose a reverse visual hierarchy (Dijkstra, Zeidman, et al., 2017; Nanay, 2023; Pearson, 2019) or a heterarchy (Liu, Zhan, et al., 2025; Spagna et al., 2024). Both models agree on the top-down imagery generation stages, starting from the initiation of the mental action in the prefrontal cortex (step 1), to the retrieval of stored information and memories from medial temporal areas (step 2), and culminating in the (re)activation of sensory and spatial representations in the visual cortex (step 3). However, the models differ on the role of early visual areas (EVA) and high-level visual areas in imagery experience. The reverse hierarchy model (Pearson, 2019) posits that imagery phenomenology results from an interplay of top-down signal strength and noise in the early visual cortex, with the absence of imagery in aphantasia possibly being caused by disruptions in the ventral visual pathway. In contrast, the heterarchy model proposes a heterarchical (i.e., horizontally- and vertically-organized) network of domain-general (FIN) and domain-preferring regions (e.g., fusiform face area for faces) on high-level visual cortex. Importantly, the model identifies the FIN



as a key hub that bridges conceptual, episodic, and domain-specific visual information to generate imagery.

The reactivation of sensory precursors in the visual cortex initiated by the IFG, corresponding to the stage 2 in our integration model (Fig 2), however, is not sufficient for imagery phenomenology. Instead, our integration model elaborates stages that occur *after* the reactivation of the visual cortex, with multi-step integration processes and subsequent amplification (stages 3 and 4 in our model respectively) marking necessary additional steps for achieving imagery experience. Furthermore, while an insufficiently strong top-down signal from prefrontal areas or a disproportionally high noise-to-signal ratio in the visual cortex, in line with Pearson, might lead to an inability to properly (re)activate sensory precursors, which would be synonymous with a failure of achieving stage 2 in our model, data from aphantasics showing stimulus-specific activity in early (Chang, Zhang, et al., 2025; Weber et al., 2024) and higher (Liu, Zhan, et al., 2025) sensory regions during imagery tasks, strongly suggest that most aphantasics do in fact achieve this stage. Instead, failure in aphantasics' imagery generation seems to occur after the reactivation of sensory precursors, namely at least at the stage where they are locally integrated into perceptual representation (stage 3 of our model).

Agreeing with the role of local integration of the FIN in the heterarchy model, the integration model extends it three important ways: First, it distinguishes between sensory precursors and perceptual representations in unconscious processing, and argues that even if the FIN integration fails to elevate precursors to a perceptual level, information can still be utilized for conceptual representation (Liu, 2025). Second, our model specifies three distinct integration stages: local, multimodal, and self-referential, which feature crucially into imagery phenomenology. Third, we highlight a parallel processing pathway with interoceptive coupling

and agency through AIC, adding an embodied dimension largely absent from the dorsal attentional processing for imagery (Liu, 2025).

Moreover, emerging evidence showed that aphantasia may have altered EVA activity reflected by higher activation and enhanced *deactivation* in different EVA subareas, and less *negative* occipital functional connectivity (Chang, Zhang, et al., 2025; Liu, Zhan, et al., 2025; Monzel, Leelaarporn, et al., 2024). This evidence suggests that rather than supporting imagery representation directly, the EVA may be implicated in impaired inhibitory control in aphantasia. Liu (2025) explains such heterogeneous imagery-related visual activity in aphantasia by pointing to deficits in top-down modulation from higher-order regions, in particular an *excitatory–inhibitory imbalance* associated with interacting attention networks. Specifically, in typical imagery, inhibitory control exerted by the VAN over posterior occipital areas suppresses interference from external stimuli and neural noise thereby stabilizing internally generated images. However, aberrant activation in the VAN observed in aphantasia may disrupt subjective imagery by either interrupting ongoing activity in more dorsal frontoparietal networks, or destabilizing excitatory–inhibitory balance in sensory cortices. A plausible mechanism is impaired filtering of noise or distracting events, which in turn interferes with internally generated models.

In summary, our model, based on the unification of the attention and interoception models, extends the mechanisms of representation in visual mental imagery theories to a multi-step mechanism of the emergence of imagery phenomenology, therefore, providing a testable hypothesis for its absence in aphantasia.

## 5.2 The integration model and other theories of aphantasia

Since we already commented on unconscious imagery theories in depth (Section 2.1-2), we will here focus on two other prominent theories of aphantasia, namely the spatial imagery (Section 5.2.1) and episodic memory (Section 5.2.2) accounts.

### 5.2.1 Spatial imagery theories (Phillips, Teng)

Phillips (2025) and Teng (2025) argue that aphantasia should be understood as retained spatial imagery abilities in the absence of object imagery. In doing so, they follow Pearson's (2019) suggestion of intact dorsal visual stream processing in aphantasia and point out that aphantasics score similarly to typical visualizers on the spatial subscale of the *Object and Spatial Imagery Questionnaire* (OSIQ; Blajenkova et al., 2006), a self-report measure that separates object and spatial imagery. Interestingly, they also argue against unconscious imagery accounts (Nanay, 2021b), on the grounds that aphantasics' self-reports indicate retained spatial imagery phenomenology, which may be overlooked by the VVIQ.

However, the spatial imagery theory, and especially a strong dissociation between preserved dorsal and impaired ventral visual stream for imagery in aphantasia (Pearson, 2019) fail to explain the preserved spatial processing in the ventral visual stream, e.g., during attempts of visualization of map, aphantasics showed retained activity in the parahippocampal place areas for processing spatial relationship between locations of cities (Liu, Zhan, et al., 2025). Furthermore, against the suggestion that aphantasics retain conscious spatial imagery phenomenology, it should be noted that while some items on the spatial subscale of the OSIQ do probe experience (e.g., "My images are more like schematic representations of things and events rather than detailed pictures"), most assess self-perceived abilities and preferences (e.g., "I was very good in 3-D geometry as a

student”; “My graphic abilities would make a career in architecture relatively easy for me”). Thus, while OSQI spatial scale scores correlate with spatial imagery abilities, such as mental rotation, inferences from OSIQ scores to spatial imagery *phenomenology* may be premature.

Viewed through the lens of the integration model, spatial imagery might be understood as a case where aphantasics successfully reactivate spatial sensory precursors in either dorsal and ventral visual streams but fail at subsequent local integration steps required for perceptual representations. Spatial templates for size, distance, and orientation may therefore remain accessible, explaining competence in spatial tasks (e.g., Pounder et al., 2022; Liu & Bartolomeo, 2023; Kay et al., 2024), but without integration with ventral stream attributes such as color and texture they cannot yield vivid perceptual imagery. Thus, their accounts can be reinterpreted as describing a case in which the reactivation of spatial precursors may be intact but integration with non-spatial features (e.g., from the ventral visual stream) fails, already at a local level.

However, contrary to Phillips and Teng, we hold that it is not only spatial precursors that remain intact in aphantasics. Liu, Cohen, et al. (2025) showed stimulus-specific representations coding for features such as colors and faces in aphantasics (although these representations, as noted above, did not show representational overlap with equivalent stimuli during perception). Since color is typically associated with object imagery, we therefore take an object-imagery distinction to be too coarse-grained to explain aphantasia.

### **5.2.2 Episodic system theories (Blomkvist)**

Blomkvist (2023) has argued that aphantasia should be understood primarily as an episodic system condition rather than as a deficit of visual imagery per se. In her model, semantic memory and episodic memory indices from each sensory modality (e.g., visual, auditory) are retrieved by separate retrieval processes and then recombined. Therefore, she posits that aphantasia might be

caused either by deficits in indexing, retrieval, or recombination. However, she discards the recombination idea since she argues that semantic memory, which is largely intact in aphantasics, also requires a recombination process.

If aphantasics would have issues with indexing or retrieval, as suggested by Blomkvist, we should not expect them to activate sensory precursors successfully (stage 2 in our model). However, since there is strong evidence for stable representations in sensory cortices in aphantasics (Chang, Zhang, et al., 2025; Liu, Zhan, et al., 2025) during imagery tasks, the reactivation of sensory precursors, which rely, at least partially, on retrieval from hippocampal areas, seems to be intact, thus pointing against indexing and retrieval deficits. If, on the other hand, the recombination process fails, this would be equivalent to a failure of multimodal integration in our model. But since the local integration may fail in aphantasia, as evidenced by the non-perceptual format of their representations, the multimodal integration step might be less determinant for most aphantasics if they do not even reach this step. Furthermore, even for those aphantasics who might achieve local and multimodal integration, self-referential integration would still be an important additional step towards imagery experience. Thus, our model goes further than mere recombination by suggesting that the multisensory integration may not be the determining effect for aphantasia. Instead, aphantasics likely already fail at local integration or, in a potential unconscious imagery aphantasia subtype, where local integration succeeds, may fail to render their representations with self-referential processing and agency.

Thus, while Blomkvist rightly emphasizes the role of episodic retrieval and recombination, the integration model both accommodates her account and extends it by clarifying which levels of integration are implicated, as well as by highlighting additional processes (i.e., self-referential integration) that her theory leaves aside.

## **6. Clinical implications of the integration model**

Last, we focus on the clinical implications of the integration model by deriving a common etiological framework for aphantasia and associated (sub)clinical conditions that may emerge from the same underlying mechanisms of the integration model (Section 6.1). In Section 6.2, we shift the perspective to potential interventions that may lead to a better integration of mental imagery in those conditions, making our assumptions more testable.

### **6.1 A common etiological model of aphantasia and associated conditions**

Given the common co-occurrence of congenital aphantasia and (subclinical) symptoms of neurodevelopmental disorders and neutral neurodivergences such as congenital prosopagnosia (Dance et al., 2023; Monzel et al., 2023), autism spectrum disorder (ASD; Dance et al., 2021; King et al., 2024), alexithymia (Monzel et al., 2024), and ADHD (Gao et al., 2025), a common etiological framework can be assumed. Sokolowski and Levine (2023) suggest that “seemingly distinct neurodevelopmental disorders share a dysfunction in long-range connections between the core and periphery of the human connectome” (p. 444). The extension of this view to aphantasia is supported by the recently found functional disconnection between frontoparietal networks and the visual cortex in aphantasics (Liu & Bartolomeo, 2025). There is comparable evidence for similarly reduced functional connectivity in the interoceptive network in alexithymia (Terasawa et al., 2021) and autism (Nomi et al., 2019) as well as in the attention network in ADHD (Bush, 2009; Metin et al., 2024). Interestingly, some approaches even discuss a failure to integrate visual information as the cause for visual agnosia (i.e., the inability to recognize or interpret visual information) (Riddoch & Humphreys, 1987), which is in line with our previous argument that mental imagery should have a similar representational geometry to perception.

The convergence of the attentional (Liu, 2025) and interoceptive model (Silvanto & Nagai, 2025) through the AIC and salience network provides a unified etiological framework for understanding aphantasia and its associated conditions. Extensive evidence demonstrates that bodily signals and their abnormal processing play crucial roles in these conditions (Silvanto & Nagai, 2025), with autonomic dysregulations producing abnormal interoceptive input and disrupted interoceptive attention in the AIC (Wang et al., 2019) further disrupting the attention-interoception coupling necessary for conscious imagery. The high comorbidity between the above mentioned conditions reflects these shared disruptions in the attention-interoception integration that transforms non-phenomenal representations to mental imagery (Mørch et al., 2024; Kvamme et al., 2024). For example, individuals with ASD show reduced interoceptive accuracy—diminished ability to detect bodily signals—combined with exaggerated interoceptive sensibility, or high subjective confidence in their abilities (Garfinkel et al., 2015). This represents a decoupling of objective internal states from subjective awareness. Conversely, acquired aphantasia, which correlates with higher scores in inattention (a core feature of ADHD), is associated with an impaired ability to integrate bodily and cognitive information (Gao et al., 2025). This may result from a deficit in attentional control, which prevents individuals from sustaining focus on subtle interoceptive signals (Silvanto & Nagai, 2025). Both patterns prevent representations from reaching consciousness, though through different mechanisms.

Traditional studies have recognized acquired forms of aphantasia since the 1900s (Charcot & Bernard, 1883), which has been more recently highlighted by Zeman's seminal case study of a patient who lost his imagery ability following heart surgery (Zeman et al., 2010). Zeman's recent review (2024) brings acquired aphantasia into focus, noting that it can arise from neurological injury (*neurological aphantasia*), psychological trauma (*psychogenic aphantasia*), or both (see

Bartolomeo, 2025, for a discussion). Recent research reveals that 62% of individuals with acquired aphantasia reported psychological triggers, including depression, anxiety, PTSD, or severe stress, while 41% cited neurological events and 30% identified pharmacological factors, with nearly half describing multiple contributing factors. These individuals showed significantly higher levels of childhood trauma and increased supra-diaphragmatic autonomic reactivity compared to typical imagers (Gao et al., 2025). Through the integrated lens, trauma simultaneously overwhelms both systems: the autonomic nervous system becomes hypersensitized (disrupting interoceptive processing) while attentional control becomes impaired (fragmenting top-down imagery generation). The resulting dissociation pushes representations below to a non-phenomenal state—they continue to exist but cannot be integrated to conscious mental imagery due to the breakdown in attention-interoception coupling (Silvanto & Nagai, 2025).

Recent findings on the salience network in depression reveal a mechanism through which attention-interoception integration becomes disrupted. Lynch et al. (2024) found that the frontostriatal salience network expands to nearly twice its normal territory in individuals with depression. The expansion was stable over time, unaffected by mood state changes, and detectable in children before the subsequent onset of depressive symptoms in adolescence. The expansion likely reflects dysregulation in both systems simultaneously: the expanded network may attempt to compensate for weakened attention-interoception integration by recruiting additional cortical territory. However, this compensation paradoxically results in a "hypervigilant" state where negative unconscious content floods consciousness while positive imagery remains inaccessible—the expanded network loses its selectivity in determining what should become conscious. Similarly, in PTSD, traumatic imagery may bypass normal conscious control by overwhelming both the attentional capacity and interoceptive integration simultaneously. The salience network,



unable to properly gate these representations, allows traumatic content to intrude into consciousness as flashbacks. Conversely, in dissociative states, the salience network may become hypoactive, preventing even adaptive imagery from reaching consciousness.

## **6.2 Mindfulness as attention-interoception integration**

The integrated view of mental imagery has immediate therapeutic implications. For individuals with aphantasia and associated conditions interventions targeting only one system may be insufficient. Instead, treatments need to address the coupling between attention and interoception—strengthening their coordination rather than enhancing either in isolation.

Mindfulness-based interventions offer a compelling example of how therapeutic approaches can target the attention-interoception interface. The MBSR program employs both focused attention meditative techniques (body scan and yoga) and open monitoring techniques (sitting meditation), with all three requiring different attentional styles while modulating the insula and interoceptive network (Gibson et al., 2019). By simultaneously training interoceptive awareness (through body scanning) and attentional control (through focused attention practices), mindfulness directly strengthens the coordination between both systems (Farb et al., 2013). The body scan practice specifically involves "moving a focused spotlight of attention from one part of the body to another", teaching practitioners to control attention even when focusing on aversive sensations and to notice how familiar body sensations fluctuate from moment to moment (Kerr et al., 2013).

The 8-week Mindfulness-Based Stress Reduction (MBSR) program increases gray matter density in brain regions including the hippocampus, posterior cingulate cortex, and temporo-parietal junction (Hölzel et al., 2011). Farb et al. (2013) found that mindfulness training predicted

greater interoceptive attention-related activity in anterior dysgranular insula regions, consistent with greater integration of interoceptive sensation with external context. A systematic review and meta-analysis by Pernet et al. (2021) identified the right anterior ventral insula as the only significant region with consistent structural effects across mindfulness studies with medium to strong effect sizes (Cohen's  $d \sim 0.8$  in the right insula).

Improvements in interoception through mindfulness may reduce autonomic overreactions, which in turn frees attentional resources previously consumed by managing physiological dysregulation. This creates a positive feedback loop where better interoceptive regulation enables improved attention, which further enhances the ability to consciously access mental imagery. This aligns with findings showing that attention control mediates the relationship between imagery vividness and emotion regulation (McKenzie et al., 2024), with imagery vividness predicting greater levels of healthy reappraisal when paired with attention control. Rather than creating imagery where none exists, mindfulness may work by improving access to preconscious representations that were always present but disconnected from conscious experience due to poor attention-interception coordination. This view is supported by a study by (Kjaer et al., 2002), showing that mental imagery vividness increased during Yoga Nidra meditation which involves principles of mindfulness. Additional studies on the association between mindfulness-based interventions and improvements in mental imagery may thus be an opportunity to verify the assumptions of the integration model of mental imagery.

## **7. Conclusion**

Mental imagery is often viewed as a reactivation of perceptual representations in sensory cortices. In line with this view, aphantasia has been explained either as insufficient reactivation of

perceptual representation or as successful reactivation that remains unconscious. We have argued for a different perspective where perceptual representations underlying imagery experience are not simply reactivated, but are the interim result of a multi-level integration process that binds more elementary representations, which we refer to as sensory precursors. Only if these precursors are integrated into perceptual representations, and subsequently bound with interoceptive and self-referential signals, can imagery experience occur.

Our integration model, which builds on recent attention- and interoception-based accounts, thus reconceptualizes imagery as the fragile outcome of multi-level integration rather than mere sensory reactivation. In doing so, it extends contemporary theories of mental imagery and aphantasia, reframes the debate on unconscious imagery, and opens new pathways for linking imagery research with broader work on consciousness and clinical conditions. Furthermore, by offering a conceptual framework that accommodates competing theories of aphantasia and outlines multiple stages at which integration might fail, it provides testable hypotheses that enable a mapping of distinct aphantasia subtypes.

More broadly, aphantasia itself can be seen as a model condition for investigating the neural mechanisms of multi-stage integration. Because imagery is uniquely fragile and heavily dependent on integration, its absence provides a window onto the processes by which distributed sensory and bodily signals, in general, are transformed into conscious experience.

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## Author contributions

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## Declaration of interests

None

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