

Review

Aphantasia and hyperphantasia: exploring imagery vividness extremes

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The vividness of imagery varies between individuals. However, the existence of people in whom conscious, wakeful imagery is markedly reduced, or absent entirely, was neglected by psychology until the recent coinage of 'aphantasia' to describe this phenomenon. 'Hyperphantasia' denotes the converse – imagery whose vividness rivals perceptual experience. Around 1% and 3% of the population experience extreme aphantasia and hyperphantasia, respectively. Aphantasia runs in families, often affects imagery across several sense modalities, and is variably associated with reduced autobiographical memory, face recognition difficulty, and autism. Visual dreaming is often preserved. Subtypes of extreme imagery appear to be likely but are not yet well defined. Initial results suggest that alterations in connectivity between the frontoparietal and visual networks may provide the neural substrate for visual imagery extremes.

Introducing imagery vividness extremes

Most of us can readily summon visual **imagery** (see [Glossary](#)) to the mind's eye – as when we imagine the look of an apple, the appearance of our kitchen, or the smile of our best friend. We can equally imagine the sound of distant thunder, the feel of velvet, and the exertion of running for a bus. Although the British psychologist Sir Francis Galton recognised in the 1880s that for some people the 'power of **visualisation** was zero' [1], this phenomenon was largely neglected until a recent surge of scientific and popular interest. This was triggered in part by the creation of convenient terms with which to refer to imagery extremes: **aphantasia**, the marked reduction or absence of conscious, wakeful imagery; and **hyperphantasia**, visual imagery 'as vivid as real seeing' [2,3]. This review focuses on the growing evidence that subjective reports of aphantasia and hyperphantasia have distinctive behavioural, physiological, and neural correlates.

Nature and mechanisms of visual imagery

Imagery involves the experience of the sensory properties of items or activities in the absence of the items themselves. In the following brief sketch of the psychology of imagery, I focus on the visual **modality**, but similar principles are likely to apply to other forms.

In perception, despite vital contributions from expectation and prediction [4], information flows from the periphery of the nervous system to the centre, thereby allowing recognition of things in the world and appropriate behavioural responses: the process is 'bottom-up'. In a typical imagery experiment, in which a participant must visualise an absent object, the flow of information is reversed: deliberate imagery is summoned 'top-down'. Visual imagery is therefore sometimes described as 'vision in reverse' [5]. In keeping with this view, visual cortices are activated during visual imagery, with a reversal of the dynamics of the perceptual feed-forward cascade [6,7]. Eye movements play a role in imagery, indicating another similarity to perception [8]. The precise extent of the overlap between perception and imagery, in particular the role of early visual areas in imagery, is controversial [5,9–11]: studies using techniques to decode brain activity suggest that

Highlights

Aphantasia and hyperphantasia are recently coined terms that refer to the absence and superabundance of imagery, respectively.

Growing evidence indicates that they have distinctive behavioural, physiological, and neural correlates.

Aphantasia is associated variably with a reduction in both autobiographical memory and face recognition, as well as with autism and a tendency toward scientific occupations. Imagery in dreams is typically preserved.

Despite the profound contrast in subjective experience between aphantasia and hyperphantasia, the effects on everyday functioning are subtle – lack of imagery does not imply lack of imagination.

Differences in neural connectivity may explain the existence of these extremes of imagery.

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imagery can be decoded from early visual areas [12,13], while the clinical literature questions the role of this activity in the experience and functionality of imagery [14].

The 'top-down' component of voluntary imagery is reflected in the involvement of frontoparietal regions implicated in attention and cognitive control [9,10,15]. These guide the key phases of visualisation identified by Stephen Kosslyn – the generation, maintenance, inspection, and transformation of visual images [16]. It is noteworthy, however, that visual imagery is unlikely to be always governed by these processes – imagery during dreams provides a possible counterexample [17], and the images triggered by reading a novel lie in the interesting middle ground between voluntary and involuntary **phantom vision** [18].

In addition to calling on systems involved in sensation and cognitive control, typical imagery tasks require the use of language, memory (short- or long-term, semantic or episodic, depending on the task), and disengagement from our immediate surroundings. Visualisation is therefore a complex process in cognitive terms and is associated with the activation of a widely distributed 'imagery network' (Figure 1A). Studies specifically examining the neural correlates of imagery **vividness** have implicated modal and supramodal regions including higher-order visual cortices and parts of the default mode network [19]. The degree of resemblance between brain activation during imagery and perception probably contributes to the experienced vividness [19,20].

Visual imagery is, for most of us, a ubiquitous feature of conscious experience (Box 1). Indeed, **descriptive experience sampling** suggests that it is the most common conscious content, and even surpasses direct perceptual awareness [21]. Against this background, the observation that a substantial minority of healthy people appear to lack imagery entirely is striking.

The rediscovery of visual imagery extremes

Galton's 'breakfast table questionnaire' from 1880, which invited his participants to rate the 'illumination, definition and colouring' of imagery of their breakfast table and its contents, was the first systematic attempt to measure the characteristics of imagery [1] (Box 2). Galton noted a wide variation in what we would now call the vividness of mental imagery. To his surprise, some of his participants, often 'men of science', protested that 'mental imagery was unknown to them ... they had no more notion of its true nature than a colour-blind man, who has not discerned his deficit, has of the true nature of colour' [1]. Galton's cousin, Charles Darwin, was an exception: his image of the breakfast table included some objects 'as distinct as if I had photos before me' [22].

Although loss of imagery due to neurological and psychiatric disorder was described in a series of case reports from the 1880s onwards [14,23–27] (Box 3), the lifelong absence of imagery, as implied by Galton's description, received relatively little scientific attention over the following century (but see [28–30]). The recent surge of interest was provoked when a scientific report of imagery loss in a middle-aged man [27] was described in *Discover Magazine* [28]. This led to spontaneous contact with the author from 21 individuals who recognised themselves in this description [28], with the difference that they had 'never' been able to visualise [2]. They gave broadly similar accounts of this intriguing variation in their experience. We coined the term 'aphantasia' to provide a convenient label for this phenomenon, borrowing Aristotle's term for the mind's eye ('phantasia') [29] and adding the prefix 'a' to denote its absence. This term and the phenomenon it described attracted much public interest. Among the numerous (~17 000) subsequent contacts of members of the public with my laboratory, some described the opposite variation, imagery 'as vivid as real seeing'. We termed this 'hyperphantasia' [3]. The warmth of the response of people with extreme imagery to the resulting work has been notable, perhaps reflecting the prior neglect of this significant but elusive feature of their inner lives.

Glossary

Aphantasia: an absence or near-absence of conscious wakeful imagery; the term was initially used to refer to an absence of 'visual' imagery but is now often used to refer to an absence of imagery in other/all sense modalities.

Autobiographical memory: our memory of our personal past, including our memory for salient past events such as our first kiss.

Binocular rivalry: the experience that typically occurs when different stimuli are presented to each eye and what we see consciously alternates between the two stimuli.

Depersonalisation: a psychiatric disorder involving a sense of alienation from oneself and one's feelings that is often associated with derealisation – the sense that one's surroundings are no longer fully real.

Descriptive experience sampling: a method of probing for introspective reports of experiences as soon as possible after they occur.

Hyperphantasia: the converse of aphantasia – imagery rivaling the vividness of perceptual experience.

Imagery: the experience of the sensory properties of items or activities in their absence, such as the look of an apple or the sound of thunder

Imagery generation: the cognitive/neural process by which images are called to mind.

Imagery strength: this term is used technically in the context of binocular rivalry experiments to refer to the degree of the priming effect of imagery.

Introspection: our ability to detect and report our own conscious experience.

Metacognition: our ability to make accurate judgements about our own cognitive states and processes, for example whether we do or do not experience mental imagery.

Mind pops: mental contents, such as an image of a place or person, that appears to come to mind spontaneously.

Modality: refers to a particular sensory channel; for example, we can experience imagery in both visual and auditory modalities.

Object imagery: imagery depicting objects and their characteristics such as shape and colour; such imagery is markedly reduced in aphantasia.

The prevalence of extreme imagery

Several recent studies have used versions of the **vividness of visual imagery questionnaire (VVIQ)**, originally developed by David Marks [30], to estimate the prevalence of aphantasia. The VVIQ typically asks participants to visualise 16 scenarios which they rate on a five-point scale from 1 ('no image at all, you only know that you are thinking of the object') to 5 ('perfectly clear and vivid as real seeing'). Mean and median values in large samples are around 55–60/80 [3,31,32]. These studies suggest that ~1% of the population experiences profound aphantasia and have the lowest possible VVIQ score (16/80), whereas 2–6% experience imagery that is at best 'vague and dim', and have VVIQ scores of 32/80 or less [3,32–36]. Hyperphantasia, which has received less attention in recent studies, appears to be more common than aphantasia. We found that 2.6% of 1288 participants from a community biobank scored 80/80, whereas 11.2% scored 75–80/80 [3]. Of 5010 participants recruited online, 5.9% scored 75–80/80 [33]. Evidence for the influence of age on imagery vividness extremes is conflicting [33,37]. There is no apparent gender bias in aphantasia [3,32], but women may be more prone to hyperphantasia [3].

Subjective reports and objective correlates

The VVIQ depends entirely on **introspective** reports – **metacognitive** judgements about the vividness of mental images. Given the well-recognised pitfalls of introspection [38], a sceptic could reasonably question whether aphantasia and hyperphantasia reflect authentic individual differences or are instead artifacts of noisy or biased metacognition. The detailed, consistent first-person accounts given by people with aphantasia and hyperphantasia, including their experience of imagery in dreams (discussed below), argue against this sceptical view [39,40]. More persuasively for the sceptic, perhaps, behavioural, physiological, and neural data are beginning to corroborate these accounts.

Three sets of objective correlates with subjective reports of aphantasia have emerged in recent work [41–43]. First, a **binocular rivalry** technique that was developed to measure **imagery strength** has been applied to people with aphantasia [41]. In participants with imagery, imagining one of two binocular stimuli before their presentation biases perception toward the previously imagined stimulus, and the extent of the priming effect can be used to estimate imagery strength. This priming effect proved to be virtually absent in participants with aphantasia. Second, whereas normal participants listening to (extremely) scary stories show a hike in their galvanic skin response (GSR) (they sweat!), people with aphantasia do not [42]. By contrast, people with aphantasia show a normal GSR in response to aversive pictures, demonstrating that they generally do not lack this capacity. The natural interpretation is that, in the absence of imagery, the impact of emotive language is reduced because imagery typically mediates between verbal description and emotional response. A third approach [43] builds on the observation [44] that the pupils of the eye react to imagined illumination, constricting when one imagines looking into a sunny sky, and dilating when one imagines looking into a darkened room. By contrast, people with aphantasia did not show evidence of an imagery-driven pupillary response, despite normal responses to (real) light and cognitive load.

These data validate subjective reports with objective measures, and fall into place alongside behavioural evidence that reports of extreme imagery are indicative of broader psychological phenotypes, as discussed in the following section, further supported by the brain imaging studies discussed later.

Associations and dissociations with extreme imagery

The marked differences in subjective experience caused by imagery extremes might be expected to have major behavioural effects. However, in general, people at both extremes of the vividness

Phantom vision: another term for visual imagery; the term is used to emphasise its varied manifestations (e.g., voluntary and involuntary).

Prosopagnosia: a 'clinically significant' difficulty with face recognition.

Rapid eye movement (REM) sleep: a type of sleep that is strongly associated with dreaming.

Sibling recurrence risk: the elevation in the likelihood that a trait or condition is present among siblings in comparison to the general population.

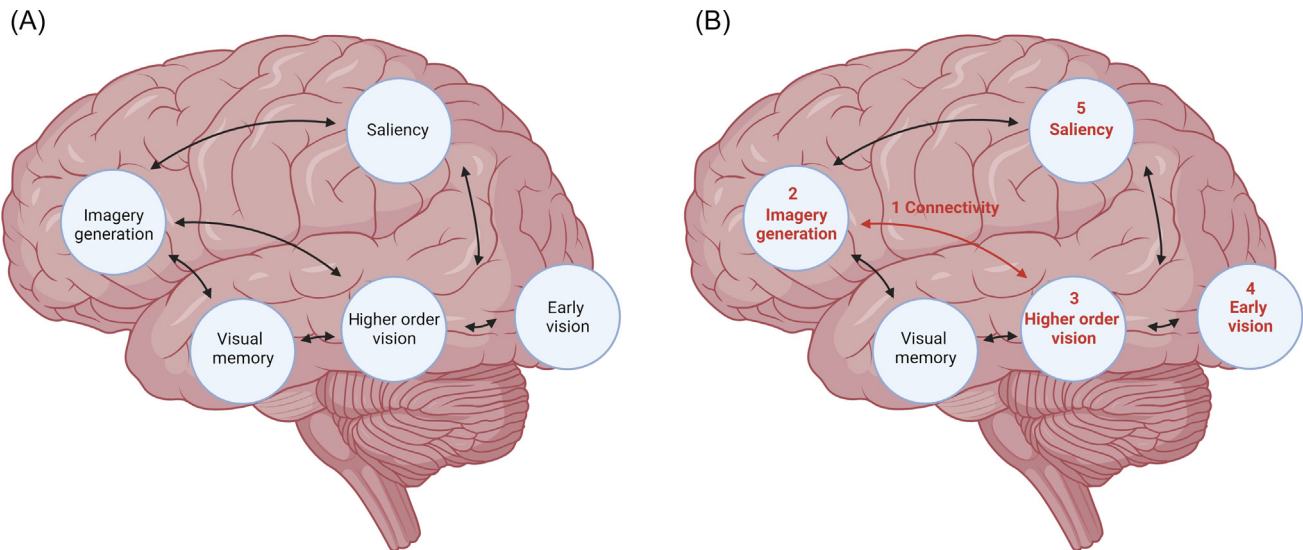
Spatial imagery: imagery for spatial relationships that is often spared in aphantasia.

Synaesthesia: the phenomenon whereby 'one quality of experience is accompanied by an involuntary unrelated secondary experience' (e.g., experiencing specific colours when hearing specific words).

Visualisation: the process by which we call visual images of things to mind in their absence, whether deliberately or not.

Vividness: the extent to which imagery experience resembles perceptual experience e.g., vivid visual imagery closely resembles 'real seeing'.

Vividness of visual imagery questionnaire (VVIQ): a widely used measure of imagery vividness [30].



Trends in Cognitive Sciences

Figure 1. The neural bases of typical and extreme imagery. (A) A highly schematic rendering of key regions involved in voluntary visual imagery. Frontal regions initiate **imagery generation** in response to task demands; parietal regions interact with frontal regions to generate imagery, mediating attentional and spatial aspects of imagery ('saliency'); temporal regions including limbic structures enable access to the semantic and episodic memories that determine what we visualise; whereas activity in higher-order visual areas, including the recently described fusiform imagery node (FIN), gives imagery its 'visual feel'. However, the extent to which activation in early visual cortices, including primary visual cortex (V1), is required for imagery remains controversial. Imagery often also calls on areas involved in language and eye movements, not shown here. (B) Candidate mechanisms for aphantasia and hyperphantasia. (1) variations in connection strengths between higher-order regions involved in cognitive control and modality-specific areas activated by sensory imagery. (2) Variations in the volume or efficacy of frontal regions involved in imagery generation. (3) Variations at the level of higher-order visual cortices including the FIN. (4) Anatomical variation in early visual areas including V1. (5) Variation of function in the parietal arm of the frontoparietal control system for imagery.

spectrum manage their everyday lives perfectly well. But while neither aphantasia nor hyperphantasia is disabling, a range of behavioural associations has come to light.

Occupation

As Galton might have predicted [1,45], aphantasia is over-represented among people working in mathematical, computational, and scientific roles, whereas people with hyperphantasia are more likely to work in traditionally 'creative' industries [3,34]. These results are in keeping with prior evidence that levels of **object imagery** are elevated among people working in visual arts, whereas high levels of **spatial imagery** characterise people working in the sciences, engineering, and architecture [46,47].

Autobiographical memory and future thinking

On a range of standard experimental measures of memory (short- and long-term, verbal and visual), people with aphantasia perform normally or show, at most, mild to moderate impairment [33,48–54]. Average IQ is, if anything, marginally higher in people with aphantasia than hyperphantasia [50]. However, one aspect of memory, that is typically neglected in standard assessments, is more markedly reduced in aphantasia, as suggested by anecdotal reports [39,40]. Both subjective [3,55] and objective [50,56] measures of the richness of **autobiographical memory** are reduced in aphantasia but increased in hyperphantasia [3,50]. In keeping with the close relationship between recollecting the past and imagining the future [57], the richness of descriptions of imagined scenes is also reduced in aphantasia [50,56]. Aphantasia similarly reduces the detail of eye-witness testimony [58].

Box 1. Contexts and functions of imagery

Mental imagery, as defined in the main text, occurs in a wide range of everyday contexts including dreaming and daydreaming, autobiographical recollection, contemplation of future possibilities, planning and problem-solving, visual memory tasks, navigation, mental practice, and reading descriptive prose. It also occurs in unusual or pathological mental states, for example in hallucinations, in association with cravings, and in the flashbacks of post-traumatic stress disorder. Many creative people have described the important role apparently played by imagery in their thinking [109–111]. Although correlation does not in itself prove causation, these examples, and experimental work that they have inspired, suggest that imagery at least sometimes supports related psychological processes including memory (both short- and long-term), future-thinking, mental practice, creativity and problem-solving, and motivation. Aphantasia prompts us both to reassess the role of imagery in these domains and to consider the potential for individual differences – the fact that some of us use imagery to perform a particular task does not imply that everyone does so; conversely, the fact that others do 'not' use imagery in a particular context does not imply that no one does so.

In some people with aphantasia, the reduction in autobiographical memory is sufficiently severe to suggest the coexistence of the recently described 'syndrome of severely deficient autobiographical memory' (SDAM) [59]. People with SDAM lack vivid recollections of personally experienced events from a first-person perspective despite normal functioning in everyday life. At least one of the three individuals described in the first report of SDAM is also aphantasic [39]. Given the important role played by imagery in autobiographical memory [60], the relationship between SDAM and aphantasia should be investigated further.

Box 2. Methods to quantify imagery

There are four broad approaches for quantifying imagery: questionnaires that rely on first-person reports, behavioural approaches utilising tasks that require (or are assumed to require) imagery, physiological methods that measure bodily responses associated with imagery, and neuroimaging techniques that directly track the neural correlates of imagery.

Questionnaires

Galton's breakfast table questionnaire, that invites participants to self-rate aspects of their own imagery, has many descendants, including the VVIQ [30] and OSIQ [112] that are discussed in the main text. These include questionnaires that assess imagery across the range of sense modalities, such as the Plymouth sensory imagery questionnaire [113], and questionnaires that assess the use made of imagery in everyday life, such as the spontaneous use of imagery scale [114]. These scales, which essentially quantify introspection, are a natural point of departure for imagery research, but, given the pitfalls of introspection, they should where possible be combined with more objective approaches.

Behavioural measures

Numerous behavioural measures have been developed to probe imagery processes [115], including the binocular rivalry method discussed in the main text [41]. Measures of 'object imagery' often utilise questions that most of us would answer with the help of imagery (which is darker, the green of grass or the green of a pine tree? Which is larger – a hazelnut or a walnut?), whereas measures of spatial imagery utilise tasks such as cube rotation and mental map scanning [16]. Accuracy and reaction times are measured to quantify performance. Studies of aphantasia have identified a shortcoming in some measures of object imagery: patient MX with acquired aphantasia [27] and at least two groups of participants with lifelong aphantasia [3,66] perform accurately on these measures (albeit with prolonged reaction times in [66]). Conscious imagery does not appear to be necessary to answer 'high imagery questions' accurately. Whether this reflects the use of unconscious imagery or purely semantic cognition ('just knowing') remains unclear.

Physiological measures

Two examples of physiological measures linked to imagery are discussed in the main text – measurement of the pupillary response to imagined scenes [43] and measurement of the GSR in response to scary stories [42] (which appears likely to be mediated by visual imagery).

Neuroimaging measures

To date, neuroimaging has been used primarily to identify the neural correlates of imagery performance which has been assessed by other methods, often questionnaires. Knowledge of the neural basis of imagery through neuroimaging could in principle be used to determine the presence or absence of conscious imagery. Studies of the neural correlates of aphantasia are discussed in Box 4 and the main text.

Box 3. Imagery lost and found: acquired aphantasia and imagery acquisition

Neurological and psychiatric cases of imagery loss secondary to injury or disease have been reported sporadically since the 1880s [14,23–27]. A landmark review of the available literature distinguished three types of neurogenic imagery loss: loss due to failure of ‘imagery generation’ (with intact perception) or to impairment of ‘long-term visual memory’ or visual ‘inspection’ (both associated with visual agnosia) [25]. Based on lesion location, this review identified the posterior left hemisphere as being crucial for the imagery generation process. A survey of the more recent literature [14] concludes that higher-order visual areas play a more important role in imagery than lower-order regions, a conclusion supported to some extent by functional imaging studies [4,19,20]. In parallel with the slowly growing neurological literature on acquired aphantasia, psychiatrists have reported the occurrence of aphantasia in the context of depression [26], **depersonalisation** [26,116,117], and psychosis [24,26].

Cases of imagery loss are relevant to the study of lifelong aphantasia for two key reasons. First, cases caused by well-defined brain lesions have the potential to illuminate the underlying neurology of visualisation and complement evidence obtained by functional imaging in people with typical and extreme imagery. Second, the existence of ‘psychogenic’ causes of aphantasia is important in its own right [118], but should also be borne in mind in recruiting participants to future studies and in interpreting the results (see [119] for related insights).

The question of whether imagery can be acquired by people with lifelong aphantasia has been raised repeatedly, although our participants mostly describe curiosity about conscious imagery rather than a sense of handicap from its absence. This question awaits systematic study, although anecdotal evidence suggests that it will be difficult for people with aphantasia to learn to visualise. Potentially relevant leads include evidence that direct current brain stimulation modulates the vividness of imagery in people who possess it [93], and two single case reports on the effects of hallucinogens in individuals with aphantasia [120,121].

Face recognition, perception, and synaesthesia

Around 40% of people with aphantasia describe difficulty with face recognition, more than twice the frequency among people with average imagery vividness or hyperphantasia [3]. This is reflected in increased scores on a **prosopagnosia** questionnaire [36,50]. It is in keeping with the prior observation that people with prosopagnosia have, on average, reduced imagery vividness [61]. Two studies have examined face perception objectively using the Cambridge face memory test [62,63]. Both detected an impairment in face recognition among people with aphantasia. One [63] also obtained evidence for impairment of face-matching. However, in [62] the reduction in face recognition was matched by subtle impairment on an object recognition task, suggesting that the impairment may not be face-specific. As discussed later, aphantasia is probably heterogeneous: one subgroup might be characterised by coexisting prosopagnosia.

Other evidence (Table 1) points to subtle interactions between aphantasia and perception more generally. Such effects might be predicted given that imagery can influence perception [4,5], but current data are conflicting. Several studies have suggested that people with aphantasia process visual tasks more slowly than people with normal imagery [64–67]. These interactions offer another fruitful area for future study.

Synaesthesia, in which ‘one quality of experience is accompanied by an involuntary unrelated secondary experience (e.g. hearing sounds gives rise to seeing colours)’ is, like aphantasia, an intriguing phenomenon with profound effects on subjective experience but more subtle effects on performance [68]. Questionnaire data suggest an increased rate of synaesthesia among people with hyperphantasia [3], consistent with the prior observation that average imagery vividness is increased in people with synaesthesia [69]. However, questionnaire data must be interpreted cautiously because first-person reports of synaesthesia are not always supported by objective evidence [70]. Using the gold-standard method for diagnosing grapheme–colour synaesthesia, one study found no reduction in its frequency overall among people with aphantasia [70]. However, synaesthesia among people with aphantasia had a more ‘associative’ and less ‘projective’ quality than among non-aphantasic synaesthetes (the distinction is between ‘knowing that’ the letter ‘K’ is green and ‘seeing’ it green).

Table 1. Studies of perception in aphantasia

Author	Number of aphantasic participants	Aspect of perception	Result
Dance <i>et al.</i> [72]	164	Sensory sensitivity Pattern glare effects	Reduced Reduced
Konigsmark <i>et al.</i> [126]	33	Flicker induced illusions	Reduced
Monzel <i>et al.</i> [64]	531 (Experiment 1) 325 (Experiment 2)	Moriya visual search task Spontaneous use of imagery visual search task	Normal Reduced
Milton <i>et al.</i> [50]	24	Face recognition	Normal (ceiling effect)
Keogh <i>et al.</i> [127]	10 (Experiment 2) 15 (Experiment 3)	Sensory strength of attentional templates	Reduced
Monzel <i>et al.</i> [65]	104	Visual search speed (hidden-object pictures)	Reduced
Monzel <i>et al.</i> [62]	65	Face recognition Car recognition	Reduced Reduced
Dance <i>et al.</i> [63]	52	Face recognition Face composite creation Face composite rating	Reduced Normal Reduced
Liu <i>et al.</i> [66]	44	Perceptual comparisons involving shape, colour, faces, letters, spatial relationships	Normal accuracy, but reduced speed for shape, colour, and faces
Cabbai <i>et al.</i> [67]	11 (Experiment 1) 97 (Experiment 3)	Operation of attentional templates assessed by contingent capture effect	Normal (experiment 1) Normal, but aphantasic responses were slower, overall less efficient, and the reported strategy was non-visual (experiment 3)

Imagery in other sense modalities

Ten of 21 individuals first described with aphantasia reported that imagery in 'all' sensory modalities was absent or faint [2]. In a sample of 2000 individuals with aphantasia and 200 with hyperphantasia, 54.2% and 47.8% respectively reported that all modalities were affected [3]. Other studies [36,55,70,71] have confirmed the finding that imagery extremes are often multi-modal. However, some individuals describe 'dissociations' between imagery types, for example, visual aphantasia combined with a vivid 'mind's ear', suggesting that both modality-general and modality-specific factors are in play.

These observations have led to debate over terminology, and it has been proposed that specific terms should be coined to refer to the lack of imagery in each sense modality, for example 'anauralia' for lack of the mind's ear [71]. 'Dysikonesia' has been suggested as a term for lack of imagery across the range of sense modalities [72]. We suggest that it is simpler to preserve the established terms aphantasia and hyperphantasia, and to qualify these as required – for example as 'visual', 'auditory', or 'global' [73,74].

One important but less conspicuous – and perhaps less 'conscious' [75] – imagery modality may be preserved in aphantasia as hitherto defined. A double dissociation between object and spatial imagery was identified using a scale, the object and spatial imagery questionnaire (OSIQ), that distinguishes them – people with aphantasia scored, as expected, much lower than controls on 'object' imagery (e.g., 'My images are very colourful and bright') but numerically higher than

controls on the spatial element of the task (e.g., 'I can easily sketch a blueprint for a building that I am familiar with') [41]. A larger study [55] confirmed that spatial imagery scores on the OSIQ did not distinguish people with aphantasia from controls. The finding that spatial accuracy is preserved when people with aphantasia reproduce drawings is consistent with these results [51]. A study of participants recruited from the general community revealed distinct varieties of 'object' and 'spatial' aphantasia occurring with similar frequencies – 3.1% and 3.5%, respectively [35]. This distinction between object and spatial imagery may map onto the contrast between ventral ('what') and dorsal ('where' or 'how') streams of visual processing within the visual system [35,75,76].

Imagery in dreams

Of the original group of aphantasic participants, 17 of 21 described dreaming visually. This observation has been borne out by subsequent reports [3,33–35,55]. However, although the majority of people with aphantasia have dreams with visual qualities, avisual dreams, with variable narrative, textual, conceptual, auditory, and emotional content are described more often by people with aphantasia than by control or hyperphantasic participants, as is absence of dream recall [3]. The dissociation between wakeful and dreaming imagery is counterintuitive at first sight, but, given that the neurochemical milieu and regional activation of the brain differ markedly between wakefulness and **rapid eye movement (REM) sleep** [77], it is neurologically plausible. Many of our own aphantasic participants have also mentioned that hypnagogic imagery is preserved.

The frequency of forms of imagery intermediate between deliberate wakeful and dreaming imagery, such as **mind pops** and imagery evoked by reading, has not yet been studied extensively in the context of extreme imagery [55]. However, a recent study suggests that aphantasia influences how a story is experienced, and there is a reduction in the amount of evoked visual and bodily imagery and in absorption by the story world, although not in liking for the story [78]. There may be a comparable impact on the experience of music [79].

Autism

Following anecdotal reports of autism from several participants, we found that those with aphantasia had higher scores on the autism spectrum quotient questionnaire (AQ) than controls, although no one in the aphantasic group had received a formal diagnosis of autism [50]. This was confirmed by a subsequent study [70]. Because there is an association, at a group level, between mathematical and scientific occupations and autism [80], this result meshes with the association between these occupations and aphantasia. It may also be relevant to the neuroimaging results described below. Elevated levels of introversion among people with aphantasia [50] are in keeping with the presence of autistic traits. However, autism has sometimes been associated with 'thinking in pictures' [81], and the relationship between imagery vividness and autism is likely to be complex.

Mental health and well-being

Imagery extremes may have important implications for mental health, but these have not yet been well explored. Imagery is known to function as an 'emotional amplifier' [82]. Previous work suggested that elevated levels of imagery vividness may be a risk factor for hallucinations in schizophrenia [83] and Parkinson's disease [84], as well as for visual intrusions of the type that occur in post-traumatic stress disorder (PTSD) [85]. The lack of a somatic response to frightening stories in people with aphantasia [42], together with a recent unpublished report of reduced visual intrusions among aphantasic participants in an experimental analogue of PTSD [86], support the – intuitive – suggestion that aphantasia may be protective against forms of emotional disorder linked to aversive imagery (*cf* [55]). Anecdotal accounts that aphantasia is conducive to living 'in the here and now' by lowering the likelihood of distressing craving and rumination merit further study.

The cognitive, neural, and genetic basis of imagery extremes

At least four cognitive explanations could be offered for reports of extreme imagery [87]. First, people might have identical imagery experiences, but describe them differently. Second, people with aphantasia may in fact have the experience of imagery but fail to recognise this because of faulty introspection. Third, people with aphantasia may lack the experience of imagery but nevertheless have – and make use of – unconscious or 'sub-personal' imagery. Fourth, people with aphantasia may both lack the experience of imagery and the sub-personal, imagistic representations on which imagery depends.

The persuasive first-person accounts provided by people with aphantasia, together with the behavioural and physiological associations of extreme imagery described above, make the first and second proposals implausible, at least as a general account. The third proposal, that suggests a disconnection between unconscious representations subserving imagery and conscious awareness, is in keeping with the relatively normal performance of people with aphantasia on a range of measures that might appear to depend on imagery (e.g., visual short-term memory tasks, high-imagery questions), their experience of imagery in other contexts (e.g., during dreams), and anecdotal accounts from people with aphantasia of imagery that feels 'just out of reach'. The fourth proposal is consistent with evidence that people with aphantasia use alternative strategies in tasks that are performed using imagery by people to whom this is available [49]. Given that imagery extremes are likely to be heterogeneous, it may be that the third and fourth mechanisms are both relevant, but in different individuals.

Brain imaging studies have the potential to adjudicate between these two proposals by providing evidence for the presence or absence of imagistic representations and by exploring their connectivity with other relevant brain regions. Studies on this topic are at an early stage, but suggestive evidence has begun to accumulate (Box 4).

The first functional imaging study to compare people with aphantasia, hyperphantasia, and average imagery [50] did not identify clear differences in brain activation during the imagery tasks, thus providing tentative evidence against the fourth proposal. The key positive findings of the study were an increase in resting state functional connectivity between prefrontal regions and visual regions in people with hyperphantasia by comparison with people with aphantasia, and greater activity in anterior parietal cortices in hyperphantastic and control participants than in aphantastic participants during visualisation when compared to perception. The first finding of a difference in brain connectivity receives support from an unpublished study using 7T fMRI to compare neural activity during perception and imagery [11]. During imagery tasks, people with aphantasia activated visual cortices similarly to controls, but a key region in the left fusiform cortex, the 'fusiform imagery node' (FIN) was functionally disconnected from left frontoparietal regions engaged by imagery tasks. An alteration in 'connectivity', in keeping with the third proposal, is therefore a promising candidate for the underlying neural source of aphantasia. This hypothesis is supported by the association between aphantasia and autism because autism is characterised by a reduction in long-range connectivity within the brain [88]. The proposal also points to a potential analogy between aphantasia and other neurodevelopmental phenomena that are linked – arguably – to weakened connectivity between higher-level control networks and lower-level perceptual processing modules [89]. Two further unpublished brain imaging studies using multivariate decoding techniques [90,91] are broadly consistent with the studies described above; however, one raises the plausible possibility that the low-level imagery representations found in people with aphantasia, although detectable, may be atypical [91]. The neural basis of the prominent association between imagery vividness and autobiographical memory should be explored in future research.

Box 4. The neural basis of extreme imagery: candidate mechanisms

Defining the neural basis of the contrast between aphantasia and hyperphantasia is a key future challenge. There is a range of theoretical possibilities – not mutually exclusive – informed by some early evidence (Figure 1B).

- (i) Are there differences in 'brain structure' between people with aphantasia and hyperphantasia? None has emerged to date [50]. Despite evidence that primary visual cortex surface area and prefrontal cortex volume correlate with imagery strength and vividness, respectively, in participants with typical imagery [92], these relationships have not yet been explored in people with extreme imagery.
- (ii) Are there differences in the intensity of 'regional brain activity' at rest and during relevant tasks? Stronger imagery in participants with typical imagery is associated with lower levels of resting activity/excitability in early visual cortex [93]. More vivid imagery in people with typical imagery is reflected in widespread patterns of activation and deactivation across the brain [19,20]. Brain activity was reduced in occipitotemporal regions during imagery in acquired aphantasia patient MX [27]. Anterior parietal activation is reduced in participants with lifelong aphantasia during imagery compared to during perception [50].
- (iii) Are there differences in the 'dynamics' of regional brain activity? There is so far no reliable information about the dynamics – frequency bands, temporal evolution – of brain activity in extreme imagery. An interesting event-related potential (ERP) single case-study is difficult to interpret in the absence of control data [122]. Investigation with electroencephalography (EEG) or magnetoencephalography (MEG) could help to fill this data gap.
- (iv) Are there differences in the 'connectivity' of brain activity? Two functional imaging studies have pointed to reduced connectivity in aphantasia [11,50]. Further replication will be necessary to be entirely confident of this finding, but it is a promising direction to pursue. Future studies should determine whether there are any corresponding structural differences in long-range connections.

It may not be a coincidence that these four candidates – structure, activity, dynamics, and connectivity – have often been highlighted in discussions of the distinction between conscious and unconscious processes [123]. Both the dominant theories of consciousness in contemporary neuroscience emphasise the importance of connectivity and global integration [124,125]. Is aphantasia primarily a disorder of visual consciousness, as recently suggested [11], that is linked to altered connectivity? The pattern of associations described in the main text suggest that it is not 'simply' a disorder of phenomenal awareness with fully preserved behavioural function, but studies on extreme imagery may nevertheless help to inform the developing science of consciousness.

Other potential candidates for the neural origins of extreme imagery should also be examined in future work. For example, an inverse relationship between the area of primary visual cortex and imagery strength estimated using the binocular rivalry technique has been reported [92]. The volume of prefrontal cortex correlated directly with imagery vividness (however, [19] provides evidence that some prefrontal activations are negatively correlated with imagery vividness). Lower levels of resting activity and excitability in early visual cortex have also been shown to predict stronger imagery [93]. These findings raise the possibility of variation in cortical volumes and cortical excitability in imagery extremes, and call for clarification of the relationship between the overlapping but distinct concepts of imagery strength and imagery vividness [94]. The role of dopamine as a modulator of imagery vividness is being assessed by a preregistered study [95].

Whether hyperphantasia simply reflects the upper end of the normal imagery vividness distribution or includes distinct populations remains unclear, as is its relationship to 'eidetic imagery'. This describes the intriguing phenomenon, seen predominantly in children, of vivid visual imagery persisting for some minutes following the presentation of a visual stimulus. It is typically perceived in its original location where it can be scanned as if the stimulus were still present [94,96].

A **sibling recurrence risk** of 9.6 in aphantasia, indicating a roughly 10-fold increase in the likelihood of aphantasia in siblings versus the general population, may point to a genetic basis for imagery extremes [3]. This would be consistent with the associations between imagery extremes and conditions such as autism and prosopagnosia which themselves have a genetic component. Future studies involving large biobanks have the potential to shed light on the genetic architecture of imagery extremes, although the likely existence of subtypes of extreme imagery will somewhat complicate this work.

Concluding remarks

Aphantasia and hyperphantasia are recently coined terms that refer to the absence and superabundance of visual imagery. In their extreme, lifelong forms they occur with a prevalence of ~1% and ~3%, respectively, and often run in families. These notable variations in conscious experience can occur in a range of contexts and are likely to have subtypes which await full definition. For most people with extreme imagery, other modalities of imagery are also affected, but dream imagery and spatial imagery are typically retained in aphantasia. Autobiographical memory and face perception are often reduced. Aphantasia can be associated with autism but may offer some protection from some mental health conditions. The emphasis on aphantasia in this review reflects the concentration of recent research on this end of the vividness spectrum.

Are aphantasia and hyperphantasia best regarded as disorders or as examples of neurodiversity – non-pathological individual differences in cognition, personality, and behaviour? A recent study [97] concluded that aphantasia does not affect activities of daily living or mental well-being to the degree that justifies classification as a mental disorder. Both aphantasia and hyperphantasia are likely to have their advantages and disadvantages [3]. They belong among a group of comparable differences in experience and behaviour, including congenital synaesthesia and prosopagnosia, which easily 'escape attention' [98,99].

The serendipitous rediscovery of extreme imagery has opened a new window onto the fascinating set of interrelationships between imagery and perception, memory, neurodevelopment, and mental health (although not discussed here, it has also excited interest from philosophy and the arts [87, 100–108]). Key directions for future research are outlined in the [Outstanding questions](#).

Two general conclusions emerge from work on aphantasia to date. First, conscious sensory imagery is not a prerequisite for human cognition – Aristotle was wrong when he wrote 'the soul never thinks without a phantasma' [29]. Second, creative achievements among people with aphantasia [100,102] indicate that sensory imagery is not a prerequisite for creative imagination – the richer capacity to represent, reshape, and reconceive things in their absence.

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Declaration of interests

The author declares no conflicts of interest.

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Outstanding questions

Are there subtypes of extreme imagery? Further analysis of first-person, behavioural, neural – and possibly genetic – data should illuminate the answer to this question.

What are the relationships between aphantasia and SDAM? Current evidence suggests that they are overlapping phenomena, but clarification is needed.

How is extreme imagery related to autism? Aphantasia is associated with autism, but both imagery extremes may co-occur with autism.

What is the neural basis of imagery extremes, and how does this explain its associated features? Altered connectivity between frontoparietal and sensory regions is a promising candidate, but other differences in brain structure and activity may contribute.

Is there a genetic basis for extreme imagery? This question can in principle be answered by using data from large biobanks.

Do people with aphantasia have – and use – 'unconscious imagery'? This could help to explain the modest behavioural effects of aphantasia: functional brain imaging has the potential to yield insights.

To what extent are imagery extremes associated with perceptual differences? Current evidence points to subtle associations that will require further study.

How does hyperphantasia relate to 'eidetic imagery', 'photographic memory', and perception itself? Objective investigation of hyperphantasia is in its infancy.

How is vividness best defined? This intuitive concept will benefit from further scientific clarification.

How does the experience of imagery differ from the experience of dreaming and hallucination? Philosophers debate whether dreams and hallucinations involve imagery; cognitive neuroscience is well placed to shed light on their relationships.

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Does a single main dimension of variation underlie the contrasting associations of aphantasia and hyperphantasia? If so, should this be characterised as episodic versus semantic, experiential versus abstract, or narrative versus systematic?

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