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Chapter 15

Aphantasia: The science of visual imagery extremes

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Abstract

Visual imagery allows us to revisit the appearance of things in their absence and to test out virtual combinations of sensory experience. Visual imagery has been linked to many cognitive processes, such as autobiographical and visual working memory. Imagery also plays symptomatic and mechanistic roles in neurologic and mental disorders and is utilized in treatment. A large network of brain activity spanning frontal, parietal, temporal, and visual cortex is involved in generating and maintain images in mind. The ability to visualize has extreme variations, ranging from completely absent (aphantasia) to photo-like (hyperphantasia). The anatomy and functionality of visual cortex, including primary visual cortex, have been associated with individual differences in visual imagery ability, pointing to a potential correlate for both aphantasia and hyperphantasia. Preliminary evidence suggests that lifelong aphantasia is associated with prosopagnosia and reduction in autobiographical memory; hyperphantasia is associated with synesthesia. Aphantasic individuals can also be highly imaginative and are able to complete many tasks that were previously thought to rely on visual imagery, demonstrating that visualization is only one of many ways of representing things in their absence. The study of extreme imagination reminds us how easily invisible differences can escape detection.

VISUAL IMAGERY

Visual imagery typically refers to the voluntary creation of the conscious visual experience of an object or scene in its absence, commonly referred to as "seeing with the mind's eye." Visual imagery has been tied to many healthful everyday cognitive processes, such as spatial navigation, memory, reading comprehension, and planning (Just et al., 2004; Guariglia and Pizzamiglio, 2007; Pearson and Keogh, 2019). In contrast, imagery can play a core role in the symptomology of many mental, psychiatric, and neurologic disorders such as anxiety disorders, depression, schizophrenia, and Parkinson's disease (Holmes and Mathews, 2010; Clark et al., 2015; Shine et al., 2015). Mental imagery has also increasingly been used as a tool for psychological treatment (Pearson et al., 2015) through imaginal exposure and imaginal rescripting (which involve repetitively imagining anxiety inducing stimuli to extinguish fear or imagining traumatic scenarios with better outcomes, respectively). Despite the very substantial role visual imagery plays in cognition and disorders, large individual differences exist in our ability to visualize, ranging from those who experience incredibly lifelike imagery (hyperphantasia) to those who experience a complete lack of any power to visualize (aphantasia). Understanding how these variations in imagery ability contribute to human cognition and mental health is an important endeavor.

History of imagery research

Quantitative research into visual imagery was inaugurated by Sir Francis Galton's classic paper from the 1800s (Galton, 1880). His "breakfast table questionnaire"

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invited participants to score the illumination, definition, and coloring of the recollected image of your "breakfast table as you sat down to it this morning" (Galton, 1880, p. 301). Galton noted that a small handful of those he surveyed claimed to have "no power of visualising" (p. 306). One such participant responded: "My powers are zero…I recollect the breakfast table but do not see it" (p. 306). Galton himself was a weak visualizer and believed that faint imagery was common among "men of science" (p. 302) (this was later challenged (Brewer and Schommer-Aikins, 2006)). Interestingly, Charles Darwin, Galton's cousin, wrote that his imagery of the breakfast table included some objects "as distinct as if I had photos before me" (Brewer and Schommer-Aikins, 2006, p. 140).

Mental imagery research went through a major disruption during the early to mid-20th century, due to the behaviorist revolution in psychological research, which denied the existence of internal representations like visual mental images. A quote by the eminent behaviorist John Watson exemplifies this absolute rejection of imagery from the behaviorist community: "What does a person mean when he closes his eyes or ears (figuratively speaking) and says, "I see the house where I was born, the trundle bed in my mother's room where I used to sleep I can even see my mother as she comes to tuck me in and I can even hear her voice as she softly says goodnight"? Touching, of course, but sheer bunk. We are merely dramatizing. The behaviourist finds no proof in imagery in all this. We have put these things in words long, long ago and we constantly rehearse those scenes verbally whenever the occasion arises." This extreme stance detrimentally impacted visual imagery research and left lingering doubt about the viability of imagery research for decades after (Pearson, 2014). However, in the 1970s, there was a strong reemergence of visual imagery research, provoking the "imagery debate" (Pearson and Kosslyn, 2015). This debate centered on the representational format of a visual image (Pylyshyn, 2003; Kosslyn, 2005; Pearson and Kosslyn, 2015).

One side of the debate proposed that there is nothing distinctive about visual images in the brain—they share a propositional/symbolic representational format with language. Those on the other side of the debate argued that imagery includes depictive representations that have distinctive properties within the brain. A depictive representation is one in which the spatial characteristics of the represented object are maintained in its representation in the brain (Pearson and Kosslyn, 2015). This can be understood through thinking of these representations as of comprising of a XY coordinate system, where spatial properties are maintained in a two-dimensional plane. Depictive representations are often referred to as pictorial due to pictures also being typically represented on a two-dimensional plane. The debate lasted for several decades, with each side providing new empirical data for their case, only to be countered by the opposing viewpoint showing that the data could be explained with an alternative hypothesis (Pylyshyn, 1973, 1981, 2003; Kosslyn, 1981, 2005; Kosslyn et al., 2006).

A key limitation to the study of visual imagery has been its inherently private nature. Historically, this has made the empirical investigation of visual imagery difficult; indeed, this difficulty of measurement has served to keep the imagery debate alive. For many years, self-report questionnaires remained the gold standard tool used to measure imagery abilities, due to their ease of administration and scalability. The advent of functional brain imaging in the 1990s allowed for a new source of data, providing evidence that visual images could be depictive and involved recruitment of sensory cortex. Behavioral research has recently provided further strong evidence that visual images can indeed be depictive (for example, see Pearson et al. (2008), Bergmann et al. (2016), and Section The Cognitive Basis of Imagery). The new wave of decoding-based functional magnetic resonance imaging work has further advanced the field of visual imagery and the depictive nature of mental imagery, indicating that visual images are represented across visual cortices. For example, studies have shown that the content of what an individual is imagining can be identified from the pattern of BOLD activity in early visual cortex (e.g., V1 (Lee et al., 2012; Albers et al., 2013)) and higher visual regions (e.g., occipital place area and lateral occipital complex (Stokes et al., 2009; Lee et al., 2012; Johnson and Johnson, 2014)) using multivoxel pattern decoding and support vector machines. Work using encoding models has provided further evidence for the depictive nature of mental imagery (Naselaris et al., 2015, see Fig. 15.1). These models go further than just showing that the content of imagery can be decoded from visual regions of the brain, they demonstrate that visual imagery contains some low-level depictive visual features. This approach uses brain response to low-level perceptual features (e.g., Gabor wavelets of varying in spatial frequency, orientation, and location) to model the expected neural response to complex imagined images (e.g., paintings). These models are able to predict neural activity both when participants view and imagine paintings, providing evidence that low-level features are represented when participants imagine images in much the same way as when they perceive the images. Due to this new behavioral and neuroimaging data, strong claims have since been made that the imagery debate is over: visual images can indeed take on a number of different representational formats (Pearson and Kosslyn, 2015), and in some cases, imagery is akin to weak perception.

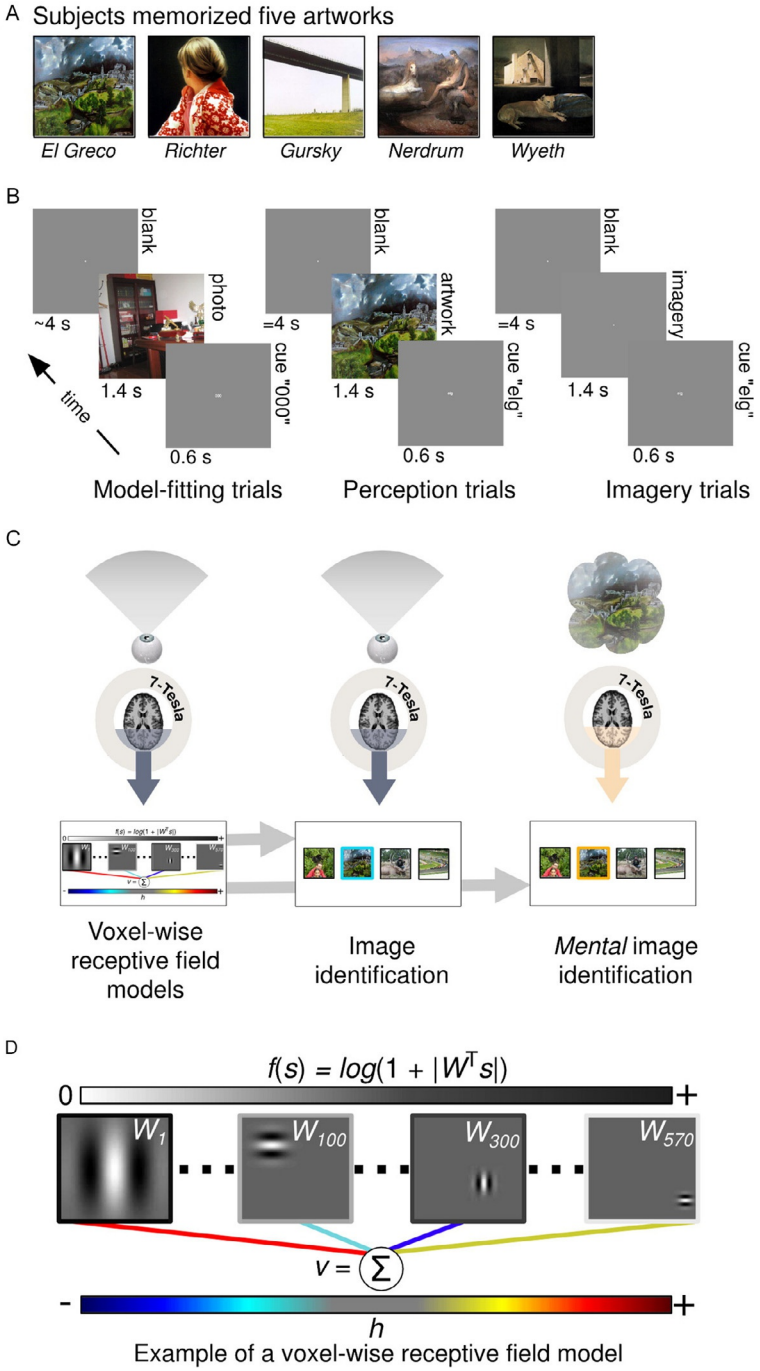


Fig. 15.1. This figure shows an example of encoding models that can be used to decode visual imagery. Voxel-wise receptive field models are created using Gabor wavelets (D) which are then used to identify the content of perception and/or imagery of scenes. Adapted from Naselaris T, Olman CA, Stansbury DE, et al. (2015). A voxel-wise encoding model for early visual areas decodes mental images of remembered scenes. Neuroimage 105: 215–28.

The cognitive basis of imagery

While questionnaires, such as the vividness of visual imagery questionnaire (VVIQ), have been the most commonly used measure of visual imagery, more objective task-based measures of imagery have been developed over the past century. The Perky experiment was one of the first recorded studies to assess the interaction between imagery and visual perception (Perky, 1910). In this experiment, participants were required to visualize an object on a screen, and while they did so, a faint

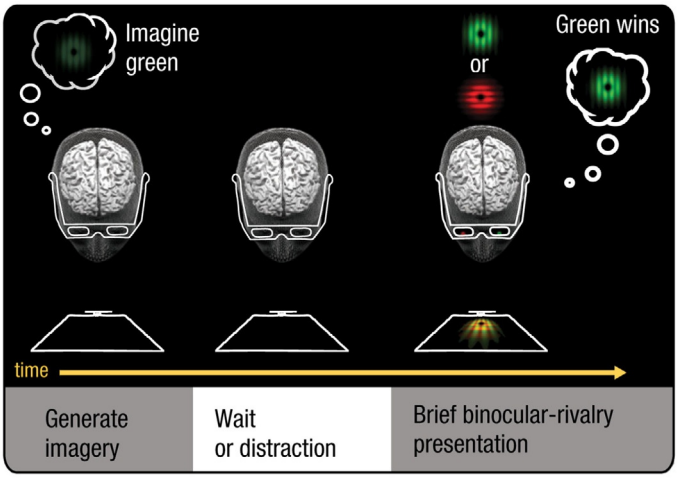
color that matched their visualization appeared. Participants failed to notice the presentation of the color, and it was proposed that the reason for this was that the color of their mental image was of similar strength or stronger than this very weak perception, resulting in either the perceived color being incorporated into their visualization or their imagery actually reducing their ability to see this weak image. This work resulted in more research investigating the effect of visual imagery on perception, with the decrement in perceptual performance due to concurrent imagery being referred to as the "Perky Effect." However, subsequent research into visual attention has shown that visual attention can also have similar effects (Carrasco et al., 2004), making it difficult to disentangle the effects of visual attention and imagery on concurrent perception.

Work during the 1970s by Stephen Kosslyn provided an interesting way to measure one's internal imagery by having participants mentally scan across a mental image (Kosslyn, 1973). In a typical task, individuals would be asked to visualize a map with landmarks and answer questions about their location. It was found that when landmarks were further apart it took longer to answer questions than when they were closer together on the map.

Later work demonstrated that imagery and perception can interact in facilitative, rather than disruptive ways. For example, one study found that when a Gabor patch was imagined next to a perceptually presented Gabor patch, the imagery could have a facilitative (make perception better) effect on perceptual detection tasks (Ishai and Sagi, 1995). Work using binocular rivalry, showed that visualizing one of two rivalrous stimuli tends to bias subsequent perception in the binocular rivalry display (Pearson et al., 2008), and this effect could be dissociated from visual attention, see Fig. 15.2. This task reveals the depictive nature of visual imagery but also provides a means of measuring imagery strength objectively. Pearson has written an extensive review on this topic (Pearson, 2014).

Recent research has also used physiological measures to assess visual imagery in a more objective manner. Research has demonstrated that visual imagery results in content-specific pupil responses, known as the pupillary light reflex—such that when an individual visualizes a bright image, pupils show greater constriction in comparison to visualization of dark images (Laeng and Sulutvedt, 2014). This provides evidence that visual imagery is similar to weak perception, although the exact

A



B

Characteristic / Process	Pictorial/ retinotopic	Orientation specific	Disrupted by luminance	Individual differences	Intra-individual differences	Metacognition	Time sensitive
Mental imagery	✓	✓	✓	✓	✓	✓	✓
Perception	✓	✓	X	✓	✓	✓	✓
Attention	✓	✓	X	✓	✓	✓	X

Fig. 15.2. (A) Schematic of the binocular rivalry imagery paradigm. Participants are instructed to imagine either a red or green Gabor patch for several seconds, after which they are presented with a binocular rivalry display comprising both the red and green Gabor patch. Participants are asked to indicate which image was dominant in the binocular rivalry task, and visual imagery strength is measured as percent of trials primed. (B) Table demonstrates the characteristic of visual imagery vs perception and feature-based attention. Adapted from Pearson J (2014). New directions in mental-imagery research: the binocular-rivalry technique and decoding FMRI patterns. Curr Dir Psychol Sci 23: 178–183.

neural mechanisms underlying this pupillary light reflex and other facilitative and disruptive effects of imagery can perhaps be better understood through assessing neuroimaging studies of visual imagery.

The neural basis of imagery

The neural basis of visual imagery has been studied for over 25 years using noninvasive brain imaging techniques. This work has demonstrated that visual imagery involves activity across a large neural network spanning frontal areas to primary visual cortex working in concert to create voluntary visual imagery (Pearson et al., 2015). Such a distributed network might be predicted on theoretical grounds: imagery tasks involve a process of decision making (I will visualize an apple), require the allocation of attention (to maintain the apple in mind), often involve language processing, draw on long-term memory of the content and spatial components of the image, ultimately leading to something akin to a visual sensation. In keeping with this prediction, a recent activation likelihood meta-analysis revealed focal activations in superior and inferior prefrontal regions and the superior parietal lobe as well as in primary visual cortex and the inferior temporal lobe (Winlove et al., 2018).

Much of the experimental neuroimaging work has focused on imagery responses in primary visual cortex, mainly due to the imagery debate. In general, the data support the hypothesis that visual imagery is based on information retrieved from memory in a reverse hierarchy process (see Fig. 15.3). That is both perception and visual imagery engage very similar brain regions; however, the temporal activation or "flow" of information can be thought of as occurring in opposite directions (Dijkstra et al., 2020). Perception is mostly driven by bottom-up stimulation (although top-down and lateral connections are also very important in perception) with the flow of information occurring from posterior to anterior regions, while the opposite is true of visual imagery.

The specific involvement of primary visual cortex was a major subject of controversy from the early 1990s to late 2000s. Over 20 studies investigated the role of primary visual cortex in imagery (Pearson et al., 2015). Early work used sustained elevated BOLD activity to assess whether early visual cortex was involved in visual imagery, with inconsistent findings. More recent studies using the pattern of fMRI response, a type of decoding approach where the content of imagery can be decoded using the *pattern of voxel activity*, rather than the *amplitude of blood-oxygen flow averaged over multiple voxels*, have provided strong evidence that a representation of the image is evident in primary visual cortex, even for imagery of complex scenes (Stokes et al., 2009; Naselaris et al., 2015; Pearson et al., 2015). It is important to note that successful decoding in early visual cortex can occur without any significant univariate changes in BOLD activity (Harrison and Tong, 2009), potentially explaining the earlier inconsistent findings when only BOLD was used to assess the role of visual cortex in visual imagery. These inconsistent findings in relation to sustained elevated BOLD in early visual cortex could also potentially be due to natural variations in imagery strength, such that individuals with stronger imagery demonstrate differential engagement of visual cortex (see Section Neural Correlates of Visual Imagery Vividness and Strength), as well as the types of imagery tasks participants are performing.

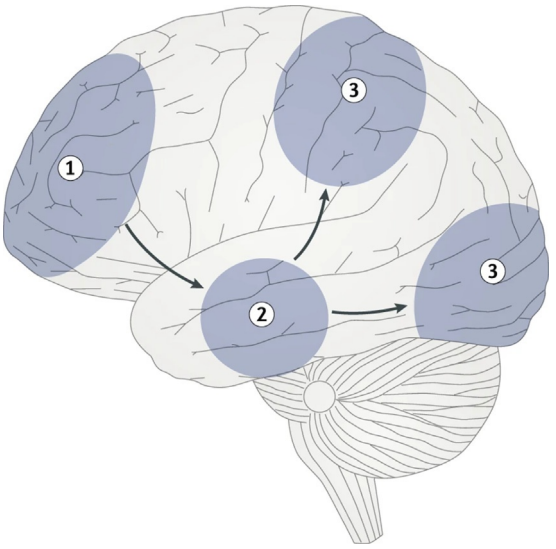


Fig. 15.3. The visual imagery network spans prefrontal (1), temporal (2), visual (3), and parietal cortex (3). Creating a visual image "begins" high in the cortical processing hierarchy, in the frontal cortex (step 1). This leads to a cascade of neural events, going "backward" compared to the flow of information when images are perceived, such that information from posterior regions such as medial temporal areas are activated (step 2), followed by the forming of sensory and spatial representations of the image held in mind (steps 3). Adapted from Pearson J (2019). The human imagination: the cognitive neuroscience of visual mental imagery. Nat Rev Neurosci 20: 624–634.

Forming or manipulating a mental image also demonstrates significant sustained averaged BOLD activity in frontal areas (Ishai et al., 2000; Yomogida et al., 2004; Ranganath and D'esposito, 2005; Schlegel et al., 2013); however, this activity does not demonstrate content specific modulations in relation to the stimuli imagined in the same way that visual regions do (Goebel et al., 1998; Ishai et al., 2000). More recent work using dynamic causal modeling has shown that category-selective effects in extrastriate cortex (due to imagery of faces, places or objects) are mediated by connectivity with prefrontal cortex. Thus, while frontal cortex does not show evidence of maintaining or holding the content of the image itself, feedforward activity form frontal cortex modulates the

content-specific activity in the sensory regions that do represent the image in mind (Ishai, 2010). These data suggest that frontal areas play more of a general organizational or executive role in coordinating spatial and sensory areas; that is, rather than holding any low-level imagery representations of content per se, the frontal regions control what, and how, memories and features are activated in downstream visual regions.

Neural correlates of individual difference in imagery

To date, only a few studies have examined the neural correlates of imagery vividness specifically. An early study found that BOLD activity in early visual cortex, when visualizing, correlated with the individual's self-reported vividness (Cui et al., 2007). In line with this, a more recent study found that intraindividual differences in trial-by-trial vividness are tied to the similarity between imagined and perceived neural activity in the entire visual system (Dijkstra et al., 2017). Another study demonstrated that activity in higher-order visual regions (Brodmann areas 18, 19, 37) and memory/default mode related regions (Brodmann areas 7, 30, 31, and the medial temporal lobes) is tied to the vividness of visual imagery (Fulford et al., 2018). Fulford et al. (2018) also conducted an informal meta-analysis of previous studies investigating the brain regions (BOLD fMRI) involved in the vividness of visual imagery and found evidence for regions spanning frontal, temporal, parietal, and occipital cortex (see Table 15.1).

With regard to objectively measured sensory strength of imagery (using the binocular rivalry paradigm, see Fig. 15.2), a study investigating the size of visual cortex found that participants with smaller visual cortices (surface area of V1 and V2) had stronger visual imagery (Bergmann et al., 2016). A recent study also found the excitability of early visual cortex (measured in two separate studies, using TMS-induced phosphene thresholds and resting state BOLD) correlates negatively with visual imagery strength, that is, participants with less excitable visual cortices had stronger imagery (Keogh et al., 2020). This study also found that imagery can be improved using noninvasive brain stimulation (transcranial direct current stimulation) to decrease the excitability of visual cortex (Keogh et al., 2020). Taken together, these data suggest that the anatomy and excitability of early visual cortex likely both contribute to individual differences in imagery strength.

An important question surrounds the exact relationship between experienced vividness of visual imagery and objectively measured visual imagery strength, both at a phenomenological and mechanistic level. Research suggests that self-reported vividness and objectively measured visual imagery strength (using the binocular rivalry paradigm) are positively correlated (Pearson et al., 2011; Keogh et al., 2020); however, they do not hold a one-to-one relationship. For example, while anatomical (visual cortex surface area) and functional (resting state BOLD and phosphenes) measurements of the visual cortex correlate with objective imagery measures, they do not significantly correlate with subjective measures of imagery vividness (Bergmann et al., 2016; Keogh et al., 2020). This raises the question as to whether imagery strength and vividness rely on two separable, but interrelated neuronal mechanisms or whether subjective reports are less reliable than objective ones due to some participants having worse metacognition than others and/or being more liable to contextual bias effects during testing, such as demand characteristics (i.e., responding in the way that they think the experimenters want them too).

Relating voluntary to involuntary visual imagery and the use of imagery in treatment

Hallucinations, intrusive imagery, synesthesia, perceptual filling-in, and many illusions are conscious visual experiences that occur without corresponding retinal stimuli—just like mental imagery. These two types of visual experiences, one voluntary (e.g., mental imagery) the other involuntary (e.g., visual hallucinations, illusions, synesthesia), have been described collectively as "phantom visual experiences" (Pearson and Westbrook, 2015). The distinction between voluntary and involuntary imagery is analogous to the two types of visual attention: endogenous and exogenous (Egeth and Yantis, 1997). We can voluntarily choose to attend to something, for example, a pen lying on our desk, as the result of an "endogenous" decision, but if someone knocks at the door, attention will immediately shift there, captured by an exogenous stimulus. Similarly, we can voluntarily imagine a sunset to relax us, while the wafting smell of a perfume might make us experience a spontaneous image of our mother, who wears that perfume.

The relationship between voluntary and involuntary visual imagery has been studied mostly in the realm of clinical psychology, psychiatry, and neurology as involuntary visual imagery is involved in many psychological and neurologic disorders. For example, intrusive involuntary mental imagery occurs as a core symptom in posttraumatic stress disorder. There is tentative experimental evidence for a correlation between the vividness of imagery generally and the risk of such intrusive images (Morina et al., 2013). Empirical data has demonstrated that the strength or vividness of visual imagery is related to organic hallucinations in a number of disorders, providing further evidence linking voluntary and involuntary forms of phantom vision. For example, a study of Parkinson's disease found that both objectively

Table 15.1

Brain regions showing positive correlations with imagery vividness in five comparable fMRI studies

	Frontal							Cingulate					Temporal				Parietal			Occipital		
	BA	10	9	8	6	45	46	32	24	31	29	30	21	22	37	MTL	7	40	39	17	18	19
Study																						
(1)												>			~	~ 35 and 36				>	>	<
(2)		~	<		~	~		<	>	<		<						~			<	~
(3)		<						<			>	>					~	~	~			~
			~	~	<					>												
(4)							> *						> *		~ *	>						
(5)		<										~			<	~ 30	>			~	~	~
(6)						<	<			>	<	<	<	>	~	~36	~		~			~
N		3	2	1	2	2	2	2	1	3	2	5	2	1	4	4	3	2	2	2	3	5

Adapted from Fulford J, Milton F, Salas D, et al. (2018). The neural correlates of visual imagery vividness—an FMRI study and literature review. Cortex 105: 26–40.

BA, Brodmann area.

*BAs inferred from paper.

> left sided activation, < right sided activation, ~ bilateral activation.

N = number of studies reporting activation in this Brodmann area.

MTL, medial temporal lobe.

Study references: (1) Amedi et al. (2005), (2) Belardinelli et al. (2009), (3) Daselaar et al. (2010), (4) Zvyagintsev et al. (2013), (5) De Araujo et al. (2012), (6) Fulford et al. (2018).

measured (binocular rivalry paradigm) and subjective reports of visual imagery are elevated in patients with Parkinson's disease who experience visual hallucinations, in comparison to patients who do not experience hallucinations (Shine et al., 2015). Elevated subjective reports of visual imagery have been associated with schizophrenia (Sack et al., 2005), in addition to objectively measured visuospatial imagery (Benson and Park, 2013; Matthews et al., 2014). Sensory visual imagery (measured using the binocular rivalry paradigm) has also been shown to be elevated in schizotypal personality disorder (Marothi and Keri, 2018). Visual imagery has also been tied to cravings for food and addictive substances (Harvey et al., 2005; Connor et al., 2014; Kemps and Tiggemann, 2014). These data provide evidence that phantom visual experiences, whether they be voluntary or involuntary, are interrelated, suggesting that people with stronger imagery might be at higher risk of developing disorders characterized by involuntary visual imagery. However, as these studies are not longitudinal, it could also be the case that these disorders are associated with enhancements of both voluntary and involuntary visual imagery.

While involuntary visual imagery is a symptom of many mental and neurologic illness, voluntary visual imagery is also being used to treat mental illness. Mental imagery is employed in exposure therapy for phobias where participants repeatedly imagine the fear inducing stimuli to reduce the fear response. Imaginal rescripting has also been used in the treatment of anxiety and mood disorders (Holmes et al., 2007). During imaginal rescripting, the practitioner has the client imagine an anxiety inducing or traumatic experience, as vividly as possible, and then reimagine the scenario with a different outcome to change the emotional meaning of the memory. In eye movement desensitization and reprogramming, an approved but controversial treatment for PTSD, visualization is combined with eye movement (moving the eyes from side to side) to reduce the distressing visual qualities of intrusive memories. Individual differences in the ability to imagine will likely influence the efficaciousness of these treatments.

EXTREMES OF VISUAL IMAGERY

Aphantasia

Although we have known that there are individuals who claim to have no visual mental imagery at all since Francis Galton's seminal paper (Galton, 1880), this finding has not been expanded on until recently. In 2015, the term congenital aphantasia was coined to describe the phenomenon of lifelong absence of the mind's eye. The preceding literature had used unwieldy phrases for the same purpose such as "defective visualisation" (Botez et al., 1985) and "visual irreminiscence" (Nielsen, 1946): a new term was needed. The term comes from Aristotle, who described the capacity for visual imagery as "phantasia" (Aristotle, 1968): the "a" denotes the absence of imagery in Aphantasia, by analogy with "aphasia," which is the absence of language. Aphantasia was used to initially to describe a specific small group of individuals who had *never* been able to visualize ("congenital" aphantasia). More recent research puts this phenomenon's prevalence at approximately 1%–4% of the population, making it a relatively common condition or variation of experience (whether lifelong aphantasia should be regarded as a "condition" or "disorder" is a moot point). Prior to the recent fascination with lifelong aphantasia, numerous reports had described the loss of the mind's eye due to neurologic or psychiatric disorder, which we refer to as acquired aphantasia. This literature is reviewed first, before returning to congenital aphantasia.

Acquired aphantasia—Neurogenic

One of the earliest cases of "acquired aphantasia" was described in 1883 by the "Father of French Neurology," Jean-Martin Charcot—"*On a case of sudden and isolated suppression of the mental vision of signs and objects*" (Charcot, 1889). This description was written 3 years after Galton's work, on *Statistics of Mental Imagery*, of which Charcot was aware. Charcot's patient, Monsieur X, had abruptly developed aphantasia, prosopagnosia, topographical agnosia (difficulty recognizing familiar landmarks), a visual memory impairment, and avisual dreams or "restriction of visual dream imagery" (Solms, 2009). Some commentators have raised the possibility that it was in fact caused by a psychiatric illness, specifically depersonalization disorder (Zago et al., 2011). While the sudden onset, neuropsychological associations, and persistence of Monsieur X's symptoms make it more likely that his presentation was the result of a neurologic event, we will never be certain as to what caused Monsieur X's "sudden … suppression of … mental vision." There is no doubt, however, that psychological state—as well as neurologic condition—can markedly affect visual imagery. A second classical case of imagery loss was described by Willbrand 4 years later. This case included *global* cessation of dreaming and prosopagnosia, due to occipitotemporal infarction. The term "Charcot-Willbrand syndrome" is sometimes used to refer to the cessation of dreaming as a consequence of focal brain damage due to these two cases. As Solms has noted, this term blurs the distinction between avisual dreaming (i.e., the patient still dreams but they are

nonvisual nature) and total cessation of dreaming (Solms, 2009), which are likely dissociable disorders.

Before the advent of accessible and affordable neuroimaging techniques, such as PET and fMRI which became popular in the 1990s, the neural correlates of visual imagery were studied through case studies of patients who had lost their ability to imagine. In 1984, Martha Farah summarized 37 of these cases, specifically looking into neurogenic (i.e., caused by some acute brain damage) cases of aphantasia (Farah, 1984). She classified these cases in terms of an information processing model of imagery, which was based on Kosslyn's theoretical approach (Fig. 15.4). This model proposes that, when imagining, information about the image is retrieved from long-term memory and is then used to generate imagery, "held" in an active store—known as the visual buffer (which is an amorphous short-term memory store, although some researchers posit that this buffer is early visual cortex). While held in the "visual buffer," this mental image can be inspected and manipulated. In Farah's (1984) paper, 10 of the cases had insufficient detail to be confident of nature of the imagery deficit. In eight cases, there was evidence for a "*generation deficit*," such that imagery was impaired but perception was not. Thirteen cases displayed a "*long-term visual memory deficit*" affecting both imagery and perception (in one case Farah was unable to decide between a generation and a visual memory deficit). Five cases evinced a disorder of the "*inspection process*" required to "identify parts and relations within the image," whether this was perceptual or imaginary in nature. Farah argued that Kosslyn's theoretical model helped to make sense of the diversity of these disorders, and that in turn the disorders supported his model of visual imagery with multiple separable components. She observed that posterior left hemisphere lesions were primarily found in those patients with generation deficits, and that the neural processes involved in imagery, visual recognition, and dreaming share at least some common neural architecture. In line with this, several cases had deficits in both perception and imagery: for example, an inability to imagine faces might also present with acquired prosopagnosia (inability to recognize faces). Indeed, consistent with this finding, research has indicated that congenital prosopagnosics demonstrate very weak and, in some cases, absent, visual imagery of faces (Gruter et al., 2009).

Several of the studies published since Farah's review have highlighted the ambiguity of the concept of the "visual buffer" in her model, providing evidence that impairments of perception are not always accompanied by impairments of imagery. Thus, some cases with substantial damage to parts of the visual cortices, which might be expected to contribute to the "visual buffer," can occur with preservation of imagery. For example, Behrmann et al. (1992) describe a patient, CK, who has profound object agnosia following a closed head injury. CK, however, was still able to perform "standard measures of imagery," such as answering questions about the shape of letters, naming the color of common objects, as well as being able to answer questions pertaining to the shape and size of animals, the distance between cities and the angle clock hands make at given times. Chatterjee and Southwood (1995) reported on three patients with extensive damage to visual cortices resulting in cortical blindness; two of whom were able to perform well on similar tests of imagery to those used by Behrmann et al. (1992) (e.g., questions on animal, letter and mouth shapes, object colors, city distances, and clock hand angles). Servos and Goodale (1995) report a well-studied patient with object agnosia following carbon monoxide poisoning (DF), who experienced "vivid and well-structured dreams." They also tested DF on some clinical imagery tasks such as mentally comparing the size of objects, the shape of animals ears and letters, and some mental rotation task: she performed normally on these. Shuren et al. (1996) describe a patient with achromatopsia following bilateral temporo-occipital strokes, who was able to perform some "colour imagery" tasks. These involved identifying the color of common objects, as well as comparing the color of objects mentally and preforming an "odd one out" task, e.g., which two items have the same color: lettuce, cucumber, and

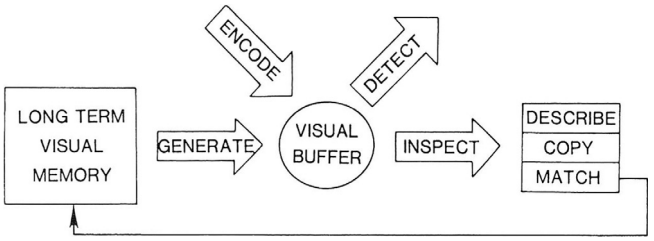


Fig. 15.4. Farah's (1984) model of neurogenic visual imagery impairments distinguished cases arising from (i) loss of representations in long-term visual memory, (ii) disorders of the "generate" step, and (iii) disorders of the "inspect" step (see text for further details). Adapted from Farah MJ (1984). The neurological basis of mental imagery: a componential analysis. Cognition 18: 245–272.

strawberry? Bartolomeo et al. (1998) report a case of "multidomain dissociation" between impaired visual perception and preserved mental imagery in a patient with bilateral extrastriate lesions due to successive bilateral temporo-occipital strokes. The tasks used here to measure visual imagery involved answering questions about the size and color of objects, the shape of letters, and question about the characteristics of famous faces. Bridge et al. (2012) showed that, despite a severely damaged primary visual cortex, their patient (SBR) had significant BOLD responses in intraparietal sulcus as well as fusiform and parahippocampal gyri when imaging houses, face, and checkerboards. SBR was also able to perform tasks comparing the size and shape of animals and letters, as well as reporting the color of objects. Conversely, Moro et al. (2008) describe two cases of patients with evidence of damage to the left temporal lobe, with additional damage to the superior parietal lobes in the second case, following closed head injury. Despite intact perception and intact primary visual cortex, these patients showed impaired visual imagery (tested on questions pertaining to the shape and size of animals and letters, as well as the colors of objects). Many of these papers are discussed in Bartolomeo's thoughtful reappraisal of the topic (Bartolomeo, 2002): a critical role for "the inferotemporal cortex, the mesio-temporo-limbic regions and perhaps the temporal pole" (Bartolomeo, 2002, p. 373) has been proposed due to these findings.

Other relatively recent contributions to this literature include a case report of a patient presenting with bilateral posterior cerebral artery stroke experiencing a total loss of visual images during dreaming, despite preservation of REM sleep (Bischof and Bassetti, 2004), and a report of an architect who lost the ability to visualize after a bilateral posterior cerebral artery stroke (Thorudottir et al., 2020). A "visual variant" of Alzheimer's disease with posterior cortical atrophy, associated with visual imagery impairment (Gardini et al., 2011), has also been reported. Patients with neglect of the left-hand side of space sometimes also neglect the left-hand side of mental visual images, as shown originally by Bisiach (Bisiach and Luzzatti, 1978; Bisiach et al., 1981). A series of papers on the relationship between perceptual and imaginal neglect indicated that just as perception and visualization are sometimes dissociated, so too can visual and imaginal neglect occur in isolation (Beschin et al., 2000).

The large, varied, and somewhat confusing literature on neurogenic aphantasia allows for a few confident conclusions. First and most obviously, brain damage can undoubtedly impair the ability to visualize. Second, deficits of imagery and perception often occur in parallel, for example, face recognition and visual dreaming, noted in Charcot's original report. However, they can dissociate: for example, some patients with severe central perceptual impairments, often associated with damage to visual cortices, may remain capable of imagery, while some patients are unable to visualize despite intact perception.

It should be noted here that clinical tests of visual imagery are often very different to those used in a laboratory setting and are highly variable (see the earlier examples and Pearson et al. (2013)) for a review of the large range of clinical visual imagery tasks). For example, one common test of imagery in the clinic is to ask participants to answer questions about images—e.g., is a strawberry darker or lighter red than a cherry or does a kangaroo or pig have a longer tail in comparison to their body? Although visual imagery can be used to complete these tasks, other nonimage-based strategies—for example, the use of semantic memory—are also available. Another common "visual imagery" task is to ask participants about the shape of letters, which is particularly problematic as kinesthetic imagery could provide the answer. Mental rotation tasks are also often used to test visual imagery: however, there is evidence that people with strong and weak imagery use different strategies, and call on different neural regions, to perform the same mental rotation tasks (Logie et al., 2011). Further, preliminary evidence and self-reports in congenital aphantasic individuals demonstrate that they appear to be unimpaired on these tasks, in keeping with the suggestion that spatial and object imagery dissociate (Keogh and Pearson, 2017; Pounder et al., 2018; Keogh and Pearson, 2018a). Thus, in the interpretation of the results of "imagery tasks," it is important to bear in mind the possibility that multiple strategies and hence brain regions could be used, and the likelihood that "visual imagery" itself is a heterogeneous nontrait-like process, reliant on different brains regions for different content.

Future studies of neurogenic aphantasia should seek to reconcile the findings described previously with what we know now of the underlying neurobiology of imagery in the healthy brain (see sections "The neural basis of imagery" and "Neural correlates of visual imagery vividness and strength"). Researchers should also ensure that the phenomenologies of imagery—both voluntary and involuntary—are fully described in future cases, a striking omission in many previous reports, alongside appropriate behavioral and neural measures.

Acquired aphantasia—Psychogenic

In the same decade that Galton recognized the phenomenon of the lifelong absence of the mind's eye and Charcot recorded its loss in Monsieur Z, the occurrence of aphantasia as an outcome of *psychiatric* disorder was described by Jules Cotard (a French psychiatrist who trained in part under Charcot). He is famed for his eponymous syndrome, Cotard's Syndrome, the "delire des

negations" [roughly translated as "nihilistic delusions"]. This syndrome leads the sufferer to declare that they, their organs, or the entire world have died and/or no longer exist (Cortard, 1882). This syndrome most often occurs in association with psychotic depression. In 1884, Cotard described 2 patients who complained of what we would now term acquired psychogenic aphantasia ("perte de la vision mentale" ["loss of mental vision"]). This loss of imagery presented in the context of "melancolie anxieuse" ["anxious depression"] (Cotard, 1884). One of the patients also described the *delire des negations*, which led Cotard to speculate that his nihilistic delusions might be secondary to his "loss of mental vision."

Since Cotard similar cases have been reported, particularly in France and Italy, although not as commonly as neurogenic aphantasia. In 2011, Zago et al. tabulated 25 cases in which they believed that a psychological or psychiatric explanation for the patients' imagery loss was more appropriate and revealing than a neurologic account (Zago et al., 2011). The psychiatric context of the onset of imagery loss in cases is typically one in which "depressive and anxious symptomatology coexist with depersonalisation and derealisation" (defined, respectively, as "an alteration in the perception or experience of the self so that one feels detached from and is if one is an outside observer of one's mental processes or body" and "an alteration in the perception or experience of the external world so that it seems strange or unreal"). A dedicated study aligns with the hypothesis that depersonalization disorder might result in Imagery loss. This study found a significant impairment of imagery vividness on both visual and movement imagery questionnaires in 28 patients with depersonalization disorder (Lambert et al., 2001).

Thus, just as brain damage can dim the mind's eye, it appears psychiatric conditions can also result in acquired aphantasia. However, everything that is functional is organic: that is, psychological and psychiatric processes and disorders arise from the brain and as such have a biological basis and this should be true too of psychogenic aphantasia (de Vito and Bartolomeo, 2016; Zeman et al., 2016). While what at a neural level drives the loss of imagery in psychogenic aphantasia has not yet, to our knowledge, been studied specifically, it is interesting that a study of depersonalization revealed altered BOLD activity in Area 19 (part of the extrastriate cortex) of the left hemisphere (Phillips et al., 2001). This region has recently been implicated in imagery vividness in a recent meta-analysis using normal populations (Fulford et al., 2018).

Congenital aphantasia

Despite the fact that we have known about the existence of individuals who claim to have no visual mental imagery at all since Francis Galton's 19th Century paper (Galton, 1880), and visual imagery attracting much interest from psychologists during the imagery debate, this finding was largely ignored until recently. There was one notable exception: the American psychologist Bill Faw, himself a "wakeful non-imager," reported that 2.1%–2.7% (out of 2500 participants) lacked visual imagery. However, Faw does not provide the details of his study's methods or results (Faw, 2009). One other study conducted prior to the naming of aphantasia in 2015 exists. In this study, the authors reported on two individuals with a lifelong absence of imagery (what we would now call congenital Aphantasia) and how this impacted their memories of the past. They found that these two individuals reported a markedly reduced sense of "reliving" during autobiographical recall, an observation that aligns with some recent findings, discussed later, on the relationship between visual imagery and autobiographical memory (Greenberg and Knowlton, 2014).

The reemergence of this interest in an inability to imagine can be tied to a popular report in *Discover* of a scientific description of a patient with acquired imagery loss (Zeman et al., 2010; Zimmer, 2010), patient MX. MX had lost the ability to visualize following a cardiac procedure. An articulate and highly visual man, a retired surveyor, he had previously relished his visual imagery and used it to aid his work. Soon after this procedure he realized that he could no longer call images to mind; when he read novels, he did not enter a visual world. He continued to dream, but his dreams were now avisual. He performed normally on tests thought to be sensitive to visual imagery (e.g., which is darker, the green of grass or the green of a pine tree?), explaining that he "just knew" the answers, but could not call upon imagery to answer these questions. A functional imaging task (fMRI) was performed to investigate whether there might be a neural correlate for MX's imagery loss. In this study, MX was presented faces perceptually and was also asked to visualize these faces. When MX *looked* at faces, he activated visual regions, including the fusiform gyrus (which contains the fusiform face area), as expected. However, when he visualized faces, occipitotemporal regions, including part of the fusiform gyrus, were hypoactivated. Further, there was hyperactivation of part of the anterior cingulate gyrus (see Fig. 15.5). His case can be thought of as one of "blind imagination," analogous with "blindsight." People with blindsight lack conscious vision, either in one half or all of visual space yet they can make accurate guesses about visual events occurring despite to being consciously aware of any visual stimuli. Similarly, MX lacked any conscious experience of visual imagery but was able to answer questions that previous researchers have proposed require it.

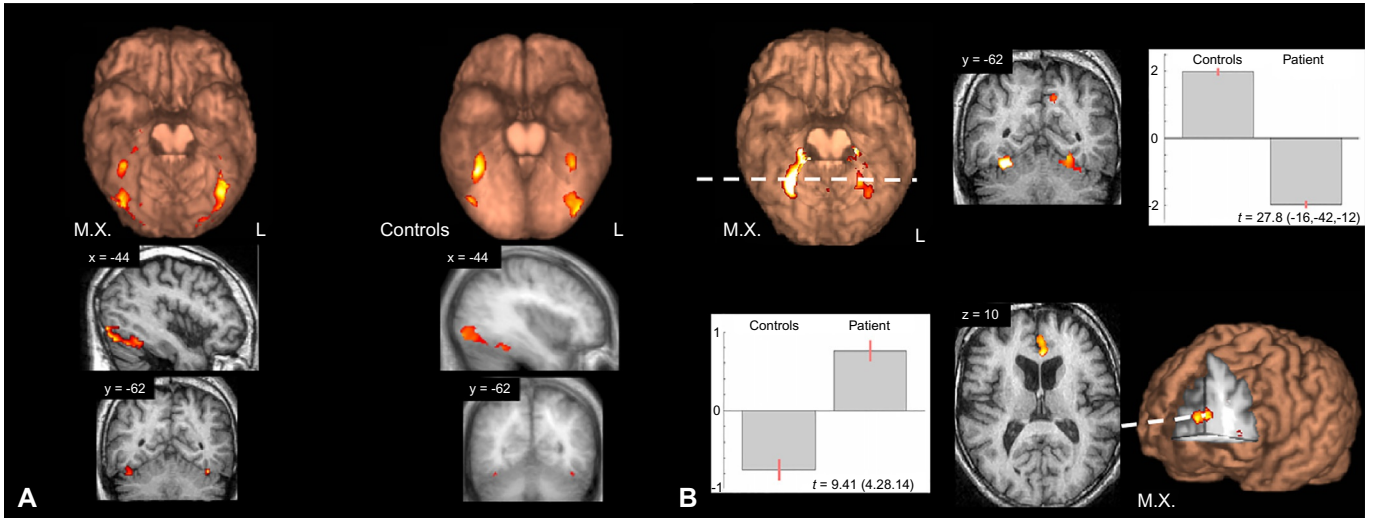


Fig. 15.5. (A) Brain activation in participant MX and matched controls, while *viewing* famous faces, these were not significantly different. (B) Brain activation in participant MX by comparison with matched controls while *imagining* famous faces. The upper images demonstrate *hypoactivation* in occipitotemporal regions, including left fusiform cortex (shown at far right) in MX, with *hyperactivation* of right anterior cingulate cortex in the lower images. Adapted from Zeman, A. Z., Della Sala, S., Torrens, L. A., et al. (2010). Loss of imagery phenomenology with intact visuo-spatial task performance: a case of 'Blind Imagination'. Neuropsychologia, 48: 145–55.

After the publication of the article in Discover, 21 individuals contacted one of the authors (AZ) as they recognized themselves in Zimmer's description, but with one salient difference: their minds' eyes had *always* been blind and they could not tie their lack of imagery to any neurologic or psychological illness, i.e., it was not acquired (Zeman et al., 2015). Their scores on the Vividness of Visual Imagery Questionnaire were at or close to floor (16/80), compared to a median score of 58/80 in a student control group, similar to results from other control groups (McKelvie, 1995). The majority of these 21 individuals realized at some point in their teens or 20s that when others spoke of "visualising" or "seeing in the mind's eye" they were speaking literally, not metaphorically as they had up until that point supposed. This realization excited curiosity as much as regret, although some individuals indicated that they felt they had been "cheated."

The 2015 study proposed the term congenital aphantasia to describe these individuals who are completely "blind" in their visual imagination. This term, and the phenomenon it describes, has garnered great public interest. News outlets around the world have covered stories of this intriguing variation in human experience, which had mostly escaped attention over the decade since Galton's breakfast table experiment. Since then, research has shown that this phenomenon is relatively common (approximately 1%–3% of the population (Zeman et al., 2020)), with individuals often being genuinely surprised to discover that others have rich visual imagery in their mental lives.

A skeptic might reasonably question whether we can be sure that the absence of the mind's eye is truly "congenital" or life-long. As this judgment currently relies entirely on the memory of those who describe the phenomenon: all we can say with confidence is that these individuals cannot recall ever being able to visualize. Whether this incapacity is strictly congenital awaits further investigation, although the prevalence among families (see the next section) might make a compelling argument for there being a congenital form of aphantasia.

Subsequent research has gone beyond self-report methods to show that along with floor scores on imagery vividness questionnaires, self-identified aphantasic individuals demonstrate significantly lower scores on the sensory binocular rivalry measure of visual imagery (Keogh and Pearson, 2018a). This suggests that aphantasia cannot be explained simply as poor metacognition of visual imagery but reflects a more basic difference at the sensory level, likely driven by an inability to alter activity in visual cortex in such a way as to experience a conscious visual image. Interestingly, in this same study, while aphantasic individuals scored much lower than controls on measures of object imagery, they actually score slightly higher on spatial imagery questionnaires (Keogh and Pearson, 2018a), although this was not statistically significant. This, in keeping with other recent work (Blazhenkova and Kozhevnikov, 2010) and the two-stream hypothesis of visual perception (Goodale and Milner, 1992), suggests that imagery, often discussed as a unitary phenomenon, is separable into "object imagery" (imagery about how things look) and "spatial imagery" (describing its spatial coordinates). These new data suggest that aphantasia may specifically involve an attenuation of object rather than spatial imagery.

We suspect that aphantasia itself will prove to be heterogeneous. For example, while some aphantasic individuals report that they lack all forms of mental imagery (i.e., no auditory, tactile, gustatory, olfactory, kinesthetic imagery), others report only an inability to visualize, retaining a rich ability to imagine in other sensory modalities (Dawes et al., 2020; Zeman et al., 2020). The presence or absence of associated face recognition difficulties, autobiographical memory disorder, autistic spectrum disorder, and variations in dreaming may also reflect heterogeneity within aphantasia, as discussed further later.

The characteristics of extreme imagery vividness

Since 2015, thousands of people have reached out, most of them reporting a form of "extreme imagery," either aphantasia or its opposite, hyperphantasia (imagery "as vivid as real seeing"). We (AZ in the UK, RK and JP in Australia) have responded to this bonanza of interest by asking those who have contacted us to complete the VVIQ and other questionnaires probing aspects of the experience of aphantasia and hyperphantasia, with some additional laboratory testing. Here, we give a brief account of some key outcomes and findings.

The first, striking, feature of the response to the coinage of aphantasia and our research effort was the warmth and excitement elicited. It was clear that a great many people had identified this quirk in their psychological nature but had been frustrated by the lack of a term with which they could describe it when speaking to family, friends, and colleagues. One participant told us that this was "the greatest mystery of my life explained," while many others spoke of their relief at discovering that they were not alone and of their general curiosity to learn more about what the implications of their aphantasia might be. We have yet to do justice to the eloquent descriptions that many participants have provided regarding aphantasia and the alternative modes of avisual, and even a-sensory, forms of representation they use in daily life. Some examples include: "the shape of an apple if you felt it in your hands in the dark"; "like painting with

jet-black paint on a jet-black canvas, you can see it in the movement"; "thinking only in radio"; "being the object"; "I excel in mental rotation—I can't begin to describe how."

More recent work has investigated how extreme imagery might impact vocation (Zeman et al., 2020). These data point to an association between hyperphantasia and careers in "creative" industries, whereas aphantasia is linked to professions associated with mathematics and computing. Interestingly, this his aligns well with Galton's early observation that "the men of science to whom I first applied (his breakfast table imagery task), protested that mental imagery was unknown to them, and they looked on me as fanciful and fantastic in supposing that the words "mental imagery" really expressed what I believed everybody supposed them to mean." This was, however, challenged by later work (Brewer and Schommer-Aikins, 2006). It should be noted, however, that while having aphantasia might predispose to careers in analytic sectors, having aphantasia does not preclude someone from becoming an artist. A substantial number of aphantasic visual artists contacted us: they realized in the course of their development that they differed from the majority of their artist peers in lacking a mind's eye. Some told us that they relied on having their subject in front of them as they worked, while others described using their paper or canvas *as* their mind's eye, deploying a kind of imagination-in-action. Aphantasic novelists and architects also described their distinctive experience as compared to their peers. These participants brought home to us the important distinction between visualization and imagination: it should be made clear that aphantasia does not imply a lack of creativity. The creativity of these artists at both extremes of the vividness spectrum was celebrated in a 2019 exhibition, "Extreme Imagination—inside the Mind's Eye" (Zeman et al., 2019).

Around one-third of participants also describe difficulty in recognizing familiar faces and in recollecting autobiographical events. Reports of autistic spectrum disorder have also been a recurring theme—however, this has not yet been examined systematically. The opposite associations in participants with hyperphantasia have not been found: their first-person testimony does, however, suggest an elevated frequency of synesthesia (Zeman et al., 2020).

The data suggest variability in two other respects. First, in line with the original, much smaller, sample, data from both the United Kingdom (Zeman et al., 2020) and Australian (Dawes et al., 2020) aphantasia research groups provide evidence of differences in dreaming in the aphantasic population as compared to the general population. Interestingly, this seems to be specific to the sensory qualities of the dreams, with many of those who do not dream visually report dreams with narrative, "conceptual," and emotional content. By contrast, visual dreaming is almost universal among those with hyperphantasia. Second, many participants with aphantasia report the absence of imagery in all modalities: however, some also report experiencing imagery in at least one other modality, suggesting that there are both modality-general and modality-specific influences of imagery vividness and potential subgroups within aphantasia.

It is possible that genetic factors influence extreme imagery, with participants with aphantasia and hyperphantasia reporting that first degree relatives share their imagery extreme more often than would expect by pure chance. A survey of over 1000 individuals in a local Biobank suggests a prevalence of extreme aphantasia (16/80 on VVIQ) of 0.7%, while extreme hyperphantasia (corresponding to a score of 80/80) is more common at 2.6%. It is too early to conclude firmly that this reflects a genetic influence, but a genetic contribution is plausible.

Experimental studies of aphantasia

Although measuring mental imagery objectively has historically been difficult, great strides are currently being made through innovative behavioral, physiological, and neuroimaging paradigms. In addition to our recent work showing aphantasic individuals lack sensory visual imagery, as measured using the binocular rivalry paradigm (Keogh and Pearson, 2018a), we have shown distinctive physiological responses to stories that normally evoke visual imagery. Specifically, aphantasic individuals do not show the same change in skin conductance in response to reading fearful scenarios (Wicken et al., 2019), and preliminary work based on research by Laeng and Sulutvedt (2014)) demonstrates that they show a lack of a content-specific pupillary response to imagined images of differing brightness (Keogh et al., 2019). These objective measures further substantiate self-reports of a lack of visual imagery in this population.

Of all our cognitive abilities, memory perhaps has been the most tied to visual imagery. From a personal perspective, many of us experience very intense and sometimes movie like imagery when we recall memories of the past. Imagery's role in memory has been tied to both short-term memory (i.e., visual working memory (Keogh and Pearson, 2011, Albers et al., 2013, Keogh and Pearson, 2014, Pearson and Keogh, 2019)) as well as long-term memory, such as autobiographical memory (Greenberg and Knowlton, 2014; Sheldon et al., 2017; Aydin, 2018) and eye-witness reports (Dobson and

Markham, 1993). One might expect therefore that aphantasic individuals may have serious impairments in their visual memory. However, from the data collected thus far, this does not seem to be the case.

Preliminary work from our lab (Keogh and Pearson, 2018b) and a previous case study (Jacobs et al., 2018) have found that visual working memory performance in people with aphantasia is unaffected by their lack of visual imagery. What differs between them and the general population appears to be the strategy they use to remember visual images. While a large majority of the general population report imagining images to remember them, individuals with congenital aphantasia report using what we have come to refer to as a "labelling" strategy, where they very quickly pick out key characteristics of an image and create a verbal label that they remember for later use. This labeling strategy appears to be automatic for many of the participants. Preliminary work indicates that even on very difficult visual memory tasks with a large number of items, or very small changes, in the visual working memory task this labeling strategy works well for these individuals. It appears that although they do not have a mind's eye, they are exceptional at labeling and picking up details in a display to later remember. We suspect that this technique has been learnt and perfected over the period of their life, and as such we might expect that acquired aphantasics would demonstrate an impairment in visual working memory due to previously having access to visual imagery as a tool for cognition. However, an alternative explanation for the preservation of visual working memory is that aphantasic individuals have unconscious access to visual imagery and that their reported labeling strategy is epiphenomenal. Future research will allow us to tease out the strategies and memory stores employed by aphantasic individuals in such tasks. As discussed further later, the reports by many aphantasic individuals that their autobiographical memory is poor, together with limited empirical results (Greenberg and Knowlton, 2014), suggest that aphantasia may have a greater impact on this domain than on working memory, but further studies are required to clarify this.

Directions for future research

While the popular media has shown a fascination with aphantasia (Grinnell, 2016; Clemens, 2018), scientific research on the topic is still in its infancy. We highlight some key questions we think will be fruitful and the field should focus on.

Subtypes of Aphantasia. It is unclear as yet whether aphantasia represents the extreme of a continuous spectrum or is somehow a discrete state—or indeed a set of states, if, as seems likely, it has several subtypes. The existence of subtypes appears likely, mapping onto alterations in different parts of the complex brain network that subserves visualization. For example, as mentioned earlier, people with aphantasia vary as to whether there is a reduction of visual imagery alone or of imagery in all sensory modalities. We may need to develop a new terminology to distinguish these varieties of experience, and further behavioral and neural studies are required to validate and understand these variations. The presence or absence of accompanying face recognition difficulties, autobiographical memory impairment or autistic spectrum disorder may—or may not—point to discrete subtypes of aphantasia: further research should clarify this question.

Autobiographical Memory and Aphantasia. Many aphantasic participants have reported an impoverishment of autobiographical memories (Dawes et al., 2020; Zeman et al., 2020). Preliminary (unpublished) data provide some objective evidence that aphantasic individuals have less detailed sensory memories. These findings dove-tail with the recently reported syndrome of severely deficient autobiographical memory (SDAM), which describes a group of individuals who have a "self-reported selective inability to vividly recollect personally experienced events from a first-person perspective" (Palombo et al., 2015). A number of aphantasic individuals have also reported that they too experience SDAM. The relationship between aphantasia and SDAM awaits further clarification.

Aphantasia and Emotion. In addition to the association between extreme imagery vividness and aspects of cognitive function, it may be that imagery strength and vividness is linked to our emotional lives and behavior. This may be reflected in the—to date, anecdotal—reports of some Aphantasic individuals who describe a relative immunity to emotions such as nostalgia, disgust, and fear at recollected images as well as an enhanced ability to "live in the present." Indeed, the finding described previously of reduced skin conductance changes when imagining fearful scenarios (Wicken et al., 2019) is consistent with these claims, as are preliminary data obtained from a questionnaire on emotional response to trauma. Specifically, aphantasic individuals report far fewer involuntary intrusive memories than control participants, despite experiencing mood changes in relation to stressful life experiences (Dawes et al., 2020). Conversely, visual imagery is often used to relive past and visualize future positive situations.

Many individuals with aphantasia regret that they cannot visualize the faces of friends and loved ones. Further research on the emotional associations of aphantasia is required.

Hyperphantasia

Hyperphantasia, which overlaps with what has previously been called eidetic imagery, lies at the other end of the imagery spectrum, conferring strong and sometimes photo-like imagery. Early in the 20th century, the topic of eidetic imagery attracted much attention: highly detailed imagery was experienced directly after seeing the object, but also days or weeks later (Allport, 1924; Stromeyer and Psotka, 1970; Gray and Gummerman, 1975; Haber, 1979). A rather higher proportion of individuals score at the high than the low end of the imagery vividness spectrum, while true eidetic imagery may be relatively rare, with frequency estimates from 0% to 11% in children (Gray and Gummerman, 1975). Eidetic imagery has been described in individuals with autism such as Stephen Wiltshire who is able to draw cities from memory, even after only briefly seeing a cityscape once, and Temple Grandin who describes herself as a visual thinker with incredible abilities to visualize, stating that "When I design equipment for the cattle industry, I can test run it in my imagination similar to a virtual reality computer program" (Grandin, 2009). As stronger visualization abilities have been observed in children and autistic individuals it has been suggested that the development of language might reduce the ability to visualize (Hudmon, 2009). Due to the difficulties of measuring visual imagery, the idea of a truly photographic or eidetic memory has remained controversial and it requires further objective study.

Although little experimental research has been undertaken with hyperphantasic individuals one would expect them to show the opposite/extreme effects we see with aphantasia, i.e., we should see heightened binocular rivalry priming scores as compared to control participants, as well as enhanced physiological responses to imagined scenarios and stimuli. There may be an association between hyperphantasia and an enhancement of autobiographical memory. In contrast to severely deficient autobiographical memory (as mentioned in Section Autobiographical Memory and Aphantasia) there is also a syndrome termed highly superior autobiographical memory (HSAM), with these individuals being able to recall personal and public events with remarkable accuracy and vividness. People with HSAM tend to score highly on imaginative-absorption and fantasy proneness, suggesting a possible link to imagery ability (Patihis, 2016). Contemporary methods including psychophysics, fMRI and TMS should be brought to bear on the neglected topics of eidetic memory, hyperphantasia, and their interrelationships.

IMPLICATIONS AND UNANSWERED QUESTIONS

The discovery—or rather the rediscovery of aphantasia, and, importantly, its naming, has attracted from a wide range of audiences: most notably interest from people outside the academy, from artists (see Aldworth, 2018, MacKisack, 2018, novelists (Miller, 2017), philosophers (D'aloisio-Montilla, 2017), and scientists (Clemens, 2018). It is likely that this level of interest reflects the centrality of imagination to many of our everyday and mental lives. As Robin Dunbar has written, "What sets us apart is a life in the mind, the ability to imagine" (Dunbar, 2004). Our capacity to detach ourselves from the present and to enter the virtual worlds of memory, prospection, the fictional world of a novel—or just to daydream during a Zoom meeting—defines many hours of our daily existence. For many us, visualization is a key facet of imagination making it difficult for us to comprehend that some people function perfectly fine in the absence of visualization, and indeed, apparently in some cases, without *any* sensory imagery.

While our ability to represent things in their absence—or "displaced reference" (Bickerton, 2014)—is crucial both to our inner experience and to our social exchanges, sensory imagery does not seem to be required to represent something internally. It would appear Aristotle was incorrect when he wrote that "The soul never thinks without a phantasm," perhaps seduced by the strength of his own imagery. In the same way, it appears many scientists positions on the imagery debate were also informed by the strength of their own imagery (Reisberg et al., 2003). It would appear that rather than visual imagery being the only representational format in the human brain, we have access to many forms of representation, visual, avisual, and even asensory. After all, language itself provides a powerful a medium for mental representation.

Visual imagery is clearly a powerful sensory cognitive tool for creating simulations, of events or objects in their absence, and holding these in mind, but it is not the only one. For those that have strong imagery, it is likely ubiquitous and often involuntary. Aphantasic individuals on the other hand can perform many of the tasks one might have assumed can only be performed using imagery, providing further evidence of the multirepresentational nature of the brain.

Further, while visualization is, for many of us, an eye-catching ingredient of a more general imagination, we should not conflate the ability to visualize with imagination. There are many highly imaginative or

creativity aphantasic individuals, as evinced by the prolific neurologist, Oliver Sacks (who we would now say was likely aphantasic, though he could hear music in his mind's ear). The leader of the first team to decode the human genome, Craig Venter, attributes his scientific prowess at least partly to his aphantasia and his lack of visual imagery. Blake Ross, the cocreator of Mozilla Firefox and an entrepreneur, shared a "viral" Facebook post that alerted many to the existence of aphantasia through describing his own "blind mind" (Ross, 2016).

What does the future hold for visual imagery research? We will highlight some broad questions we think will likely be fruitful future avenues:

1. What is a mental image? Why are mental images generally weaker than normal everyday perception? For example, imagining an apple is less clear and vivid than seeing an apple. This question relates to the underlying brain mechanisms of imagery. How are perceptual images and mental images represented in the brain? What are the differences? Answering this question would inform us as to why some have stronger imagery than others and what limits imagery from becoming realistic and indistinguishable from perception.
2. What is the relationship between voluntary and involuntary phantom vision? Answering this question has important implications of mental and neurological disorders. It might be able to inform us as to how we control our imagery, which will inform new treatment options for those with uncontrollable traumatic imagery.
3. Why do such large individual differences exist in our experience of visual imagery? Why are some people more predisposed to using visualization, while others prefer language-based formats?
4. Does aphantasia exist? If you have read this contribution, you may be persuaded that it does, but there remains some scope for skepticism: how accurately can people describe their imagery experience? Can we be sure that someone describing aphantasia really lacks imagery? We are of course unable to observe the imagery of others directly, and introspection can be unreliable. Edging toward an answer to these questions requires a gradual triangulation: if this distinctive variation in experience exits, we should expect it to have both behavioral and neural correlates. We and others are seeking to measure these.
5. Why do some people lack mental imagery? (aphantasics) It seems that some people are born without any imagery and others lose it, but we do not yet know why in any detail. What is different in an aphantasics brain when compared to someone with imagery? Answering these questions would shed light not only on the causes of aphantasia and if it is reversible, but also on the brain mechanisms of mental imagery more generally.
6. Can aphantasia be understood using the model of predictive coding that is being widely employed in models of both perception and action? Our capacity for visualization is a natural outcome of a predictive model of perception: its absence in some individuals poses an interesting challenge for such models.
7. Can we "cure" or reverse aphantasia? This question has been posed by many of our research participants. While it appears to be entirely possible to lead a normal, fulfilling and indeed creative life with aphantasia, many people with aphantasia would like to be able to experience visual imagery at will. This reasonable question deserves careful study: a recent report (Keogh et al., 2020), showing that it is possible to modulate imagery strength in people with "average" imagery, using transcranial electrical stimulation, may point the way. However, whether this would generalize to those with no imagery at all remains an open question. Perhaps a form of training would be required, in addition to any brain stimulation. Answering these questions would not only inform us about the underlying mechanisms of mental imagery but also lead to a practical product to enable self-training of imagery abilities. Some with aphantasia have said that they would like to have it; hence, the understanding and development of such a training protocol would have great impact for such individuals.

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