Progress in Neurobiology xxx (2014) xxx–xxx

Contents lists available at ScienceDirect

Progress in Neurobiology

journal homepage: www.elsevier.com/locate/pneurobio

Tricks of the mind: Visual hallucinations as disorders of attention

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ARTICLE INFO

Article history:
Received 9 September 2013
Received in revised form 29 January 2014
Accepted 30 January 2014
Available online xxx

Keywords:
Hallucinations
Common neural mechanism
Attentional Control Networks
Dorsal Attention Network
Default Mode Network
Ventral Attention Network

ABSTRACT

Visual hallucinations are common across a number of disorders but to date, a unifying pathophysiology underlying these phenomena has not been described. In this manuscript, we combine insights from neuropathological, neuropsychological and neuroimaging studies to propose a testable common neural mechanism for visual hallucinations. We propose that 'simple' visual hallucinations arise from disturbances within regions responsible for the primary processing of visual information, however with no further modulation of perceptual content by attention. In contrast, 'complex' visual hallucinations reflect dysfunction within and between the Attentional Control Networks, leading to the inappropriate interpretation of ambiguous percepts. The incorrect information perceived by hallucinators is often differentially interpreted depending on the time-course and the neuroarchitecture underlying the interpretation. Disorders with 'complex' hallucinations without retained insight are proposed to be associated with a reduction in the activity within the Dorsal Attention Network. The review concludes by showing that a variety of pathological processes can ultimately manifest in any of these three categories, depending on the precise location of the impairment.

Contents

1. Introduction

The presence of visual misperceptions and hallucinations is characteristic across a wide range of disorders that have different underlying pathophysiological causes (Teeple et al., 2009), making the identification of a unifying mechanism difficult. Previous work has suggested that the origin of these symptoms is likely to be multifaceted, with a range of pathological changes that impact on the capacity of neural circuits to successfully integrate information (Diederich et al., 2009). In this manuscript, we performed a targeted survey of the relevant literature in an attempt to provide a neuroanatomical framework underlying visual hallucinations, regardless of the specific disorder in which they occur. It is hoped that such a framework may help to provide the basis for integrated research across disciplines facilitating novel therapeutic strategies.

Dysfunction in the complex neural networks underlying sensory perception of the surrounding environment can lead to a wide spectrum of abnormal phenomenological experiences, ranging from illusions and misperceptions to frank well-formed hallucinations. Hallucinations have been described as "involuntary sensory experiences perceived as emanating from the external environment, in the absence of stimulation of relevant sensory receptors" (Epstein et al., 2002), whereas misperceptions refer to situations where an actual external stimulus is misperceived or misinterpreted (Barnes and David, 2001). Impaired perception can occur in any sensory domain and has been related to various loci including disturbances of primary sensory input, the midbrain and

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http://dx.doi.org/10.1016/j.pneurobio.2014.01.004

thalamus as well as within higher cortical regions (Adachi et al., 2000; Anderson and Rizzo, 1994; Behrendt and Young, 2004; Behrendt, 2003; Ffytche et al., 1998; Frith and Dolan, 1997; Imamura et al., 1999; Matsui et al., 2006; Mocellin et al., 2006; Nagano-Saito et al., 2004; Nashold and Wilson, 1970; O'Brien et al., 2005).

Visual hallucinations are often further divided into two main categories: 'simple' hallucinations, which lack recognisable form, such as a "menacing shadow"; and 'complex' hallucinations, where well-formed images, such as faces, people, animals, inanimate objects or complex scenes are perceived (Santhouse et al., 2000). Simple visual hallucinations are often related to pathology within primary visual processing systems, such as the primary visual cortex (Anderson and Rizzo, 1994) (e.g. migraine and epilepsy (Manford and Andermann, 1998)); whereas more complex hallucinations are thought to arise due to impaired integration between the neural mechanisms underpinning perception and attentional processes (Collerton et al., 2005; Ffytche, 2008), or due to dysfunction in the cortical sites responsible for the direct reception and processing of sensory information, such as the visual associative cortices and the medial temporal lobe (Manford and Andermann, 1998; Santhouse et al., 2000) (the putative "What" pathway (Milner and Goodale, 1995)).

Whilst the dichotomy of simple versus complex percepts may help to characterise the experiential quality of different hallucinatory phenomena, the demarcation between the two classes of hallucinations is not always clear. For example, patients with migraine often experience a relatively simple image with no obvious categorical content (such as a jagged line through their visual field), but the image is appreciated in exquisite detail. In contrast, patients with Parkinson's disease (PD) and Dementia with Lewy bodies (DLB) often suffer from hallucinations in which the visual percept is of extremely poor clarity, yet it is readily identified (incorrectly) as a threatening object. In addition, the perception of complex hallucinations is not constrained to disorders with widespread pathology and can occur in disorders with discretely localised pathology, such as macular degeneration in the case of the Charles Bonnet Syndrome (CBS) (Manford and Andermann, 1998).

These examples suggest that the phenomenological character of hallucinations is not simply due to pathology within particular regions (such as specific cortical sites or sensory end organs), but rather to dysfunction across the networks integrating and assessing information (Ffytche, 2008; Shine et al., 2011). Indeed, previous hypotheses to explain hallucinations, such as the Activation, Input and Modulation model (Diederich et al., 2005), the Perception and Action Deficit model (Collerton et al., 2005) and models that implicate dysfunctional thalamocortical systems (Behrendt and Young, 2004), have implicated dysfunction within both sensory and attention systems. Whilst these models have broadly suggested potential neuroanatomical loci, precise pathophysiological mechanisms remain poorly understood. In order to integrate the observations regarding the sensory, attention-related and neurotransmitter mechanisms underlying the manifestation of hallucinations, it is important to appreciate the role of large-scale neural networks and the manner in which they co-ordinate their activity to facilitate veridical perception.

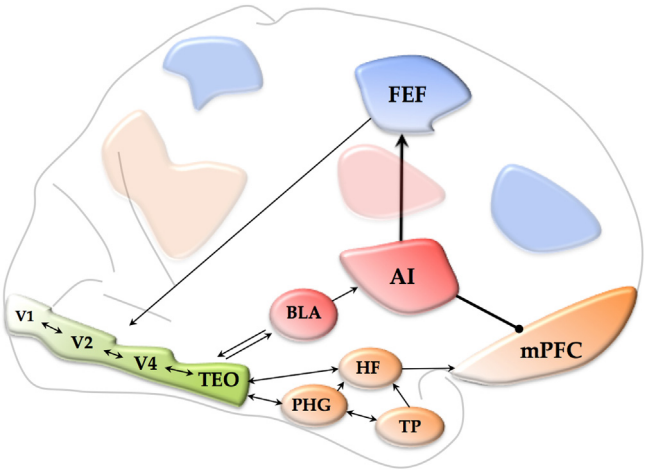


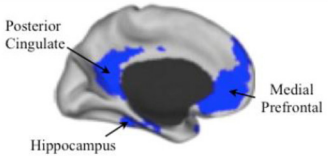
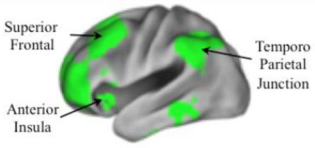
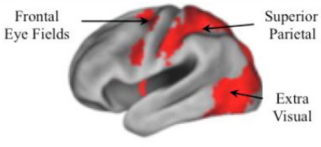
Fig. 1. Large-scale neuronal networks interact with the ongoing processing of visual information (via the ventral visual pathway; Green) and underlie conscious perception. Visual stimuli that are either salient or ambiguous alert the Ventral Attention Network (Red) through the basolateral amygdala, which leads to activity in the Dorsal Attention Network (Blue), which interacts with the ventral visual pathway to clarify the contents of visual attention. The Default Mode Network (Orange), which interacts with the ventral pathway to clarify the categorical content of perception, is inhibited by activity in the Ventral Attention Network. Key: V1, visual area 1; V2, visual area 2; V4, visual area 4; TEO, medial temporal lobe; BLA, basolateral amygdala; HF, hippocampal formation; PHG, parahippocampal gyrus; TP, temporal pole; AI, anterior insula; mPFC, medial prefrontal cortex; FEF, frontal eye fields. Arrows represent excitatory connections whereas closed circles represent inhibitory connections.

2. Attentional networks

Recent work has led to the discovery of a number of key neural networks that co-activate in response to specific internal and exogenous demands (Fornito et al., 2012; Laird et al., 2011; Smith et al., 2009) and also in the absence of an external task (Biswal et al., 1995). The dynamic interaction between these networks has been shown to correlate with performance on complex behavioural tasks (Fornito et al., 2012; Menon, 2011; Menon and Uddin, 2010; Spreng and Schacter, 2012; Spreng et al., 2010) and has been strongly linked with the processes underlying perception and consciousness (Chica et al., 2012; Marchetti, 2012) (Fig. 1).

The Dorsal Attention Network (DAN; Table 1) is group of neural regions that direct attention and the encoding of neural signals related to the behavioural significance of salient stimuli, particularly

Table 1
Attentional Control Networks, their associated areas and function.

Default Mode Network	Ventral Attention Network	Dorsal Attention Network
		
Task-independent thought Mind wandering	Mediate activation of other networks Engages attention to salient stimuli	Voluntary orienting Cognitive information processing

in the visual domain (Asplund et al., 2010; Kincade et al., 2005). The DAN is composed of neural hubs within the frontal eye fields, the dorsolateral prefrontal and posterior parietal cortices as well as specific areas of the striatum (e.g. caudate nucleus). Many of these regions also contain strong white matter connections with the superior colliculus (Bisley and Goldberg, 2010; Mesulam, 1981), a region that is responsible for directing behavioural responses towards specific points in egocentric space. Indeed, the structural and functional connections between the DAN and the superior colliculus are likely to play an important role in saccadic eye and head movements (Kustov and Robinson, 1996; Perry and Zeki, 2000; Pierrot-Deseilligny et al., 2003), as well as the re-orienting of attention (Kustov and Robinson, 1996; Müller et al., 2005). As such, appropriate functioning within the DAN allows for the dynamic re-direction of eye and head movements, focussing attention onto exogenous stimuli and ultimately priming the perception of objects in the external visual field (Knapen et al., 2011).

The Default Mode Network (DMN; see Table 1) is characterised by activation throughout medial temporal, medial prefrontal, posterior cingulate, lateral parietal cortices and the precuneus (Greicius, 2002; Raichle et al., 2001). In contrast to the DAN, the DMN has been correlated with periods of autobiographical 'task independent', self-referential internal thought (e.g. 'mind wandering'), during which the retrieval and manipulation of episodic memories and semantic knowledge occur (Ballard et al., 2011; Binder et al., 1999; Mazoyer et al., 2001). Key hubs within the DMN are also strongly connected with the hippocampus (Andrews-Hanna et al., 2010b) and the ventral visual pathway, implicating the network in the recognition of the object contents of conscious perception (the putative "What" pathway) (Milner and Goodale, 1995). The core hubs within the DMN are strongly related to autobiographical processes, leading some researchers to suggest that the core of the DMN is responsible for the creation of our sense of "self".[6,25-27] These findings further strengthen the role of this neural network in the incorporation of self-referential information into waking consciousness.

A third network of neural regions, the Ventral Attention Network (VAN; Table 1), comprising of the anterior insula and the dorsal anterior cingulate cortex, has been proposed to co-ordinate relative activity between the DAN and the DMN. The VAN has been shown to monitor the flow of sensory information and selectively respond to increases in salience, leading to a rapid engagement of the DAN towards the salient stimulus (Corbetta and Shulman, 2002). Indeed, 'bottom-up' information in the sensory systems that trigger the VAN will often require more 'top-down' processing thus the DAN and VAN are frequently functionally interactive (Asplund et al., 2010; Spreng et al., 2010) and are commonly associated with a concomitant deactivation of the DMN (Fornito et al., 2012; Fox et al., 2005). While the mechanism underlying the successful transition between these competing attentional networks is poorly understood, the basolateral amygdala has been implicated as playing a key role in the communication of environmental salience to the anterior insula region (Menon and Uddin, 2010; Nelson et al., 2010), which appears to be responsible for the effective 'switching' of activity between the different networks. It is also likely that the basal ganglia nuclei (Hikosaka and Isoda, 2010), the cerebellum (D'Angelo and Casali, 2012) and cortical cholinergic interneurons (Xiang et al., 1998) are involved in these capacities.

3. Attentional network dysfunction

Recent work has suggested that the coordinated activities within and between these attentional networks may underlie conscious experience (Posner, 2012), and as such, veridical perception (Bartolomeo et al., 2012; Dehaene and Changeux, 2011). For example, the DAN and the VAN are functionally coupled in a wide array of neuroimaging experiments that explore the neural patterns underlying cognitive control and set shifting (Cole and Schneider, 2007; Wager et al., 2004). Despite some contention regarding the precise relationship between attention and consciousness (Tallon-Baudry, 2011), there is little doubt that activity within these networks has an important impact on the contents of conscious experience (Chica et al., 2012; Marchetti, 2012). As such, an exploration of the phenomenological sequelae arising from the targeted impairment of the neural structures underlying these interactions should help to improve our understanding of disorders of consciousness and perception. Furthermore, neurological dysfunction within different regions of these Attentional Control Networks could ultimately relate to distinct hallucinatory phenomena and visual misperceptions.

In the case of disorders with simple hallucinations, there is strong evidence to suggest that the presence of hallucinatory phenomena are associated with dysfunction at primary sensory cortical sites, such as the occipital lobes (Rogawski, 2008). For example, the visual scotomas experienced by sufferers of migraine headaches are strongly correlated with waves of Cortical Spreading Depression over the occipital lobes (Hadjikhani, 2001). Similarly, hypersynchronous cortical activity in epilepsy localised in the occipital cortex is commonly experienced as flashes of light (Rogawski, 2008). In each case, the perceptual experience of the abnormal neural activity is not recognised as having a categorical aspect, presumably due to a lack of interaction with the cortical regions of the brain that are specialised to 'interpret' sensory stimuli, such as the ventral temporal lobe (20) (see Table 2). Therefore, simple misperceptions and hallucinations can be thought of as disorders of perceptual inference without content modulation from attentional control structures.

It is also possible, however, for patients with dysfunction of primary sensory pathways (such as the retina and the parieto-occipital cortices) to experience complex hallucinations (see Table 2). For example, impairments in primary reception of visual sensory information can lead to incorrect 'pattern matching' in the ventral temporal lobe of the DMN (Kivisaari et al., 2012; Milner and Goodale, 1995); an interpretation that is supported by evidence from functional neuroimaging experiments (Adachi et al., 2000; Ffytche et al., 1998; Kazui et al., 2009). Indeed, even high functioning attention systems would be unable to 'over-ride'

Table 2
Predicted attentional network activities in three classes of hallucinations and the associated disorders: increased (+) or decreased (−).

Attentional network function	VIS Visual perception	DMN Object interpretation	VAN Salience detection	DAN Exogenous attention	Disorders
Simple	+	−	−	+	Epilepsy; migraine; SD
Complex + insight	+	+	−	+	CBS; PCA; AS; Med/Drug
Complex − insight	+	+[a]	+[a]	−	PD/DLB; PH; SZ; PTSD; PS

Key: VIS, visual network; DMN, Default Mode Network; VAN, Ventral Attention Network; DAN, Dorsal Attention Network; SD, sensory deprivation; PD, Parkinson's disease; DLB, Dementia With Lewy bodies; PH, peduncular hallucinosis; PS, parasomnic; PTSD, post-traumatic stress disorder; AS, Anton syndrome; PCA, posterior cortical atrophy; CBS, Charles Bonnet Syndrome; Med/Drug, medication and drug-induced; and SZ, schizophrenia.
[a] Dependent on time-course (see Fig. 2).

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these visual perceptual errors, as there is no mechanism by which to improve the fidelity of the incoming visual information. As such, the presence of visual hallucinations might vary along a spectrum of visual inputs depending on factors such as ambient lighting and familiarity with the environment. Despite the potentially vivid nature of these hallucinations, a high degree of insight would be expected given the preserved functioning that exists across the attentional networks. This 'paradox' is commonly observed clinically within CBS, where patients do not react negatively to the presence of visual hallucinations despite their well-formed, complex visual imagery (Ffytche, 2007). This presumably reflects an appreciation by the intact attentional networks that such images are inconsistent with expectations and can therefore be dismissed as meaningless.

In contrast, patients with conditions that impair attentional processing are more likely to be afflicted by troublesome misperceptions and hallucinations, due in part to impaired insight. For example, patients with PD and visual hallucinations are more likely to suffer from impairments in visuoperceptive functions (Ramírez-Ruiz et al., 2006; Makin et al., 2013), inhibitory control (Barnes and Boubert, 2008), as well as deficits in attentional set-shifting (Shine et al., 2012), suggesting that the well-formed complex visual hallucinations in PD are due to dysfunction within more widespread brain circuits than simple visual perceptual processing pathways. However, many of the disorders associated with complex hallucinations also display pathology within primary receptive systems, such as retinal dysfunction in PD and DLB (Altintaş et al., 2008; Moreno-Ramos et al., 2013), and occipital cortex hyper-activity, as evidenced by experiments using transcranial magnetic stimulation (Taylor et al., 2011).

Many lines of research have also implicated impaired levels of a variety of neurotransmitters in the pathophysiology of hallucinations, including dysfunction within monoaminergic (Goetz et al., 1998), cholinergic (Francis and Perry, 2007) and serotonergic systems (Geyer and Vollenweider, 2008). For example, the ratio of dopaminergic to cholinergic activity has been shown to relate to the severity of visual hallucinations in PD (Diederich et al., 2009) and is also thought to underlie the pathophysiology of peduncular hallucinosis (Benke, 2006), as well as the hallucinatory effects of methamphetamines (Hermens et al., 2010). Interestingly, recent research has shown that dopaminergic levels modulate the connectivity between attentional networks and their striatal and midbrain targets (Cole et al., 2012), suggesting that improved information processing efficiency in the attentional networks may underlie this benefit. This interpretation is also consistent with a mechanistic understanding of antipsychotic medications used to treat schizophrenia, in which the blockade of dopaminergic receptors could potentially restore the delicate balance of neurotransmitter populations in key neural hubs (Cole et al., 2012).

4. A common neural mechanism for visual hallucinations

A recent synthesis of these abnormalities has led to the proposal that visual hallucinations in PD are due to an inability to recruit the DAN in the presence of ambiguous percepts, leading to decreased activity within the superior colliculus, thereby impairing the delicate process of feed-forward and feed-back activity between the cortical and brainstem structures mediating perception (Shine et al., 2011). Impairments in the neural communication between the external world and the DAN would therefore lead to an over-reliance on other attentional networks, such as the DMN, to interpret ambiguous stimuli. Although perceptual insight is likely to be mediated through more widespread networks, such as those encompassing the frontal pole (Shad et al., 2006) and the posterior cingulate cortex (van der Meer et al., 2010), it is possible that these regions will mediate perceptual insight through functional interactions with the DAN. Therefore, when the DAN is underactive, exogenous perceptual information cannot be utilised to update the interpretation of ambiguous percepts. This hypothesis has received initial support from behavioural experiments (Shine et al., 2012), along with magnetic resonance spectroscopy (Lewis et al., 2012) and fMRI imaging studies (Shine et al., 2013). In addition, the model is well aligned with other findings in the field, including both structural (Ibarretxe-Bilbao et al., 2011) and functional imaging (Meppelink et al., 2009; Ramírez-Ruiz et al., 2008; Stebbins et al., 2004) experiments, and also with pathological studies that reveal the presence of severe Lewy body pathology in the basolateral amygdala, insula and claustrum (Yamamoto et al., 2007). Together, these results suggest that the phenomenological symptoms of visual hallucinations in Lewy body disorders may be operating through impaired coordination of the attentional networks as they attempt to overcome impairments of primary sensory systems (Shine et al., 2011).

While this attentional network model has previously focussed solely on dysfunction within Lewy body disorders, the themes are also consistent with other disorders that are characterised by hallucinations and attentional impairment, such as schizophrenia (Bleich-Cohen et al., 2012). A hallmark of schizophrenia is the presence of constant and unnecessary neural responses to environmental and internal salience, leading to over-activation of the VAN (Dichter et al., 2010) and subsequent recruitment of the DAN to stimuli that have no ecological salience, having been incorrectly attributed salience by the dysfunctional attention system (White et al., 2013). Furthermore, schizophrenia has recently been associated with a decrease in the synaptic density within the anterior insula cortex (Bennett, 2011), along with decreased grey matter in temporal cortical regions associated with the processing of auditory information (Allen et al., 2012; Shergill and David, 2006; Silbersweig et al., 1995). As such, hallucinations in schizophrenia (which are predominantly auditory in nature) may reflect an inability to appropriately orient attention (through the DAN) to salient phenomena, leading to the inappropriate interpretation of poorly attended auditory signals as being externally created. Interestingly, the DMN has been shown to contain altered connectivity profiles in schizophrenia (Whitfield-Gabrieli and Ford, 2012), suggesting that the incorrect interpretation of stimuli may occur via a mechanism similar to that seen in other pro-hallucinatory disorders. Importantly, these mechanisms can occur independent of the specific disease process, and may help to explain the presence of extra-visual hallucinatory phenomenon in PD (Fenelon et al., 2000), which may reflect an 'over-interpretation' of impaired sensory information in auditory and somatosensory domains.

The commonalities between the different disorders suggest the possibility of a unifying neural mechanism underlying all visual hallucinatory phenomena (see Fig. 2). Indeed, it is possible that hallucinations simply represent impairments within the normal engines of perception, albeit in specific ways due to the particular pathophysiological mechanism of each disorder. Perception is an active process that requires integration across space and time, and within and between multiple levels of the nervous system, along with a number of complimentary yet competing neurotransmitter systems (Noe, 2004). Indeed, previous studies have shown that medication targeting dopaminergic depletion can have contrasting effects on cognitive performance (Cools and D'Esposito, 2011; Lewis et al., 2005). This observation has been related to the differential loss of dopamine that occurs across the striatum with neuropsychological performance obeying an inverse U-shaped curve response to local neurotransmitter levels (Cools and D'Esposito, 2011). It is interesting to note that in clinical practice, hallucinations are usually improved by the reduction of dopaminergic therapy or by the use of anti-psychotic medications that target dopamine receptors, regardless of the underlying disease

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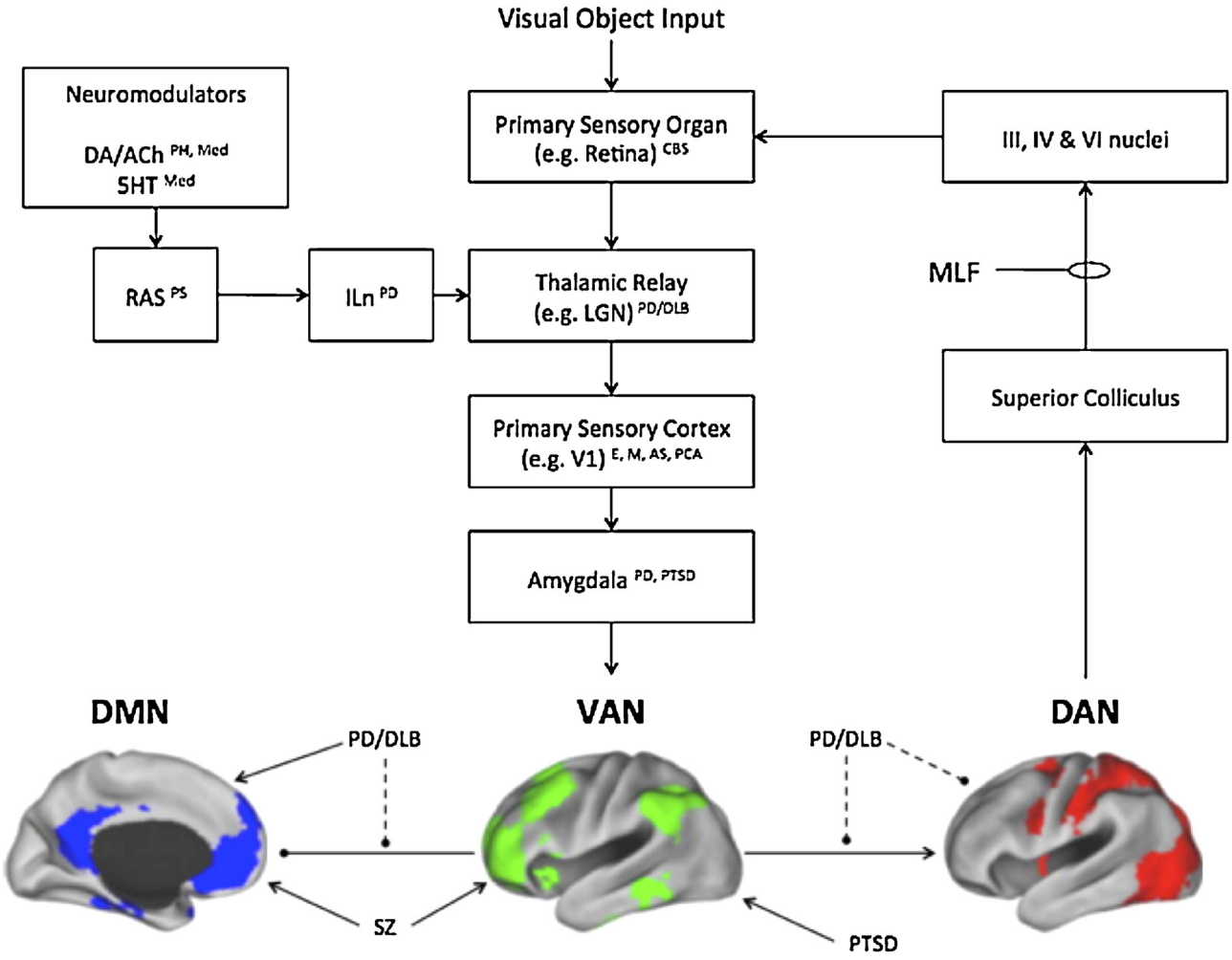

Fig. 2. A common neural mechanism for visual hallucinations. Pathological impairment at different regions along the visual perception pathway ultimately manifest as visual misperceptions and hallucinations due to impaired communication between Attentional Control Networks, such as the Default Mode Network (DMN), the Ventral Attention Network (VAN) and the Dorsal Attention Network (DAN). Key: RAS, reticular activating system; ILn, intra-laminar nuclei of the thalamus; LGN, lateral geniculate nucleus; V1, visual region I; MLF, medial longitudinal fasciculus; PD, Parkinson's disease; DLB, Dementia with Lewy bodies; PH, peduncular hallucinosis; PS, parasomnic; PTSD, post-traumatic stress disorder; E, epilepsy; M, migraine; AS, anton syndrome; PCA, posterior cortical atrophy; CBS, Charles Bonnet Syndrome; Med, medications; and SZ, schizophrenia.

pathology. Thus, impaired switching across the attentional networks may be modulated by local neurotransmitter levels and should be investigated in future studies. However, it is not clear from current research whether high concentrations of dopamine are causative of visual hallucinations or merely a mitigating factor that worsens pre-existing impairment due to other pathological processes (Barnes and David, 2001).

Specific impairments at any point in the ongoing processing of information can lead to a loss of the quality of perception, and ultimately lead to hallucinations. For example, pathology in the primary sensory organ (such as the retina in CBS), in the primary sensory cortex (such as to the occipital lobe in Anton's syndrome, Posterior Cortical Atrophy or in epilepsy) or in subcortical structures that are only indirectly involved in visual perception, such as the amygdala (as in DLB and PD), is associated with the presence of visual misperceptions and hallucinations. Global disturbances in arousal (such as in the transition from the awake to the sleep state) and disorders of awareness (such as in peduncular hallucinosis (Benke, 2006)) can also modulate the attentional system to produce a less efficient visual search. However in each case, the pattern of attentional network dysfunction differs and as a result, so does the phenomenological characteristics of the hallucination (see Table 2).

Abnormal synchronous neural activity confined to sensory cortices appear to manifest as simple hallucinations, often experienced in the primary visual cortex as flashes of light or as simple geometric patterns across the visual field (Rogawski, 2008). In contrast, the mechanisms underlying complex misperceptions and hallucinations are driven by abnormalities in the co-ordination within and between Attentional Control Networks. In the case of complex hallucinations with retained insight, ventral temporal structures within the DMN become activated in an attempt to 'pattern-match' the ambiguous sensory information arriving from the damaged sensory organ and its cortical relays (Ffytche, 2007). Indeed, the hallucinatory content in these disorders is often bizarre (Ffytche, 2007), suggesting that the co-ordination of this capacity is not under conscious control. In complex hallucinations without insight, failure to recruit the DAN during periods of perceptual ambiguity would result in the interpretation of those perceptual targets by neuronal networks poorly suited to the task, such as the VAN and DMN (Shine et al., 2011).

In this model, the difference between simple and complex hallucinations is therefore related to the degree of DMN and VAN influence over the contents of perception. If the VAN is involved in the perceptual experience, the 'misinterpretation' would likely occur due to the over-ascribing of salience to a misperceived stimulus (e.g. misperceiving a hose as a threat, such as a snake)

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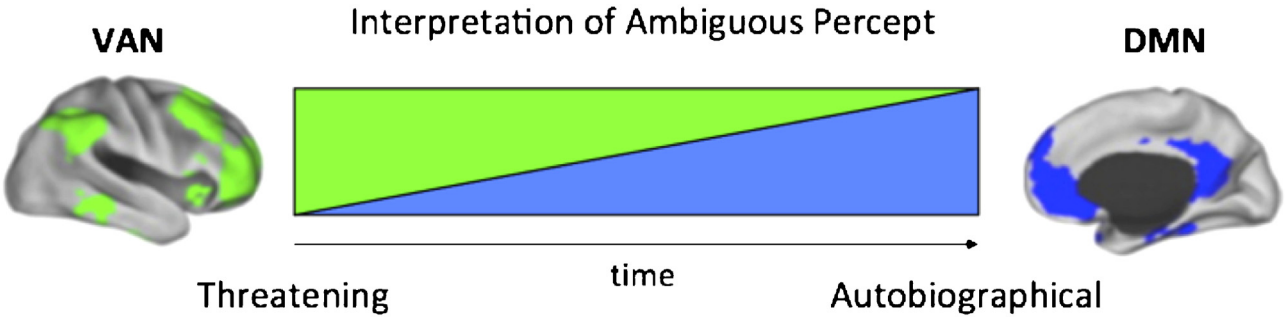

Fig. 3. In the presence of an ambiguous visual percept and decreased activity within the Dorsal Attention Network, interpretation of the ambiguous percept is proposed to occur through neural circuitry that is poorly prepared for such a task. Under significant time pressure, the Ventral Attention Network (VAN; Green) is more likely to interpret the stimulus as threatening. When there is little time pressure, the Default Mode Network (DMN; Blue) is more likely to incorporate autobiographical information into the interpretation.

(Menon and Uddin, 2010; Seeley et al., 2007). If the DMN is utilised to interpret an ambiguous percept, the misperception is more likely to incorporate autobiographical information (e.g. misperceiving a hose for a snake because the patient might associate a snake with the garden environment) (Spreng et al., 2010). This mechanism also suggests that perceptual errors would be manifested through different networks, depending on the specific time-course of the hallucinatory percept (see Fig. 3). For example, if a decision is required under temporal pressure, it is likely that the VAN will interpret the percept as a salient object. The paradigm example of this VAN-mediated interpretation is post traumatic stress disorder (PTSD), which is characterised by overactivity of the VAN (Daniels et al., 2010; Fani et al., 2012) and impaired amygdala function (Khoury-Malhame et al., 2011). In contrast, if there is no temporal pressure placed on the interpreter, it is more likely that the core regions of the DMN will incorporate specific autobiographical information into the hallucination (Andrews-Hanna et al., 2010a; Laird et al., 2011; Spreng et al., 2010; Spreng and Schacter, 2012), a situation that is often the case in PD (Fenelon et al., 2000; Ffytche, 2008; Shine et al., 2011).

Despite the qualitative distinctions between the different categories of visual hallucinations, it is likely that the phenomenological characteristics of the different disorders exist instead along a perceptual spectrum. For example, the experience of epileptic activity is strongly related to the neural region in which the activity occurs and is most commonly perceived as flashes of light, presumed to occur secondary to activity in the occipital cortices (Rogawski, 2008). However, epileptic syndromes are also associated with the presence of complex hallucinations, possibly related to oscillatory epileptiform activity in higher visual processing centres, such as the occipitotemporal junction in the case of palinopsias (Meadows and Munro, 1977). Similarly, PD patients with visual hallucinations regularly experience both simple and complex misperceptions (Fenelon et al., 2000). In each of these cases, the specific pathophysiological mechanism underlying the disorder manifests as either a simple or complex percept, depending on which precise neuroanatomical regions are involved in each unique misperception. As such, greater clarity may be achieved by reconceptualising visual misperceptions and hallucinations with respect to the dysfunctional interactions within and between the Attentional Control Networks, rather than by their perceptual content per se (see Table 2).

Although each of the clinical entities that manifest visual misperceptions and hallucinations can be incorporated into this neuroanatomical model (see Fig. 2), a number of outstanding questions remain. For example, the neural correlates of veridical perception remain unclear (Blake and Sekuler, 2006; Blake and Wilson, 2011; Noe and Evan, 2004), with little consensus as to whether perception arises from focussed activity in one neural region, such as the claustrum (Crick and Koch, 1990), or as a more distributed process occurring over widespread regions of the brain (Blake and Sekuler, 2006; Blake and Wilson, 2011; Noe, 2004). Furthermore, much collaborative work across conditions must be performed in order to test the specific predictions of the 'common neural mechanism' hypothesis. Functional and structural neuroimaging offer one potential means for testing a number of these speculations in the near future. If the patterns of neural dysfunction in the different hallucinatory disorders do in fact conform to the predictions of this hypothesis, the potential for improving therapeutics and diagnostic markers is immense.

5. Conclusions and future directions

The proposed mechanism has the potential to act as a 'common ground' between the studies of hallucinatory phenomena in different neuropsychiatric disorders, allowing the exploration of the precise time-course of hallucinations along with their response to different combinations of therapeutics. The model will also allow for the creation of disease-specific predictions that can be formally tested with neuropsychological measures that are able to objectively evoke misperceptions and hallucinations in a clinical environment, such as testing of visual perception while viewing degraded images (Schwartzman et al., 2008) or tasks that probe bistable perception (Shine et al., 2012). Combined together, these mechanisms can lay the framework for improving our understanding of misperceptions and hallucinations in a wide range of disorders.

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Please cite this article in press as: Shine, J.M., et al., Tricks of the mind: Visual hallucinations as disorders of attention. Prog. Neurobiol. (2014), http://dx.doi.org/10.1016/j.pneurobio.2014.01.004

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Please cite this article in press as: Shine, J.M., et al., Tricks of the mind: Visual hallucinations as disorders of attention. Prog. Neurobiol. (2014), http://dx.doi.org/10.1016/j.pneurobio.2014.01.004

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Please cite this article in press as: Shine, J.M., et al., Tricks of the mind: Visual hallucinations as disorders of attention. Prog. Neurobiol. (2014), http://dx.doi.org/10.1016/j.pneurobio.2014.01.004