The Functional Roles of Attention

Bob K & Brit B

Abstract

Though it is widely agreed that attention and consciousness are distinct phenomena with functionally and anatomically distinct, underlying neural substrates (Crick & Koch, 2003; Kentridge et al. 2004; Koch and Tsuchiya, 2007), there is less consensus about what the functional role of attention is and how it relates to consciousness. Here we argue that two main functions of visual attention are to enhance the precision of visual representations and to modulate the informational input to the visual system, leading to visual representations with a greater chance of tracking properties in the external world.

Keywords: attention, informational overload, psilocybin, type-2 blindsight

1. Neural Architecture and Neural Coding of Information

When confronted with a new visual scene we need to interpret it in terms of objects in the world, not the retinal image. How might attention help us achieve this? Let us consider some implications of broad principles of the neural architecture and neural coding of information in the visual system.

The architectural principle we want to stress concerns systematic changes in the receptive field properties of cells as we move from primary visual cortex along the ventral visual stream moving into the temporal lobe towards the temporal pole (see e.g. Gross, Rocha-Miranda & Bender, 1972). In primary visual cortex receptive field sizes are small and the properties that receptive fields are tuned for are simple. As we move along the ventral stream receptive fields become larger and tuning properties more complex. Towards the rostral end of the temporal lobe we might find cells which are tuned to respond selectively to specific body parts which have receptive fields spanning nearly an entire hemifield. Consider the size of a hand, a face or indeed any other object that these cells might be tuned for. These objects almost always subtend visual angles much smaller than a hemifield. How then can a cell with a much larger receptive field respond selectively to an object when it seems it should be driven, directly or indirectly, not only by signals from the object but also by signals from a large area of space surrounding the object? We can only achieve this if, at levels of the visual system where receptive field are small, signals from the space surrounding the object are suppressed. Is it possible that this is what attention is for?

Consider what we know about the effects of attention on the response properties of cells in the visual system. In the earliest and best known experiment on the effects of attention on the receptive field properties of cells in the visual system (Moran & Desimone, 1985) the effect of attending to a particular object is to effectively shrink an initially large receptive field around the attended object so another object nearby in space no longer affects the cell's responses. Changes induced by attention on the effective size, shape and position of receptive fields have been demonstrated repeatedly since. Reynolds & Desimone (1999) suggest that attentional shrinkage of receptive fields around attended objects serves to solve the binding problem. As only features in the region of space specifically occupied by the object of attention can induce responses in receptive fields shrunk around the object this means that errors attributing features belonging to other objects in nearby space to the object of attention (illusory conjunctions) are no longer possible. The problem of illusory conjunctions is essentially the same as the problem of ensuring that a cell with a large receptive field and complex tuning is not subject to interference from features within its receptive field other than those belonging to the object of attention.

What then should we make of the consequences of attending for the sensitivity of cells that appear to have been demonstrated behaviourally (e.g. Carrasco et al, 2004) or electrophysiologically (e.g. McAdams & Maunsell, 1999)? There is little doubt that attention alters the gain (but probably not sharpness of tuning) of cellular responses to stimuli. One might anticipate that the perceptual consequence of this would be a change in apparent properties of an attended object. This need not, however, be the case. The broad principle of neural coding I want to stress is that representations are generally encoded in a distributed fashion. It is the relative strength (or even relative timing) of responses across multiple cells that encodes information, not the response of individual cells. So, if we increase the gain of all cells within a selected region of space then we do not necessarily affect the property coded across those cells. The relative activities elicited by a stimulus should remain similar even if the absolute amounts of activity all increase. It is *possible* that the increase in gains might improve the signal to noise ratio of responses to the attended object. The most likely consequence, however, is, that by virtue of inhibitory interactions, the response of cells whose gain is not enhanced by attention will be suppressed by a 'winner take all' mechanism (see e.g. Desimone & Duncan, 1996; Lee, Itti, Koch & Braun, 1999). What then might be happening in behavioural tasks that claim to show that the appearance of attended items is altered? One possibility is that the receptive fields of cells responding to the non-attended items remain large and so their responses continue to be influenced by aspects of the stimulus other than the object of interest. With a uniform surrounding background the effect is to 'dilute' the effect of matches between the properties of the unattended object and cells' tuning. The appearance of attended and unattended items may therefore truly differ but there is no ambiguity about the veridical properties of stimuli. Ned Block (2010) suggested that we adopt a view of phenomenal experience dubbed 'mental paint' in order to deal with the uncertainty over what constitutes a veridical phenomenal experience that Carrasco's experiments on the alteration of experience by attention might imply. If, as we suggest here, attention serves to increase the veridicality of experience then we can discard mental paint.

In the account above attention serves to optimise the veridicality of responses of cells involved in coding complex properties of stimuli. Such optimised responses might be used to engage action, but there is no necessity for this. We might need to know about an object for many reasons that do not involve directing actions towards it (e.g. in order to remember it, something it is hard to credit as being 'action'). Similarly, once the identity of an object has been established as best it can, it may be that it is deemed irrelevant and is not subject to any further processing in terms of memory, planning or action. If consciousness is associated with this deeper processing (as global workspace and allied theories hold) then it is clear that it might be necessary to attend to an object for it to enter consciousness but attention is not sufficient - not all attended items need enter consciousness.

2. Further Evidence for Selection for Precision

Further evidence for the idea that a function of attention is to facilitate enhanced perceptual precision comes from the case of type-2 blindsight. Blindsight is a kind of residual vision found in people with lesions to V1. Subjects with blindsight typically report no visual awareness, but they are nonetheless able to make above-chance guesses about the shape, location, color and movement of visual stimuli presented to them in their blind field. Type-2 blindsight is a kind of residual vision found in patients with V1 lesions in the presence of some residual awareness (see e.g. Foley & Kentridge, 2015).

Studies indicate that the attributes experienced in type-2 blindsight are less determinate than the properties experienced in ordinary vision. When shown different letters, blindsight subject DB would sometimes report being aware of the direction of the stimulus and having a feeling of whether the stimulus was 'smooth' (the O) or 'jagged' (the X) (Weiskrantz, et al., 1974). When strongly encouraged to provide an answer, blindsight subject KP described experiencing 'a very faint flash' in response to stimuli (Weiskrantz, 1980), and blindsight subject JP reported being aware of ill-defined and poorly formed 'blobs' when words were flashed in her blind field (Shefrin, et al. 1988). Several subjects have reported having feelings of 'something' or 'something happening' (Weiskrantz, 1986). These reports testify to the hypothesis that the properties that subjects are aware of in their blind field in type-2 blindsight are determinables (as opposed to determinate properties, such as *red* and *square*), sometimes of the most general kind ('something', 'something happening').

The reason type-2 blindsight differs from ordinary visual experience in this respect is likely that it is generated by an alternative visual pathway that bypasses V1. Morland, et al. (1999) investigated blindsight subject GY's ability to make luminance matches in his hemianopic field and between both hemifields. They found that GY was able to make matches when the stimuli were presented in the blind field but was unable to establish matches based on luminance when the stimuli were presented in opposing fields. A plausible explanation of this observation is that the perceived luminance of the stimuli in his blind field (perception of brightness) is derived from direct projections from subcortical areas to extrastriate areas bypassing V1, whereas the perceived luminance of the stimuli in his intact field originate in the normal visual pathway that

includes V1. This would make it possible for him to compare stimuli on the basis of luminance when both are presented in the hemianopic field, but when the stimuli are presented to opposing fields, the distinct pathways would yield different kinds of percepts, making lawful matching difficult. This indicates that V1 plays a crucial role in generating brightness perception. And if GY's type-2 blindsight vision fails to be fine-grained, this further suggests that brightness perception is required for generating conscious awareness of determinate properties.

The latter observation allows us to draw some connections between enhanced perceptual precision and attention. Neurophysiological studies have shown that attentional modulation and changes in the luminance of a stimulus can create identical modulations of firing rates (Carrasco, et al., 2004). It has also been found that attentional modulation and the intensity of the brightness of a stimulus may have a shared underlying mechanism (Treue, 2001). This points to a neural mechanism according to which attention modulates the strength of a stimulus by altering its perceived luminance, or brightness.

But if attentional modulation and changes in luminance have a shared neural mechanism, then we can provide an argument for the view that attentional modulation yields a difference in the determinacy of the perceived attributes. Neurophysiological evidence points to a defect in brightness perception in type-2 blindsight. Brightness perception is likely compromised because type-2 blindsight takes place via a visual pathway that bypasses V1, a region that may be associated with generating brightness percepts. It also appears that only highly determinable (as opposed to determinate) properties are consciously represented in type-2 blindsight. This then suggests that when the mechanism for generating brightness is compromised, then visual experience cannot consciously represent very determinate properties.

These considerations suggest that a possible function of attention may be to modulate the strength of a stimulus by altering its perceived luminance, or brightness, and thereby making the perceptual experience more precise.

3. Prevention of Informational Overload

Modulating the strength of a stimulus by altering its perceived brightness is only one function of attention. Another function appears to be to prevent informational overload by selecting relevant information and filtering out irrelevant information from crowded visual scenes. For the case of vision, it has been found that attention can modulate responses in extrastriate and striate visual cortex and even LGN (O'Connor, et al., 2002) and in that way affect the nature of our visual experiences. It has been reported to do this in three different ways. Attention enhances neural responses to attended stimuli, attenuates responses to ignored stimuli and increases the baseline activity in the anticipation of visual stimulation (Koch and Tsuchiya, 2007).

Independent evidence that a role of attention is to prevent informational overload comes from the case of hallucinogens (psilocybin, LSD, mescaline). The mushroom-derived hallucinogen psilocybin--one of the most selective hallucinogenic drugs studied--has been shown to

significantly reduce subjects' attentional tracking ability, although it has no significant effect on spatial working memory (Carter, et al. 2006). A possible mechanism for how psilocybin affects attentional tracking can be identified by looking closer at how this chemical generates drug-induced hallucinations.

It is by now fairly well established that psilocybin is a potent partial agonists at serotonin 5-HT1A/2A/2C receptors, with serotonin 5-HT2A receptor activation directly correlated with hallucinogenic activity (Glennon, 1990; Vollenweider et al., 1998; Nichols, 2004; Presti and Nichols, 2004). Though the mechanism of action varies for different hallucinogens, it is believed that 5-HT2A receptor activation of cortical neurons is responsible for mediating the signaling pattern and behavioral response to hallucinogens (Presti and Nichols, 2004; González-Maeso et al., 2007).

The activation of the cortical serotonergic system does not fully explain the perceptual effects of psychedelic drugs, as not all 5-HT2A agonists have an excitatory mechanism of action and not all 5-HT2A agonists have psychedelic effects (e.g., methysergide). So, this raises the question of what other factors need to be present for drug-induced hallucinations to occur when subjects are under the influence of psilocybin.

A promising suggestion for how psilocybin and other similar hallucinogenic drugs is associated with visual hallucinatory effects is that it activates layer V pyramidal neurons in the visual cortex, which engage in gating functions in communication between the cortex and the thalamus (Barkai and Hasselmo, 1994; Brogaard, 2013). When a hallucinogen binds to 5-HT2A serotonin receptor on layer V pyramidal neurons, this gives rise to an excitatory response via increased release of the excitatory neurotransmitter glutamate (Scruggs et al., 2003; Nichols, 2004; Ceglia et al., 2004; Torres-Escalante et al., 2004).

Now, 5-HT2A serotonin receptors are found on both layer V glutamatergic neurons and GABAergic interneurons that connect the visual cortex to the thalamus (Lee and Roth, 2012). There is also a direct activation of GABAergic interneurons through the synapses of pyramidal cells onto the interneurons (Markram et al., 2004). So, a large excitatory response in a pyramidal neuron will lead to a large inhibitory response in the interneuron. As GABAergic interneurons inhibit the brain regions they project to, this will inhibit the normal gating mechanisms of the thalamus, allowing too much information to enter the visual cortex.

This points to a mechanism where psilocybin affects attentional tracking by allowing an informational overload of neural information to enter the visual cortex. The additional neural information available to the visual cortex includes both irrelevant information that is normally filtered out as well as random neural activity generated by the thalamus. Drug-induced hallucinations are generated when the brain attempts to make sense of random or irrelevant activity from the thalamus. If this mechanism for how hallucinations may occur under the influence of psilocybin is correct, then that suggests a possible role of selective attention as a modulator of informational input. A function of attention may be to prevent informational overload from the thalamus to sensory cortical areas. This sort of overload would lead to a loss

in the tracking abilities of perceptual representations, as the perceptual representations would be a partial result of the brain's attempt to make sense of random and irrelevant information. So, another way of formulating this role of attention is in terms of veridicality. We might say that attention serves to optimize the veridicality of conscious perceptual representations.

4. Attention for Action?

Wayne Wu (2014) has argued that attention is for action. More precisely: attention is selection that guides task performance. According to Wu, for some modality M (perceptual, cognitive),

> If a subject S M-selects X to guide performance of task T, then S M-attends to X.

Wu refers to this as an 'empirically sufficient condition'. Action suffices for attention. Although Wu also argues that action is necessary for attention, this condition by itself is perfectly consistent with the view that attention has more than one function. One may think of 'attention' as semantically on a par with 'tool'. To a first approximation, the meaning of 'tool' is 'a device held in the hand used to carry out a particular function'. But there are many different functions tools can play. The same goes for attention. As far as we are concerned, one function/purpose of attention could, in principle, be 'for action' (task-related). This does not rule out that attention can also be selection for precision or modulation of informational input. Increased precision and modulation of informational input may be used to engage action or improve action performance but there is no requirement that action is engaged as a result of the optimized responses. A precise perceptual representation of an object may be generated only to be judged to be irrelevant and as a result never undergo further processing for memory, thought, planning or action.

Here, of course, it is important that we don't read the suggestion that attention is selection for precision and modulation of informational input as the claim that enhanced perceptual precision and modulation of visual input *require* attention. If your spouse turns on the light in the room, this will increase the precision of your visual representation. But this sort of increase in precision does not require any additional attentional selection on your part. Likewise, if your spouse replaces most items in the room with uni-colored patches, she will in some sense have modulated how much visual information enters into your visual system. But this type of modulation does not require any additional attentional modulation on your part.

How then are we to understand the claim that attention functions to select for precision and modulate informational input? For the case of vision, the claim is best understood in terms of top-down influences on neural processes in the visual cortex and on neural regions to which the visual cortex projects or from which it receives neural input. Without attentional top-down influences on the visual processes, brightness perception will be remarkably reduced and any conscious visual representation will be notably less precise. Likewise, without the top-down attentional selection of input from the thalamus to the visual cortex, the conscious visual

representation that is generated in the visual cortex will be intermingled with hallucinatory elements that do not track reality.

It may be argued that one could, in principle, artificially mirror the effects of selective attention by directly manipulating the computation of brightness and the informational flow from the thalamus. This is no doubt correct but it doesn't affect the functional claim we are making. A functional role of a mental process is a set of causes and effects that surround a mental process. Specifying a functional role of a mental process thus does not require that we identify a set of mental processes for which the target mental process is required. The aim is to identify a set of causes and effects in a normal brain in normal conditions. As such, it can be a functional role of attention to increase precision and modulate informational input, even if not every *conceivable* enhancement of perceptual precision or modulation of informational input requires attention.

Now, it is plausible that a functional role of attention could be given, where attention simply is understood as the underlying processes that satisfy the specified functional roles. This is a stronger claim, however. It commits us to a conception of radically different underlying processes that satisfy the functional roles as attentional selectional processes. For example,we can imagine that a microchip could be inserted into the visual system of people whose neural systems don't prevent informational overflow and play the role that attention plays in neurotypical individuals. On a functional analysis of attention, this microchip would then not simply play the role of attention. The microchip would be (part of) the attentional system. Whether we ought to make this further and stronger claim is beyond the scope of the debate in this volume.

References

Barkai, E., and Hasselmo, M. E. (1994). Modulation of the input/output function of rat piriform cortex pyramidal cells. *J. Neurophysiol.* 72, 644–658.

Block, N. (2010). Attention and mental paint. *Philosophical Issues* 20, 23-63.

Brogaard, B. (2013). Serotonergic Hyperactivity as a Potential Factor in Developmental, Acquired and Drug-Induced Synesthesia, *Front. Hum. Neurosci.* 7:657, 2013.

Carrasco, M., Ling, S., & Read, S. (2004). Attention alters appearance. *Nature Neuroscience*, 7, 3, 308–313.

Ceglia, I., Carli, M., Baviera, M., Renoldi, G., Calcagno, E., and Invernizzi, R. W. (2004). The 5-HT receptor antagonist M100, 907 prevents extracellular glutamate rising in response to NMDA receptor blockade in the mPFC. *J. Neurochem.* 91, 189–199.

Ciranna, L. (2006). Serotonin as a modulator of glutamate- and GABA-mediated neurotransmission: implications in physiological functions and in pathology. *Curr. Neuropharmacol.* 4, 101–114.

Crick, F. C. & Koch, C. 2003 A framework for consciousness. Nat. Neurosci. 6, 119–126.

Foley, R. & Kentridge, R.W. (2015) Type-2 Blindsight: Empirical and Philosophical Perspectives. *Consciousness & Cognition* 32, 1-5.

Glennon, R. A. (1990). Do classical hallucinogens act as 5-HT2 agonists or antagonists? *Neuropsychopharmacology* 3, 509–517.

Gross, C.G., Rocha-Miranda, C.E. & Bender, D.B. (1972) Visual properties of neurons in inferotemporal cortex of the Macaque. *Journal of Neurophysiology* 35, 96-111.

González-Maeso, J., Ang, R. L., Yuen, T., Chan, P., Weisstaub, N. V., López-Giménez, J. F., et al. (2008). Identification of a serotonin/glutamate receptor complex implicated in psychosis. *Nature* 452, 93–97.

Kentridge, R.W., Heywood, C.A. & Weiskrantz, L. (2004). Spatial attention speeds discrimination without awareness in blindsight. Neuropsychologia 42: 831-835.

Klimesch, W. (2011). Evoked and early access to the knowledge system: the P1 inhibition timing hypothesis. *Brain Res.*1408, 52–71. doi: 10.1016/j.brainres.2011.06.003

Koch, C. and Tsuchiya, N (2007), Attention and consciousness: two distinct brain processes, Trends Cogn Sci. 11(1):16-22

Kometer, M., Schmidt, A., Jäncke, L., and Vollenweider, F. X. (2013). Activation of serotonin 2A receptors underlies the psilocybin-induced effects on oscillations, N170 visual-evoked potentials, and visual hallucinations. *J. Neurosci.* 33, 10544–10551

Lee, H.-M., and Roth, B. L. (2012). Hallucinogen actions on human brain revealed. *Proc. Natl. Acad. Sci. U.S.A.* 109, 1820–1821. doi: 10.1073/pnas.1121358109

Markram, H., Toledo-Rodriguez, M., Wang, Y., Gupta, A., Silberberg, G., and Wu, C. (2004). Interneurons of the neocortical inhibitory system. Nat. Rev. Neurosci. 5, 793–807.

McAdams, C.J., Maunsell, J.H.R. (1999) Effects of attention on the orientation tuning functions of single neurons in macaque area V4. Journal of Neuroscience. 19:431-441.

Moran, J., & Desimone, R. (1985). Selective attention gates visual processing in the extrastriate cortex. Science, 229, 782–784.

Morland AB, Jones SR, Finlay AL, Deyzac E, Le S, Kemp S. (1999). Visual perception of motion, luminance and colour in a human hemianope. Brain 122(Pt 6): 1183–98.

Nagel, E. (1961). The Structure of Science. Indianapolis, IN: Hackett.

Nichols, D. E. (2004). Hallucinogens. *Pharmacol. Ther.* 101, 131–181.

O'Connor, DH, Fukui, MM, Pinsk, MA & Kastner, S (2002). Attention modulates responses in the human lateral geniculate nucleus, Nature Neuroscience 5, 1203 - 1209.

Pesenti, M., Zago, L., and Crivello, F. (2001). Mental calculation in a prodigy is sustained by right prefrontal and medial temporal areas. *Nat. Neurosci.* 4, 103–107.

Scruggs, J. L., Schmidt, D., and Deutch, A. Y. (2003). The hallucinogen 1-[2,5-dimethoxy-4-iodophenyl]-2-aminopropane (DOI) increases cortical extracellular glutamate levels in rats. *Neurosci. Lett.* 346, 137–140.

Shefrin SL, Goodin DS, Aminoff MJ. (1988). Visual evoked potentials in the investigation of "blindsight". Neur ology 38(1): 104-9.

Torres-Escalante, J. L., Barral, J. A., Ibarra-Villa, M. D., Perez-Burgos, A., Gongora-Alfaro, J. L., and Pineda, J. C. (2004). 5-HT1A, 5-HT2, and GABAB receptors interact to modulate neurotransmitter release probability in layer 2/3 somatosensory rat cortex as evaluated by the paired pulse protocol. *J. Neurosci. Res.* 78, 268–278.

Trudeau, L. E. (2004). Glutamate co-transmission as an emerging concept in monoamine neuron function. *J. Psychiatry Neurosci.* 29, 296–310.

Vollenweider, F. X., Leenders, K. L., Scharfetter, C., Maguire, P., Stadelmann, O., Angst, J. (1997). Positron emission tomography and fluorodeoxyglucose studies of metabolic hyperfrontality and psychopathology in the psilocybin model of psychosis. *Neuropsychopharmacology* 16, 357–372.

Weiskrantz, L. (1980). Varieties of residual experience. Quart. J. exp. Psychol. 32, 365-386.

Weiskrantz, L. (1986). Blindsight: A case study and implications. Oxford: Oxford University Press.

Weiskrantz, L., Warrington, E.K., Sanders, M.D., Marshall, J., 1974. Visual capacity in the hemianopic field following a restricted occipital ablation. Brain 97, 709–728.

Wu, W. (2014). Attention, London: Routledge.