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CEREBRAL CORRELATES OF VISUAL AWARENESS

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Abstract—While it may be a long time before we can specify the mechanisms through which a brain process achieves awareness, it may be possible to determine as a first step whether awareness is limited to the products of certain *kinds* of processing. In the domain of vision, for example, perceptual awareness might only be attainable in association with object-centred coding, configural representations of space, and other such forms of abstracted (non-retinocentric) coding. It appears that these forms of visual coding are anatomically restricted to telencephalic structures, and indeed it has been argued that they may be peculiar to, or at least visually dependent upon, the 'ventral stream' of visual areas with the cortex. It is suggested here that such a brain process would still not be able to enter visual awareness unless it was selectively amplified through neuronal gating of the kind that has been shown to be correlated with selective spatial attention. The present paper explores the extent to which this putative dual requirement for visual consciousness might form a basis for understanding the various phenomena of "covert vision" seen in patients suffering from hemianopia, apperceptive agnosia, and unilateral spatial neglect.

Key Words: consciousness; vision; cortex; neglect; agnosia; blindsight.

INTRODUCTION

I shall not attempt in this paper to discuss the nature of visual awareness itself, either in normal or brain-damaged individuals. These matters are not amenable to direct empirical investigation. I shall only be concerned here with phenomena that are reportable, and with questions that are, in principle, answerable. My operational assumption is that in general when a patient disclaims awareness, there is no awareness. Since one can never be in a position to verify or refute the patient's reports, neuropsychology can only operate within an operational framework of this kind. Thus I use the term "unawareness" as a shorthand for "absence of reportable awareness" (using "reportable", of course, in a broad sense, not in a purely verbal sense). My usage of such other words as "conscious", "unconscious", and "experience" is based on a similar shorthand.

BLINDSIGHT

The basic phenomena of blindsight are well-known: patients who have large field defects as defined by conventional perimetry may nevertheless be capable of performing a number of visual and visuomotor tasks, including saccadic and manual localization, motion detection, and even colour discrimination [18, 79, 80]. By definition, these tasks are performed without the aid of any veridical visual experience of the stimuli concerned, and in general without any visual experience at all. The interpretation of blindsight, however,

has been a matter of controversy. I put forward here one particular interpretation, which has been documented more fully elsewhere [48]. It is argued that most if not all of the phenomena of blindsight can be understood within a novel functional interpretation of the two quasi-separate "cortical visual systems" that were first delineated by Ungerleider and Mishkin [76].

There are good anatomical, behavioural, and physiological reasons for believing that corticocortical projections from the primary visual cortex (V1) are segregated into two broad pathways or streams, one located dorsally and terminating in posterior parietal cortex, the other coursing ventrally and terminating in inferior temporal cortex [1, 19, 53, 76, 83]. It has recently been proposed that the patchwork of visual areas constituting the dorsal stream operates to provide a visual substructure for the guidance of a range of motor acts, and does so without *ipso facto* furnishing visual awareness of any aspect of the visual array. This proposal is consistent with a wide range of physiological, behavioural, and neuropsychological observations [28, 47]. Of course, it is not being denied that in general we are visually aware of an object when (for example) we are picking it up. What is proposed is that our visual awareness is being provided by *different* visual processes from those which guide the act itself. Our visual awareness, it is suggested, is generated separately, as a result of activity within the ventral stream.

On this hypothesis, to the extent that the dorsal stream can function in the absence of V1, it could provide the principal neurobiological basis for blindsight. And we know that many of the properties of single neurones in dorsal-stream areas such as MT (V5) and V3A do survive the destruction or inactivation of area V1 [25, 27, 72]. Therefore, there is a substantial *prima-facie* case for arguing that the dorsal stream, along with its associated subcortical structures in the midbrain and posterior thalamus, could mediate a wide range of visual abilities in the absence of area V1 [48].

In marked contrast, the visual responsivity of cells in the inferior temporal cortex is completely eliminated following lesions of the striate cortex [71]. Other ventral-stream areas too are silenced during inactivation of V1, including V4 [26]. Furthermore neurones in area STP, lying in the superior temporal sulcus adjacent to the inferior temporal cortex, lose their selectivity for form and motion in the hemifield corresponding to a unilateral V1 removal [12]. The evidence as a whole indicates that processing in the ventral stream is almost entirely dependent on visual input from the geniculostriate pathway, whereas processing in the dorsal stream depends substantially on inputs from the superior colliculus (probably via the pulvinar nuclei of the thalamus). In the theory of Goodale and Milner, the ventral stream of cortical areas is argued to provide the neural basis for visual perception and recognition. Therefore, these perceptual functions—whether performed consciously or unconsciously—should not be possible in patients with extensive damage to the striate cortex.

The proposal that such lesions prevent visual perception directly challenges descriptions of blindsight as a form of "unconscious perception". But a form of visual processing that it never—and can never be—associated with *recognition*, let alone with visual awareness, bears no useful resemblance to what is normally understood by "perception". Perception is part of a process that permits the economical storage of information about the relatively permanent world of three-dimensional objects, people, and places, with the primary purpose of achieving identification and understanding. Almost none (if any) of what the blindsight patient can do comes into this category. Rather than describing blindsight in terms of "unconscious perception", therefore, it is more appropriate to characterize it as a

set of visual skills. These skills allow the patient, for example, to reach toward objects in the "blind" visual field, to make saccadic and pursuit eye movements with respect to such stimuli, to locomote around obstacles, and even to orient the hand in anticipation of grasping an "unseen" object [43, 56]. The unconscious perception that is available to normal individuals under certain circumstances, for example outwith the focus of attention or under tachistoscopic and/or masked presentation conditions [45], is not akin to blindsight.

There is, however, one major exception to the rule that V1 destruction abolishes visual perception. It has long been known [67] that a moving stimulus can often be detected in the absence of shape or colour perception after occipital brain injury, and recent studies suggest that in some patients this appreciation of motion (which can include both speed and direction) is *conscious*. That is, in the present terminology, some blindsight patients possess "motion perception" [2] rather than merely "motion blindsight" [8, 7]. For example, Barbur *et al.*'s patient G.Y. has a rather complete lesion to the primary visual cortex of the left hemisphere, causing subjective blindness in the right hemifield, yet he is able to perceive the occurrence and direction of motion [2].

This report has important implications for an understanding of the neural mediation of visual awareness. There has been a general assumption until now that V1 is a *sine qua non* for visual awareness [e.g. 18, 84]. For example, Cowey and Stoerig [18] recently suggested that it might be through a reciprocal synchronous activation between V1 and extrastriate visual areas that the latter come to mediate conscious visual events. But G.Y. has a large occipital lesion, which appears to include a complete unilateral loss of V1, on the basis of both imaging evidence and the subjectively dense hemianopia for static stimuli that it causes. Accordingly, since this patient can consciously perceive movement, V1 cannot be a *necessary* component in the circuitry leading to visual awareness.

The report of Barbur *et al.* [2] also, at first sight, challenges the present suggestion that residual visual processing in the dorsal stream after V1 lesions would always be "unconscious". But their finding can be accommodated if one bears in mind that a clean lesion restricted to V1 will very rarely be encountered in the clinic. Neighbouring cortical areas will inevitably be damaged, with the pattern of damage varying from patient to patient. It must, of course, be assumed that the lesion which damages V1 spares the visual areas of the dorsal stream such as MT sufficiently to allow information about visual motion to be received at the cortex via tectopulvinar pathways. This could then provide the raw material for *either* motion perception *or* motion blindsight. But which of these two possibilities actually occurs may depend upon whether the occipital damage *also* spares ventral-stream areas such as the human homologue of V4. If V4 is spared *as well as* MT (as may be the case in G.Y.), visual information arriving in dorsal areas could be transmitted to the ventral stream for further processing, which in turn could yield conscious motion perception. There is good evidence for cross-connections between the two streams [see 24] which could mediate this.

The idea then, that blindsight depends on the activity of visuomotor circuits in the dorsal stream (along with linked subcortical "slave" systems), is not incompatible with the appreciation of motion being conscious in some blindsight patients. It should be noted here that patients who have undergone hemidecortication generally retain motion detection, but appear to lack an appreciation of either speed or direction [55, 64]. Since these patients have lost *all* cortical visual areas on one side of the brain, it would be predicted that their motion detection would also always be unconscious: in other words, in

the present terms, it would always be a form of blindsight rather than a form of visual perception. It is assumed, then, that the activation of circuits in the ventral steam is necessary for perception; but it is not being implied here that this is *sufficient* to yield a visual experience, as will be discussed further in a later section.

APPERCEPTIVE AGNOSIA

Damage to the striate cortex is not the only means by which temporal-lobe areas can become visually deafferented. Selective disconnections *within* the ventral steam, between the occipital and temporal lobes, can also occur.

Patients with visual object agnosia, by definition, have a fair degree of preserved visual perception: they show this by their ability to report colour and motion, to detect spots of light within the visual field, and to distinguish fine gratings. Yet in severe apperceptive agnosia, patients may be quite unable to identify or describe the simplest of shapes, and can only guess at the nature of an object by making inferences based on its colour, reflectance or visual texture. It has recently been proposed that such "visual form agnosia" [3, 50] results from a selective disconnection of ventral-stream circuits from their striate cortical input channels [28, 47]. While patients of this kind have often suffered diffuse brain damage, recent MRI studies indicate that the cortical damage tends to be heavily concentrated in lateral prestriate areas, with little detectable necrosis in the striate cortex itself [50, 74].

Patient D.F. [50], for example, although able to detect high-spatial-frequency gratings or name colours with ease, is unable to distinguish squares from rectangles with a 2:1 aspect ratio, or to report the orientation of a horizontal, oblique, or vertical slot correctly. Yet this failure of shape and orientation perception does not prevent her from adeptly reaching out and inserting her hand, or a card, into the same slot [50], or to form her finger–thumb posture perfectly in anticipation of grasping rectangular blocks of different width [29]. This dissociation exemplifies clearly the distinction drawn above between visual perception and the visual guidance of action. It is neatly consistent with the assumption that contour information is unable to access processing systems for shape perception in the temporal lobe of D.F., while remaining well able to access systems for controlling hand and finger movements in the parietal lobe.

It is argued that this profound disorder of shape recognition, with a concomitant loss of *awareness* of shape, results from an occipito-temporal disconnection. An analogous disconnection can be created artificially: the severe deficit in shape discrimination that follows bilateral inferior temporal ablations in monkeys [31] can be mimicked by combining a unilateral optic tract section with a contralateral inferior temporal lesion plus callosal section [21]. A similar result follows the use of a unilateral striate removal instead of optic tract section [51]. These experiments achieved a complete visual isolation of the remaining temporal lobe by directly removing ipsilateral geniculostriate inputs, and also indirectly removing contralateral ones (by sectioning the corpus callosum). But of course one can only guess as to the contents of the monkey's visual experience in these experiments.

One can also only surmise as to the range of visuomotor skills these cross-lesioned monkeys would have had. But we do know that, despite a profound disorder of shape recognition analogous to visual form agnosia, monkeys with bilateral inferior temporal lesions can readily catch flies [63], pursue and grasp moving food objects (C. R. Butler,

cited in [31]), and can orient the hand grip accurately to pick up elongated food items [13]. Indeed Klüver and Bucy [42] informally described several intact visuomotor skills in the presence of a profound "visual agnosia" in their classic studies of large bilateral temporal-lobe resections over 50 years ago. Almost certainly, these preserved skills are mediated by intact tissue in the parietal areas of the monkey's dorsal stream, where damage causes impairments in these same visuomotor abilities [13, 23, 33].

Unlike the human syndrome of visual form agnosia, these animal models involve a destruction or disconnection of the inferior temporal cortex as a whole, affecting not only shape discrimination but also other aspects of visual perception such as colour [37]. In relation to shape discrimination, however, inferior temporal lesions provide a good model of human apperceptive agnosia. It is therefore arguable that if a different kind of human pathology were to cause a *complete* occipito-temporal disconnection, the resulting apperceptive agnosia would be a rather comprehensive one, in which the patient might report no conscious visual perception at all. In other words, the patient's subjective state would be little different from that experienced by a cortically-blind patient. Of course, in such an imaginary patient the intact striate cortex might be able to furnish the visuomotor systems of the parietal lobe with rather more refined information than the tecto-pulvinar system could provide alone: in other words, the visual resolution available for the guidance of actions might be somewhat superior to that available in blindsight. In both cases, however, one would expect that visual awareness, at least of static items in the visual field, would be absent. That is, in the limit, apperceptive agnosia would become subjectively indistinguishable from cortical blindness.

ATTENTION, AWARENESS, AND THE VENTRAL STREAM

The hypothesis of Milner and Goodale [28, 47, 48] assumes that only visual processing in the ventral stream, with its assumed function of synthesizing percepts, could ever reach awareness. But presumably a good deal of processing proceeds to a high level without reaching awareness—for example during focal attention to another source of information. The first question, therefore, is what characteristics of visual coding in the ventral stream might make it qualitatively distinct from visual coding elsewhere, and second, what differentiates the particular ventral stream processes which do culminate in visual awareness from those which do not.

One possible answer to the first question would be that brain processes corresponding to selective attention might characterize the mode of operation of the ventral stream, but not the operation of dorsal stream areas. For example, the currently dominant circuits active in inferior temporal cortex might tend to be boosted at the expense of other parts of the system, and if the activation of such a selected subset of elements exceeded some threshold value relative to the rest of the system, this modulation might confer visual awareness. There is now good evidence for the operation of spatially-selective gating at the neuronal level in both areas V4 and inferior temporal cortex [16, 52] in the monkey. In human subjects similarly, spatially focused attention during a shape discrimination task is reflected in the activation of an area within the contralateral ventral stream (the fusiform gyrus) [36].

Unfortunately for this hypothesis, however, attentional gating at the neuronal level is certainly not restricted to areas in the ventral stream: indeed such phenomena were first described at the cortical level in areas within the *dorsal* stream [15]. In fact, it can be argued

[28, 47] that selective processing would be just as necessary in visual areas dedicated to the guidance of action as in those dedicated to the elaboration of visual perception. That is, there would be a need to intensify the processing of relevant parts of the array at the expense of irrelevant ones, whether the purpose is to select a particular item for action or to select it for perception. Human brain metabolic (PET) studies support this argument: the spatial allocation of visual attention activates different areas depending on the subject's task. Thus when the task requires the subject to discriminate visual symbols, ventromedial occipitotemporal regions are activated [36], but when s/he merely has to detect a visual symbol using a key-press response, superior parietal regions are activated [17].

Indeed it is arguable that even when striate cortex damage prevents conscious visual perception, a blindsight patient may still be able to deploy attentional processes within visuomotor control systems, for example when reaching for an object in the hemianopic field. Independent support for this idea is the finding that an irrelevant visual stimulus in the "blind" field can appreciably lengthen saccadic reaction times made to stimuli in the "good" hemifield [65]. This apparent diversion of visual attention into the hemianopic field—probably saccade-related—may be directly analogous to the selective spatial attention exercised by the frog in preparation for prey capture [38], and indeed both may be mediated by tectal mechanisms. In any event, if selective visual attention can be attributed to a cortically-blind patient, let alone to a frog, then it is clear that it neither implies awareness nor is it likely to be mediated solely by ventral-stream activity.

These considerations indicate that while selective attention might operate within the ventral stream to confer consciousness on an active processing network, there must be something else that prevents this happening in the dorsal stream. One distinction that might be crucially important is that while viewer-based coding is required for visuomotor control, visual coding for the purposes of perception must deliver the identity of the object independent of any particular viewpoint. This could be accomplished by constructing a network of multiple views: from this, object identity could be accessed by transforming or interpolating any particular view of an object with respect to that network (e.g. [14, 75]), or by exploiting broad tolerances in the selectivity of specific view representations [58]. Alternatively, a particular view of an object could be transformed to some sort of canonical or prototypical view (e.g. [54]). Whatever the particular coding mechanisms might be (and they could vary between different classes of objects), the essential problem for the *perceptual* system is to code (and later recover) object identity—thus approximating what Marr [44] called an "object-centred" description. It is objects, not object views, that the perceptual system has evolved to deliver. It might, then, be this "object-centred" aspect of ventral stream processing that uniquely permits its products to enter our experience.

Given this need for two qualitatively different kinds of visual coding in the brain, some means may have evolved for preventing the two from competing with each other in the control of behaviour. For example, storing the successive orientations of an object from moment to moment as separate visual memories would not generally be useful at a later time; yet this is precisely the kind of information needed for the on-line control of an action such as catching a frisbee. The temporally-varying and intrinsically viewer-centred coding needed for us to perform skills of this kind must have to be kept separate from the coding of object-based invariants needed for the purposes of recognition and planning. The association of consciousness with only one of these two different kinds of coding

might have been sufficiently useful during mammalian evolution to exert a powerful selective pressure in its favour. Furthermore it is possible that similar considerations may apply to consciousness in the context of perceptual systems other than vision. Indeed cognition in general may have consciousness as its hallmark precisely because it has to deal with enduring rather than transient aspects of the world.

In this discussion, I have simplified matters by arguing as if visual coding was always of intrinsically unchanging, inanimate, "objects". But of course much of our visual processing is concerned with less stable items—namely other organisms. Perhaps prey and predators could be responded to relatively stereotypically using the visual coding available in the dorsal stream and its associated subcortical networks. But neurones responding to "social" stimuli, such as the face or the actions of a conspecific, have been found not in those areas but instead in the inferior temporal cortex, in medial temporal structures like the amygdala, and most especially in area STP [59]. Although the movements of others are crucially important in understanding and predicting their future behaviour, it is often not their motion in relation to oneself that is important. Instead, systems for organizing emotional and social behaviour will often need visual information about conspecifics which is abstracted from its precise viewer-centred coordinates and coded in terms of its goal-directedness. Perrett and his colleagues, for example, have identified cells in area STP which appear to code not just the nature of a seen action but also the location of the goal of the action [60]. This "action-centred" coding may be regarded as a form of invariance akin to object-centred coding. Nevertheless, it cannot be denied that an egocentric view of what a conspecific is doing may also be important—whether or not the other's gaze is directed at oneself, for example. The ventral stream, therefore, if that can be assumed to be the source of visual information for social as well as cognitive purposes, will need to preserve egocentric information as well as abstracting invariants from it [60].

If, as proposed earlier, the perceptual system's activities only enter awareness in respect of objects on which attention is being focused, then the focusing of attention on some other perceptual system (e.g. the auditory perceptual system)—or on internal cognitive operations—may temporarily take awareness away from vision altogether. This could presumably cause the kind of conflict between the two forms of visual coding that I have proposed visual awareness would normally eliminate. In other words, a possible prediction from this theory is that visuomotor skills might be prone to interference from the visual perceptual system under extraneous distraction conditions. One way of testing this prediction might be to use a visual illusion such as the Titchener size illusion. Goodale *et al.* [30] have recently shown that although the visual perception of size is subject to this illusion, visually-guided grasping of the same objects is not. This observation nicely illustrates the need for a visuomotor system to over-ride the geometrical illusions to which our perceptual system can be prey, and shows that the two systems must use separately processed visual information. Therefore, if the grasping task could be shown to *become* vulnerable to the Titchener illusion under suitable auditory distraction conditions, it could be inferred that the diversion of attention caused a breakdown of this separateness. If such an improbable prediction could be confirmed, the present proposals would gain considerable support.

Of course any theory which proposes a set of necessary and sufficient conditions for a brain process to be conscious (say the conjoint conditions of object-centred coding and attention) is not the same as a theory of consciousness. It says nothing about any causal

links whereby the particular set of conditions *should* confer that particular mental quality. Nonetheless, a proposal of this kind probably comes as close as it is possible to come to such a theory of consciousness, within the confines of empirical science. This, in my view, makes such theories deserving of experimental investigation.

VISUAL SPACE, EXTINCTION AND NEGLECT

Brain damage can, of course, cause altered states of visual awareness without any absolute perceptual deficit of the kinds discussed so far. In visual extinction, the patient can report a left-side stimulus when presented alone, despite being unable to do so when it is presented with a similar right-side stimulus. And a patient with visuospatial neglect may be able to demonstrate high-level (semantic) knowledge about a picture presented on the left through a priming paradigm, despite being unable to report that information when requested to do so directly [4].

Several CT-scan studies have established beyond doubt that most patients with neglect have lesions which include the inferior parietal lobule and neighbouring temporal lobe tissue, usually in the right hemisphere [35, 77]. In contrast—although less extensive evidence is available—a recent CT-scan study demonstrates that visual extinction (in the absence of neglect) is *not* associated with such inferior parietal damage [78]. Conversely, extinction—but not visuospatial neglect [77]—can be caused by lesions of the *superior* parietal lobule [62]. Posner *et al.* [62] tested 13 patients with unilateral parietal lesions (some right, some left) on an extinction-like task, in which the subject had to respond to a lateralized visual stimulus, having been cued on the same or opposite side immediately previously. They found that an "invalid" visual cue in the good field caused catastrophically poor detection of a visual target flash immediately afterwards in the impaired field. The patients who showed the greatest deficit were those whose lesions encroached most into the superior parts of the parietal lobe. There was no relationship with the extent of damage in the inferior parietal lobule, and indeed little indication of spatial neglect was associated with the extinction seen in these patients.

The upshot of this evidence is (a) that visuospatial neglect and visual extinction are caused by damage to different functional systems; (b) that neglect is associated primarily with the inferior parietal lobule; and (c) that visual extinction is associated with a range of areas *excluding* the inferior parietal lobule (IPL) but *including* the superior parietal lobule (SPL). At the beginning of this century, Brodmann [10] proposed on cytoarchitectonic grounds that the human SPL corresponded to areas 5 and 7 in the monkey [9]: this is the region now identified as the endpoint of the monkey's dorsal visual stream. Brodmann's comparative anatomy fits well with the fact that the impairments of visually guided action of arm, hand and fingers that constitute optic ataxia are associated with damage to the SPL [39, 40, 57, 66], just as similar deficits are associated in the monkey with damage to areas 5 and 7 [22, 23, 33].

Taken together, these two sets of localization data suggest that visual extinction may be associated with unilateral damage to the endpoint of the human dorsal stream (among other sites, most of which are themselves connected with dorsal stream areas)—while visuospatial neglect is generally associated with damage to the right IPL, which lies in neither visual stream. In agreement with this formulation, visual extinction has been described in the monkey following damage to the posterior parietal cortex [e.g. 69] but deficits resembling human spatial neglect have not [46].

According to the "premotor" theory of visuospatial attention proposed by Rizzolatti and his colleagues [e.g. 70], attentional shifts are generated within systems concerned with the visual control of spatially-directed action. In support of this, a detection task requiring shifts of visuospatial attention has been found to cause increased metabolic activity in the SPL of healthy human subjects [17]. If the premotor theory is correct, visual extinction might be predicted to occur following the unilateral disruption of dorsal stream circuits. And it would account for the fact that extinction and visuomotor incoordination seem to be associated with damage to the same areas in both monkeys and humans. A discussion of how the known characteristics of extinction could be generated from such damage can be found in reference [68].

In contrast, neglect seems to affect complex spatial representations of visual scenes and patterns [e.g. 5], and therefore is not readily understood in terms of damage to visuomotor systems. For example, neglect may affect only one class of patterns, such as faces [82]; and it may be associated with the left side of objects (even when they are rotated) rather than just the left side of retinocentric visual space [20]. Neglect also affects imagined visual scenes [6], and causes spatial and perceptual distortions [34, 49]. In other words, although neglect is intrinsically egocentric (its leftwardness would have no meaning except with reference to an observer), nevertheless its coordinates seem not typically to be retinally-based but rather based on figural frames of reference. I would argue, therefore, that damage to the IPL causes neglect by disrupting a system for representing highly-processed figural information, a system therefore in large part dependent on visual inputs from the ventral stream.

It is worth nothing that only ventral-stream inputs would permit the IPL to code spatial relationships among objects, because such relational coding would be logically impossible without reference to object identities. In humans, it appears that the right hemisphere's ventral stream dominates visual perception and recognition, as least at a global perceptual level, since disorders such as topographic agnosia, and probably also prosopagnosia, are associated much more with right ventral occipito-temporal lesions than with left [32]. It should therefore not be surprising that a system for representating spatial relationships that depended for important visual inputs on the ventral stream would also be more heavily represented in the right hemisphere.

According to the hypothesis proposed here, then, the asymmetrical disruption of visual consciousness seen in spatial neglect may follow from damage to networks dependent on the ventral stream. But in the case of extinction, I have argued that *dorsal* stream structures (and/or related visuomotor systems) are damaged. Such damage should not directly impair the awareness of visual stimuli, according to the assumption made earlier in this paper that attentional gating operates within these systems without their products entering consciousness. Yet clearly unilateral damage to these systems does, in causing extinction, affect visual awareness. This raises the question as to how the physiological gating processes observed in the two streams (defined broadly to include related subcortical structures) inter-relate. The obvious answer is to propose that there must be a non-reciprocity between the two streams, such that a locus selected through the dorsal stream controls the gating processes seen in the ventral stream—but not vice-versa. Such an influence could be exerted through the well-documented pathways that exist between the two streams [24]. In other words, attentional gating may originate in posterior parietal cortex, but its effects then generalize to networks within the ventral stream, with consequent effects on visual awareness. If this is correct, then it could explain why damage

to the dorsal stream could cause failures of stimuli to reach awareness in tests of extinction.

This interpretation could be tested empirically, by examining whether the "attentional" phenomena seen in V4 and inferior temporal cortex [16, 52] survive lesions or inactivation of the posterior parietal cortex. A dorsal-stream domination of spatial attention (see also Perrett *et al.* [61]) would also predict that only those objects or loci selected for potential action—construed broadly, so as to include, for example, saccadic and pursuit eye movements—could be selected for special processing in the ventral stream. Of course, a parieto-temporal control function of this kind only has plausibility when focal attention is being considered. It is unclear how it could bring about a diffuse or divided attentional state, such as the de-focused visual awareness one may experience when regarding a landscape. This must remain one of the many problems that the present proposals do nothing to resolve.

CONCLUSIONS

I have sketched an approach to a neuropsychological understanding of visual awareness which is a long way from a detailed, let alone a definitive, account. It is, however, data-led, in the sense that it has emerged from a consideration of a wide range of physiological and neuropsychological findings. In fact, the ideas set out here have been reached in an attempt to make sense of this array of discoveries in the visual neurosciences, rather than in an attempt to "explain visual consciousness". The general approach may lend itself to attempts to understand awareness in other sensory modalities. In fact the present proposals already have implications beyond the purely visual, since the spatial representational network postulated to be located in the inferior parietal lobule is likely to be polysensory, given the different routes through which one can construct spatial reality. This putative network may have its evolutionary roots within the superior temporal sulcus of the monkey, which is known to receive polysensory inputs [11, 41]. Anatomical studies have shown that parts of this region receive visual inputs from the inferior temporal cortex [73], and physiological studies show that its neurones depend for their form and motion selectivity on the striate cortex, just like other ventral stream areas [12].

There is a curious corollary of the present suggestion that the potential of visual processing to achieve conscious status depends upon its ability to access object- (or action-, or relation-) based coding: In principle, it might be possible for brain damage to selectively impair access to such coding, while leaving intact some measure of view-specific coding, even within the ventral stream itself. For example, one could conceive that a proportion of the viewer-centred face cells described by Perrett *et al.* [58] in STP might still be activated by familiar still photographs of faces despite brain damage that prevented recognition of those faces and access to the identity of their owners. If so, there might be the potential for "unconscious" access to episodic "snapshot" representations of particular faces, yet without access to the identity of those faces via "head-centred" (let alone person-centred) representations. There is now well-established evidence for a measure of preserved "unconscious" recognition of famous people in some patients with prosopagnosia, when they are shown facial photographs of those people [e.g. 81]. For example, despite the absence of any conscious recognition, such patients may demonstrate the presence of some form of recognition of the photograph by means of electrodermal

responses or other indirect means. Could it be that it is the photograph that is "recognized", rather than the person depicted? If so, then unfamiliar photographs of such famous people should never elicit such "covert recognition".

Of course this idea could not explain instances of covert recognition where a patient has access to a person's occupation or other semantic associations, despite being at chance on forced-choice discriminations of the same stimuli [81]. Nonetheless, a careful analysis of familiar versus unfamiliar photographs of famous faces may be of general value in such research.

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