NOTE

The Motion Vision of the Blind

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INTRODUCTION

Can patients blinded by lesions in area V1, the primary visual receiving area in the cortex, see anything in their blind fields and, if so, what is the characteristic of their so-called residual vision and what does it tell us about the visual brain? In 1917, Riddoch (1917) recorded the largest number of such patients ever assembled and, in an observation since confirmed (Pöppel et al., 1973; Blythe et al., 1987; Mestre et al., 1992), reported that some are able to perceive motion in their otherwise blind fields. Riddoch did not have a well-formulated and plausible explanation for his observation, which was therefore dismissed (Holmes, 1918) and relegated to oblivion for over half a century (Zeki, 1991). But our current view of the visual brain as consisting of many visual areas (Zeki, 1978; Van Essen, 1985) of which are specialized for visual motion (Zeki, 1974; Van Essen, et al., 1981; Albright, 1984), allows us to account for this phenomenon in terms of the sparing of specific pathways leading to the specialized areas. And, given the multiplicity of visual areas, we can raise the question of whether other specialized pathways, leading to other visual areas, can be similarly spared and lead to a residual vision for other visual attributes. I thus define residual vision, in a broad sense, as the visual left after damage to area V1 and enquire into the insights that we may draw from studying one kind of residual vision, motion vision. Two characteristics of residual vision may be derived directly from Riddoch’s description: (1) an ability to detect the presence of motion without being able to characterize the other attributes of the stimulus or to see an object if it is stationary, implying that a functionally specialized pathway is spared; (2) the conscious awareness of having seen movement, implying that the activity of a specialized pathway, without the parallel activity of V1 or of other visual pathways, is sufficient to lead to conscious awareness. This second characteristic distinguishes residual vision from the controversial syndrome of blindsight (Campion et al., 1983; Weiskrantz, 1986; Celesia et al., 1991; Ruddock, 1991; Fendrich et al., 1992) when subjects are said to be able to discriminate visual stimuli which they are not consciously aware of having seen.

THE NEURAL PATHWAYS OF RESIDUAL MOTION VISION

To what neural mechanism can one attribute this capacity? It is unlikely to be due to spared tissue within V1, as Riddoch imagined, because the layers of V1 which contain the motion detecting (directionally selective) cells are located in layer 4B and layer 6 (Lund et al., 1975; Fries et al., 1985; Shipp and Zeki, 1989a). It is improbable that these layers are specifically spared by bullet wounds or vascular accidents that damage the rest of the visual cortex. A more plausible explanation lies in the cortex surrounding area V1. This cortex was for long considered to be a single visual “association” cortex and the repository of “higher” visual functions with the vague role of “interpreting” and “understanding” the visual image “received” by V1 (Zeki, 1993), implying a separation between the cortical areas involved in “seeing” and “understanding.” But this association cortex is in fact made up of multiple visual areas, the most interesting of which, in this context, is area V5, specialized for motion in both monkey and man (Zeki et al., 1991; Watson et al., 1993; Tootell et al., 1995). Input to it and to another prestriate area, V3, is dominated by a specialized group of retinal ganglion cells, well suited to register motion and which reach layer 4B of V1 via the lower two magnocellular (M) layers of the subcortical visual center, the lateral geniculate nucleus (LGN). V5 and V3 receive their input from layer 4B of V1 and from specific subcompartments of another area, surrounding V1, area V2 (DeYoe and Van Essen, 1985; Shipp and Zeki, 1989b) with both of which V5 and V3 are reciprocally connected (Zeki and Shipp, 1988; Shipp and Zeki, 1989a). If V1 is destroyed, all of these cortically relayed signals to V5 are lost. But V5 also receives
a less prominent and well studied visual input that by-passes V1 and reaches it directly from subcortical visual centers such as the LGN (Fries, 1981) and the pulvinar (Standage and Benevento, 1983). The latter nucleus is connected with a midbrain visual center, the superior colliculus, which in turn receives input from the retina. It was indeed the superior colliculus that was considered to be the center mediating residual visual capacities in monkeys after removal of V1 (Keating, 1980; Pasik and Pasik, 1982).

Because residual vision in man has a conscious dimension, it becomes intuitively more appealing to suppose that it is a cortical component, fed by the pathway reaching the prestripate cortex directly from the thalamus, that is able to mediate a crude, but conscious, perception of visual motion. Indeed, recordings from V5 in monkeys whose V1 had been inactivated (Rodman et al., 1989; Girard et al., 1992), and which consequently had no input into V5, show that the characteristic of the latter area, directional selectivity, is maintained, although the cells lose their crispness and sharpness of tuning, suggesting that V5 is not totally dependent upon V1 for its specialization. Even if activity restricted to V5 is not, in itself, a sufficient condition for conscious motion perception, can activity in a V5 disconnected from V1 contribute to a conscious visual experience without pre- or postprocessing by V1? Animal studies have concluded that it does not (Bugler et al., 1994). But conclusions about consciousness are not easy to reach from animal experiments and the issue is not trivial for, if activity in a V5 disconnected from V1 has a conscious dimension, the implication would be that individual specialized visual areas may be able to contribute directly and explicitly to conscious, if crude, vision, without the need to act in concert with V1, either through pre- or postprocessing by the latter. In brief, V1 which, explicitly in the early literature (see Zeki, 1993, for a review) and implicitly in the current theorizing about blindsight (Cowey and Stoerig, 1991), has been considered to be the only perceptually effective gateway into conscious vision, may not in fact be so. Moreover, given that there are other specialized visual areas, there may be many more or less separate consciousnesses for different attributes of the visual world, based on activity in separate visual areas.

THE CONSCIOUS DIMENSION OF RESIDUAL VISION

Fortunately, there is now a direct way of studying the problem because both V5 and V3 (Sereno et al., 1995; Shipp et al., 1995) have now been defined in the human brain; they, together with a region in the parietal lobe, are activated when humans view moving visual stimuli. This makes it possible to address, in the human brain, the question of whether activity can occur in these areas when V1 is destroyed and when all signals relayed to them from V1 are consequently lost. If so, one can address the further question of whether the activity in these areas, and especially in V5, correlates with a conscious visual perception. Our activation studies (Barbur et al., 1993), using the method of positron emission tomography (PET), showed that these same prestripate areas, but not V1, were active when patient GY (Weiskrantz, 1990), blinded by a lesion to V1 since childhood, reported verbally, with 100% accuracy, whether a stimulus presented to his blind field was moving to the left or the right. His vision was of course much impoverished; he could only detect high contrast, fast moving stimuli. But he was conscious of the direction of motion of the visual stimuli, which gave him a very elementary and crude, although nevertheless useful, knowledge about his world in motion. GY told me that, through this crude vision, he was able, on three occasions at least, to avoid colliding with an oncoming car. This suggests that the very crude but conscious perception of visual stimuli is possible without V1 and that activity in the specialized areas defined above is an essential part of the conscious process of perceiving visual motion. In this context, it is worth emphasizing that the point raised raised by Fendrich et al. (1992), that blindsight itself might be due to islands of spared tissue within a largely destroyed V1, does not apply to the residual visual motion of patient GY, since the relevant part of his V1 was completely destroyed and, correspondingly, did not "light up" in our PET experiments.

THE PARALLEL PATHWAYS TO V1 AND V5

What are the characteristics of the signals that reach V5 without passing through V1 and endow activity in it, without the participation of V1, with a conscious dimension? Patient GY, with a lesion in V1, can only detect high contrast, fast-moving (>10° s⁻¹) stimuli; in comparison, the studies that we and others have done with patient LM, with bilateral lesions in V5, have shown that she can only detect very slowly moving stimuli (<10° s⁻¹) (Hess et al., 1989; de Jong et al., 1994). It is safe to assume, therefore, that the direct pathway to V5 is better suited to signal fast motion. A second characteristic, derived from theoretical calculations and experimental observations, is that the direct pathway is able to deliver signals at latencies of about 30 ms. The technique of transcranial magnetic stimulation, through which one can inactivate human visual areas briefly and selectively (Beckers and Hümberg, 1992), shows that a stimulus moving at 22° s⁻¹ cannot be perceived if a magnetic pulse, lasting 800 μs, is used to inactivate V5 at any time during a period of 10 ms.
before to 20 ms after the 28-ms appearance of the visual stimulus, implying that signals from fast moving stimuli reach V5 at latencies of about 30 ms (Beckers and Zeki, 1995). By contrast, the milder motion imperception produced by inactivation of V1 occurs when the magnetic pulse is delivered at 60–70 ms after the appearance of the same visual stimulus. This not only shows that V5 is more critical in the perception of fast signals, already implied by the clinical evidence, but also leads to the surprising conclusion that signals from fast moving stimuli use a fast pathway to reach V5 before they reach V1 and that they reach human V5 at far higher speeds than had been supposed. The inference can be confirmed by direct measurements from the scalp of normal humans in whom V5 had previously been identified with PET, using the electroencephalography (EEG) technique supplemented by magnetoencephalography (MEG) (ffytche et al., 1995). This combined method, which gives latencies of 37 ms for the first arrival of signals, shows directly that signals from fast moving stimuli reach V5 first; by contrast, those from slow moving ones reach V1 first and then V5. It follows that activity in prestriate cortex occurs in parallel with or even precedes that in V1. Because the latter recordings are from normal humans, it follows further that the activation of the fast, direct pathway to V5 does not become operational only when a lesion compromises the more classic pathway; rather, the two parallel pathways can be simultaneously active or not, depending upon the stimulus. We therefore speak of a dynamic parallelism (ffytche et al., 1995).

These general conclusions about the timing of arrival of signals in visual cortex and the parallel input to V1 and V5 are confirmed by the results of direct recordings from the cortex itself, which show that signals can reach the visual cortex in 20–30 ms in monkey (Petersen et al., 1988; Maunsell and Gibson, 1992; Kawano et al., 1994) and 30 ms in man (Wilson et al., 1983; Ducati et al., 1988). Moreover, comparative monkey studies, whose implications in terms of parallel inputs to V1 and V5 are not commented on by their authors (Raiguel et al., 1989), show that the earliest signals are picked up from V5 not V1. Why, then, has the EEG method not detected early motion induced activity in prestriate cortex and supposed instead that visual cortex outside of V1 may not receive motion signals at all (Maier et al., 1987; Van Dijk et al., 1987) or that prestriate cortex is always activated after V1, in sequential manner, with peak deflections occurring at intervals of 160–220 ms after delivery of the motion stimulus (Drasdo et al., 1993; Frobst et al., 1993). The answer to the problem lies in the type of stimulus used. Almost all previous EEG studies have used very slow motion (<0° s⁻¹), the very component which passes first through V1, and have thus not detected the fast component (ffytche et al., 1995).

THE DETECTION AND MEASUREMENT OF EARLY COGNITIVE PROCESSES

Hence the initial cortical process associated with conscious vision, or the ones that trigger the processes leading to conscious vision, can be of very short latency and duration and thus beyond the temporal resolution of the most sophisticated imaging technique currently available, that of functional magnetic resonance imaging (fMRI). The latter has a theoretical temporal resolution of 500 ms (Cohen and Bookheimer, 1994), when the initial cortical processes that trigger conscious perception may well be over. In fact, the time relationship between cortical activity and conscious appreciation is far from clear (Glynn, 1990) and I do not mean to imply that the conscious processes involved in vision are limited to the first 100 ms or less after delivery of the visual stimulus. But to measure the early triggering process of conscious vision a much better time resolution is needed, possibly that provided by a technique such as MEG in association with PET or fMRI.

THE DIRECT CONTRIBUTION OF INDIVIDUAL AREAS TO CONSCIOUS VISION

The above evidence suggests that, provided a visual input is relayed to them, individual visual areas may contribute directly and explicitly to conscious vision according to their capacities, without the necessity for pre- or postprocessing by V1. It also shows that there can be a conscious dimension to the activity of the specialized visual areas and that that dimension does not necessarily become available only after all the processing in all the visual areas is over. Finally, it shows that there is no sharp distinction between seeing and understanding, since activity in these areas leads to both simultaneously. Does this mean that the appropriate activity in any visual area, without parallel activity in V1, can lead directly to a conscious visual experience, however impoverished and, by extension, that activity in V1 alone, without the prestriate cortex, can lead to crude but conscious visual awareness?

It is possible that activity in other specialized visual areas disconnected from V1 can also have a conscious dimension. There is at least one report of a V1-blinded patient who was nevertheless able to discriminate consciously the colors of spectral lights (Blyth et al., 1987), implicating area V4 which, in monkey at least, has a direct subcortical input by-passing V1, much like V5 (Fries, 1981; Standage and Benevento, 1983). There is another instance of a surprisingly sophisticated residual vision mediated by another area acting without a V1. This relates to the ability of a V1-blind patient to perceive consciously optic flow (Ceccaldi et al., 1992), an activity that, in the human, depends upon motion-related cortex outside of V5 (de Jong et al., 1994). Presumably activity in these areas depends upon whether the lesion, which usually invades white matter, has
spared the direct input to them. The question is more
difficult to answer with V1. Experimental evidence
suggests that massive, but not total, removal of pre-
striate cortex does not lead to blindness in monkeys
(Ungerleider and Mishkin, 1982), whereas human evi-
dence suggests that prestriate cortex may be neces-
sary for even rudimentary vision (Bodis Wollner et al.,
1977). On the other hand, subjects rendered achro-
matoptic following a lesion in V4 (Kennard et al.,
1995), or monkeys with experimental lesions in V4
(Walsh et al., 1993), have difficulties with color con-
stancy tasks, although they are able to discriminate
wavelengths surprisingly well (Fries and Zeki, unpub-
lished results; Heywood et al., 1987; Vaina, 1994), the
latter almost certainly an activity of V1 (Zeki, 1983),
but possibly of V2 as well. As well, although agnostic
patients with large prestriate lesions may be unable to
recognize an entire pattern, they may nevertheless rec-
ognize the line segments that constitute the pattern
(Humphreys and Riddoch, 1987), raising the possibility
that it is activity in V1 and possibly V2 that is con-
tributing directly to the limited visual capacities that
they have. In general, the functions of V1 can be sum-
marized in anatomical and physiological terms as that of
conducting a piccemeal analysis of the visual field (Hu-
bel and Wiesel, 1977) and of parcelling signals related
to different attributes to the different areas of the pre-
striate cortex (Zeki, 1975). The residual visual capaci-
ties of achromatopic and agnostic patients raise the
question of whether, in performing these functions, V1
has some form of rudimentary perceptual output, in-
dependent of the prestriate areas and independent of
the return input from the latter areas to V1 (Zeki and
Shipp, 1988).

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