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THE MOTION VISION OF THE BLIND AND THE MODULARITY OF CONSCIOUSNESS

BY PROFESSOR S. ZEKI*

Humans blinded by lesions of the primary visual receiving cortex, area V1, can sometimes see certain attributes of the visible world, and in particular motion, crudely but consciously. This article examines why and enquires into the insights to be drawn from such a study.

Introduction
Over a century of intensive work has established beyond doubt the pre-eminent role of the primary visual cortex, area V1, in vision. It receives a massive input from the retina through the lateral geniculate nucleus and lesions in it lead to blindness, total if the damage is total and partial if the damage is incomplete. It also is a critical distribution centre, parcelling out visual signals in parallel to the different areas within the visual association cortex, which are therefore deprived of much of their input when V1 is damaged.1 But work on the visual brain in the past 25 years has led to another and no less interesting insight—that the so-called visual “association” cortex consists of many visual areas, specialized for different attributes of vision.2 In addition to receiving a visual input from V1, these areas also receive another visual input that by-passes V1, either directly from the lateral geniculate nucleus (L.G.N.)4 or from the pulvinar nucleus5 or from both. This anatomy raises an interesting physiological question—to what extent can the visual areas of the “association” cortex act independently of V1? In practice, this amounts to asking whether patients who are blinded by lesions in area V1 can see anything in their blind fields and whether this vision is the result of the direct input to the visual areas of the prestitiate cortex. Allied to this is the no less interesting question whether the vision conferred on patients by an anatomical input that bypasses V1 has a conscious correlate. It has been generally considered that conscious vision is not possible without V16 and that such patients can only discriminate correctly what they neither see nor have any conscious experience or awareness of. But is this so?

In 1917, Riddoch* recorded the largest number of such patients ever assembled and, in an observation since confirmed10,11,12 he reported that some are able to perceive motion in their otherwise blind fields. No one has commented upon one word—conscious—that Riddoch

*Professor Zeki was educated at U.C.L., where he received a B.Sc. in Anatomy and a Ph.D. (1967). After periods at St Elizabeth’s Hospital, Washington, D.C. and the University of Wisconsin, he returned to U.C.L. in 1969 as a Lecturer in Anatomy. From 1975 to 1980 he was the Henry Head Research Fellow at The Royal Society and was appointed to his current position as Professor of Neurobiology at U.C.L. in 1981. He has held Visiting Professorships in this country and abroad. He is a former Chairman of the Wellcome Trust Vision Panel and is a member of the Board of Scientific Governors of the Scripps Research Institute in California. Professor Zeki has received numerous international prizes and his publications include A Vision of the Brain (1993) and many articles, in professional journals, on vision and the brain. In 1990, he was elected a Fellow of The Royal Society.
used at least four times in his paper, almost certainly without realizing its significance. He is not to be blamed for this; I have read and quoted Riddoch but as late as this decade\(^1\) did not realize the significance of the word, or the condition that it described, either. Riddoch did not have a well formulated and plausible explanation for his observation; he tried to account for it in terms of spared tissue within V1, an explanation that seems more implausible today that it must have seemed in 1917. His view, that motion can be specifically spared after lesions of area V1 which are sufficiently to render subjects blind or at least scotomatosus, was therefore dismissed\(^4\) and relegated to oblivion for over half a century.\(^9\) Holmes\(^4\) considered that Riddoch must have been “certainly incorrect...since in all my cases the blindness was total”. This is surprising because in that very same paper Holmes gives an account of a patient blinded by a lesion in V1. This patient was “...generally conscious only of the movement of the white test object”. Holmes chose not to allude to this patient in concluding that “occipital lesions do not produce any dissociation of function with intact retinal sensibility”.

Our current view of the visual brain as consisting of many visual areas\(^5,14\) some of which are specialized for perceiving motion\(^9,16,17,18\) 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, 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 vision 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 motion 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\(^9,16,20,21,22\) syndrome of blindsight\(^23\) when subjects are able to discriminate visual stimuli which they are not consciously aware of having seen. It is noteworthy that the blindsight literature has described how subjects who may not see what they can correctly discriminate can nevertheless feel something that is triggered by the visual stimulus.\(^25,26\) But in terms of conscious experience and awareness, that literature makes an unsatisfactory distinction between seeing and feeling. I consider both to be conscious experiences triggered by a visual stimulus, however crude and degraded the latter may be with regard to the former. The statement of one blindsight subject, D. B. is compelling enough in this regard.\(^23\) His verbal commentary to his discrimination of a moving vertical line was, “I did not see the line...but I could feel the movement, and I was absolutely sure of it” (original emphasis). That certainty implies a conscious dimension and when a subject like D. B. reports “having a ‘feeling of something moving’ but asserts firmly that he does not ‘see’ anything”,\(^24\) one is hard put to understand why the former (feeling) is not also a conscious experience since certainty is indicative of a conscious dimension. However, a feeling is not what Riddoch or Holmes were describing. They were instead talking of conscious sight and my examination of patients blinded by lesions in V1 has convinced me that patients blinded by lesions in V1 can see sometimes actually see rather than just feel and that the same patient may see a stimulus on some occasions and feel it on others. In summary, activity in a neural system that is deprived of much of its normal structure can and does have conscious experience as correlate, whether subjects see or merely feel something that is triggered by the appearance of a visual stimulus in their blind field.

**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,\(^9\) because the layers of V1 which contain the motion-detecting (directionally selective) cells are located in layer 4B and layer 6.\(^22,26,27\) 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, implying a separation between the cortical areas involved in “seeing” and “understanding”. It is in fact made up of multiple visual areas. The most interesting of these in this context is area V5, which is specialized for motion in both monkey and man. The input to it and to another prestripate area, V3, is dominated by a specialized group of retinopelvic ganglion cells, well suited to register motion, and which reach layer 4B of V1 via the lower two magnocellular (M) layers of the sub-cortical visual centre, the lateral geniculate nucleus (L.G.N.). V5 and V3 receive their input from layer 4B of V1 and from specific sub-compartments of another area, surrounding V1, area V2, with both of which V5 and V3 are reciprocally connected. If V1 is destroyed, all these cortically relayed signals to V5 are lost but V5 also receives a less well studied visual input that by-passes V1 and reaches it directly from subcortical visual centres such as the L.G.N. and the pulvinar. The latter nucleus is connected with a mid brain visual centre, the superior colliculus, which in turn receives input from the retina. It was indeed the superior colliculus that was considered to be the centre mediating residual visual capacities in monkeys after removal of V1.

Residual vision in man has a conscious dimension and it is therefore interesting 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 and which consequentially had no input into V5, show that the characteristic of the latter area, directional selectivity, is maintained although the cells lose their 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 perception of motion can activity in a V5 disconnected from V1 lead to a conscious visual experience? Animal studies have concluded that it does not, but conclusions about consciousness are not easy to reach from animal experiments and the subject 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 post-processing by the latter. In brief, V1 which, explicitly in the early literature and implicitly in the current theorizing about blindsight, 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. In brief, visual consciousness itself may be modular, reflecting the processing and perceptual modularity of the visual brain.

The conscious dimension of residual vision
Fortunately, there is now a direct way of studying the problem because both V5 and V3 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 consider, in the human brain, 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 further consider whether the activity in these areas, and especially in V5, correlates with a conscious visual perception. Our activation studies, using the method of positron emission tomography (PET) showed that these same prestripate areas, but not V1, were active when patient G.Y., 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. This conscious dimension of G.Y.’s vision has now been confirmed by the studies of Weiskrantz and his colleagues, who have also
shown convincingly that G.Y. is capable of discriminating to a very high degree of accuracy, without conscious awareness, if the visual stimuli are appropriately scaled, that G.Y., in other words, can have both conscious and unconscious vision. It will be especially interesting to learn whether there are particular stimuli for demonstrating blindsight which would suggest that blindsight must be distinguished sharply from the Riddoch phenomenon, or whether, with the same stimulation conditions, conscious awareness itself waxes and wanes, resulting in blindsight in some test sessions and in conscious awareness of the same stimuli in others. The former would suggest that a highly specialized visual pathway, distinct from that capable of mediating a conscious correlate, is used. The latter would suggest that blindsight and conscious residual vision for motion are two aspects of the Riddoch phenomenon and would raise the interesting question whether the brain switches from one neural system to another during the waxing and waning of consciousness—a rich if difficult question for imaging studies. Riddoch’s subject’s vision was of course much impoverished; he could detect only high contrast, fast moving stimuli but he was also conscious of the direction of motion of the visual stimuli, which gave him a very elementary and crude, though nevertheless useful, knowledge about his world in motion. I asked G.Y. on two separate occasions, separated by about two years, to describe to me what he saw. In a description similar to that given by Riddoch, who described what his patients had seen as “vague and shadowy”, G.Y. told me on the first occasion that his vision is similar to that of a normal person who shuts his eyes, looks out of the window, moves his hand in front and sees the shadows. On the second occasion, he told me it was a “feeling” of having seen something but when high contrast, rapidly displaced stimuli were used he was quite sure that something had occurred in his blind field, whether he saw it or felt it. It had, in other words, a conscious dimension. His equivocation may reflect a genuine linguistic difficulty or it may represent a fluctuating capacity of the direct input to the prefrontal cortex, a notion that merits further consideration. The conscious dimension of vision in his blind field is not only confirmed by the previous descriptions of Riddoch and of Holmes, but also by the more recent description given of a patient blinded by a lesion in V1 (patient M.M.) who has a conscious experience of optical flow stimuli. 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. I have recently had occasion to examine patient (M.M.) with my French colleagues and can confirm their findings and add to them the results of our more recent enquiry, that his conscious vision is either specific or most easily manifested for a specific kind of motion, optical flow. M.M. does not see the coherently moving stimuli that G.Y. can experience nor can he report anything when his blind field is stimulated with biological motion, possible reflecting the fact that visual cortex related to motion shows sub specializations, in both monkey and man. If so, this would be a strong argument in favour of the notion that I am presenting here, that activity in separate systems, even ones that are subspecialized for a particular subattribute (for example optical flow in the motion system) can have a conscious correlate, thus leading to the view that consciousness itself is modular.

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 G.Y., with a lesion in V1, can detect only high contrast, fast-moving (>10°sec⁻¹) stimuli; in contrast, patient L.M. with bilateral lesions in V5, can detect only very slowly moving stimuli (<10° sec⁻¹). 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 30ms. The technique of transcranial magnetic stimulation, through which one can inactivate human visual areas briefly and selectively, shows that a stimulus moving at 22° sec⁻¹ cannot be
perceived if a magnetic pulse, lasting 800μs., is used to inactivate V5 at any time during a period of 10ms. before to 20ms. after the 28ms. appearance of the visual stimulus, implying that signals from fast moving stimuli reach V5 at latencies of about 30ms. By contrast, the milder motion imperception produced by inactivation of V1 occurs when the magnetic pulse is delivered at 60–70ms. 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, it 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 P.E.T., using the electroencephalography (E.E.G.) technique supplemented by magnetoencephalography (M.E.G.).

This combined method, which gives latencies of 37ms. 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 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.

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 20–30ms. in monkey and 30ms. in man. Moreover, comparative monkey studies show that the earliest signals are picked up from V5, not V1. Why, then, has the E.E.G. method not detected early motion induced activity in prestriate cortex and supposed instead that visual cortex outside V1 may not receive motion signals at all or that prestriate cortex is always activated after V1, in sequential manner, with peak deflections occurring at intervals of 160–220ms. after delivery of the motion stimulus. The answer to the problem lies in the type of stimulus used. Almost all previous E.E.G. studies have used very slow motion (<6° sec⁻¹) and have thus not detected the fast component.

The direct contribution of individual areas to conscious vision

The above evidence suggests that, provided that 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 post-processings 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 a crude but conscious visual awareness?

It is possible that activity in other specialized visual areas disconnected from V1 can also have a distinct conscious dimension, reflecting the specialization of the area and the pathway that is spared by the lesion. This in itself suggests that consciousness, too, is modular and not exclusively dependent either upon a single cortical area or areas, or upon the healthy functioning of the entire system. There is at least one report of a V1-blinded patient who was nevertheless able to discriminate consciously the colours of spectral lights, implicating area V4 which, in monkey at least, has a direct subcortical input by-passing V1, much like V5. There is another instance of a surprisingly elaborate 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, an activity that, in the human, depends upon motion-related cortex outside of V5. Presumably activity in these areas depends upon whether or not 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 prestripate cortex does not lead to blindness in monkeys whereas human evidence suggests that prestripate cortex may be necessary for even rudimentary vision. On the other hand, subjects rendered achromatopsic following a lesion in V4 or monkeys with experimental lesions in V4 have difficulties with colour constancy tasks though they are able to discriminate wavelengths surprisingly well, also Fries and Zeki (unpublished results). The latter is almost certainly an activity of V1, but possibly of V2 as well. As well, though agnosic patients with large prestripate lesions may be unable to recognize an entire pattern, they may nevertheless recognize the line segments that constitute the pattern, raising the possibility that it is activity in V1 and possibly V2 that is contributing directly to the limited visual capacities that they have. In general, the functions of V1 can be summarized in anatomical and physiological terms as that of conducting a piece-meal analysis of the visual field, of parcelling signals related to different attributes to the different areas of the prestripate cortex. The residual visual capacities of achromatopsic and agnosic patients raise the question of whether, in performing these functions, V1 has some for of rudimentary perceptual output, independent of the prestripate areas and independent of the return input from the latter areas to V1.

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