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The Riddoch syndrome: insights into the neurobiology of conscious vision*

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Summary

We have studied a patient, G.Y., who was rendered hemianopic following a lesion affecting the primary visual cortex (area V1), sustained 31 years ago, with the hope of characterizing his ability to discriminate visual stimuli presented in his blind field, both psychophysically and in terms of the brain activity revealed by imaging methods. Our results show that (i) there is a correlation between G.Y.'s capacity to discriminate stimuli presented in his blind field and his conscious awareness of the same stimuli and (ii) that G.Y.'s performance on some tasks is characterized by a marked variability, both in terms of his awareness for a given level of discrimination and in his discrimination for a given level of awareness. The observations on G.Y., and a comparison of his capacities with those of normal subjects, leads us to propose a simple model of the relationship between visual discrimination and awareness. This supposes that the two independent capacities are very tightly coupled in normal subjects (gnosopsia) and that the effect of a V1 lesion is to uncouple them, but only slightly. This uncoupling leads to two symmetrical departures, on the one hand to gnosanopsia (awareness without discrimination) and on the other to agnosopsia (discrimination without awareness). Our functional MRI studies show that V5 is always active when moving stimuli, whether slow or fast, are presented to his blind field and that the activity in V5 co-varies with less intense activity in other cortical areas. The difference in cerebral activity between gnosopsia and agnosopsia is that, in the latter, the activity in V5 is less intense and lower statistical thresholds are required to demonstrate it. Direct comparison of the brain activity during individual 'aware' and 'unaware' trials, corrected for the confounding effects of motion, has also allowed us, for the first time, to titrate conscious awareness against brain activity and show that there is a straightforward relationship between awareness and activity, both in individual cortical areas, in this case area V5, and in the reticular activating system. The imaging evidence, together with the variability in his levels of awareness and discrimination, manifested in his capacity to discriminate consciously on some occasions and unconsciously on others, leads us to conclude that agnosopsia, gnosopsia and gnosanopsia are all manifestations of a single condition which we call the Riddoch syndrome, in deference to the British neurologist who, in 1917, first characterized the major aspect of this disability. We discuss the significance of these results in relation to historical views about the organization of the visual brain.

Keywords: agnosopsia; gnosanopsia; V5; conscious vision; functional MRI

Abbreviations: BOLD = blood oxygenation level dependent (contrast); fMRI = functional MRI; SPM = statistical parametric mapping

Introduction

In 1917, George Riddoch published a remarkable paper. He had been a temporary officer in the Royal Army Medical Corps and had had the occasion to examine soldiers who had fallen victim to enemy fire and been consequently blinded by gunshot wounds affecting the calcarine cortex (area V1). Of the 10 patients he described, the most interesting for this

*This article is dedicated to Keith Ruddock,who was tragically killed in a car accident on December 19, 1996. He brought many unusual and interesting visual syndromes of cerebral origin to the attention of the neurological world, and his many studies include that of patient G.Y., first described by him and his colleagues.

study are the first five, none of whom had become unconscious after being struck and all of whom, save one, had soldiered on in the immediate aftermath of the injury. All had been wounded overseas and had been subsequently repatriated to England where they were examined between 3 and 5 months after injury at the Empire Hospital for Officers in London. Riddoch's perimetric studies showed that all were able to detect the presence of motion within their scotomatous fields, without being able to characterize the other attributes of the stimulus. For Patient 1, 'The 'moving things' have no distinct shape, and the nearest approach to colour that can be attributed to them is a shadowy grey'. For Patient 2, 'The 'moving something' had neither form nor colour. It gave him the impression of a shadow'. Patient 3 could detect the movement of feet in the street '. . . though they had no shape'; Patient 4 '. . . declared he could distinguish no object . . . but he knew that something had moved through his blind field', while Patient 5 said, 'They [the moving objects] don't appear to have any colour or shape. They look like shadows. Sometimes I can tell if the moving things are white'. Thus one feature of the syndrome of residual motion vision as described by Riddoch is the crude ability to detect motion within the hemianopic field, without being able to assign any other attribute to the moving object or stimulus.

A phenomenon never remarked on, though one which is of cardinal importance, is that Riddoch's patients were conscious of having seen movement in their blind fields. Riddoch himself did not emphasize this point explicitly but made repeated references to it in his paper, almost certainly without realizing its true significance. Thus he writes of 'The frequency with which patients with restricted visual fields from occipital wounds . . . were immediately *conscious* of 'something' moving when the object was oscillated', of Patient 1 in whom '. . . the *consciousness* of 'something moving' kept up a continual desire to turn the head' and of Patient 4 who '*knew* that something had moved through his blind field' (our emphases). But he also writes that conscious awareness was restricted to movement: all were '. . . quite sure that neither shape nor colour can be attributed to [the movement]' but also, and significantly, 'The patients have great difficulty in *describing* the nature of the movement that they see: it is so vague and shadowy' (our emphasis). Thus another feature of the Riddoch syndrome is the ability to perceive motion in the blind field crudely but consciously.

Riddoch did not have a plausible explanation for his phenomenon (see Zeki, 1991) and his observations were therefore dismissed by Holmes in 1918. This is surprising because, in the very same paper, Holmes discusses a patient (his case 11) who was ' . . . in general only *conscious* of the movement of the white test stimulus' (our emphasis). In fact, the Riddoch phenomenon has been confirmed more recently by Mestre *et al.* (1992), Ceccaldi *et al.* (1992) and by our studies (Barbur *et al.*, 1993) on patient G.Y., who is the subject of this study. But this confirmation does not render Riddoch's implicit explanation for his phenomenon, that it is due to spared tissue within V1, any more plausible (see also Fendrich *et al.*, 1992; Kentridge *et al.*, 1997). We now know that, when the activity in the brain of at least some patients suffering from such a syndrome is imaged, it occurs outside V1, without any trace of active tissue within it (Barbur *et al.*, 1993). Nor can the syndrome be explained by the sparing of specific layers within V1, because the cells that are critical for motion vision are located in clusters within layer 4B and upper layer 6 (Lund and Boothe, 1975; Shipp and Zeki, 1989) it would seem unlikely that such clusters within individual layers would be selectively spared by gunshot wounds that destroy everything else within the area. The explanation that we have given to account for Riddoch's observations is therefore related to activity outside V1 and, more specifically, within prestriate cortex (Barbur *et al.*, 1993; ffytche *et al.*, 1996). In the work reported here, which is an extension of our previous studies, both on G.Y. and on the direct input to V5, we explore the characteristics of motion vision without involvement of area V1.

G.Y. has been reported to have good conscious vision when moving stimuli of the appropriate characteristics are used (Barbur *et al.*, 1993; Weiskrantz *et al.*, 1995), in other words he exhibits the Riddoch phenomenon. But using other stimuli, he has also been shown to be able to discriminate with high accuracy in the absence of all acknowledged awareness (Weiskrantz *et al.*, 1995). He therefore provided us with an ideal opportunity of learning about the neural bases of conscious versus unconscious vision, and thus of distinguishing neurologically between the Riddoch syndrome and the condition known as 'blindsight'. The latter capacity has been considered to be due to the functioning of a separate system (Weiskrantz, 1995; Weiskrantz *et al.*, 1995), even though the precise neural pathways involved have never been ascertained, the capacity being attributed at times to sub-cortical stations such as the superior colliculus (e.g. see Weiskrantz *et al.*, 1974; Keating, 1980; Pasik and Pasik, 1982) and at others to the activity produced by a direct input to V5, thought not to reach consciousness (Rodman *et al.*, 1989; Bullier *et al.*, 1994; Stoerig, 1996). One way of deciding the issue of whether conscious and unconscious vision use separate neural systems was to study the activity in the brain of G.Y. when his blind hemifield was stimulated in ways which would result in conscious vision on the one hand and unconscious vision on the other. In this work, we have restricted ourselves to patient G.Y., partly because he has been so extensively studied by others (e.g. Barbur *et al.*, 1980; Blythe *et al.*, 1987; Weiskrantz *et al.*, 1995), and partly because we ourselves have studied him in sufficient detail to know that the pathway to the prestriate cortex is intact in him, and produces activity that is detectable and measurable by both imaging and evoked potential methods (Barbur *et al.*, 1993; ffytche *et al.*, 1996). The final question that we have addressed, though

with some diffidence, is whether we can titrate levels of conscious awareness against cerebral activity. That we managed to do so and thus gain some insight into the relationship between brain activity and conscious awareness, however little, was both surprising and gratifying.

A brief account of these results has already been published (Zeki and ffytche, 1997).

Material and methods

The visual characteristics of G.Y., as well as the pathology of his brain, have been described in detail (Barbur *et al.*, 1980; Blythe *et al.*, 1987; Weiskrantz, 1990; Barbur *et al.*, 1993). His hemianopia with macular sparing is the consequence of a lesion in the left occipital lobe that spared the pole; it was sustained during a car accident at the age of 8 years. We first examined him at the age of 36 years (Barbur *et al.*, 1993) and have continued studying him since; the results reported here are the most recent ones and were collected over five sessions when he was 38–39 years old, each session on a separate day. We started our investigations with a series of psychophysical tests, derived from the studies of Weiskrantz *et al*. (1995) and considered as definitive tests to demonstrate blindsight (Cowey, 1996). The main difference between our tests and those of Weiskrantz *et al.* (1995) is that we used a TV monitor rather than a laser beam projected onto a screen. In view of our results, we do not think that this difference influenced the outcome of our study. We also collected data from normal subjects, to provide a baseline against which we could compare G.Y.'s performance. Informed written consent was obtained from all subjects and the study was approved by the joint National Hospital for Neurology and Neurosurgery and Institute of Neurology Ethics Committee.

Psychophysical testing

Stimuli were presented on a 17-inch computer monitor (frame rate 66 Hz; pixel resolution 640 × 480) driven by a Macintosh 7500/100 computer. After a period of adaptation lasting ~5 min, G.Y. was asked to fixate on a small black square while resting his chin on a support 30 cm from the screen and given a few practice trials. Eye movements were monitored by a video camera and trials in which fixation was not maintained were removed from further analysis. The screen luminance to the left, and at $<3.5°$ to the right, of the fixation square was held constant at a level that, as shown by Weiskrantz *et al*. (1995), masked any light scattered from the blind, right hemifield (95 cd/m^2 for two-direction and 172 cd/m^2 for four-direction experiments). His task was to discriminate the direction of motion of targets presented to his blind hemifield and to report whether or not he was aware of anything. Catch trials, when no target appeared or in which the target was stationary, were included in some four-direction experiments. We investigated the effect of target direction along the vertical and horizontal axes, and of size, velocity, trajectory, spectral content (by making the targets on the screen red instead of white) and contrast on G.Y.'s awareness and discrimination scores. The range of stimulus parameters employed are detailed in Table 1. The targets were white or red circles with an approximately Gaussian luminance distribution, as in the experiments of Weiskrantz *et al.* (1995). The luminance at the target centre was fixed at 150 cd/m^2 (43 cd/m^2 for the red target), decreasing to the level of background luminance at its outer edge. Target contrast was varied by adjusting the luminance of the background (i.e. the part of the computer monitor extending from 3.5° to the right of fixation). The mid-point of all 20° target trajectories was 15° to the right and 10° above the fixation point. The same coordinate was used as the starting point of the 10° trajectories.

Experiments were performed in blocks of 50 trials. In the two-direction experiments, the target size, trajectory, velocity, colour and background luminance were held constant in any given block. In the four-direction experiments, backgrounds of differing luminance were randomly interleaved. In some blocks G.Y. initiated each trial himself while in others he was warned verbally when it was about to begin. The target appeared after a random delay of 1–2 s from the onset of the trial and was followed by a prompt to indicate that the trial was over. The total trial-time varied for different velocities (the slower moving targets require more time to traverse 20° of visual angle than fast moving ones) but it was constant for any given block (~4 s for a 15°/s target).

Normal control subjects

Eight control subjects (mean age 31 years), with normal corrected vision, were tested with a two-direction discrimination task. By reducing the luminance contrast between target and background (see Table 1) we were able to manipulate the level of awareness from 0 to 100%, thus matching the range found in G.Y. This enabled us to compare the overall performance of G.Y. with that of normal subjects, in tasks matched for level of awareness. The target size, location and trajectory were identical to those used in the experiments with G.Y., as was the left hemifield luminance mask. Contrast was varied by presenting different luminance targets (41.1–44.1 cd/m^2) against a constant luminance background (37.5 cd/m^2). For five subjects, the different contrasts were randomly interleaved, while for the remaining three, each contrast was presented in a separate block.

The commentaries in relation to conscious experience

G.Y. and normal subjects responded in one of two ways, in separate blocks of trials; they either indicated their choice verbally to the experimenter, who then entered their choice into the computer, or they entered their choice into the computer directly themselves, via a customized keypad. All

Table 1 *Stimulus parameters*

Directions	Target	Radius	Trajectory	Velocity (°/s)	Background (cd/m^2)	No. of blocks	Session
Up and right	White	41′	20°	16	4	5	2 and 5
					15	3	2 and 5
					72	3	2 and 5
	White	22′	20°	16	4	5	4 and 5
					24	9	4 and 5
					95	5	4 and 5
	White	22′	20°	2	4	3	4 and 5
				7.5	4	1	4
				20	4	1	4
Up and down	White	22′	20°	15	24	2	4
Up, down, right and left (blank and static)	White	42′	10°	16	1	2	1 and 5
					42	2	1 and 5
					143	2	1 and 5
	White	3°	10°	16	1	1	1
					42	1	1
					143	1	1
	White	6.5°	10°	16	1	1	1
					42	1	1
					143	1	1
Up, down, right and left	White	22′	20°	2	4	2	4
Up and right	Red	22′	20°	16	4	1	3
					15	1	3
					72	1	3
Normal subjects							
Up and right	White	22′	20°	15	37.5 (background)		
					44.1	1	
					42.9	1	
					41.1	1	

subjects were required to make two separate responses; one was to identify the direction of motion, e.g. up or right, while the other was to indicate awareness. The latter was done by pressing (or naming) one of four keys: 1 indicated that they were unaware of anything occurring in their stimulated field (blind field in the case of G.Y.) while 2–4 indicated increasing levels of awareness (see Table 2).

Psychophysical model

In order to establish whether G.Y.'s psychophysical performance deviated from that expected by chance, we first had to generate a model of the relationship between awareness and discrimination. We argued that perfect observers would (i) discriminate the direction of motion correctly when aware and (ii) discriminate at chance when unaware. The theoretical relationship can be summarized as follows:

$$D = A + \frac{U}{Directions}$$

$$D_{low\alpha} = A + Ulow_{\alpha}$$

$$D_{high\alpha} = A + Uhigh_{\alpha}$$

where D is the total number of correct motion discriminations in an experiment with A 'aware' trials and U 'unaware' trials, assuming the unaware trials are guessed perfectly at chance ($U/2$ in the two-direction experiments and $U/4$ in the four-direction experiments). $Ulow_{\alpha}$ and $Uhigh_{\alpha}$ are the minimum and maximum number of correct responses that one might expect to get from U unaware trials at a particular statistical threshold α. These terms are calculated from the binomial distribution of U trials with a 1 in 2 probability of being correct by chance for the two-direction experiments and a 1 in 4 probability of being correct by chance in the four-direction experiments. When they are added to the number of aware trials A, they provide an estimate of the minimum ($D_{low\alpha}$) and maximum ($D_{high\alpha}$) number of correct discriminations expected at threshold α.

Imaging studies

The stimuli were generated on an Amiga computer and projected along the bore of the scanner to a vertically oriented, translucent screen. G.Y. viewed the screen via a front silvered mirror angled at 45°. The screen and mirror were mounted in a blackened box, resulting in a total screen–eye distance of 25 cm, and a stimulus that subtended 25° × 22° of visual angle. Fixation was not monitored during the scans; this was not deemed necessary as G.Y. is such an experienced subject. In fact, the absence of any activity within his normal striate cortex showed that his eye movements had not been of sufficient magnitude to stimulate his good hemifield.

Table 2 *Awareness responses*

Response	Details	Score
Unaware	There was no feeling of something being there. A total guess.	1
Aware	There was a feeling that something was there and guessed the direction.	2
	Fairly confident of the direction.	3
	Certain of the direction.	4

Similarly, the absence of a signal from the striate cortex which corresponds to the non-stimulated hemifield in the normal control subject showed that our precautions had been adequate. As in the psychophysical tests described above, G.Y. fixated on a small square and was asked to report the direction of motion in his blind field, in an area that extended from 3.5° to 15° in his right hemifield and 11° above and below the horizontal meridian. The region to the left of the stimulated area was masked to reduce the effects of scattered light, as in the psychophysical experiments. We initially used single spots moving either vertically or horizontally but the results were unsatisfactory. In further imaging experiments, we therefore used a medium contrast (<54%) random checkerboard composed of 27′ checks moving in one of four directions at 4°/s in the slow motion condition or 20°/s in the fast motion condition, a stimulus that we had found to be effective in our previous evoked potential studies (ffytche *et al.*, 1996). The checkerboard was restricted to the blind hemifield (see above). Each trial lasted 7 s, and G.Y. was required to give his awareness (button press) and direction (joystick) response in the 1-s inter-scan interval (each scan lasted 6 s). Fast motion and slow motion trials were presented in blocks of five trials, each lasting 35 s. Every third block consisted of a grey screen of the same mean luminance as the checkerboard. Ten blocks of each of the three conditions were presented in a single experiment.

Image acquisition

Functional images sensitive to blood oxygenation level dependent (BOLD) contrast were acquired on a Siemens 2-T Vision Scanner with a head radio-frequency resonator, using a gradient echo planar imaging sequence (TR = 7.01 s TE = 40 ms). The scanner was triggered by the Amiga to acquire one volume for each trial. Each volume consisted of 64 transverse slices (3 × 3 × 3 mm voxels in a 64 × 64 × 64 matrix) and 150 such volumes were acquired in a single experiment. T_1-weighted structural images were obtained in the same session. Analysis was performed using the statistical parametric mapping (SPM) software, modified for functional MRI (fMRI) and developed in our Department (Friston *et al.*, 1995*b*). After realigning each volume to remove motion artefact, images were smoothed with a 4 mm FWHM (full width at half maximum) Gaussian filter. Changes in BOLD contrast produced by different experimental conditions were assessed at each voxel using the general linear model and theory of Gaussian fields (Friston *et al.*, 1995*a*), modelling the haemodynamic response function as a 6 s delayed box-car and assessing significance with a cluster-level analysis (Friston *et al.*, 1996). Low frequency variations in the BOLD signal were included in the model as co-variates of no interest. The T_1 structural image was co-registered with the mean realigned functional image to help identify the exact anatomical location of significant activations.

Statistical analysis

Our studies allowed us to analyse our results in four different ways. Essentially our design was aimed at learning whether any brain areas were activated with fast and with slow motion compared with a control (grey) and whether any areas were better activated by fast than by slow motion, or vice versa. These four comparisons were effected by making fast motion, slow motion and grey stimulus conditions the co-variates of interest and by setting up appropriate contrasts (i.e. fast motion versus grey, slow motion versus grey, fast motion versus slow and slow motion versus fast) in the design matrix. In another analysis, we wanted to remove the confounding effects of motion and learn whether activity in any brain area correlated with conscious awareness alone; this was done by making the aware and unaware trials the co-variates of interest and including fast motion, slow motion and grey stimulus conditions as co-variates of no interest. In a related analysis, we used a factorial design consisting of four conditions (aware fast motion and slow motion versus unaware fast motion and slow motion) to isolate the effects of awareness for fast and slow motion. The grey control was included in the design as a co-variate of no interest. Finally, we wanted to identify all the areas in which activity co-varied with V5; in this analysis the co-variate of interest was the BOLD signal in V5 derived from the fast versus slow analysis described above.

Results

G.Y.'s description of his visual experience

Over the years, G.Y. has given us a varied account of what he experiences when his blind field is stimulated. When we first asked him the question in 1993, he told us, just as he had told Barbur *et al.* (1980) previously, that his experience resembles that of a normal person when, with the eyes shut, he looks out of the window and moves his hand in front of his eyes. It was, he said, like a 'shadow', a term reminiscent of Riddoch's description of what his subjects saw as 'dark and shadowy' (Riddoch, 1917). When we asked him again in 1994, his account had changed slightly. He now said that he has a 'feeling' of something happening in his blind field and, given the right conditions, that he is absolutely sure of the occurrence. When we pointed out the discrepancy between this and his earlier statement, he replied that he had, on the previous occasion, been using language that he thought a

normally sighted person would understand. Asked again in 1996, he described his experience as that of 'a black shadow moving on a black background', adding that 'shadow is the nearest I can get to putting it into words so that people can understand'. He also volunteered the information that he was much better at seeing vertical and horizontal movements than oblique movements. Because of a chance remark he made at the end of the present set of experiments, we wondered whether the four levels of awareness that we used were sufficient to characterize his levels of consciousness. When we presented him with a low contrast stimulus, he spontaneously remarked that the awareness score here should be 'minus one or minus two', implying that there might be, for him, degrees of unawareness. In spite of the variability in his verbal description of his experience, we were left with little doubt that he was able to experience consciously stimuli of certain characteristics when they were presented to his blind field, regardless of whether he described his experience as seeing or merely feeling the stimulus.

Psychophysical model derived from normal subjects

We begin by describing the performance of normal subjects on the same tasks as those performed by G.Y., though with stimuli of lower contrast, to compensate for the better vision of normal subjects (see Material and methods section). The aim was to obtain a standard against which G.Y.'s performance could be compared. The overall performance of normal subjects in discriminating the two directions of motion of a spot subtending 22′ and presented at varying contrasts is shown in Fig. 1, where each point represents a block of 50 trials. For Fig. 1A and C, subjects were judged to be aware if they pressed keys 2–4 and the distribution of the awareness levels for all subjects and for all blocks of trials was as follows: level 1 = 42 ± 16% of trials; level 2 = 11 ± 6%; level 3 = 8 ± 4%; level 4 = 39 ± 17%. However, the graphs of Fig. 1A and C do not distinguish between different levels of awareness, since the distinction being made is solely between aware (2–4) and unaware (1) trials.

Figure 1A describes the discrimination performance of subjects during trials when they reported themselves to be aware (levels 2–4) of the presence of the moving stimulus. It shows that they score correctly when aware and that their performance resembles very closely that of a theoretical perfect observer (continuous black line). Figure 1B shows the discrimination performance of the same subjects when they report themselves to be unaware (level 1) of the presence of the stimulus; the solid line predicts the performance that would fall exactly at chance level (1 in 2) while the outer dotted lines on either side give the maximum and minimum scores predicted by chance at a threshold of $P < 0.01$ and the inner dotted lines give the range of scores at a threshold of $P < 0.05$. Pooled together (Fig. 1C), the results show what one might expect, that in normal subjects there is a high correlation between awareness and discrimination (correlation coefficient 0.96; $P < 0.01$) and that almost all the points fall within the limits of the model (see Material and methods section). The graph of Fig. 1C thus provides us with a 'baseline' against which the capacities of G.Y. can be compared.

The performance of G.Y.

The overall performance of G.Y. in all two-direction experiments is shown in the graph of Fig. 2A, which is identically prepared to that of Fig. 1C. Once again, scores 2–4 have been pooled together as aware responses, since the distinction is between aware and unaware responses (the distribution for G.Y. in the equivalent set of experiments as that of normal subjects was as follows: level 1 = 76% of trials; level 2 = 16%; level 3 = 8%; thus, the only difference between G.Y. and the control subjects is that G.Y. never responded with a 4). The graph shows the relationship between awareness and discrimination in G.Y.; it has a correlation coefficient of 0.58 ($P < 0.01$) which suggests that the association between discrimination and awareness is still present in G.Y., though weaker than that found in normal subjects. As with normal subjects, most of the points (70%) fall within the boundaries described by the model (cf. Figs 2A and 1C). The remaining 30% that fall outside the boundaries can be divided into two groups that we interpret below to represent two different states of G.Y.'s visual system. To the left are three points (10%) where G.Y. scored less than would be expected from his level of awareness; these three points are derived from three separate blocks of trials at two different contrasts (41′ target moving at 16 °/s; against a background of 15 cd/m^2 and 4 cd/m^2), all performed on the same day (session 2). To the right are seven points (20%), derived from different tasks (22′ target moving at speeds of 2–16°/s against backgrounds of 4 and 24 cd/m^2, with directions upwards/rightwards and upwards/downwards) done on the same and different days, corresponding to session 4 (six blocks) and session 5 (one block); here G.Y. scored better than might be expected from his level of awareness. One feature of these latter points, six out of seven of which were collected on the same day, is that they are all clustered around 0% awareness.

The cumulative representation of the scores for all tests on the same graph, as in Fig. 2A, obscures some features of G.Y.'s performance. Chief among these is the variability in both his level of awareness and his discriminatory performance for the same task. This variability is shown in Fig. 3A–F. The scores for eight repeats of an upward and rightward discrimination of a 22′ spot moving against a 24 cd/m^2 background at 16°/s, on the same day and on different days (sessions 4 and 5), are shown separately in Fig. 3A; except for the use of the TV monitor and of a white spot (instead of a red laser), this task is identical to the one described by Weiskrantz *et al.* (1995) and Cowey (1996). While G.Y.'s discrimination was constant between 70 and 80%, his awareness level varied between 0 and 80%, making it impossible to relate his performance on this task to a given level of awareness. These results are shown

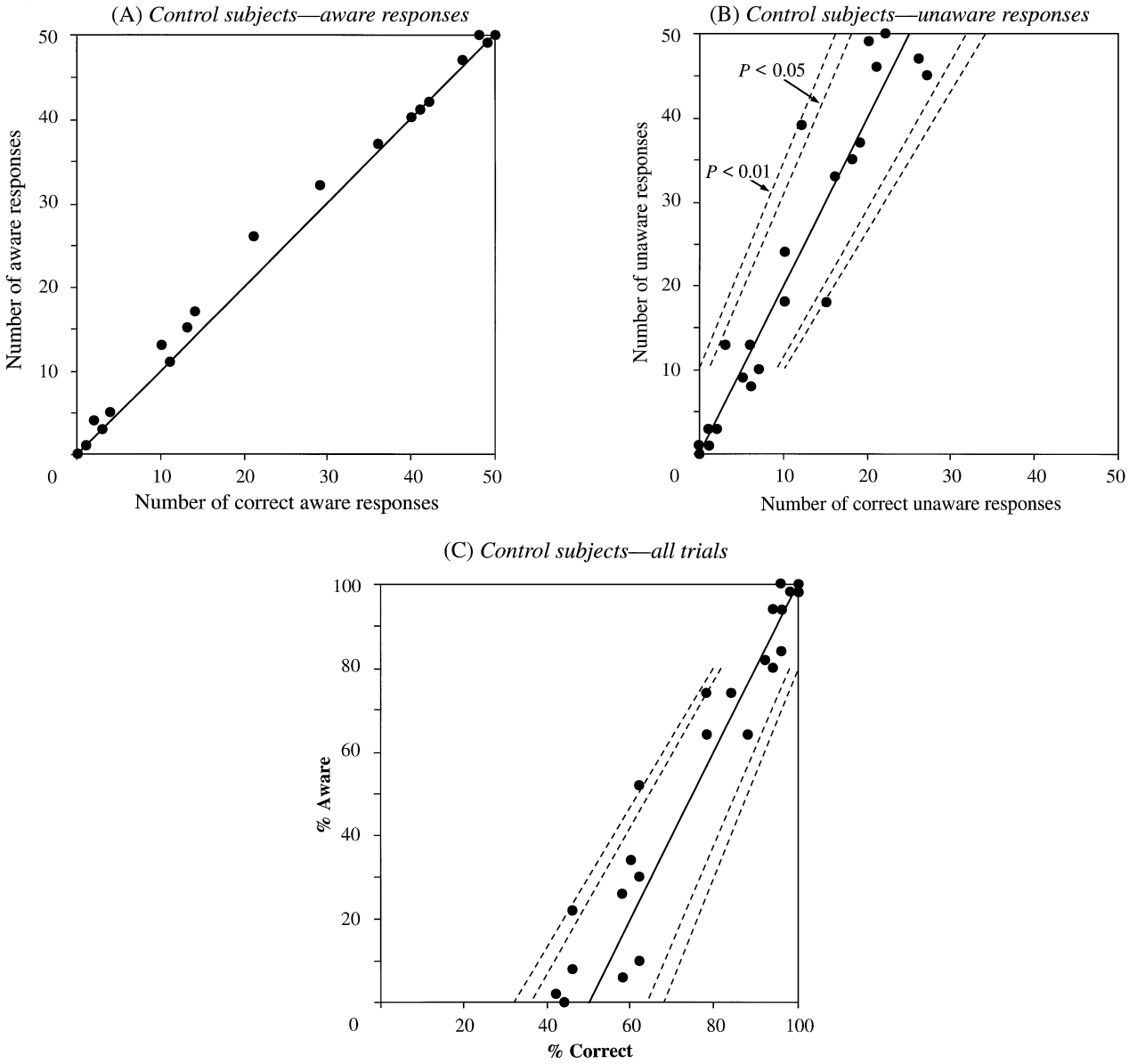

Fig. 1 The performance of eight control subjects on two-direction motion-discrimination tasks at three contrasts. Each point represents a block of 50 trials, three points being contributed by each subject (where two points are the same only one is plotted). (**A**) The total number of aware responses in a given block is plotted against the number of correct, aware motion discrimination responses. For example, the point in the top right-hand corner of the graph shows a block of trials in which the subject reported being aware on 50 trials, each of which was accompanied by a correct direction discrimination. Awareness responses of 2, 3 and 4 have been pooled together to produce a single estimate of awareness for each block (see Table 2). The black line shows the hypothetical performance of perfect observers that discriminate the direction of motion correctly each time they are aware. (**B**) The total number of unaware responses in a given block is plotted against the number of correct, unaware motion discrimination responses. The continuous black line shows a discriminatory performance exactly at chance (1 in 2) while the inner and outer dotted lines show the limits of chance performance under the binomial distribution at $P < 0.05$ and $P < 0.01$, respectively. The binomial distribution has not been calculated for small numbers of unaware trials (<10). (**C**) A combination of the data shown in **A** and **B**. The percentage of aware responses in each block is plotted against the percentage of correct motion discriminations, regardless of whether they were aware or unaware. The thick and dotted lines represent the psychophysical model (see Material and methods section). The thick line is the sum of (i) the number of aware trials in each block and (ii) the score expected by chance for the remaining unaware trials, the dotted lines represent the boundaries of the model under the binomial distribution at $P < 0.05$ and $P < 0.01$, respectively. The limits of the model have not been calculated for small numbers of unaware trials (<10).

in the context of his more general psychometric function, derived from three different contrasts, in the graph of Fig. 3D; the variability in his level of awareness is obvious. It is because of this variability that some of the scores fall well within the theoretical envelope predicted by our model and represent points where discrimination correlates with awareness; others

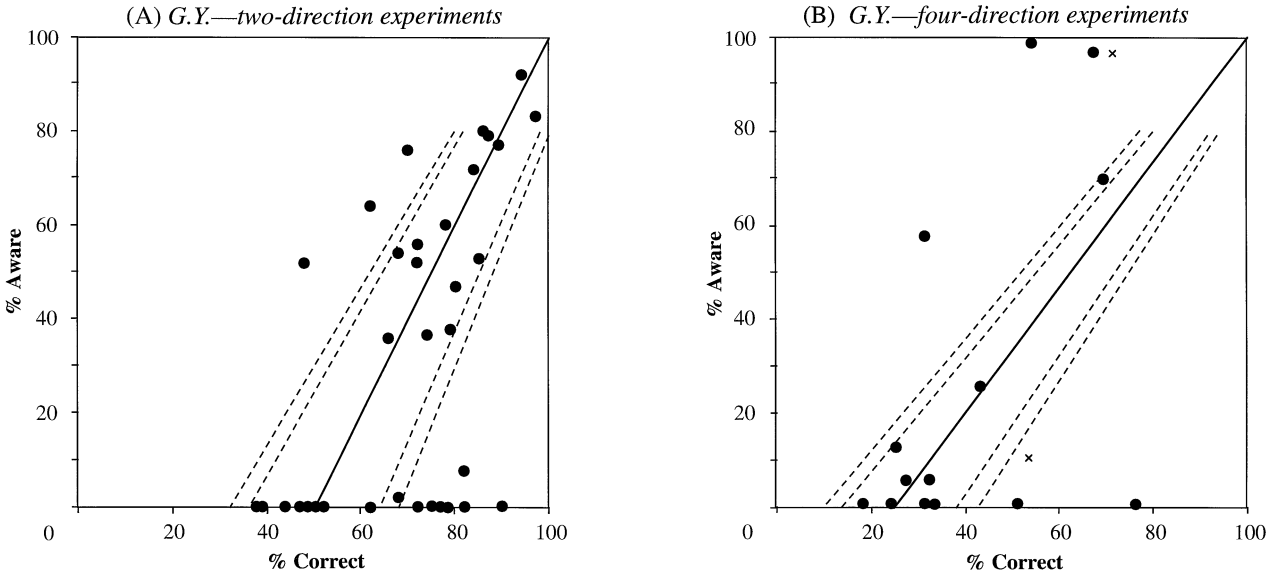

Fig. 2 (**A**) G.Y.'s performance on two-direction motion-discrimination tasks. Conventions as in Fig. 1C. (**B**) G.Y.'s performance on four-direction motion-discrimination tasks. The model parameters have been adjusted to take into account the change in discriminatory performance expected by chance (1 in 4). The crosses show G.Y.'s psychophysical performance during fMRI experiments.

fall outside, and represent points when he was able to discriminate correctly while being unaware. Figure 3B shows that, just as awareness during a given task can vary for a given level of discrimination, so discrimination can vary for a given level of awareness, though with a smaller variability. This graph shows his performance on four separate repeats of the same task—discrimination of a slow moving (three repeats at 2°/s and one at 7.5°/s), 22′ stimulus at a fixed high contrast (background: 4 cd/m^2)—in session 4 (three blocks) and 5 (one block). Here his level of awareness remained constant (at 0%) while his performance varied, from 50 to 70% correct; the consequence is that only one of the points (the 70% point) represents his ability to discriminate in the absence of awareness; all the remaining points fall within the envelope and thus do not differ from the scores expected by chance. Figure 3C shows a variability in both discrimination and awareness, the task being similar to that described for Fig. 3A, although the spot is larger (41′ instead of 22′) and the contrast higher (15 cd/m^2 background instead of 24 cd/m^2). In this graph, two of the points relating awareness to discrimination fall outside the theoretical envelope, showing that G.Y.'s discrimination was worse than might be expected from his level of awareness.

Similar results were obtained when G.Y. was tested with four directions instead of two (Fig. 2B), when the correlation between discrimination and awareness was 0.57 ($P < 0.05$). This was similar to his performance with two directions, showing again an association between awareness and discrimination in G.Y. Once again, some points (18%) appear to the left, outside the bounded area, while some (25%) appear to the right. The former represent a worse discriminatory performance than would be expected from his degree of awareness while the latter represent a better one. Catch trials were introduced into the four-direction experiments to find out how often G.Y. would indicate that he was aware when no target had appeared; his rate of false positive responses was $<0.01\%$.

In summary, taking a range of different discrimination tasks related to motion and spread over several sessions, G.Y.'s overall capacity to discriminate correlates with his awareness and is largely predictable by the model. But the results also show a variability in G.Y.'s performance, with the consequence that, using the same tasks, he is able to discriminate better than would be predicted by his level of awareness on some occasions and worse on others.

We noticed an interesting feature in the errors made by G.Y. when he was given a four-direction task, as shown in Fig. 4. Chance performance dictates that incorrect responses should be distributed evenly between the three incorrect options, giving each a 33% proportion of the total errors. But our analysis of G.Y.'s incorrect responses ($n = 513$) showed that, regardless of whether his response was accompanied by an awareness or not, the errors were not distributed evenly amongst the three options. He chose a response that was +90° to the true direction of motion more often than one might expect by chance (38%; $P < 0.01$) and chose a response exactly opposite to the true direction of motion less often than one might expect by chance (26%; $P < 0.001$). Thus, if the direction of the target were upward (towards 12 o'clock), his incorrect responses were unlikely to be 6 o'clock and tended to be 3 o'clock. The significance of this finding is taken up in the Discussion.

Variables that might affect the performance of G.Y.

Delay

We wanted to learn whether the introduction of a delay between the disappearance of the stimulus and G.Y.'s response

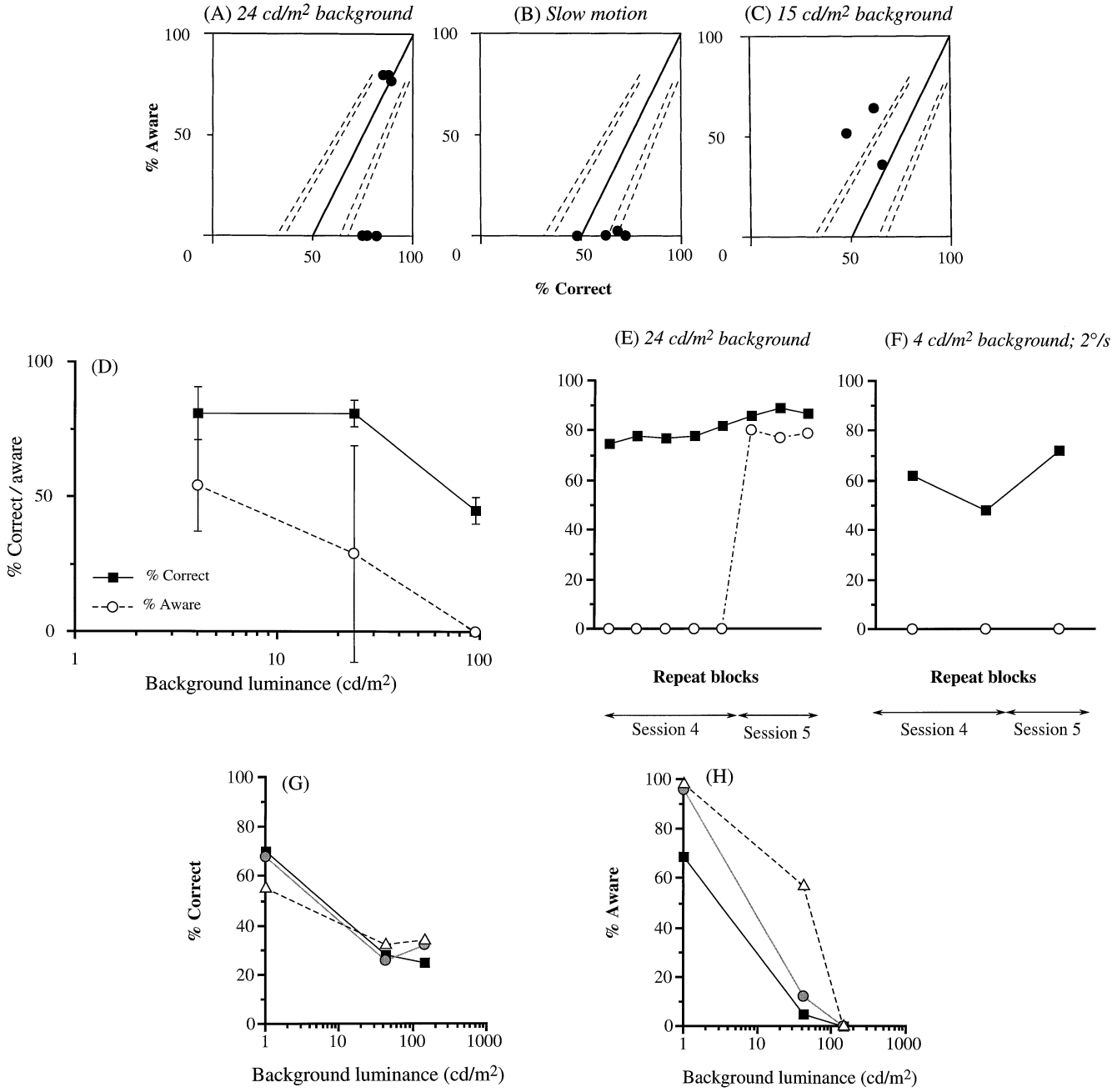

Fig. 3 The variability of G.Y.'s psychophysical performance. (**A**) Variability in awareness. Task: 22′ target, upwards/rightwards motion, 16°/s, with a 24 cd/m^2 background. Each cluster of blocks represents G.Y.'s performance on a single day (sessions 4 and 5). (**B**) Variability in discrimination. Task: 22′ target, upwards/rightwards motion, 2 and 7.5°/s, with a 4 cd/m^2 background (sessions 4 and 5). (**C**) Variability in awareness and discrimination. Task: 42′ target, upwards/rightwards motion, 16°/s, with a 15 cd/m^2 background. The conventions in **A**–**C** follow those of Fig. 1C. (**D**) Psychometric function. Task: 22′ target, upwards/rightwards motion, 16°/s, with a 4, 24, 95 cd/m^2 background. The mean awareness (open circles) and discrimination (closed squares) scores for repeated blocks of the same task are plotted against the background luminance. The error bars represent the standard deviation of the scores (n = 5 for 4 and 95 cd/m^2 and n = 9 for 24 cd/m^2). (**E**) Effect of time. Task as in **A**. The awareness and discrimination scores for each of eight repeated blocks of trials are plotted in sequence along the abscissa. Conventions as in **D**. (**F**) Task: 22′ target, upwards/rightwards motion, 2°/s, with a 4 cd/m^2 background. (**G**) Effect of target size on discrimination. Task: targets of 42′ (black squares), 3° (grey circles) and 6.5° (white triangles), upwards/downwards/rightwards/leftwards motion, 16°/s, and backgrounds of 1, 42 or 143 cd/m^2. (**H**) Effect of target size on awareness. Task as in **G**.

would influence his performance. Overall, this seemed not to be an important variable in our tests. Figure 5A shows the results of two such experiments. In the first, G.Y. was required to discriminate a spot of 41′, moving at a speed of 16°/s against a background of 4 cd/m^2; two repeated blocks of trials with a mean response delay of 1.3 s are shown as

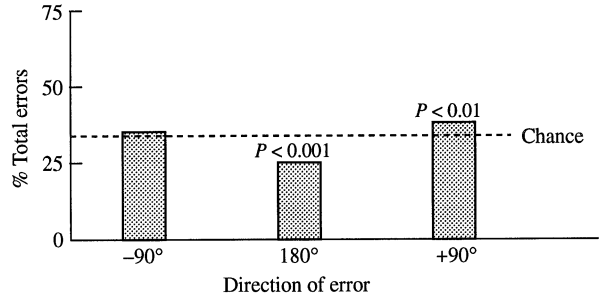

Fig. 4 The distribution of errors in the four-direction experiments. Both aware and unaware trials have been included ($n = 513$). Errors are divided into those occurring at –90°, 180° and +90° to the true direction of motion. The dotted line shows the number of errors expected by chance (one in three).

crosses while two repeated blocks with a mean delay of 4.1 s are shown as open circles. The introduction of a delay obviously made no difference to G.Y.'s discrimination or awareness scores. For the second experiment, which required G.Y. to discriminate a spot of 22′ moving at a speed of 16°/s against a background of 24 cd/m^2, his awareness but not his discrimination seemed to be affected by the introduction of a delay (mean delay: 1.5 s, filled circles; 4.2 s, star), changing from 0 to 38% during the two blocks of 50 trials recorded on the same day (session 4). There seemed little point, given these results, in exploring the influence of delay on discriminatory ability further.

Verbal versus non-verbal responses

We thought it interesting to compare his performance when his responses were communicated to us verbally (the simplest indicator of conscious awareness) and when he used the keypad to log them directly, especially since this is reported to have an effect on correct reaching responses to oriented lines in the absence of an awareness of their orientation (Goodale and Milner, 1992). The results obtained from four repeats of three different tasks (spot of 22′, moving at 16°/s against backgrounds of 4, 24 and 95 cd/m^2, all collected on the same day (session 4), are shown in the graph of Fig. 5B. Each cluster of crosses and circles is the result of a single task (bottom left: 95 cd/m^2; bottom right: 24 cd/m^2; top: 4 cd/m^2), two of the repeats utilized a verbal response (shown as crosses) and two the keypad (circles). The graph shows that his performance was unaffected by the mode of response.

Variations in other properties of the stimulus

The target size for the same task (four directions, 16°/s motion presented against backgrounds of 1, 42 and 143 cd/m^2) was varied, using sizes of 42′, 3° and 6.5°. The size did influence the pattern of results in that G.Y. was more likely to discriminate less well than one might expect from his level of awareness, as the size increased (see Fig. 3G–H; target size influences G.Y.'s awareness but not his discrimination). We also used a red spot in session 3, although, because of the phosphor limits of the computer monitor, the target was of a much lower luminance than the white one. G.Y. was not aware of the stimulus at any contrast and his discrimination was at chance levels, a change in performance that might be due to the spectral composition of the target or its absolute luminance. Finally, in session 3 we used a red laser spot that was projected onto a large screen; however the speed of the laser spot was too high and we have therefore not included these results here.

Effects of time and repeated testing

We considered whether repeated exposure to the same tests, administered by us and others, might have altered G.Y.'s sensitivity and improved both his discrimination and awareness of stimuli presented in his blind field. The graphs of Fig. 3E and F show that this is not so. Figure 3E shows that his awareness for the 22′ target moving at 16°/s presented against a background of 24 cd/m^2 improved between sessions 4 and 5; however, his awareness for another task (22′ spot moving at 2°/s against a background of 4 cd/m^2) presented in the same sessions remained constant. This, together with the points we take up in the Discussion, leads us to believe that the variability is not the result of repeated testing.

Capacities for discriminating vertical and horizontal motion

In some blocks of trials we noted that G.Y. was more likely to report that he was aware if the target was moving up rather than to the right. In order to establish whether this was a consistent finding across different sessions, we pooled together all experiments in which G.Y. had been required to discriminate upward and rightward motion ($n = 1681$). We found that G.Y. was twice as likely to be aware of the upward motion than the rightward motion (41% aware versus 23% aware; $\chi^2(1) = 61$; $P < 0.00001$) and concluded that his threshold of awareness was different for the two directions, a finding that we discuss below.

Imaging the brain of G.Y.

The analytical tool that we use to interpret the functional significance of the brain images in our studies is the SPM method, as modified for fMRI (see Friston *et al.*, 1995*a*, *b*). At its most conservative, the method assesses the significance of a particular activation in terms of its *Z*-score and the size (number of voxels in the activated region) in relation to the size and number of voxels expected by chance, using the theory of Gaussian fields (Friston *et al.*, 1996). This allows one to state, without any *a priori* knowledge or hypothesis, that a particular area is activated by a particular stimulus. This is a rather stringent method which has to be used with discretion; if one has an *a priori* hypothesis, the stringency can be relaxed and a lower *Z*-score accepted as indicative of

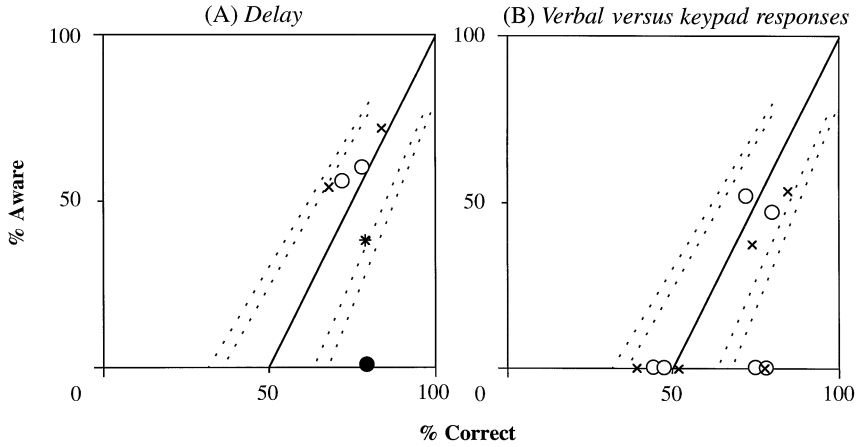

Fig. 5 (**A**) The effect of response delay. Two different experiments are shown. Experiment 1 [task: 41′ target, upwards/rightwards motion, 16°/s, with a 4 cd/m^2 background (crosses and open circles)]. All four blocks of trials were performed on the same day (session 2). The crosses show his performance with a mean delay of 1.3 s (range 0.6–2.1 s), the open circles show his performance with a mean delay of 4.1 s (range 2.2–6.6 s). Experiment 2 [task: 22′ target, upwards/rightwards motion, 16°/s, with a 24 cd/m^2 background (filled circle and star)]. Both blocks of trials were performed on the same day (session 4). The filled circle shows his performance with a mean delay of 1.5 s (range 1–2 s), the star shows his performance with a mean delay of 4.2 s (range 1–7 s). (**B**) The effect of response modality. Task: see Fig. 3D. All blocks of trials were recorded in the same session (session 4). Each cluster of scores represents his performance at a single contrast, crosses denote verbal responses and open circles denote keypad responses.

significant activity even if it does not withstand a correction for multiple comparisons. We therefore separate the results below into those with and those without an *a priori* hypothesis, and we accept all the activations shown as being biologically significant.

Significant activation without a priori *hypotheses*

Comparison of fast and slow motion. We puzzled over what the fluctuation in G.Y.'s level of performance, in terms of both awareness and discrimination, might mean in neurological terms. It seemed possible that the switch from the unaware to the aware state might involve different pathways. But the transition between the aware and unaware states during repeated blocks of the same task reported above made it equally, if not more, plausible that only one pathway is involved. A relatively simple way of answering this question was to image the activity in G.Y.'s brain when he was discriminating above chance in two different states, one of which was 'unaware' and the other 'aware'. This was a risky strategy, given the fluctuation in his level of discrimination and awareness for the same task reported above. We initially opted for the two stimuli that promised the best chance of success, namely vertical and horizontal motion, since his awareness of vertical motion was significantly greater than his awareness of horizontal motion. Unfortunately, these stimuli proved to be ineffective for imaging the activity in his brain, possibly because they were relatively small in relation to the total extent of the blind hemifield; they were also present for a relatively short part of the total scanning time. At the lowest corrected threshold that we used, the many isolated voxels, especially in the frontal lobes, did not reach significance. We therefore opted for a different stimulus, derived from our previous work on visual evoked potentials in G.Y. (ffytche *et al*., 1996). This consisted of a checkerboard pattern moving either rapidly (20°/s) or slowly (4°/s); the stimulus differed from our previous study in that it moved in four directions, thus allowing us to measure G.Y.'s discrimination of motion. We knew from our previous work that the fast condition would elicit cortical activity while the slow one would not, at least in measurable physiological terms. We also knew that G.Y. was unfailingly conscious when presented with fast motion; by contrast, he was only aware of the onset of the slow motion stimulus in our previous EEG experiments, but not of its direction of motion (ffytche *et al*., 1996). Before using these two stimuli for our fMRI studies, we decided therefore to titrate the contrast of the stimuli with G.Y. in the scanner, until he reported himself no longer aware of even the onset of the slow-motion stimulus. We also determined his discriminatory capacity and his level of awareness after each trial while he was being scanned. He gave the following scores, which are illustrated as crosses in the graph of Fig. 2B: (i) for fast motion, 72% correct, 96% aware (showing that his performance was not as good as would be predicted from his awareness level) and (ii) for slow motion, 54% correct and 10% aware (showing a better capacity to discriminate than would be predicted from his awareness level). Given the variability in his discriminatory performance and his level of awareness, described above, we were very fortunate in the outcome of his performance while he was in the scanner, since the two conditions were now ideally suited to reveal whether the switch from the 'aware' to the 'unaware' mode would activate different pathways. The profile of activity in his brain when we compared the fast with the slow motion condition (Fig. 6A), showed only one area of

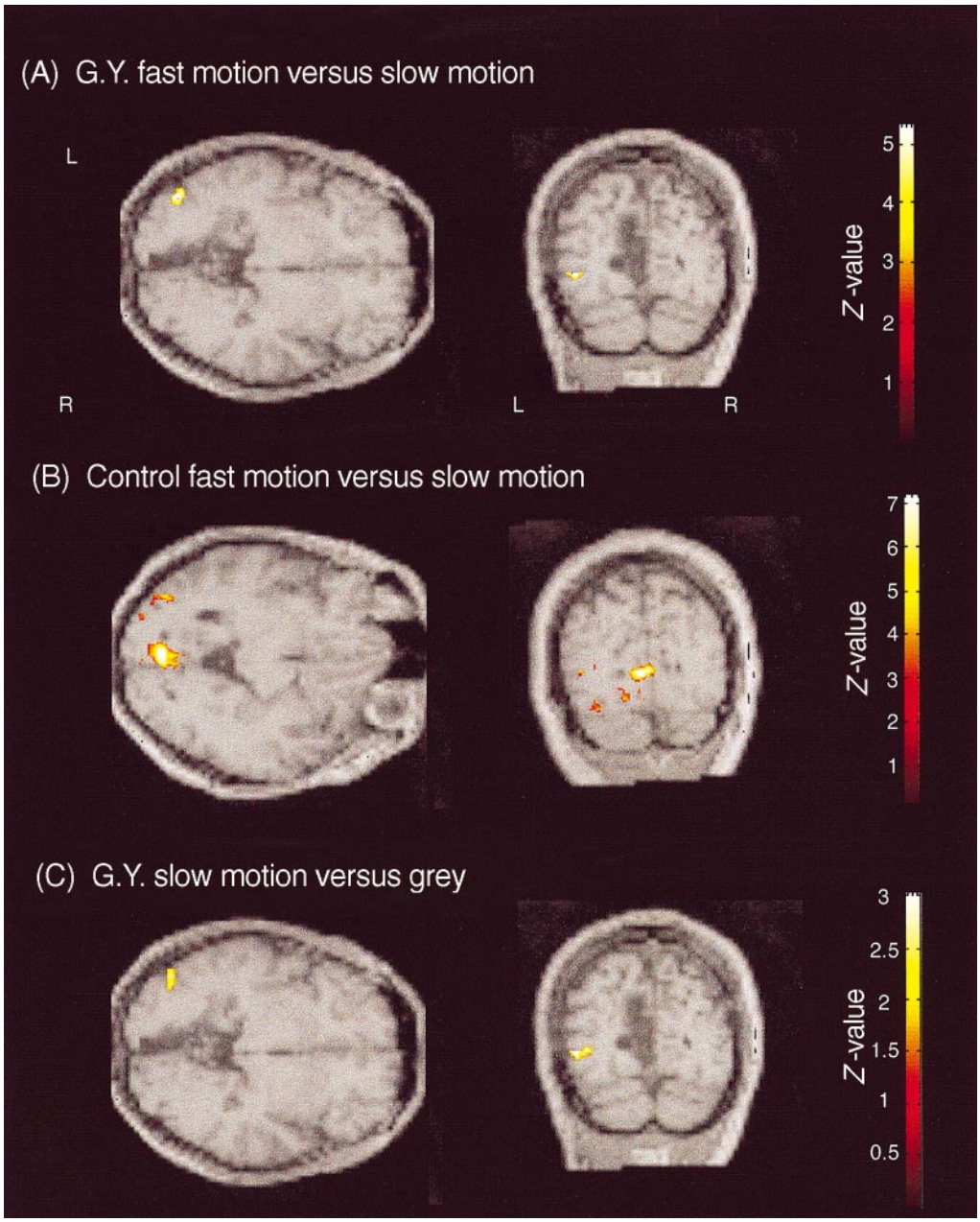

Fig. 6 Imaging experiments. Statistically significant increases in BOLD signal (shown in colour) superimposed on transverse and coronal sections of G.Y.'s brain. The size and level of significance of each region are given in Table 3. (**A**) The increases in cerebral activity comparing fast with slow motion in G.Y. (**B**) The increases in cerebral activity comparing fast with slow motion in a control subject. (**C**) The increases in cerebral activity comparing slow motion with an isoluminant grey control.

activation: a region situated ventrally in the occipital lobe, within the area identified in our earlier study as being human area V5 (Watson *et al*., 1993) (Table 3). For comparison, we tested the same stimulus in an age-matched normal male control who, like G.Y., is left handed. The activity elicited in the control brain is shown in Fig. 6B and includes area V1 (at the correct eccentricity) as well as area V5. Comparison of fast motion versus grey in G.Y. and in the normal control also resulted in an activity in V5, but a comparison of slow motion versus grey resulted in a significant activity in V5 of the normal control subject only. In both, slow motion compared with fast motion did not elicit any detectable cerebral activity, thus showing that a different set of areas is not activated with the slow motion stimulus. We conclude that, in both G.Y. and the normal control subject, fast motion activates V5 better than slow motion.

The activity in aware versus unaware trials. As shown above, G.Y. was not aware during every fast motion trial, nor was he unaware during every slow motion trial. The design of our experiment was such that we could compare the activity produced in the brain when we selected the aware

Table 3 *The* fMRI results

Subject	Comparison	Area	*Z*-score	Voxels	Corrected significance (cluster-level)
G.Y.	Fast versus slow (correlated areas)	V5	5.77	29	$P < 0.003$
		V3	4.49	4	$P < 0.001$ uncorrected
		Brodmann area 7	4.11	7	$P < 0.001$ uncorrected
		Cerebellar vermis	3.65	1	$P < 0.001$ uncorrected
		Superior temporal gyrus	4.88	5	$P < 0.001$ uncorrected
		Middle frontal gyrus	4.60	19	$P < 0.018$
	Fast versus grey	V5	7.10	32	$P < 0.001$
	Slow versus grey	V5	2.47	11	$P < 0.007$ uncorrected
	Aware versus unaware (fast and slow motion)	Brainstem	4.77	24	$P < 0.007$
	Aware versus unaware (motion confound removed)	V5	4.53	3	$P < 0.001$ uncorrected
		Brain stem	3.80	9	$P < 0.001$ uncorrected
Control	Fast versus slow	V5	5.05	25	$P < 0.002$
		V1	7.21	186	$P < 0.001$
	Fast versus grey	V5	8.74	80	$P < 0.001$
		V1	8.95	401	$P < 0.001$
	Slow versus grey	V5	8.46	34	$P < 0.001$
		V1	8.29	39	$P < 0.001$

trials with that in the unaware trials, regardless of whether the motion that produced the 'awareness' or 'unawareness' was fast or slow, and treating the grey condition, of which G.Y. could not be aware, as a confounding variable. The result of such a comparison showed that the only significant activity in the brain of G.Y. occurred inferior to the ponto-medullary junction (Fig. 7) in what we interpret later to be the reticular formation (see Discussion section). We could of course not do the same to the normal control subject since he was always aware, regardless of the trial.

Significant activation with a priori *hypotheses*

Slow motion versus grey stimulation. Because a separate set of areas was not activated with the slow motion condition, we were left with the puzzle of what areas are involved during G.Y.'s discrimination of slow motion. We formulated the hypothesis that the same areas, namely V5 in G.Y., and V5 and V1 in the normal subject, would be activated with the slow motion stimulus but at a lower intensity, requiring a less stringent statistical test for detection. A quick way of establishing this was to compare the activity in V5 during slow motion and grey stimulation. This revealed an activation of V5 in G.Y. We conclude that both fast and slow motion lead to activity in V5 but at different levels of intensity, even in the absence of V1 (see Table 3 and Fig. 6C). This result is consistent with our earlier demonstration of a fast motion input to V5 that by-passes V1 (ffytche *et al*., 1995); we discuss below why slow motion did not reveal an activity in V5 with the EEG method.

Aware versus unaware. The aware versus unaware analysis given above had not taken into account the fact that most of the fast motion trials were aware and most of the slow ones unaware. In order to reveal areas that may be activated when awareness is divorced from the confounding effects of speed, we treated speed as a confounding variable and re-analysed our results; this naturally reduced the number of usable trials and hence also the *Z*-score, but we were able to test for activity in area V5 and in the brainstem. The analysis revealed an activation of area V5 and of a region lying caudal to the ponto-medullary junction, in what we again interpret to be the reticular formation in G.Y. Once again, the absence of unaware trials in the normal subject made it impossible to undertake a similar analysis with him.

Co-variations of other cortical areas with V5

In our earlier PET study (Barbur *et al*., 1993), we found that fast motion activated areas beside V5, the most prominent among these being area V3 and the parietal cortex. We wondered whether the activity that we had seen in these other areas in our earlier study could still be observed by identifying any areas, visual or otherwise, that co-varied consistently with the activity in V5. The result of such an analysis of co-variation is given in Table 3, and shows that the areas that were activated in our previous study co-varied with V5 in this one. This study, however, also revealed a further area, not seen in our previous results, located in the right middle frontal gyrus. We do not know what significance to attach to these activations since the analysis of co-variation reveals a network of areas that act in concert, without

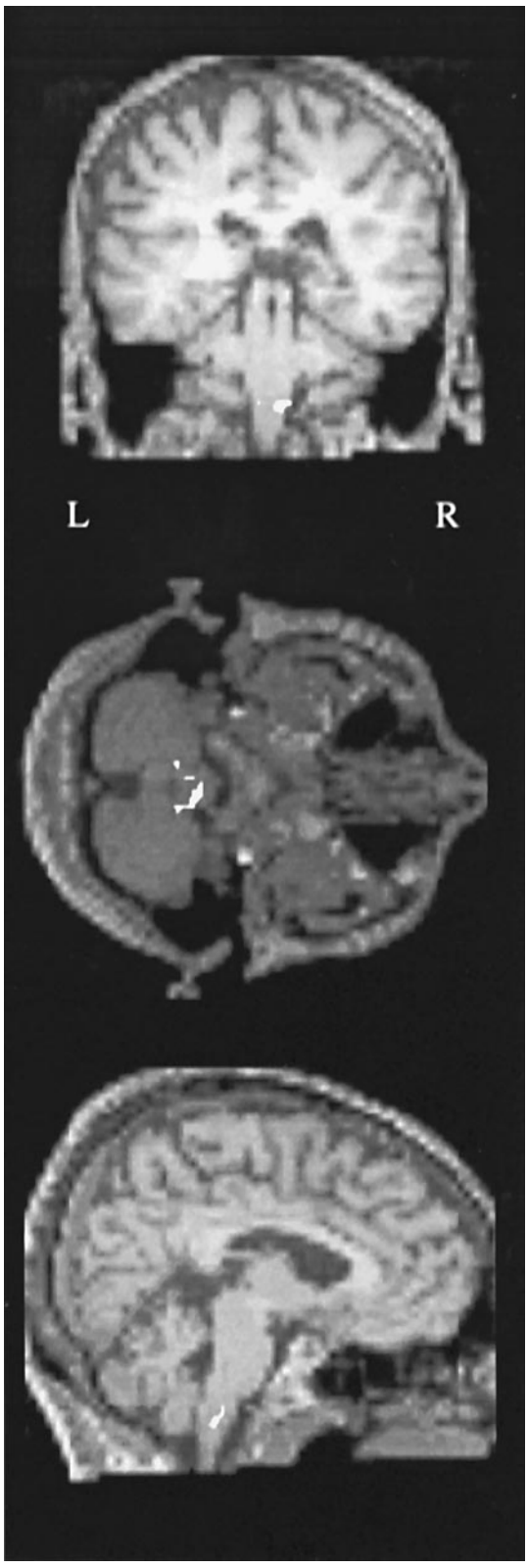

Fig. 7 Imaging of aware versus unaware trials. A factorial analysis has been performed testing for the main effect of awareness (fast aware and slow aware versus fast unaware and slow unaware). The grey control stimulus is included in the analysis as a co-variate of no interest. The statistically significant increases in BOLD signal are superimposed on coronal, transverse and sagittal sections of G.Y.'s brain and the size and level of significance of each region is given in Table 3.

reference to any of the three stimulus conditions; we are therefore not able to determine the role of each individual area within this network.

Superior colliculus and pulvinar

One area that has been implicated in unconscious vision (e.g. Barbur *et al*., 1980) is the superior colliculus. Since the superior colliculus connects with the pulvinar which in turn has direct inputs to V5 (Cragg, 1969; Standage and Benevento, 1983), it seemed reasonable to formulate the *a priori* hypothesis that one or both of these structures would be active, at least in the slow motion condition. We therefore specifically examined the superior colliculus and the pulvinar in all comparisons (fast versus slow; slow versus fast; slow versus grey; fast versus grey), at the lowest thresholds and also for co-variation with V5; we found no activity in either structure in the comparisons or in the co-variation analysis.

In conclusion, we may summarize the imaging studies as follows: (i) fast versus grey and fast versus slow activated V5 in G.Y.; (ii) slow versus grey activated V5 in G.Y. but required a less stringent threshold for demonstration; (iii) comparisons of aware trials for fast and slow motion with unaware trials for the same two speeds, which makes awareness the critical variable, led to significant activity in the medulla; (iv) using speed as a confounding variate and thus focusing on awareness, independent of motion, led to activation of area V5 and the medullary region in G.Y.

Re-examination of G.Y.'s evoked responses

The above results show a highly significant change in the BOLD signal during fast motion trials, but also an activity during slow motion trials that is sufficiently less pronounced to require a relaxation of thresholds to be detected. This made it interesting to compare these results with those obtained from our earlier evoked potential study with G.Y. (ffytche *et al.*, 1996). In that study we had devised two methods to differentiate between genuine signals related to the processing of a visual stimulus and background EEG noise. These methods showed that fast motion elicited a consistent, repeatable early response in G.Y. ($<$100 ms) which matched that of normal control subjects. On the other hand, slow motion failed to elicit a repeatable response and the pattern of early activity bore no resemblance to that of normal control subjects. We concluded that slow motion did not activate V5 in G.Y. The fMRI and the EEG studies are thus in general agreement, the only difference being that fMRI emerges from this study as a more sensitive indicator of small increases in activity than the evoked response method.

Discussion

What we had imagined would be a simple study of the pathways involved in conscious and unconscious vision ended

up immersing us in a study of the characteristics of the visual motion capacities of G.Y. and of the relationship between discrimination, awareness and cerebral activity. Our results, we believe, have provided us with some insights into the Riddoch phenomenon, its relationship to 'blindsight' and, beyond that, into the contribution that individual prestriate areas make to conscious vision.

Is activity in prestriate cortex, without parallel activity in V1, unconscious?

The most important conclusion that we reach confirms our earlier one more extensively, namely that G.Y. has a conscious experience when visual stimuli are presented in his blind field and activate area V5 without activating V1 (see Barbur *et al.*, 1993). The evidence that we have presented here and elsewhere (ffytche *et al.*, 1996) leaves us in no doubt that this capacity is conferred on G.Y. by the activity in his prestriate cortex, with V5 when motion is involved, although it could also involve the activity in the other areas that, as we have shown here, co-vary with V5.

That patients blinded by lesions in V1 can discriminate certain visual stimuli presented to their blind field consciously is not new. The first description was by Riddoch (1917), who inferred the presence of lesions in V1 from his perimetric studies; in more recent times this has been emphasized by Ruddock and his colleagues (Blythe *et al.*, 1987). Table 4 shows a number of other studies in which patients with lesions in V1 have experienced a stimulus presented to their blind fields consciously. The conscious experience is described in different ways, many of the terms being the same as those used by G.Y. Sometimes subjects have a 'feeling' but are 'absolutely sure of it' (Weiskrantz, 1986); sometimes they see 'shadows' (Riddoch, 1917; Barbur *et al.*, 1980) or 'pinpoints' of light (Weiskrantz, 1980). However described, there can be little doubt but that these descriptions refer to conscious states. Although the reference to 'feeling' something is acknowledged in the literature that emphasizes the capacity to discriminate in the absence of awareness, it is nevertheless also true that, because of the implicit assumption that 'feeling' is not the same as 'seeing' (an unexceptionable assumption), subjects with such a capacity are often considered to be 'blindsight' subjects, without exploration of the extent to which their conscious state, however described, contributes to their performance. Thus, with the exception of the patients of Blythe *et al*. (1987), Ceccaldi *et al*. (1992) and Fendrich *et al*. (1992), the subjects tabulated in Table 4 have been considered to be 'blindsight' subjects, that is to say subjects who have no awareness of anything occurring in their blind field (Sanders *et al.*, 1974; Weiskrantz *et al.*, 1974). It was only after the publication of our 1993 paper (Barbur *et al.*, 1993), in which we showed that G.Y.'s vision can be conscious, that the conscious dimension in 'blindsight' patients was acknowledged (Weiskrantz, 1995) and a systematic attempt was made to distinguish between conditions in which G.Y. could discriminate stimuli of which he was consciously aware, without distinguishing between feeling or seeing, and stimuli which he could discriminate 'without any sensation or feeling or experience of the visual event' (Weiskrantz *et al.*, 1995), the result confirming our view that a conscious experience of visual stimuli is possible without V1, itself a confirmation of the earlier work of Riddoch (1917). More explicitly, we consider a 'feeling', especially one that the subject is sure of, to be a conscious experience and our results demonstrate that this state contributes significantly to G.Y.'s capacity to discriminate, to the extent that there is, in G.Y. as in normal subjects, a positive correlation between awareness and discrimination, however the awareness is described. Taking the published evidence and our own results into account, we thus disagree with the conclusion that 'much of the non-striate capacity is 'unconscious', i.e. not accompanied by the person's awareness of the stimuli' (Weiskrantz, 1990). But this is not to say that every activity in prestriate cortex has a conscious correlate; clearly the activity in V5 elicited by slow motion does not always have a conscious correlate.

Conscious 'vision' without V1

More recently, in the light of our results and those of others (Barbur *et al.*, 1993; Weiskrantz *et al.*, 1995) the definition of 'blindsight' has been modified to include subjects who '. . . were aware of the occurrence of a visual event' though they could not see it (Sahraie *et al*., 1996); another recent modified definition considers blindsight to be the ability to discriminate in the absence of phenomenal vision (Stoerig and Cowey, 1995; Stoerig, 1996), phenomenal vision being defined as 'the lowest level of conscious vision; provides an image consisting of qualia' (Stoerig, 1996), implying that the conscious capacities described above are not visual in nature. This distinction in the phenomenology, between seeing and feeling, has led to the notion that 'conscious vision is not possible without V1' (Stoerig and Cowey, 1995; Stoerig, 1996). Whether the conscious experience of subjects like G.Y. and others provides qualia is difficult to tell, and the question is probably not worth debating because it is impossible to ascertain. Such experience as these subjects have, whether described as seeing or feeling, is triggered by a visual stimulus and is therefore a visual experience. Phenomenology, by definition, refers to knowledge derived from the senses. In this instance, it is knowledge derived from the visual sense, generated by a visual stimulus, presented to a visual apparatus and accompanied by a correct and conscious discrimination of both its presence and its characteristics. There are of course examples to show that the input from one sensory system can trigger experiences in another, synaesthesia being one well known example. But, significantly, the published evidence and our results show that the 'feeling' of the patients reviewed here, including G.Y., is closest to the visual modality and can transmute into the explicit experience of 'seeing', with the characteristics

Table 4 *Consciousness and striate lesions*

Author	Patient	Lesion	Conscious experience in blind field
Sanders *et al*. (1974)	D.B.	Occipital pole and calcarine sulcus	When presented with a vigorously moving stimulus he would sometimes report seeing 'something' but was unable to identify it.
Weiskrantz *et al*. (1974)	D.B.	Occipital pole and calcarine sulcus	If pressed, he might say that he perhaps had a ' feeling' that the stimulus was either pointing this or that way, or was 'smooth' (the O) or 'jagged' (the X).
Weiskrantz (1986)	D.B.	Occipital pole and calcarine sulcus	D.B. reported an impression of 'waves' in parts of his field defect. The experience is of a kind unlike anything in normal visual experience, and for which precise words seem to be lacking. The 'waves' can have some sort of form. They can be straight or curved, or can even have a 'squareness'.
Weiskrantz (1980)	K.P. T.H.F. E.Y.	Unspecified field defect Unspecified field defect Hemianopia	'A very faint flash' 'I just have a feeling' 'When I was certain there was a definite pinpoint of light'
Barbur *et al*. (1980)	G.Y.	Medial occipital	Subjectively, G. reports that a flashed target of near-threshold illumination appears as a 'dark shadow' located in the 'blind' hemifield. At higher illumination levels, it sometimes appears as a bright flash.
Blythe *et al*. (1987)	R.C. R.L. B.W.	Medial occipital Occipital pole Medial occipital	All three experience a sensation of a dark shadow, localised within their 'blind' fields when stimulated by transient changes in illumination of either positive or negative contrast.
Shefrin *et al*. (1988)	J.S.	Occipital lobe	When pressed, admitted to an occasional impression of seeing something ill-defined and poorly formed ('blobs') when the words were flashed in her blind field.
Ceccaldi *et al*. (1992)	M.M.	Bilateral medial occipital	He consciously perceived visual motion in the blind parts of his visual field.
Fendrich *et al*. (1992)	C.T.	Medial occipital	He occasionally had a sense that 'something happened there'.

of the 'seen' stimulus correctly and adequately defined by the subject. Moreover, none of us can decide that when a subject reports 'seeing' something 'ill-defined' such as 'blobs' (Shefrin *et al.*, 1988) or 'definite pinpoints of light' (Weiskrantz, 1980) that this does not constitute visual qualia. We do not wish to imply that there is no difference between feeling and seeing, nor that the sight that such blind people have is in any way similar to that of normal subjects. We simply affirm that it has a conscious correlate. Hence we do not think that the case for saying that 'conscious phenomenal vision is not possible without V1' has been made. In summary, we conclude that conscious vision without V1 is possible.

Fluctuations in levels of awareness and visual discrimination

Our findings show that there is a correlation between G.Y.'s capacity to discriminate and his awareness. The correlation, though significant, is not absolute. This is almost certainly due to the fluctuating level of both his visual awareness and his discrimination performance. His level of awareness under particular stimulus conditions can vary between sessions, without necessarily entailing a change in levels of discrimination. Figure 3A shows, for example, how G.Y.'s awareness for a given task varied between 0 and 80% on different occasions while his discrimination level remained unchanged. It follows that, with the same task, G.Y. can be said to have 'blindsight' with awareness and discrimination scores that are similar to those reported by Weiskrantz *et al.* (1995) on some occasions, while on others he does not show this property. Conversely, G.Y.'s discriminatory performance can vary for the same task, without necessarily entailing a parallel fluctuation in awareness (see Fig. 3B). This variability in both G.Y.'s awareness and discriminatory levels has not been reported before; it has undermined our confidence in 'blindsight' as a distinct phenomenon. The fluctuations that we refer to are not simply the normal scattering of results that might be expected for repeated presentations of the same task; this does happen with G.Y., as shown in Fig. 5B; the fluctuations are large and unpredictable and lead to an apparent uncoupling between awareness and discrimination. It is probable that it is the V1 lesion that causes this uncoupling, perhaps by increasing the overall level of background neural noise in G.Y.'s spared pathways, and,

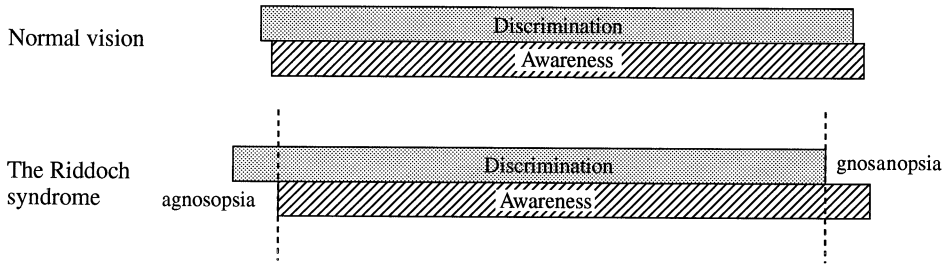

Fig. 8 The proposed relationship between discrimination and awareness in normal vision and in the Riddoch syndrome.

although there is no direct evidence of this, we speculate on it below. But we do so with respect to motion vision alone. We do not know whether a lesion in V1 also produces an uncoupling for other attributes of the visual scene, such as form or colour. The variability in the level of conscious awareness is the key to understanding the Riddoch syndrome and provides a convincing explanation of why 'blindsight' patients 'sometimes' see and experience feelings when stimulated in their blind fields, and sometimes do not (Sanders *et al.*, 1974; Shefrin *et al.*, 1988).

Whatever the cause, the looser coupling between discrimination and awareness leads to three inter-related states, all of which are observable in G.Y. The most common of these is the capacity to discriminate when aware; we refer to this state as gnosopsia (from *gnosis* = knowledge and *opsia* = vision). The second is a condition in which there is an awareness that something has happened in the blind field, without the capacity to discriminate correctly what has occurred; we refer to this as gnosanopsia (*anopsia* = blindness). The third is the capacity to discriminate correctly without having any conscious awareness; this is the variant that we call agnosopsia; it corresponds best to 'blindsight'. But unlike 'blindsight' which, as described, refers to a state always accompanied by a lack of awareness and which one can reliably elicit with a given set of stimulus parameters, agnosopsia is an unstable state which, because of the fluctuations in the level of awareness, cannot be always demonstrated; in fact it can transmute into gnosopsia.

The variable relationship between awareness and discrimination, for both normal subjects and hemianopic patients, is shown in a simplistic model in Fig. 8. Because of the results given above, we have illustrated the two visual attributes as being very closely coupled in normal subjects and much less tightly coupled in G.Y. One consequence of a lesion in V1 is to relax the coupling without abolishing it, with the result that patients with lesions in V1 can show all of the three variants described above. This leads us to conclude that 'blindsight' is simply one state in a more general condition, in which awareness and discrimination are not as tightly coupled as in normal individuals, a point taken up below.

Because of the close inter-relationship of these three conditions, we believe that they are all manifestations of a single syndrome, which we shall refer to as the Riddoch syndrome, in deference to the neurologist who first described it. We do not use the term 'blindsight' for five reasons: (i) our belief, demonstrated in the above psychophysical and imaging results, that 'blindsight' is but one manifestation of the Riddoch syndrome; (ii) that even as only one manifestation of the more general Riddoch syndrome, the fluctuations in levels of awareness prevent 'blindsight' from being reliably and repetitively demonstrated, at least in our hands; (iii) that the term 'blindsight', though easy to remember, nevertheless makes no unambiguous reference to the defining condition of the state that it purports to describe, namely the absence of awareness during correct discrimination; (iv) that, as used in the past, it has excluded 'feeling' from being a conscious experience, which is why, in spite of the certainty of these 'feelings' in response to visual stimulation in some patients (Weiskrantz, 1986), they were nevertheless considered to be 'blindsight' patients; and finally (v) that the definition of blindsight itself has changed (e.g. Stoerig, 1996; Sahraie *et al.*, 1996, see above) to acknowledge the fact that 'blindsight' patients can have awareness for visual stimuli.

It is interesting to consider briefly whether our observations may be due to the fact that G.Y. sustained his lesion many years ago and has been tested repeatedly since, thus allowing considerable recovery. We think not; monkey physiological evidence shows an immediate re-organization of field properties in V5 after sub-total lesions within it (Wurtz *et al.*, 1990). There is no similar evidence in humans, but if the nervous system is organized along similar lines in the two species, any re-organization in human V5 or the inputs leading to it would have occurred soon after the lesion. We did not detect any overall improvement in the performance of G.Y. over the period during which we tested him (e.g. see Fig. 3F) and the fundamental condition, of a capacity to be consciously aware of moving stimuli presented to the blind field, is what Riddoch observed and his patients were studied between 3–6 months after injury. The similarity between the descriptions given by the Riddoch patients and by G.Y. convinces us that his abilities are not due to repeated testing or to a slow recovery of function over the 30-year interval between his injury and our studies. Moreover, our evoked potential studies (ffytche *et al.*, 1996) show that G.Y.'s response to a fast moving stimulus presented to his blind hemifield is identical to that of normal subjects, thus arguing against a subtantial reorganization. The rarity of the Riddoch syndrome reflects, we believe, the rarity of occipital lesions

that destroy the striate cortex without the prestriate cortex and its direct subcortical input.

G.Y. is not alone in demonstrating visual awareness without V1; that other patients can be capable of conscious vision without V1 has been demonstrated by us and others (see Table 4). But it is from G.Y. that the best evidence for a relaxed coupling between discrimination and awareness, resulting from a V1 lesion, has been obtained. This uncoupling results in the two symmetrical conditions of *agnosopsia* and *gnosanopsia*, a pattern that is at present solely derived from the subjective experiences and reports of G.Y. We are less certain of how common the uncoupling is and whether, for unknown reasons, it may work more in one direction than in another, although fluctuation in the level of awareness is implicit in the variable descriptions given by hemianopic patients. It is essential that it should be validated by a study of other patients, using other paradigms and tasks, especially ones that do not rely on two choices (a binary system). This is made especially emphatic by our demonstration that G.Y. has different thresholds of awareness for up and right motion, possibly leading him to discriminate between the two by responding right when he is unaware and up when he is aware. If there are indeed fluctuations in both awareness and discrimination, as we have described them, then new patients will have to be investigated more systematically, with the level of awareness being determined after each discrimination and the repeatability of their performance for the same task on different occasions ascertained, since it is also possible that these fluctuations may be, up to a point, task dependent. It is only by the use of the right test at the right (and unpredictable) time that one may be able to uncover a condition corresponding to, say, agnosopsia.

The neural pathways in the Riddoch syndrome

Our supposition that the three states represent different aspects of one and the same syndrome, the Riddoch syndrome, receives support from the demonstration here that the same neural pathways are activated in all three states. Our imaging studies show two features that merit consideration: (i) the activation of V5 by both fast and slow motion, and the co-variation of this activity with a restricted network of other areas and (ii) the relationship between awareness and activity in V5 and the reticular activating system.

Regardless of whether the motion stimuli of which he is usually aware or usually unaware were shown, i.e. whether movement was fast or slow, the activity in G.Y.'s brain was centred on V5; the only difference between the two conditions was that the activity produced there by the slow motion was less intense. That activity in V5 can be associated with both aware and unaware states is neither new nor surprising. After all, one can record perfectly healthy responses from V5 in the anaesthetized brain, both in the monkey (e.g. Zeki, 1974; Albright, 1984) and human (our unpublished work with David Sandeman and Stuart Butler) where the conscious dimension is either minimal or non-existent. What our results show is that the switch from the unconscious to the conscious state correlates with the strength of activation in a given area, in our instance in the area that is specialized for the visual attribute that we concentrated on, namely motion. What modulates the strength of activation and the consequent change in awareness is less certain in the case of a subject like G.Y. than in anaesthetized states. Anaesthetics depress the overall activity of cells in the cortex and lead to an unconscious state but they leave the cells in an area such as V5 responsive and selective enough for one to be able to record from them. We presume that, in G.Y., it is the absence of V1 that is responsible for the variation of activity in V5 in response to the same stimulus presented on different occasions; though we do not know what this role amounts to in neural terms, we speculate on it below. A result that may favour our conclusion that consciousness for a given attribute such as motion may correlate with the strength of activation of a given visual area such as V5 is that fast moving stimuli, which reach V5 without passing through V1, elicit a stronger activation of V5 and are better able to elicit a conscious discrimination in G.Y. We naturally realize that the finding we report here for the first time, of a positive relationship between cerebral activity in a specific visual area and awareness for a correspondingly specific visual attribute, has implications that go beyond the V5 system and may be generalizeable to the whole cortex.

The activation of the reticular system

The specific activation of V5 in the cortex, with both fast and slow motion, was impressive but should not lead us to suppose that it is only the strength of activation in this area that modulates conscious awareness for a visual stimulus in motion. Not less impressive to us was the activation in the medullary region, observed when we compared the aware with the unaware condition. We interpret this activation to be centred on the reticular activating system, but our evidence in this regard is based solely on the general area; our spatial resolution was not high enough to place it there unequivocally rather than in another of the many distinct nuclei that crowd into this region. Whatever its actual location, the fact that this area showed a high activation in the aware condition leads us to propose that activity there correlates with the state of awareness. It is far less certain how this activity is related to the activity in V5 and whether it is specific to motion or represents a non-specific alerting response. Activity in the reticular activating system influences the transfer of visual signals through the lateral geniculate nucleus (see Singer, 1977) and thus might increase the level of activity in V5 above a certain threshold in aware trials. However, the medullary region did not co-vary with V5 in our analysis, arguing against this particular explanation. Another possibility is that the medullary activity alerts G.Y. to the fact that something has happened, without being related to the processing of motion signals, thus accounting for the uncoupling of awareness and discrimination.

The physiological basis of the Riddoch syndrome

Here we consider the surprising result that the errors that G.Y. made when confronted with four directions were mainly in a direction orthogonal to the actual direction of motion of the test target. This result was significant enough for us to speculate on the neurological basis of the syndrome.

It is now well established that the characteristic of V5—directional selectivity—is maintained after inactivation of V1 (Rodman *et al.*, 1989; Girard *et al.*, 1992), though cells in V5 apparently become less exigent in their directional specificity. Both the above studies have shown that signals that reach V5 without passing through V1 are sufficient to maintain the physiological characteristic of V5 but have argued that such an input does not have a conscious correlate; they suppose it therefore to be the neural pathway that leads to one of the variants of the Riddoch syndrome, namely agnosopsia. Our view is, on the other hand, that the input that reaches V5 without passing through V1 can and does lead to a conscious awareness of motion. Despite this difference in interpretation, some features of their findings can nevertheless help us understand our present results, derived from a different species. The first is the broadening of the tuning curves (Girard *et al.*, 1992). If G.Y.'s errors are due to this broadening, then one might expect him to make more of his errors in a direction orthogonal to the true direction, which would be encompassed within the broadened curve, then in a diametrically opposite direction, which would be well outside it; this is what we have observed. The second feature is the finding that the activity in V5 is diminished after removing V1 (Rodman *et al.*, 1989; Girard *et al.*, 1992). We have argued above that activity in V5 correlates with conscious awareness only if it is above a certain level. V5 receives a heavy input from V1 and is reciprocally connected with it (Cragg, 1969; Zeki, 1969; Shipp and Zeki, 1989). When V1 is removed, V5 will be deprived of much, if not all, of its cortical input. We speculate that this reduces the level of motion-related activity in V5, bringing it closer to threshold levels for awareness, and that it increases the amount of neurophysiological noise. This in turn might explain why subjects with lesions in V1 are conscious of stimuli on some occasions and not on others.

In historical context

Riddoch's work has made little impact. This may not be surprising given that, at the time that Riddoch was writing, most experiments had shown, or so neurologists believed, that area V1 was '. . . the only entering place of the visual radiation into the organ of psyche' (Flechsig, 1905) and was thus the 'sole' visual perceptive cortex (Holmes, 1945; for review see Zeki, 1990). Although Riddoch himself had not suggested that the capacity to see motion was conferred by any other cortex than the spared tissue within V1, the concept that V1 was the 'sole' perceptive cortex had been at least called into question by the demonstration of a dissociation of functions following cerebral lesions, and in particular from the finding, reported by Eperon (1884), Wilbrand (1884), Verrey (1888) and Mackay and Dunlop (1899), that colour vision could be specifically compromised following cerebral lesions located in the lingual and fusiform gyri. Any demonstration that the causative lesion might be outside V1 would suggest—in the thinking of the time—that the 'seeing cortex' is more extensive than V1, a notion firmly rejected by Holmes and others. Indeed, the lingual and fusiform gyri are located within cortex which was widely believed at the time to have 'psychic' and interpretative functions; Campbell (1905) had written that the visual cortex consists of two parts '. . . one [V1] specialized for the primary reception of visual sensations, the other constituted for the final elaboration and interpretation of these sensations'. Bolton (1900) had voiced a very similar view (for a review see Zeki, 1990). But Verrey (1888) and Mackay and Dunlop (1899) had been led by their observations to suppose that the primary 'seeing' cortex extended well beyond V1, a view that Henschen (1900) dismissed as improbable, insisting that 'the cortical retina [V1] is also a retina for colour impressions'. To Henschen, the notion of a perceptive centre for colour outside V1 was absurd for, if it were true, then '. . . with the calcarine cortex destroyed and the cortex of that other gyrus [lingual and fusiform] intact, the patient would then have to be absolutely blind and yet be able to see colours, which makes no sense' (Henschen, 1910). That scenario, which seemed so improbable to Henschen, is the very one that Riddoch described in his 1917 paper for motion and which we repeat here.

The work of Henschen, Holmes and others thus conferred on V1 the sovereign capacity of 'seeing' while the interpretation of what was seen was deemed to be the function of the then ill-defined visual association cortex, a notion that neatly separated seeing from understanding and assigned a separate cortical locus to each. Partly because of the relatively slow maturation of the visual 'association' cortex, it was deemed to be part of the higher centres capable of higher conscious functions, the *geistige Zentren* or *Cogitatzionzentren* of Flechsig (1905). If only by implication, conscious experience was therefore more properly regarded as being the function of association cortex, although the early thinkers did not deprive activity in V1 of a conscious content. In the 1970s and 1980s it became increasingly apparent, with the demonstration of multiple specialized visual areas in what used to be known as visual 'association' cortex, that the processing of the visual image is far from complete at the level of V1; it also requires the activity of the specialized areas of the prestriate cortex. The distinction between seeing and understanding became increasingly blurred and the notion of a separate cortical seat for each faculty was rendered untenable. It is an irony of history that the notion that subjects blinded by lesions in V1 cannot see consciously has led to the reverse doctrine, that the non-striate cortex (association) is 'unconscious' (Weiskrantz, 1990). Our evidence, which

shows that activity in prestriate cortex can have a conscious correlate, leads us in a different direction. We reject the strict separation between seeing and understanding; our results, as well as those of Riddoch and others, show beyond doubt that a conscious experience of seeing, accompanied by an understanding of what is seen, at least in terms of discrimination, is possible without V1, through the specialized visual areas of the prestriate cortex. We conclude that activity restricted to one of the parallel systems comprising the visual pathways can generate a conscious visual experience and that visual consciousness itself may therefore be modular, even when it excludes V1. It is an hypothesis that is worth testing.

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References

Albright TD. Direction and orientation selectivity of neurons in visual area MT of the macaque. J Neurophysiol 1984; 52: 1106–30.

Barbur JL, Ruddock KH, Waterfield VA. Human visual responses in the absence of the geniculo-calcarine projection. Brain 1980; 103: 905–28.

Barbur JL, Watson JD, Frackowiak RS, Zeki S. Conscious visual perception without V1. Brain 1993; 116: 1293–302.

Blythe IM, Kennard C, Ruddock KH. Residual vision in patients with retrogeniculate lesions of the visual pathways. Brain 1987; 110: 887–905.

Bolton JS. The exact histological localisation of the visual area of the human cerebral cortex. Phil Trans R Soc (Lond) B 1900; 193: 165–222.

Bullier J, Girard P, Salin PA. The role of area 17 in the transfer of information to extrastriate visual cortex. In: Peters A, Rockland K S, editors. Cerebral cortex Vol. 10: primary visual cortex in primates. New York: Plenum Press, 1994: 301–30.

Campbell AW. Histological studies on the localisation of cerebral function. Cambridge (UK): Cambridge University Press, 1905.

Ceccaldi M, Mestre D, Brouchon M, Balzamo M, Poncet M. Autonomie déambulatoire et perception visuelle du mouvement dans un cas de cécité corticale quasi totale. Rev Neurol 1992; 148: 343–9.

Cowey A. Visual awareness: still at sea with seeing? [Review]. Curr Biol 1996; 6: 45–7.

Cragg BG. The topography of the afferent projections in circumstriate visual cortex of the monkey studied by the Nauta method. Vision Res 1969; 9: 733–47.

Eperon. Hémiachromatopie absolue. Arch Ophthal (Paris) 1884; 4: 356–70.

Fendrich R, Wessinger CM, Gazzaniga MS. Residual vision in a scotoma: implications for blindsight. Science 1992; 258: 1489–91.

ffytche DH, Guy CN, Zeki S. The parallel visual motion inputs into areas V1 and V5 of human cerebral cortex. Brain 1995; 118: 1375–94.

ffytche DH, Guy CN, Zeki S. Motion specific responses from a blind hemifield. Brain 1996; 119: 1971–82.

Flechsig PM. Gehirnphysiologie und Willentheorien. Translated by G. von Bonin (1960). In Some papers on the cerebral cortex. Springfield (IL): Charles C. Thomas, 1905: 73–89.

Friston KJ, Holmes AP, Worsley KJ, Poline JB, Frith CD, Frackowiak RS. Statistical parametric maps in functional brain imaging: a general linear approach. Hum Brain Mapp 1995a; 2: 189–210.

Friston KJ, Holmes AP, Poline JB, Grasby PJ, Williams SC, Frackowiak RS, et al. Analysis of fMRI time-series revisited. Neuroimage 1995b; 2: 45–53.

Friston KJ, Holmes A, Poline JB, Price CJ, Frith CD. Detecting activations in PET and fMRI: levels of inference and power. Neuroimage 1996; 4: 223–35.

Girard P, Salin PA, Bullier J. Response selectivity of neurons in area MT of the macaque monkey during reversible inactivation of area V1. J Neurophysiol 1992; 67: 1437–46.

Goodale MA, Milner AD. Separate visual pathways for perception and action. [Review]. Trends Neurosci 1992; 15: 20–5.

Henschen SE. Sur le centre cortical de la vision. In: Rochon-Duvigneaud M, editors. XIII Congrés International de Médecine, Ophtalmologie. Paris: Masson & Cie, 1900: 234–45.

Henschen SE. Zentrale Sehstörungen. In: Lewandowsky M, editors. Handbuch der Neurologie. Berlin: Springer, 1910: 891–918.

Holmes G. Disturbances of vision caused by cerebral lesions. Br J Ophthal 1918; 2: 353–84.

Holmes G. The Ferrier Lecture: the organization of the visual cortex in man. Proc R Soc (Lond) B 1945; 132: 348–61.

Keating EG. Residual spatial vision in the monkey after removal of striate and preoccipital cortex. Brain Res 1980; 187: 271–90.

Kentridge RW, Heywood CA, Weiskrantz L. Residual vision in multiple retinal locations within a scotoma: implications for blindsight. J Cog Neurosci 1997; 9: 191–202.

Lund JS, Boothe RG. Interlaminar connections and pyramidal neuron organisation in the visual cortex, area 17, of the macaque monkey. J Comp Neurol 1975; 159: 305–34.

MacKay G, Dunlop JC. The cerebral lesions in a case of complete acquired colour-blindness. Scott Med Surg J 1899; 5: 503–12.

Mestre DR, Brouchon M, Ceccaldi M, Poncet M. Perception of optical flow in cortical blindness: a case report. Neuropsychologia 1992; 30: 783–95.

Pasik P, Pasik T. Visual function in monkeys after total removal of visual cerebral cortex. Contrib Sens Physiol 1982; 7: 147–200.

Riddoch G. Dissociation of visual perceptions due to occipital injuries, with especial reference to appreciation of movement. Brain 1917; 40: 15–57.

Rodman HR, Gross CG, Albright TD. Afferent basis of visual response properties in area MT of the macaque. I. Effects of striate cortex removal. J Neurosci 1989; 9: 2033–50.

Sahraie A, Weiskrantz L. Simmons A, Williams SCR. Conscious and unconscious processing of visual signals: psychophysical and fMRI studies [abstract]. Abstracts of the AVA Christmas meeting, 1996: 5.

Sanders MD, Warrington EK, Marshall J, Weiskrantz L. 'Blindsight': vision in a field defect. Lancet 1974; 1: 707–8.

Shefrin SL, Goodin DS, Aminoff MJ. Visual evoked potentials in the investigation of 'blindsight'. Neurology 1988; 38: 104–9.

Shipp S, Zeki S. The organization of connections between areas V5 and V1 in macaque monkey visual cortex. Eur J Neurosci 1989; 1: 309–32.

Singer W. Control of thalamic transmission by corticofugal and ascending reticular pathways in the visual system. [Review]. Physiol Rev 1977; 57: 386–420.

Standage GP, Benevento LA. The organization of connections between the pulvinar and visual area MT in the macaque monkey. Brain Res 1983; 262: 288–94.

Stoerig P. Varieties of vision: from blind responses to conscious recognition. [Review]. Trends Neurosci 1996; 19: 401–6.

Stoerig P, Cowey A. Visual perception and phenomenal consciousness. [Review]. Behav Brain Res 1995; 71: 147–56.

Verrey L. Hémiachromatopsie droite absolue. Arch Ophthal (Paris) 1888; 8: 289–300.

Watson JDG, Myers R, Frackowiak RSJ, Hajnal JV, Woods RP, Mazziotta JC, et al. Area V5 of the human brain—evidence from a combined study using positron emission tomography and magnetic resonance imaging. Cereb Cortex 1993; 3: 79–94.

Weiskrantz L. Varieties of residual experience. Q J Exp Psychol 1980; 32: 365–86.

Weiskrantz L. Blindsight. Oxford: Clarendon Press, 1986.

Weiskrantz L. Blindsight—not an island unto itself. Curr Dir Psychol Sci 1995; 4: 146–51.

Weiskrantz L, Warrington EK, Sanders MD, Marshall J. Visual capacity in the hemianopic field following a restricted occipital ablation. Brain 1974; 97: 709–28.

Weiskrantz L. The Ferrier lecture, 1989. Outlooks for blindsight: explicit methodologies for implicit processes. [Review]. Proc R Soc (Lond) B 1990; 239: 247–78.

Weiskrantz L, Barbur JL, Sahraie A. Parameters affecting conscious versus unconscious visual discrimination with damage to the visual cortex (V1). Proc Natl Acad Sci USA 1995; 92: 6122–6.

Wilbrand H. Ophthalmiatrische Beiträge zur Diagnostik der Gehirn Krankheiten. Wiesbaden: J.F. Bergman, 1884.

Wurtz RH,Yamasaki DS, Duffy CJ, Roy JP. Functional specialization for visual motion processing in primate cerebral cortex. [Review]. Cold Spring Harbor Symp Quant Biol 1990; 55: 717–27.

Zeki SM. Representation of central visual fields in prestriate cortex of monkey. Brain Res 1969; 14: 271–91.

Zeki SM. Functional organization of a visual area in the posterior bank of the superior temporal sulcus of the rhesus monkey. J Physiol (Lond) 1974; 236: 549–73.

Zeki S. A century of cerebral achromatopsia. [Review]. Brain 1990; 113: 1721–77.

Zeki S. Cerebral akinetopsia (visual motion blindness). A review. [Review]. Brain 1991; 114: 811–24.

Zeki S, ffytche DH. The Riddoch syndrome: insights into the neurobiology of conscious vision. Soc Neurosci Abstr 1997; 23: 302.

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