

The neuronal basis of motion perception

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Abstract. The central nervous system of humans supports a range of cognitive functions that contribute to conscious mental states. The neural systems underlying several of these cognitive functions, including perception, memory, planning and action, are proving susceptible to experimental analysis in lower primate species such as rhesus monkeys. In particular, recent investigations have generated striking new insights concerning the neural mechanisms that mediate visual perception. We briefly review the functional organization of the primate visual pathways and describe new experiments that demonstrate a causal link between neural activity in one of these pathways and a specific aspect of perceptual performance. The experiments illustrate an incisive method for linking perceptual abilities to their neural substrates. This approach may prove applicable to the analysis of other cognitive functions as well.

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All visual information reaches the brain in the form of brief electrical impulses carried by the 1.5 million nerve fibres that originate in the retina of each eye. Thus, our unified percept of the visual scene, including the rich array of objects, colours and motions that we routinely and effortlessly see, must be constructed from the fragmented bits of electrical information transmitted through the fibres of the optic nerves. In humans, this remarkable synthetic feat occurs primarily within several cortical visual areas near the back of the brain. Visual information reaches the primary cortical visual area (V1, or striate cortex) from the optic nerves via a relay connection in the thalamus. A great deal of sophisticated processing occurs in striate cortex and information is then transmitted to adjacent ‘extrastriate’ visual areas for further analysis. Near the turn of the century, the functional specialization of posterior neocortex for vision became clear to neurologists, who observed that destruction of posterior neocortex creates a state of near-blindness in humans even though the eyes and retina continue to function normally.

Complementing this observation in dramatic fashion, a number of later investigators showed that human subjects can see small patches of light when electrical stimulation is applied directly to the visual cortex. Fig. 1 shows an X-ray

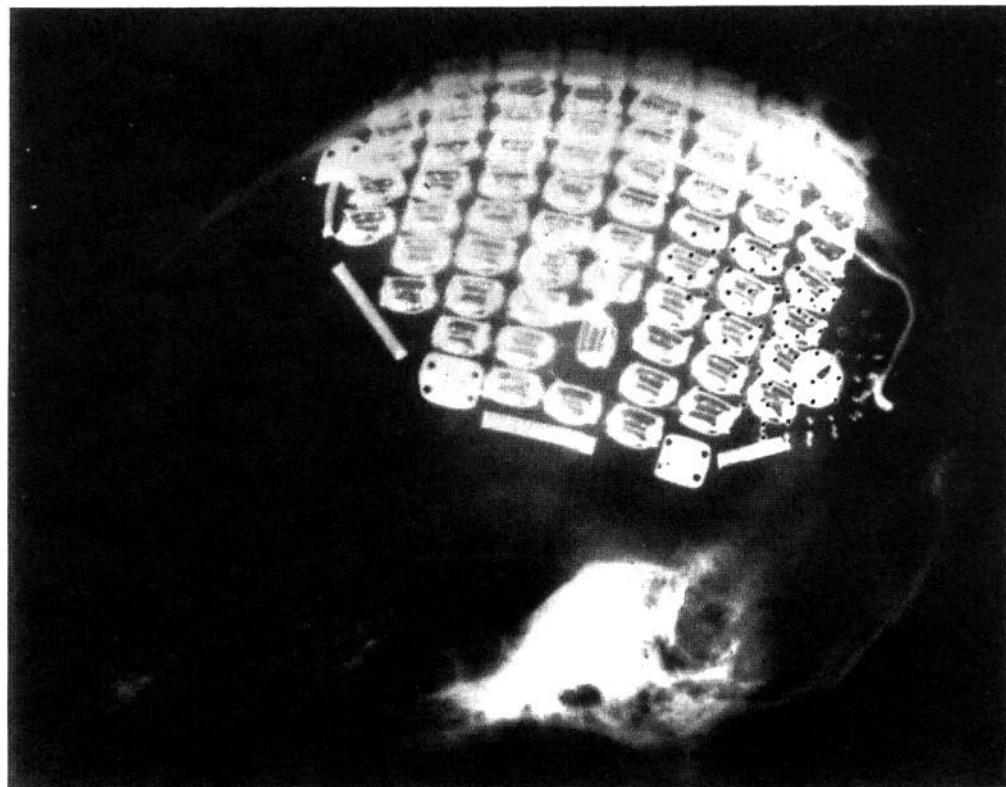


FIG. 1. A lateral X-ray photograph of the electrical stimulation implant used by Brindley & Lewin (1968). The large white objects are an array of 80 radio receivers, embedded in silicone rubber and implanted extracranially. An array of 80 stimulating electrodes (each $800 \times 800 \mu\text{m}$) was positioned intracranially against the surface of the visual cortex using a cap of silicone rubber moulded to fit the contours of the cortical surface. The electrodes appear as an array of tiny dots on the right-hand side of the implant. The shadows of most of the dots were retouched in the original photograph and appear as black dots. Each radio receiver was connected to an individual stimulating electrode by a fine, insulated wire. Radio signals could be pulsed to any receiver by means of a transmitting coil tuned to an appropriate frequency. Pulses were usually $200 \mu\text{s}$, delivered at an overall rate of 100 Hz. Reproduced with permission from Brindley & Lewin (1968).

photograph of an array of stimulating electrodes and radio receivers surgically implanted over the visual cortex of a blind human patient. Brindley and his co-workers in England undertook these experiments to assess the feasibility of developing a prosthetic device that could permit rudimentary vision in persons who were made blind by an injury to the eyes or optic nerves (e.g. Brindley & Lewin 1968); Dobelle and his colleagues have performed similar experiments in the United States (e.g. Dobelle & Mladejovsky 1974). The array of stimulating electrodes, each separated by 2.4–3.4 mm, was placed on the surface of visual cortex on one side of the brain. The stimulating electrodes appear in Fig. 1 as

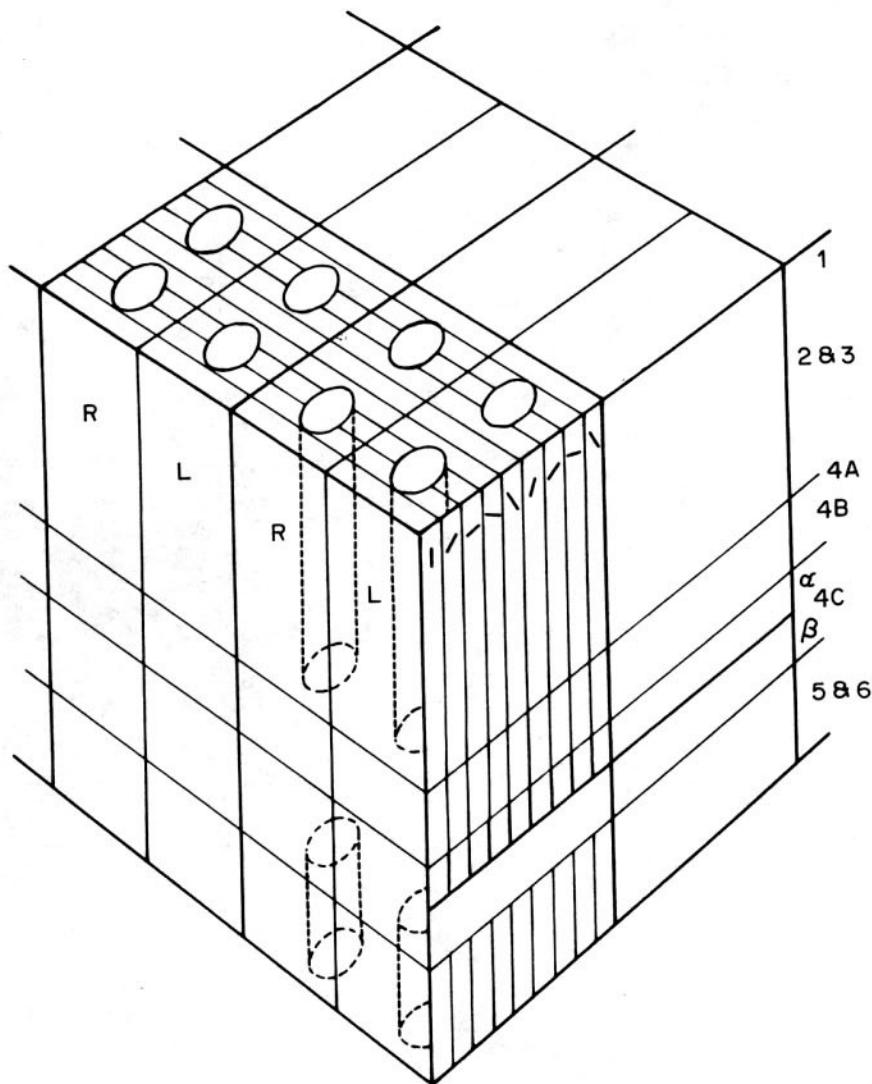
small black or white dots toward the right-hand side of the implant; the large white objects in the photograph are the radio receivers, each of which communicates with a single electrode via a small insulated wire. By activation of the appropriate receiver with a transmitting coil placed over the scalp, visual cortex could be stimulated through any electrode in the array. When stimulated in this manner, subjects reported seeing bright patches of light that were described variously as resembling stars in the sky, small bright clouds, or elongated blobs of light. These visual sensations, or 'phosphenes', commenced when electrical stimulation began and terminated abruptly when stimulation ended.

These experiments showed that visual cortex is not only *necessary* for normal visual experience, but that activation of visual cortex is *sufficient* to generate subjective visual sensations even though no corresponding activity exists in the retina or optic nerve. Besides the potential importance of these experiments for the development of visual prosthetics, the results are of substantial theoretical importance because they demonstrate a causal relationship between neural activity in the cortex and visual sensations that can be consciously perceived and described by human subjects. While the patterns of light perceived in the experiments of Brindley and Dobelle were relatively simple, the data nevertheless raise the enticing prospect of linking more complex percepts to specific patterns of cortical activity. (Penfield and his associates have shown that electrical stimulation with a single electrode can lead to subjective impressions of remarkable specificity (e.g. Penfield & Perot 1963), but the locations of his electrodes suggest that the neural circuits activated in those experiments are related more to memory of past events than to the synthesis of novel percepts).

How might this prospect be realized? One key step is to design experiments that take advantage of contemporary knowledge concerning functionally specialized circuits within the visual cortex. V1 is composed of a large number of processing modules called hypercolumns. Each hypercolumn occupies about 2–4 mm² of the cortical surface. A single hypercolumn contains specialized circuits for analysing contour, colour and motion within a small patch of the visual image. Because adjacent hypercolumns analyse adjacent patches of space, V1 contains an orderly map of the visual field.

Fig. 2, taken from the work of Livingstone & Hubel (1984), illustrates the current conception of the internal organization of a hypercolumn. A slab of visual cortex, like all neocortex, is composed of several layers of cells occupying a total thickness of 1–2 mm. A conventional numbering scheme for the layers is provided along the right-hand edge of the figure. The functional organization of a hypercolumn can be explored by lowering a microelectrode into the cortex and recording the electrical impulses (or action potentials) generated by single neurons in response to visual stimuli projected onto the retina. Experiments of this nature have shown that each hypercolumn is composed of a set of smaller columns with distinct physiological characteristics. For example, each

hypercolumn contains two large slabs (or 'columns') of neurons, each dominated by inputs from one eye ('L' and 'R' in Fig. 2). Running orthogonally to these 'ocular dominance' columns is a set of smaller 'orientation' columns containing neurons that encode information about the orientation of local contours within the visual scene. These neurons are called 'orientation selective' because they respond optimally to an edge or slit of light of a particular orientation; neurons within a single column share a common 'preferred' orientation. As indicated by the small icon at the top of each orientation column in Fig. 2, the preferred orientation shifts systematically from column to column so that all possible orientations are represented within a set of columns occupying 1–2 mm of cortical surface area (for a general review see Hubel 1988).



Specialized circuits for the analysis of motion and colour also exist within the hypercolumn. Layer 4B, for example, is dominated by direction selective cells which respond only when a visual stimulus moves in a particular direction through the region of space analysed by the neurons. Similarly, neurons within the cylindrically shaped columns in Fig. 2 appear to be specialized for analysing the wavelength composition of visual stimuli. Thus, each hypercolumn contains the neural machinery required to process inputs from both eyes and to analyse a particular patch of the visual scene for oriented contours, motion and wavelength.

Given the intricacies of local circuit structure at the level of the hypercolumn, it is not surprising that electrical stimulation yielded relatively crude visual sensations in the human subjects tested by Brindley and Dobelle. Both the electrodes and stimulating currents used in the early experiments were sufficiently large that neurons were probably activated over $1-3\text{ mm}^2$ of the cortical surface, an area that would include all of the elegantly complex local circuits of one or more hypercolumns. On the basis of current knowledge, then, it is tempting to speculate that more refined percepts of orientation, motion and colour could be generated were the local circuits stimulated in a more selective fashion.

Current techniques enhance the prospects of success in such experiments, since very small intracortical electrodes ($20\text{ }\mu\text{m}$ in length as opposed to the $800 \times 800\text{ }\mu\text{m}$ surface electrodes used by Brindley) can be used to characterize neurons physiologically and to stimulate the same region of cortex by passing pulses of

FIG. 2. The current conception of a V1 hypercolumn, taken from the work of Livingstone & Hubel (1984). The diagram illustrates an idealized slab of cortex from V1, showing the orthogonally intersecting systems of ocular dominance columns and orientation columns that constitute a hypercolumn. Neurons within the ocular dominance columns are dominated by inputs from the left eye (L) or right eye (R). Neurons within an orientation column respond optimally to local contours of a particular orientation, and the preferred orientation shifts systematically from column to column as indicated by the small icons at the top of each orientation column. A hypercolumn comprises one set of right and left ocular dominance columns and a complementary set of orientation columns; thus the diagram illustrates two hypercolumns in detail. Each hypercolumn is approximately $1-2\text{ mm}$ wide. The cylindrical structures signify regions of the cortex that stain heavily for the enzyme cytochrome oxidase. In the macaque monkey, these structures are thought to be specialized for analysing wavelength information. All neocortex consists of recognizable layers of neurons, and a conventional numbering scheme for the cortical layers of V1 is illustrated to the right of the diagram. The direct pathway from the retina via the thalamic relay nucleus terminates predominantly in layer 4C of V1, though a few fibres terminate in layer 6 and the lowest portion of layer 3. Neurons in the upper layers send information primarily to other cortical areas. A very high proportion of neurons in layer 4B are direction selective neurons, suggesting that this layer is concerned primarily with motion analysis. Reprinted with permission from Livingstone & Hubel (1984).

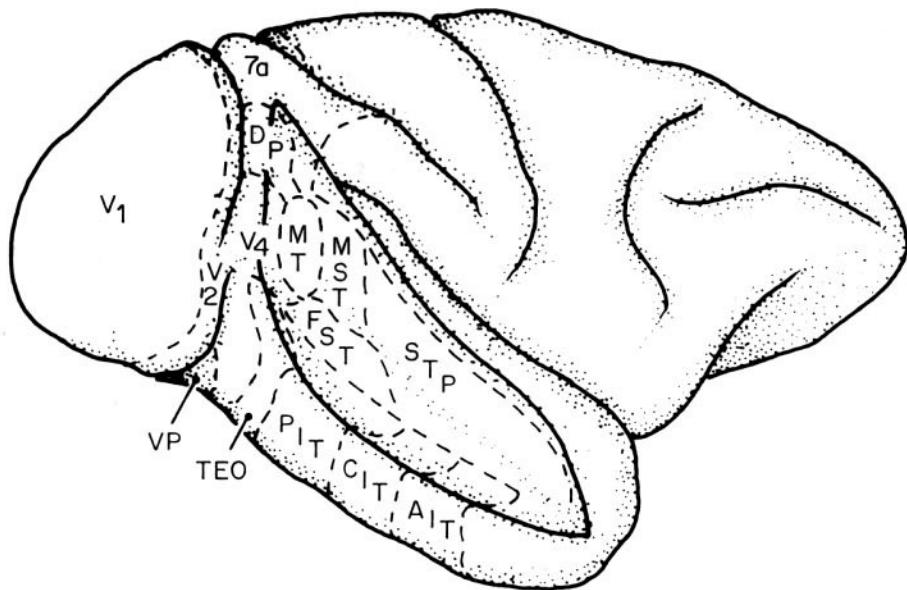


FIG. 3. A lateral view of the cerebral hemisphere of a macaque monkey illustrating the boundaries of currently identified cortical visual areas. Visual cortex in the macaque occupies a large portion of the occipital, parietal and temporal lobes, covering approximately 50% of the cortical surface in all. The primary visual area, V1, is located at the posterior pole of the hemisphere (far left in the diagram) and the middle temporal visual area (MT) resides in the depths of the superior temporal sulcus. This sulcus was artificially 'opened' in the diagram so that the location of MT and neighbouring visual areas could be seen. MT and an adjacent area, MST (medial superior temporal area), are specialized for analysing visual motion information. Other areas buried within adjacent sulci also contribute to the motion pathway. AIT, anterior inferotemporal area; CIT, central inferotemporal area; DP, dorsal prelunate area; FST, floor of the superior temporal sulcus; PIT, posterior inferotemporal area; STP, superior temporal polysensory area; TEO, TE occipital area; VP, ventral posterior area.

electrical current. Furthermore, the experiments can be performed in extrastriate cortical areas where functionally specialized circuits are more accessible than in the intricately organized hypercolumns of striate cortex. We have begun to examine the perceptual effects of applying microstimulation to extrastriate cortex by training rhesus monkeys on visual discrimination tasks so that they can report what they see. The rhesus monkey (*Macaca mulatta*) is an appropriate subject for our experiments because: (1) this species can be easily trained to perform visual discrimination tasks, (2) there exists an extensive base of knowledge concerning the structure and function of its central visual pathways, and (3) the basic perceptual capacities and the structure of its visual system are similar to those of humans.

The middle temporal area and the motion pathway

Fig. 3 depicts a lateral view of the cerebral hemisphere of a rhesus monkey; the figure indicates the boundaries of several cortical visual areas as we now

conceive them. Striate cortex is situated at the posterior pole (far left) of the hemisphere, and visual information from this area is distributed (directly and/or indirectly) to more than 20 extrastriate visual areas in nearby cortex of the occipital, temporal and parietal lobes (reviewed by Van Essen 1985, Felleman & Van Essen 1991).

Several of the extrastriate areas appear to be specialized for processing specific sorts of visual information, but the best example of functional specialization is an extrastriate pathway comprising several visual areas that preferentially analyses visual motion information (Maunsell & Newsome 1987). This pathway was originally linked to motion processing because a large majority of its neurons were found to be directionally selective in the same manner described above for cells in layer 4B of striate cortex (Zeki 1974, Van Essen et al 1981). The best studied area of the motion pathway is the middle temporal area (MT, or V5), which is situated on the posterior bank of the superior temporal sulcus (this sulcus has been 'opened' in Fig. 3 so that MT can be visualized). Fig. 4 shows that MT has a columnar structure for direction of motion similar to that in striate cortex for contour orientation (Albright et al 1984). Thus, a microelectrode that passes vertically through MT typically encounters a sequence of direction selective neurons responding optimally to the same direction of motion. If the electrode passes tangentially through MT, the direction of motion preferred by single neurons changes systematically as the electrode moves from column to column.

The columnar organization in MT allows one to position a microelectrode within a directionally specific column of neurons by advancing the electrode in small steps, recording the responses of clusters of neurons at each step and noting the points of transition from one preferred direction of motion to another. When the electrode is positioned properly within a column, surrounding neurons can be stimulated selectively using weak current pulses ($10\ \mu\text{A}$) whose spread within the cortex is limited to dimensions approximating those of a cortical column (Stoney et al 1968). If a monkey's perception of motion direction is based on the outputs of such neurons, one would expect microstimulation to cause the monkey to perceive motion in the direction encoded by the stimulated neurons.

Microstimulation of the middle temporal area

To examine the effects on perceptual performance of applying microstimulation in MT, we trained several monkeys on a direction discrimination task using operant conditioning techniques. During experimental sessions, a monkey sat in a primate chair and viewed computer-generated visual stimuli presented on a TV screen directly in front of it. The visual stimuli were flickering random dot patterns designed to activate direction selective neurons in the brain. The random dot display could take several forms in which the strength of the motion

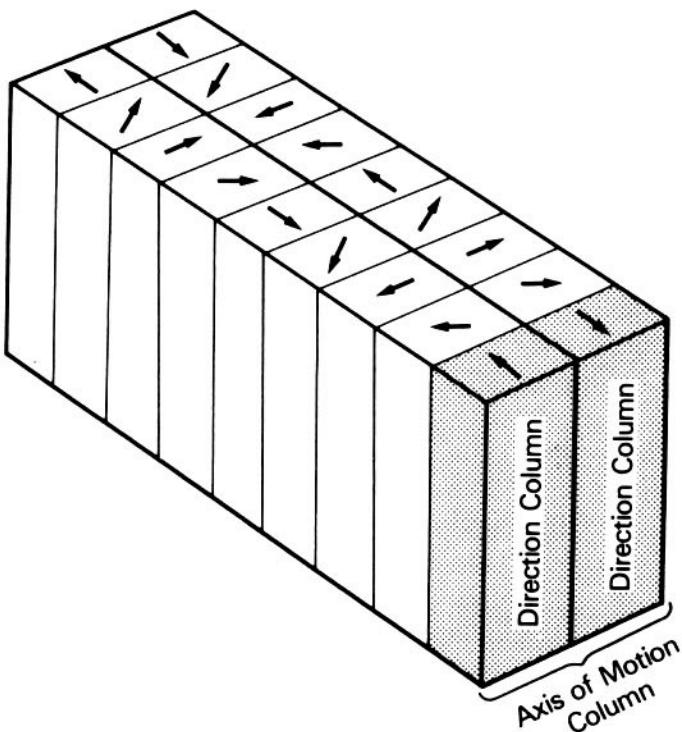


FIG. 4. An idealized diagram of the columnar structure of the middle temporal area (MT) taken from the work of Albright et al (1984). The diagram illustrates the pattern of direction columns within a slab of cortex in MT. Neurons in each column respond optimally to motion in a particular direction, and the preferred direction of motion shifts systematically from column to column as shown by the small arrows at the top of each column. A complete set of direction columns, encompassing all possible directions of motion, occupies 1–2 mm of cortex. Adjacent columns in MT may sometimes have *opposite* preferred directions of motion. In order to introduce a directionally specific signal into the cortical circuitry, therefore, one must restrict electrical stimulating current as closely as possible to a single column. Reprinted with permission from Albright et al (1984).

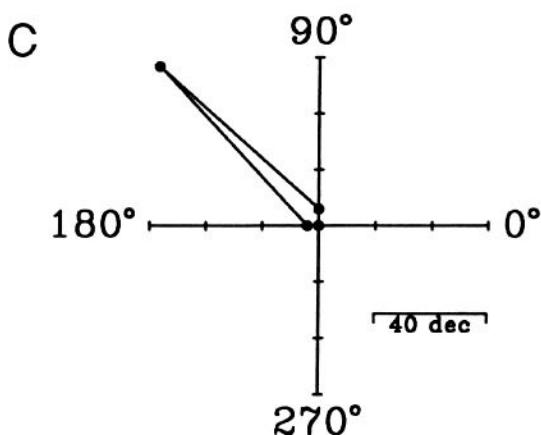
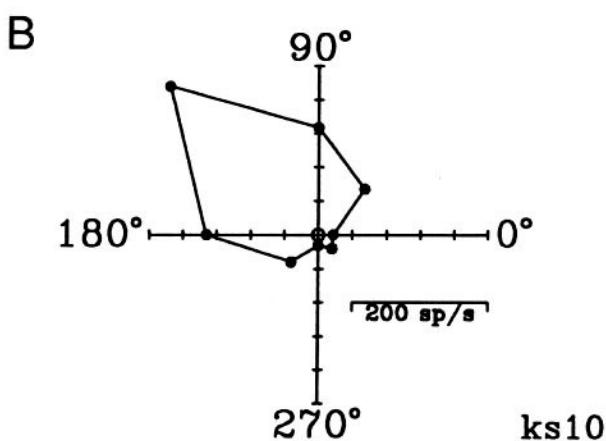
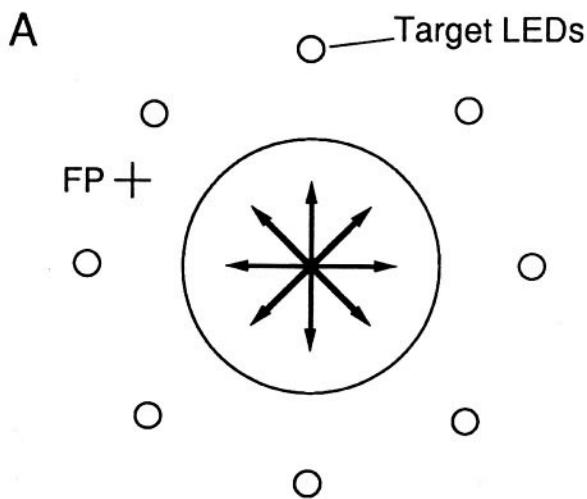
signal in the display was varied. At one extreme, the direction of motion of individual dots was entirely random and there was no coherent motion signal in the display; this form of the display looks like the visual noise on a domestic television tuned between stations. Coherent motion can be introduced to the display by specifying a small proportion of the dots to move in a particular direction while the remaining dots continue to move randomly. In this form, the entire display appears to drift in the direction of the coherent motion signal. If the percentage of dots in coherent motion is low, the global motion percept is weak and its direction of motion is frequently identified incorrectly. As the percentage of dots in coherent motion increases, the percept becomes stronger and its direction of motion is easily identified. Monkeys typically perform as well

as humans at discriminating the direction of motion in the display. This stimulus is ideal for our purposes, because the effects of microstimulation on directional judgements are most obvious when the monkey attempts to discriminate a weak motion signal whose direction is uncertain.

Our experimental paradigm is illustrated in Fig. 5A. On an individual trial, the monkey was required to fix its gaze on a small point of light (FP) while it viewed the random dot display for one second. In the present experiments, the coherent motion signal could occur in any of eight directions equally spaced around the clock at 45° intervals. The dot display was presented within a circular aperture (solid circle in Fig. 5A) that was superimposed on the region of the visual field analysed by neurons at the stimulation site. Thus, the monkey attended eccentrically to the display while maintaining his gaze on the fixation point. At the end of the one second viewing interval, the monkey reported the direction of the coherent motion signal by moving his eyes from the fixation point to one of eight light-emitting diodes (target LEDs) that corresponded to the eight possible directions of motion. The monkey sat within a magnetic search coil apparatus that provided a precise measure of eye position throughout the experiment. Correct answers were detected by the computer and reinforced with a liquid reward; incorrect answers were punished by a brief time-out period between trials. The reward contingencies were the same whether or not microstimulation was applied on a given trial.

We obtained data in blocks of trials in which motion occurred with equal probability in any of the eight directions and over a range of signal strengths (percentage of dots in coherent motion). All trial conditions were presented in random order; the monkey had no basis for predicting the direction or strength of the motion signal on a given trial. Microstimulation was applied on half the trials for each condition (chosen randomly), and the stimulating pulses (10 µA, 200 Hz, biphasic pulses) began and ended simultaneously with the onset and offset, respectively, of the random dot display. In this manner, we attempted to introduce a directionally specific signal into the cortical circuitry that would influence the monkey's perceptual judgements in a predictable way. We assessed the influence of microstimulation by comparing the monkey's choices on 'stimulated' versus 'non-stimulated' trials. (Detailed descriptions of the visual stimuli and our physiological and behavioural methods can be found in prior publications from this laboratory: Newsome & Pare 1988, Salzman et al 1992.)

Figs 5B and 5C illustrate data from one experiment. The polar plot in Fig. 5B shows the visual responses recorded from neurons near the electrode tip before the stimulation experiment began. The visual stimuli in this phase of the experiment were random dot patterns with a maximum strength motion signal (all dots moving coherently) that moved across the receptive field in each of eight possible directions. The angle of each data point with respect to the centre of the polar plot indicates the direction of motion of the visual stimulus. The distance of each data point from the centre of the polar plot shows the intensity



of the neural response recorded for that direction of motion. Thus the 'tuning curve' illustrated in Fig. 5B shows that the electrode was positioned within a group of directionally selective neurons whose preferred direction was up and to the left (135° by laboratory convention). This tuning curve, like those of most MT neurons, was somewhat broad since the neurons also responded well to adjacent directions of motion on either side of the preferred direction.

Fig. 5C illustrates the behavioural data obtained during the microstimulation phase of the experiment. Because we were primarily interested in learning how microstimulation *changes* the pattern of choices made on the direction discrimination task, we subtracted the number of choices made in favour of each direction of motion during non-stimulated trials from the number of choices made for the corresponding direction of motion during stimulated trials. Positive differences from this subtraction reveal the directions that the monkey chose more frequently as a result of microstimulation, and these positive differences are illustrated as a function of direction in the polar plot of Fig. 5C. In this experiment, microstimulation elicited a massive increase in decisions favouring motion at 135° , but little or no increase in favour of any other direction. Two aspects of the data in Fig. 5C are particularly noteworthy: (1) the preferred direction of the 'behavioural tuning curve' corresponds closely to the preferred direction of the visual tuning curve in Fig. 5B, and (2) the width of the behavioural tuning curve is much narrower than the width of the visual tuning curve. Both of these results are typical of the data obtained in 25 other experiments in which microstimulation yielded substantial effects on the monkey's behaviour in the eight-choice task (Salzman & Newsome 1991).

FIG. 5. The results of a recording and microstimulation experiment in the middle temporal area (MT). (A) Methods employed in the microstimulation experiment. The monkey fixated a point of light (FP) and motion was presented in one of eight possible directions (arrows) within an eccentric aperture (large circle). The aperture was positioned so as to cover the portion of visual space analysed by neurons in the stimulated column in MT. The monkey viewed the dot pattern for one second, then revealed its judgement of the direction of coherent motion by making a quick eye movement to one of eight light-emitting diodes (Target LEDs) corresponding to the eight possible directions of motion. (B) Multi-unit recording of visual responses from a directional column in MT. The diagram is a polar plot showing the intensity of response (in total spikes) as a function of the direction of motion of the visual stimulus. This cluster of neurons responded optimally to motion up and to the left (135°), but robust responses occurred for motion at 90° and 180° as well. (C) The results of electrically stimulating the column for which the visual responses are illustrated in panel B. The polar plot indicates the number of choices made by the monkey as a function of direction. The number of choices plotted for each direction is the *positive difference* between the number of choices made on stimulated trials and the number of choices made on non-stimulated trials. Negative differences are not shown. Twenty trials were presented in each of eight directions for both the stimulated and non-stimulated conditions (320 trials total). Microstimulation resulted in an increase of 80 choices toward the LED at 135° , corresponding nicely to the preferred direction of neurons at the stimulation site (panel B).

The consistent correspondence in preferred direction between the two tuning curves shows that the microstimulation-induced changes in the choices made by the monkey can be *predicted* from the visual tuning properties of the stimulated neurons. Thus, microstimulation introduces a directional signal into the cortical circuitry that is interpreted in a meaningful fashion by the monkey as it performs the discrimination task. The disparity in the bandwidths of the two tuning curves is also interesting. The monkey's behaviour suggests that it has access to a very precise directional signal that is computed from the activity of broadly tuned MT neurons. Neurophysiologists investigating motor systems have proposed population models in which precise movements of the eyes and limbs can emerge from the collective activity of broadly tuned neurons in motor regions of the brain (Georgopoulos 1990, McIlwain 1991). A similar population coding strategy for visual motion may occur in MT.

Concluding remarks

The primary significance of these results is that they establish a causal relationship between the activity of direction selective neurons in the cortex and a monkey's perceptual decisions on a direction discrimination task. Clearly, our success in this particular experiment raises the hope that similar links between physiology and performance can be established for other classes of cortical neurons such as those selective for orientation and colour. Our experimental approach, which combines multi-unit physiological recording, electrical microstimulation and an appropriate psychophysical task, may be applicable wherever neurons with similar physiological properties are clustered together in columns or patches within the cortex. This arrangement permits one to stimulate simultaneously enough neurons to elicit behavioural effects without sacrificing functional specificity. Ultimately, however, more sophisticated approaches will be required to identify the neural substrates for complex perceptual capacities that require simultaneous modulation of activity in large numbers of circuits.

Our experiments are similar in spirit to the brain stimulation studies of pioneering investigators like Penfield, Brindley and Dobelle, who established the principle that subjective visual sensations can be elicited by electrical activation of neural tissue within the visual cortex. The present study extends this principle significantly by showing that a specific aspect of perceptual performance can be linked empirically to activity within physiologically specialized circuits. These circuits, though small and intricately organized, can be accessed and manipulated with modern neurophysiological techniques, and the perceptual consequences of this manipulation can be measured quantitatively.

A major distinction between the earlier studies and our own is the difference in experimental subjects. Whereas human subjects can report what they see in a reasonably direct manner (through language), our inferences concerning the

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DISCUSSION

Marcel: I would like to describe one patient, the Munich patient, in whom we know the only damage is in MT and V5. That patient does not see movement; she sees 'a series of stills'. Secondly, if you do selective attention experiments,

where normal people benefit from segmentation by perceived movement, this patient does not benefit from that (McLeod et al 1989). But, if you throw a ball, she will catch it. So it does look as though, for the human, MT is giving phenomenal movement and that on which judgements are based, but actions which are direction sensitive, in the sense of intercepting a ball, can be performed in the absence of a functional MT.

Newsome: Ultimately, it would be highly desirable to analyse the functional roles of MT and other visual areas by studying human patients, and I certainly pay close attention to clinical studies such as the one you mention. It is probably premature, however, to conclude that this patient has a selective ablation of MT. As I understand it, she has damage to cortical and subcortical grey matter in both hemispheres and to white matter tracts as well. The fact that her deficits appear reasonably selective for motion vision certainly suggests that her lesions involve a pathway that is analogous to the motion pathway identified in monkeys. Even in monkeys, however, this pathway is composed of several visual areas, and it is not yet clear whether the patient's lesions actually involve a homologue of macaque MT. For the present, then, I would avoid the strong conclusion that her perceptual phenomenology gives us unambiguous information about the role of MT in motion perception in humans.

Humphrey: A monkey without the striate cortex, and therefore lacking any of these motion-detecting cells in MT, can catch a moving fly. So although I would want to describe that monkey as having blindsight and not having phenomenal experience of seeing movement, it can use movement information.

Newsome: Let me add a cautionary comment concerning the interpretation of lesion experiments. While I believe that lesion experiments are very useful, we must remember that a damaged cortex may invent new ways to solve problems that are not characteristic of a normal cortex. For example, we have shown that a monkey's thresholds on our direction discrimination task were dramatically (and selectively) elevated during the first few days following a complete unilateral lesion of MT. After three weeks of practice, however, the monkey recovered nearly to pre-lesion standards, although some permanent deficit remained. To interpret this result properly, one must remember that MT is only one visual area in an extended cortical pathway that processes motion information. For example, MT sends motion-related outputs to two higher-level visual areas, the medial superior temporal area (MST) and the ventral posterior area (VP), near the juncture of the parietal and occipital lobes. Both MST and VP, however, receive additional inputs from antecedent areas on the motion pathway other than MT, mostly notably V3 and V2. Thus MT may be a critical centre for processing motion signals in the normal cortex, but in a damaged cortex MST and VP may be able to employ inputs from V2 and V3 to accomplish many tasks in a reasonably normal way. This does not mean that MT plays only a minor role in normal motion vision; rather, it indicates that damaged cortices may be very resourceful in developing new ways to solve sensory problems.

monkey's visual experience are less certain. It remains possible, for example, that electrical microstimulation of MT influences what the monkey *decides* on a given trial without influencing what it *sees*. Several aspects of our results, which are beyond the scope of the present publication, suggest that the primary effect of microstimulation in MT is to affect the sensory representation of the stimulus rather than the 'decision process' which presumably operates at a higher level of cortical processing (Salzman et al 1992). Thus, our results are highly suggestive of a causal link between neural activity in a physiologically classified set of neurons and a particular mental process (the perception of motion), but data confirming this link cannot be obtained until equivalent experiments can be carried out in human subjects.

While our efforts are directed towards understanding the neural mechanisms underlying visual perception, other neurophysiologists are producing exciting insights into the neural substrates of cognitive functions such as memory, attention and motor planning (e.g. Moran & Desimone 1985, Miyashita & Chang 1988, Georgopoulos et al 1989, Barash et al 1991, Corbetta et al 1991, Maunsell et al 1991, Miller et al 1991). Such 'bottom-up' approaches to cognitive function will complement and extend the 'top-down' approaches of cognitive psychologists and philosophers. Ultimately, this combination of efforts may provide a clearer understanding of the self-aware and reflective mental state that we refer to as consciousness. As will be clear from other papers in this volume, it is arguable whether our most incisive approaches and our best efforts will ever yield a completely satisfactory account of our own conscious states. Nevertheless, it seems both a prudent and enjoyable goal to push these approaches as far as they will take us.

Acknowledgements

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Nick Humphrey has described a most interesting case (p 160), that of the monkey, Helen, who has a complete lesion of striate cortex yet can perform many visually guided tasks in a surprisingly normal way. I understand, however, that it took this monkey many weeks, if not months, to develop these behaviours after the lesion, a period in which a great deal of reorganization could take place. In some ways, then, a visual system that has had a great deal of time to recover from a lesion may represent a novel system that informs us more about neural plasticity than about normal visual function.

Gray: In the microstimulation experiment, the stimulation was applied on half the trials and you said that the animal was not rewarded on the stimulated trials. This puzzles me, because it means that the monkey was on a 50% reinforcement schedule. Did you see any behavioural reactions on the part of the animal that would distinguish his response on trials when he was rewarded or not rewarded?

Newsome: The animal is always rewarded for a correct choice, regardless of whether it's a stimulated trial or non-stimulated trial.

Gray: When you say correct response on the stimulated trial, the stimulation on those trials is different from the display?

Newsome: That's right. We rewarded him for detecting what was on the display.

Gray: From his point of view, that could be non-reward for a correct response.

Newsome: You raise an important point, and I want to address it in two parts. I agree that on stimulated trials the monkey is sometimes not rewarded for what is a correct response from his point of view. We were concerned at first that such trials might essentially 'de-train' the animal and cause him to answer in unpredictable ways. This seems not to be the case, however, for a couple of reasons. First, stimulated trials are randomly interleaved with non-stimulated trials, and the monkey is therefore rewarded correctly for veridical reports on half of the trials in a block. Second, the absence of a reward for an answer that was 'correct' from the monkey's point of view is precisely what happens to animals who work on a variable reinforcement schedule, and such reinforcement schedules do not appear to exert untoward influence on an animal's choice strategy.

The second thing I want to point out is that microstimulation occasionally gives the monkey extra rewards because it causes him to answer correctly on a trial on which he might otherwise choose incorrectly (if, for example, the display contains a weak motion signal in the preferred direction of the stimulated column of cells). The frequency of these two events—stimulation causing a 'mistake' and stimulation causing a 'correct' answer—is about equal in our two-alternative, forced-choice task, and the overall reward rate remains fairly stable throughout a block of trials.

In the eight-alternative task, however, the frequency of induced mistakes is greater than the frequency of induced correct responses, and the overall reward

rate on the stimulated trials decreases. We were concerned for a while that the monkey's motivation might deteriorate because of the drop in reward rate, with the unpleasant prospect that our data might be compromised. This seems not to be the case. The monkey continues to perform in a motivated fashion throughout the microstimulation experiments, and his accuracy on non-stimulated trials remains quite good. It helps in this context to realize that random performance in the eight-choice task would be 12% correct (an abysmal percentage), so the monkey has a lot to gain by performing in a motivated fashion, even if microstimulation causes a modest decrease from his usual reward rate.

Gray: Have you looked at the following two aspects of your data from this point of view? If the stimulus that you are providing is exactly like the real stimulus out there in the world, you might expect the monkey, on those trials where he's responding, from his own point of view, correctly but not getting rewarded, to show some signs of surprise. I would think in a monkey this would be easily detectable. Conversely, if the stimulation you are providing is very similar to what's coming in from the real world, but discriminably different (I suppose a more plausible hypothesis), you might expect him, over a course of trials, to begin to learn the difference. In other words, you might get a trend over trials for the monkey to perform better at the beginning than at the end. Did you see either of these two phenomena in your data?

Newsome: To answer your first question, I have not noticed subjective responses related specifically to stimulation trials. Monkeys sometimes become frustrated when they fail to get rewarded on a trial, as evidenced by a vocalization or by a sudden movement in the primate chair. (There are New York monkeys and California monkeys—New York monkeys hate to miss a single trial whereas California monkeys take a much more relaxed approach!) But I have not noticed an increase in such behaviour on stimulated trials relative to non-stimulated trials.

Your second point is an excellent one, and we were very concerned about this possibility at the beginning of the experiments. Because the monkey experiences a decrease in reward rate on stimulated trials, he might be motivated to develop a different discrimination strategy on the stimulated trials in order to return the reward rate to normal. If, as you suggest, the monkey can subjectively distinguish stimulated from non-stimulated trials, he might compensate for the effect of the stimulating current by adjusting his criterion for reporting preferred direction motion. In other words, the monkey might insist on an unusually strong sensation of motion before he is willing to report the preferred direction. If the monkey adopted such a strategy, however, we would expect our microstimulation effects to disappear with time as the monkeys became more adept at recognizing stimulated trials and applying the new strategy. We have now run three animals for 6–18 months on this experiment, and we have observed no such decrease in the frequency or quality of the

stimulation effects in any of the three animals. We believe, therefore, that the monkey cannot subjectively distinguish stimulated from non-stimulated trials, and that the existence of occasional trials with incorrect reward contingencies (from the monkey's point of view) has no significant effect on our results. More generally, it is comforting to realize that large microstimulation effects are obtained despite an overall decrease in the monkey's reward rate. If the monkeys received an increased reward rate for generating the result we wanted to see, I would suspect an artifact and probably lose considerable sleep over it.

Gray: Why did you choose the stimulus parameters that you did? *A priori*, you could have equally well made the exact opposite hypothesis. You could have said: 'If I stick an electrode in the middle of the right column and I put on a burst of high frequency stimulation, I'm going to disrupt whatever firing of those neurons would count as normal, and the system rather than use information from this column would actually stop using it.' Did you choose your stimulus parameters in the light of some knowledge of the normal firing patterns of those neurons?

Newsome: The stimulation parameters were chosen carefully. We decided on 10 μ A current pulses because a prior study in the motor cortex of monkeys by Asanuma and colleagues had demonstrated that pulses of this size would directly activate neurons within about 85 μ m of the electrode tip (Stoney et al 1968). This distance is roughly similar to the width of a cortical column, and we therefore hoped that the 10 μ A pulses would activate a small number of columns in a fairly selective manner. We selected 200 Hz as our default stimulation frequency because 200 Hz approximates the largest firing rate we have seen from MT neurons in response to our random dot patterns; we wanted a frequency that would be physiologically plausible yet give us the best chance of seeing an effect.

We have recently explored the effects of changing current amplitude and frequency. We can obtain significant microstimulation effects for current amplitudes as low as 5 μ A or with stimulating frequencies as low as 25 Hz. Interestingly, the 5 μ A figure compares favourably with current thresholds for eliciting muscular contraction from stimulation in motor cortex, superior colliculus or frontal eye fields; it is a very small amount of current. The fact that 25 Hz pulses can influence the monkey's choices shows that effects can be obtained with frequencies that are well within the normal range of firing rates for most MT neurons. If we stimulate with large current pulses—80 μ A, for example—the directional bias in the monkey's choices disappears and a simple reduction in psychophysical sensitivity (increased thresholds) occurs. The most likely explanation for this is that large current pulses activate a large array of columns encoding divergent directions of motion. Thus, the large current pulses degrade the specificity of the directional signal within the cortex and simply impair the monkey's performance on the direction discrimination task.

Your final point is quite interesting. There seemed little reason *a priori* to believe that stimulation with an extracellular microelectrode would do much more than disrupt the normal pattern of activity within a column and thus impair the monkey's performance. Cortical columns are complex processing units whose normal workings seem to involve a delicate interplay between excitatory and inhibitory inputs of multiple origins. It is remarkable to me that our relatively crude intervention with a single stimulating electrode can have large and coherent effects on the animal's behaviour. Perhaps a motor physiologist would tell us that all of this is old hat—that there is no more reason to be surprised at this result than there is to be surprised that single muscles twitch in response to microstimulation of motor cortex. But I am surprised anyway. In this context, it is probably important to remember that not all of our experiments yield positive results; we probably have about a 60% success rate overall. It may be that some of the failures result from the fact that the microstimulation sometimes shuts down the outputs from a column, perhaps by activating inhibitory circuits, rather than exciting it.

Harnad: What about selective inhibition by stimulating the MT column in one direction while the sensory stimulus is in the opposite direction?

Newsome: That situation is built into the task I described. Motion can appear in any of eight different directions and we apply stimulation to a single column during each condition. At some time during the experiment, therefore, visual stimulus motion is going downward while we are stimulating a column that signals upward motion. This creates the condition for a very interesting experiment in which we attempt to 'null' the directional signal created by stimulation of the 'up' column by gradually adding a stronger downwards signal (i.e. more correlated dots) to the visual stimulus until the monkey acts as if no motion at all is present in the stimulus (chooses either direction with equal probability). This allows us to quantify the strength of the microstimulation-induced signal by expressing its worth in units of the visual stimulus—percent correlated dots. In our largest effects, microstimulation can be 'worth' 50–100% correlated dots, an intensity that is well above the psychophysical threshold for discriminating motion direction. For many experiments, however, microstimulation is 'worth' only 3–7% correlated dots, a value that is near or sometimes below the psychophysical threshold.

Marcel: Do you observe streaming effects? You are saying that a receptive field detects dots going in non-coherent directions and in some way sums the effect to determine which way it responds. But when we consciously see the moving dots, we are aware of streams. I see a stream of dots going one way and a stream of dots going another way. It's not that I see the sum of the different streams as being movement in a particular direction, I see movement in different directions. The interesting point is: do I see movements in two different directions or do I see movement in one direction on a background, i.e. is there a figure-ground effect?

Newsome: That is an important question, but I don't know the answer to it. The monkey cannot describe to me what he sees, and I therefore don't know what it is 'like' to be the animal and be stimulated.

Marcel: Alan Cowey has proposed ways of asking monkeys, for some things, a 'what it's like' question. One example is to use the animal's choice of a reinforcement schedule; for example, to ask whether a monkey has blindsight or not you allow the monkey a choice. You train the monkey such that, if it makes no choice whatsoever it will get 75% reward. If the monkey chooses correctly, it will get 100%; if it chooses incorrectly it gets 50%.

The question is whether the monkey is experiencing something or not. The monkey can choose on the basis of how confident it is that it is seeing something, or it can choose just to receive a reward where it doesn't have to base a choice on a visual discrimination.

Newsome: There may be some approaches along those lines that we should think about. Ultimately, though, I am very sceptical about our ability to know what a monkey's internal sensory experiences are like, especially ones that are caused by 'unnatural' intervention such as an experimenter's electrode. The most satisfactory approach would be to side-step this problem by doing similar experiments in humans. If one could reproduce the behavioural result in a human, and the human in addition responded, 'By golly, I see motion upward when you stimulate', the thorniest difficulties of subjectivity would be circumvented to my satisfaction. This is not altogether a pipe dream, because electrical stimulation experiments are sometimes done with the cooperation of conscious humans who are undergoing surgery for treatment of epilepsy or other neurological conditions. I would want to have a very good idea where MT is in a human before trying such an experiment, however.

Having expressed my reservations, I will say that I believe the monkey probably experiences a sensation of motion during large stimulation effects. Perhaps the best way to think about this is to consider a perceptual illusion called the motion after-effect or waterfall illusion. If you stare at a waterfall for 30 seconds or so and then look at the cliff face to the side of the water, you get a compelling sensation that the cliff face is moving upward at a slow rate. This occurs even though you know cognitively that the cliff cannot possibly be moving. This is a very revealing observation because it shows that motion can be computed independently in the brain and, in essence, 'assigned' to objects in the environment that may not be moving at all. I suspect that something akin to this may be going on when we stimulate MT. The monkey may 'see' motion by attributing this internal sensation to the dots on the screen. If this is the case, that subjects undergoing microstimulation actually see coherent motion, I would feel justified in saying the brain pathway being stimulated plays an important role in conscious awareness.

Kinsbourne: When you stimulate the columns, do you get eye movement?

Newsome: No, microstimulation in MT seems to have no direct effect on motor circuits that control eye movements. If we apply microstimulation during the intertrial interval (for one second at 200 Hz, just as we do during the actual experiments), we do not elicit eye movements of any kind—saccadic, pursuit or other. In the context of our experimental paradigm, of course, microstimulation in MT ultimately affects oculomotor neurons, because the monkey must report the outcome of his decision with an eye movement, but that influence is indirect. In order for microstimulation in MT to affect the oculomotor system, the monkey must be performing the experimental task.

Kinsbourne: If you are getting phenomenal movement in one direction and maintain it, why would you not get optic nystagmus?

Newsome: We don't see optokinetic nystagmus in the normal experimental situation or during microstimulation. Remember that the monkey is required to fixate a stationary point and attend to the motion stimulus at a restricted peripheral location. If the motion stimulus is presented over the fovea, we do see optokinetic nystagmus, but when it is presented peripherally we generally do not.

Kinsbourne: You could set the experiment up so that the monkey should 'see' streaming in one direction; then you should get optokinetic nystagmus.

Harnad: This point is again related to hermeneutics. The reason I left the field of laterality (Harnad et al 1977) was the disproportionate importance assigned in that field to what look like trivial data. Consider, for example, a 50 ms speed advantage that may occur when a stimulus is presented in the left rather than the right visual field in some perceptual task. If the same 50 ms advantage had instead been exhibited in the running speed of one group of kids compared to another, the effect would rightly be dismissed as trivial. But when such differences can be annotated with the mystique of being related to 'the brain' in some way (even something as vague as being on the left or the right), their significance and explanatory power are immediately elevated by interpretations (in terms of grand left-brain/right-brain theories) vastly out of proportion to their actual empirical content.

By way of analogy, suppose that instead of being recorded from the brain, Bill Newsome's findings had been read off an oscilloscope stuck into a simple artificial optical recognition system that was capable only of detecting orientation; and suppose one found in this system a unit that was selectively responsive in just the way Bill has described. One would not feel one had made an inroad on the mind–body problem. So why should one feel that one has done so when one happens to find this in the brain?

I am not suggesting that Bill's finding is trivial as *neurophysiology* (as some of the laterality findings are). But it certainly does not seem to cast any new conceptual light on the mind–body problem along the lines Tom Nagel has here expressed optimism about eventually achieving, as we get closer to scientific Utopia.

compared and the decision is made in favour of the direction yielding the largest signal. An appropriate operant response is then planned and executed.

Interestingly, we know a lot now about the brain centres that encode direction of motion and about the brain centres that plan and programme saccadic eye movements. The real mystery is the decision process between the two that links sensation to action. In some sense then, that may be the most important part of the network you are asking about—this downstream comparator that selects one of the directional signals and links it in an appropriate manner to a motor response. This machinery must lie somewhere between MT and the oculomotor system, and we know roughly the anatomy linking the two. By recording in these linking structures while the monkey performs the discrimination task, we may be able to get some hints about the physiological structure of the decision process. This is perhaps the most important sequel to the present experiments.

Kihlstrom: How do you think that your results contribute more than the inferences that can be drawn from the earlier Hubel & Wiesel type of experiments?

Newsome: I would say that the key contribution of our experiments is to establish causality. We not only show that a certain type of neuron is impressively correlated with a particular aspect of perceptual performance, but that manipulation of that type of neuron *causes* predictable changes in performance.

Kihlstrom: Do you have any qualms about the Hubel & Wiesel experiments that you are still not satisfied about on the basis of your present experiment?

Newsome: Sure. Establishing an empirical link between direction selectivity and motion vision does not accomplish the same for orientation selectivity and form vision. For years, physiological (and, increasingly, computational) studies have been based on presumed connections between cortical cell types and perceptual experience. In principle, our experimental approach can be extended to these other cell types as well.

Humphrey: I would like to describe a similar situation that arose in ethology in relation to the dance of bees. Von Frisch originally noticed the correlation between the speed of wagging and the distance of the food source from the hive. It was assumed that this information was being used in a communicative way. Then some critics said that although there is a correlation, the bees don't use this information, they actually use other cues. It wasn't until people made a little mechanical bee that could waggle, and showed that they could manipulate the foraging behaviour of other bees which had observed it, that people finally became convinced that Von Frisch's conjecture is correct.

I think exactly the same thing arises with the results of Hubel & Wiesel. It looked as though all the information was there in the selective responses of individual cells, then people like Fergus Campbell said: 'The information is there, but it's not being used'. Experiments like Bill Newsome's really do suggest that the information is being used. This is very important.

Searle: All the stuff that comes in through the lateral geniculate nucleus to the cortex. Furthermore, how does the rest of the information in the cortex get into visual experience? The fascinating thing about vision is that you don't just see a body moving but, for example, you might see a Mercedes 300 driving down Portland Place. That is, in fact, how I would characteristically see something. But I don't have a special Mercedes detector in my brain. If I don't have a Mercedes detector and yet I can see a Mercedes, there must be a whole lot of information elsewhere in my brain that is being used directly in the formation of visual experiences.

Newsome: I agree that the problem of integration is one of the most important and difficult ones facing visual neuroscience. This, however, is not the problem being addressed in our experiments, and we have done everything possible to avoid that problem. We have designed the task so that the monkey can perform it simply by using information that is encoded in those columns in MT. By using a stochastic stimulus, we tried to eliminate any extraneous features or position cues that might provide the monkey with an alternate strategy for solving the problem. Thus, we are really trying to tap directly into the brain mechanisms that 'do' motion. Now, if we changed the task so that the monkey was required to discriminate a red square moving upward from a green triangle moving leftward, all hell would break loose. Then we would have an integration problem. While we are making no attempt to address this problem in our current experiments, I certainly agree that a satisfactory visual science will ultimately require a solution to the problem.

Gazzaniga: If you make a microlesion in the cell that you are affecting, my guess is that you would not see a raised threshold in that part of the visual field. This would suggest that your stimulation is sending output to some other critical structure that's actually doing the computation that's assisting discrimination.

Newsome: I suspect that a microlesion of the column we are stimulating might raise thresholds very transiently, but I don't even know whether the impairment would last long enough for us to measure it well. I believe there exist other columns in MT and other visual areas as well that can pick up the load if any single column or group of columns is damaged (we actually have evidence to this effect from MT lesion experiments).

Certainly, information must emerge from MT and affect other structures in order for stimulation in MT to influence behaviour, and we have little idea at present how large that network of affected structures might be. The question is important because its answer would ultimately include a description of the processes by which sensory signals emerge from MT, decisions are made regarding the direction of motion, and operant responses (eye movements in our case) are programmed and executed. We think loosely about decision processes in the following fashion. If a monkey is performing an up versus down discrimination, a measurement of neuronal activity is made from a group of 'up' columns and a group of 'down' columns. These two measurements are

Newsome: I don't know, I'm asking you! This is one reason I would like to do these experiments in humans. If the human reported, 'I see motion' and it impinged upon their conscious awareness and their subjective experience of who they are, then I would feel I had done something to affect the stream of awareness that we call 'mind'.

Nagel: But it must have some effect on the experience. Wouldn't the question be: what are the options? I suppose the stimulation could affect the monkey's choices, not by affecting its visual experience but by somehow affecting, as it were, the inference it makes about what is actually going on from its visual experience. Are you sceptical that these results show that the stimulation affects the monkey's experience at all?

Newsome: The stimulation certainly affects the monkey's behaviour; it affects the choices he makes very dramatically. I don't know whether the monkey feels that it saw up and reported up but we didn't reward him.

Searle: It seems to me, Bill, that you have some legitimate hesitations and I think that's commendable. But you are perhaps over-cautious with some forms of scepticism. Let me give a blunt commonsense, philosophical answer to your question. We have pretty good evidence that it's because of the impact of the stuff that comes in through the eyeball on the rest of the brain that we have visual experiences at all. We also have pretty good evidence that the visual cortex plays a special role in the production of visual experience. So I think you are too cautious, if you suppose that you really don't know anything about the vision of the monkey on the basis of this research. I think the research is terrific and I think it is exactly the kind of thing we need to know in order to understand vision.

But there are some dangers. The greatest danger is, roughly speaking, that we may be committing a combination of the homunculus fallacy and the atomistic fallacy. It's ironic that something very like this discussion took place in this very room when we had a Ciba Foundation symposium on Brain and Mind in 1978. Creutzfeldt gave a display of the pattern of firings exhibited by the Hubel-Wiesel cells in the visual cortex of a bird. When he projected a picture on the screen of the pattern of individual neurons that were firing, the atomistic points on the screen made a pattern that he thought was a representation of the bird's visual experience. It seems to me that he was making two fallacies. One was the fallacy of supposing that the pattern of individual nerve firings adds up to a picture that is seen. The second was the tacit assumption that there must be some little homunculus inside the brain that looks at this pattern of neural firings. Those are very tempting fallacies, but I don't think you are making either of them. So, if we can avoid these fallacies, what is the next step? Obviously, we want more research of the type that you are doing, but the next hard question is: how does the brain integrate all of this into a conscious visual experience?

Newsome: 'All of this' meaning . . . ?

Newsome: The information is indeed being used and in a manner that fits our intuition. After seeing these data for the first time, Torsten Wiesel commented to me, 'It is a lovely experiment, but I could have told you the answer before you did it'!

Searle: This is the answer to Stevan Harnad's scepticism. Of course, you can build an artificial system that will detect movement. You can, for example, put a unit into the system that is sensitive to lateral motion. That shows that it is *possible* that the brain works this way. Bill Newsome has given us good evidence that there is a part of the system that actually *is* responsive in the way he describes.

Marcel: If you don't have visual cortex on one side, it does not prevent you from having visual experience in the corresponding area of experienced visual field. That doesn't mean that area doesn't play a part in visual experience, it just means it's not necessary. There are visual hallucinations in cortically blind subjects where you know from the evoked potential of the cortex and from scanning that that area of cortex is dead. This is very important for interpretations of the sort that you are interested in—of what the functional role is, at least in experience, for certain areas of cortex.

Boden: John Searle asked earlier, what would it be like to understand the mind–body relation? He said it would be to have a neurophysiological theory which told us that when such and such happened in the brain, something must go on in the mind. What is the nature of this 'must'? And what sorts of things could offer it to us? Clearly, a mere correlation isn't enough. Descartes himself would have said that when you have the experience of something occurring in a certain way, there's something happening in your brain different from when you experience something occurring in a different way. If by 'must', you mean 100% reliable regularity, Descartes would have been perfectly willing to say 'must', but he wouldn't have said it was in any sense intelligible.

There needs to be some sort of mapping, not just temporal correlations of events in the two spheres, but some sort of shared abstract logical structure, like in Bill Newsome's columns in the visual cortex. Not only are the physical motion directions apparently stored in particular groups of cells in the brain, but the relationships between the different orientations and the cells in this part of the brain are themselves mapped in a very systematic way. If you have that sort of abstract relationship, as well as a mere correlation—these cells fire when we see that—then perhaps you are nearer to something which one might call an intelligible 'must', rather than what you might call a brute fact 'must', with which Descartes would have been perfectly happy.

As another example, Crick & Koch (1990) in their paper about consciousness talked about the binding problem with respect to vision. Their theory was that what makes us bind different visually discriminable properties to one and the same thing, and what makes us have a conscious percept in which these things are in some sense unified, is that the different cell assemblies in the parts of

predicts and explains. The quantum puzzles, on the other hand, are still a source of frustration and perplexity to most physicists (and philosophers) who give them any thought. The puzzles are certainly not the triumphant aspect of quantum mechanics. Rather, one reluctantly reconciles oneself with them in exchange for quantum mechanics' enormous empirical power. Physicists would no doubt be delighted to jettison all the quantum puzzles (duality, complementarity, uncertainty) for a Grand Unified Theory with all the power of quantum mechanics but none of its paradoxes. Those who would like to import those paradoxes alone to another field are trading in the weaknesses of quantum mechanics rather than its strengths.

Nagel: Margaret Boden's suggestion that the discovery of isomorphisms is an essential step on the route to intelligibility must be right. But, it raises the following question. Bill Newsome's results are very specific; of course, anything one is likely to get at this stage is going to be very specific. But even something as specific as motion perception, I would think, can exist only as part of a more global visual experience. So there is always going to be a problem with the interpretation of highly specific results like this. Bill, do you feel that a large background must be assumed to make sense of the connection that you have found? Or do you think we could do it point by point and get the large, more global picture by construction out of the points? I'm prompted here by Pat Wall's more global orientation.

Newsome: This specific experiment emerged from a substantial background of work and thought about the way the visual system is organized and might function. That background, however, is not particularly arcane. With the assistance of a few diagrams on a napkin, I can explain the key concepts to an intelligent layperson in about half an hour. One must accept (at least as a working hypothesis) that the primary function of a brain is to mediate behaviour and that this task is accomplished by normal rules of cause and effect. Given this intellectual (philosophical?) commitment, everything else that led to our experiments is empirical. We have formulated no fundamentally new concepts here. We have put together several traditional physiological ideas—receptive fields, topographic maps, cortical columns, direction selectivity—in an experiment that brings them to life and validates them in a compelling fashion. The power of the experiment comes from the repeatable changes in behaviour that follow a fairly precise manipulation of a neuronal system. This is not dissimilar to the situation in molecular biology where the power of an experiment lies in the changes in the amino acid composition of a protein following manipulation of the genome. In systems neurobiology, however, the ultimate test of a linking hypothesis lies in the realm of measurable behaviour rather than in protein sequences!

Searle: Remember the bunch of lines that we see as human face (Searle, this volume, Fig. 1). The stimulus doesn't literally look like a human face. The implication is that the contribution of the brain to the visual perception is enormous. I think that is part of what Tom was getting at.

the brain which independently respond to these particular properties come to oscillate together. Are they just saying something that would in no way have impressed or surprised Descartes? Or are they trying to say something more than that? Something with the same sort of logic Descartes himself used when he said that unity of consciousness and the senses and so forth must be in the pineal gland because it is in the centre of the body, it isn't doubled, etc. If we are ever to get this intelligible 'must' in our theory, it will have to be via some shared abstract structural properties of that sort, rather than mere correlation.

Gray: You are absolutely right. Can I set the standard one notch higher? In a sense, as Max Velmans has emphasized, we have the problem of putting together two sides of the dual aspect problem. We could call this putting together the third- and first-person perspectives. I much prefer the way John Searle approaches the problem—that is, putting together the neurophysiology on the one hand and the conscious experience on the other.

The big example we have of a dual-aspect theory that works is the duality of particle and wave in quantum mechanics. That is an abstract theory which not only works and produces an 'intelligible must', but comes up with a whole lot of predictions that could not have been made without the theory. The problem we face at the moment, in not having such a theory of consciousness, is that we can work only at the level of refining these kinds of correlations. Experimentally, we have gone beyond correlations—the beauty of Bill Newsome's experiment, which impresses me enormously, is precisely that it does go beyond correlation and into causation. But even given causation in experimental terms, there is still only the brute fact of being able to say: action here produces conscious experience of apparent motion, and action somewhere else produces apparent colour experience or whatever, and action in yet a third place, in the superior colliculus or in the optic tectum, has nothing that looks like a conscious correlate. The gathering of these kinds of data must be a major empirical issue for a long time to come. From them, one supposes that the conditions will gradually emerge which will facilitate the creation of the kind of theory that Margaret Boden has just been talking about.

Humphrey: I think the example you have just given is a very unfortunate one. The wave and particle aspects of matter are not logically isomorphic in the way that Margaret Boden seems to be asking for. As I understand, she wants a theory which says that the physiology has certain logical abstract properties and the phenomenology has the same properties, therefore they go together. That would be wonderful and would get towards what Tom Nagel describes as an objective phenomenology. But the example you gave, Jeffrey, of dual aspects where matter can be both wave and particle at the same time, doesn't seem to involve any such isomorphism. It just happens to be a brute fact of nature.

Harnad: In any case, puzzles are not resolved by further puzzles. The strength of quantum mechanics is the broad range of empirical data it successfully

Newsome: I am fully committed to the idea that perception is a constructive process. I think the Gestalt psychologists demonstrated this well. Most of the time the brain's construction is fairly accurate and we operate well within our visual world. At other times the brain makes mistakes, as we know from various perceptual illusions. We would very much like to know how this constructive process works in physiological terms, and there are a few laboratories around the world that are actually addressing this problem. The work of Rudiger von der Heydt and Esther Peterhans on the responses of visual cortical neurons to illusory contours is one example. A satisfactory visual neuroscience must certainly account for these phenomena in the long run.

Lockwood: Could I say something about isomorphism before we lose this point? I was studying psychology at the time the early Hubel & Wiesel work was published. This established the existence of cells in the primary visual cortex that fire preferentially in response to bars and edges with a particular orientation, and certain specific directions of motion. A friend of mine, who was a philosopher, told me how marvellous he thought this work was. He said: 'That's exactly what vision feels like'. This remark struck me as a perfect statement of what I did not feel about Hubel & Wiesel's work!

The trouble with this notion of isomorphism is that it's very difficult to get a firm grasp on what kind of isomorphism is required here. There's a sense in which anything that could be regarded as holding all the information that is present in one's phenomenology could be said to be isomorphic with the phenomenology itself—there would be, after all, a systematic mapping from one to the other. But it seems to me implausible to suppose that just any such isomorphism would be good enough. Suppose that when we looked at the brain, it turned out to be a digital computer, and what corresponded to any particular phenomenological state was just a bit string. There would be one sort of bit string that tends to come up, perhaps, in the auditory cortex, another type in the visual cortex. All the information would be there. Yet this does not seem to work as an explanation of the phenomenology and the differences that are to be found within it. Appealing to different arrangements of ones and zeros does nothing whatever to render the phenomenology, and the differences within it, rationally intelligible. So not just any isomorphism will do. On the other hand, the idea of an 'intelligible' isomorphism is itself not, in our present state of understanding, a particularly intelligible concept.

Dennett: Margaret talked about the Crick & Koch paper. The reason their claim about binding leaves us unsatisfied is that in their paper they do not address the question: And then what happens? The same is true when you say, 'Suppose we discover that all this information is encoded in a bit string'. What we want to know is: And then what happens? I submit that if there is an intelligible answer to this question that shows how the information in that bit string manages to accomplish the work that has to be done as revealed in the reports and behaviour of the subject, then it becomes intelligible. The fact that it's a bit string does

not disqualify it: what disqualifies it for the moment is that you don't have any answer to the question: And then what happens?

Harnad: Becomes intelligible *as what?* One of the effects of this very interesting talk is that some of the fragile alliances that had formed at this symposium have been shattered; people who thought they could make common cause are now clearly going their own ways!

When I wear my cognitive psychologist's or neuroscientist's hat, I find Bill Newsome's results very interesting, because I see them as telling me how the brain works and perhaps how to make a visual system in principle work. But if I put on a philosopher's hat, it's another story. Bill Newsome has gone 75% of the way; let's pretend he has also done the ablation study and the human study so that he has really established that this unit is necessary and sufficient in this particular mechanism for motion detection. That would be an empirical step, but it would be just a piece in a much larger puzzle, and you all agree that there are many other relevant things we need to know. One of the things Maggie Boden suggested was that it is not even enough that there should be a completed big puzzle: there even has to be isomorphism—the whole structure we eventually discover that is necessary and sufficient to do all the vision must, in addition, square with the subjective phenomenology of vision, and all the rest.

I want to question the slight epiphany that we get when we manage to capture a necessary and sufficient unit such as Bill Newsome's. I'm suggesting that our epiphany is spurious. Why should our reaction to this unit be different to our reaction to a clear non-starter, such as the computer vision system I mentioned earlier? Why do we go 'Aha' with this unit and not with a functional equivalent that has essentially the same properties, but neither aspires to be human nor resides in one? Not only do I not see any justification for an 'Aha' in the one case when it is so clearly unjustified in the other, but I don't even see anything suggesting the road leading to an 'Aha' in anything along those lines (including Maggie's isomorphisms and Dan's heterophenomenology).

Newsome: I agree. I believe our data are important from the point of view of neuroscience or cognitive psychology, but I doubt that they raise any fundamentally new questions, or answer any fundamental problems, from a philosophical point of view. When I was invited to this conference, I wanted to come because I wanted to hear what philosophers talk about at meetings of this nature. I doubted, however, whether I had anything to say that was particularly important for a philosopher to hear. I personally believe that the philosophical issues raised and addressed by our experiments are essentially the same as those raised and addressed by the work of Giles Brindley, who elicited subjective sensations of light in blind patients by stimulating visual cortex, or by the work of Wilder Penfield, who elicited organized, complex sensations by stimulating temporal cortex. The important point of those experiments was that focal activation of brain tissue can cause meaningful conscious phenomena even if the activation is achieved by artificial means. At least some conscious

phenomena, then, seem to arise in a fairly direct manner from the activity of systems of neurons in the brain. If we assume for the moment that our monkeys actually 'see' motion when we stimulate MT, our experiments simply extend this principle by linking a more specific type of sensation to more precisely defined circuits within the brain.

Hopefully, this incremental approach will bring more and more of our subjective experience within the domain of empirical investigation as research continues. I must admit that I am less optimistic about obtaining really satisfying answers to other questions about sensory experience. For example, why are the subjective sensations that accompany stimulation of visual and auditory cortex so radically different? Why is it that the first cause in me the subjective quality associated with 'seeing' whereas the second cause the quality of 'hearing'? I think this is a very difficult issue and I am always embarrassed when an undergraduate or first-year medical student asks this question, because I have no good answer. Neurophysiologists tend to mumble things about labelled lines in reply to such questions because that is the best we can do, but I've never been convinced that that emperor is well clothed.

Gray: There is one further point—you are doing these experiments in a monkey. If you end up getting, as you will, evidence that is compatible with the hypothesis that the monkey perceives motion in the sense that we see motion—maybe you could show the waterfall illusion being affected by your stimulation—then we would have strong grounds to say monkeys have conscious experience. I'm not sure that you can at present demonstrate that.

Searle: The point is, Bill, philosophically, one is never satisfied until one knows exactly how it works. That's why your work is philosophically important.

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