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Toward a Theory of Visual Consciousness

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The visual brain consists of several parallel, functionally specialized processing systems, each having several stages (nodes) which terminate their tasks at different times; consequently, simultaneously presented attributes are perceived at the same time if processed at the same node and at different times if processed by different nodes. Clinical evidence shows that these processing systems can act fairly autonomously. Damage restricted to one system compromises specifically the perception of the attribute that that system is specialized for; damage to a given node of a processing system that leaves earlier nodes intact results in a degraded perceptual capacity for the relevant attribute, which is directly related to the physiological capacities of the cells left intact by the damage. By contrast, a system that is spared when all others are damaged can function more or less normally. Moreover, internally created visual percepts—illusions, afterimages, imagery, and hallucinations—activate specifically the nodes specialized for the attribute perceived. Finally, anatomical evidence shows that there is no final integrator station in the brain, one which receives input from all visual areas; instead, each node has multiple outputs and no node is recipient only. Taken together, the above evidence leads us to propose that each node of a processing-perceptual system creates its own microconsciousness. We propose that, if any binding occurs to give us our integrated image of the visual world, it must be a binding between microconsciousnesses generated at different nodes. Since any two microconsciousnesses generated at any two nodes can be bound together, perceptual integration is not hierarchical, but parallel and postconscious. By contrast, the neural machinery conferring properties on those cells whose activity has a conscious correlate is hierarchical, and we refer to it as generative binding, to distinguish it from the binding that might occur between the microconsciousnesses.

INTRODUCTION

A dire vrai, il n'est pas possible, dans l'état actuel de nos connaissances, de formuler une théorie définitive du plan architectural et fonctionnel du cerveau . . . Il est inutile de dire que nous ne prétendons pas donner a notre hypothèse un caractère dogmatique; nous savons trop bien que des faits imprévus modifient ou renversent, du jour au lendemain, nos conjectures scientifiques. Tous ce que nous pouvons souhaiter, c'est qu'il reste de notre conception quelques-uns des principes sur lesquels nous l'avons basée.

Santiago Ramon y Cajal,
Histologie du Système Nerveux, 1911

Different workers have approached consciousness for different reasons, with different tools and in different experiments. Our approach is dictated in part by a philo-

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sophical view of the functions of the brain and in part by the unexpected results of two sets of experiments; these, interpreted against the background of what we have learned about the visual brain in the past 25 years, have led us ineluctably in the direction which we expose here. Our philosophical view can be summarized by saying that the function of the visual brain—and indeed of much of the brain—is to acquire knowledge about the world. But since humans acquire knowledge mainly in the conscious state and since consciousness is an omnipresent product of human brain function—whether as main product or as unavoidable byproduct, as precondition or as consequence—any physiological study of the visual brain, and indeed of the whole brain, becomes unavoidably a study of consciousness. The unexpected results of two sets of experiments gave urgency to this view and enabled us to bring under one heading—that of consciousness—diverse results from different fields of neuroscience. The evidence derived from each one of these sources or from individual experiments, on their own, may seem unsatisfactory and leave many questions unanswered. But when all the results are viewed together, the evidence for the view that we propose here becomes compelling.

The first unexpected result was derived from an experiment which we undertook to study the visual capacities of a subject deprived of vision by a lesion in V1 sustained during childhood. We had supposed from the published literature on ''blindsight'' (Weiskrantz, 1990) that the patient would be able to discriminate the direction of motion of stimuli in his visual field without having a conscious awareness of having seen anything. We wanted to learn what areas of the brain become activated during this ''sightless view'' (Shakespeare), when he apparently ''sees'' without being aware of having seen. But our results (Barbur, Watson, Frackowiak, & Zeki, 1993; Zeki & ffytche, 1998), gave a different picture—the subject was not only able to discriminate correctly the direction of motion of fast moving stimuli presented to his blind field but was also conscious of having seen them. This immediately showed that pre-processing or post-processing of visual stimuli by V1, or reciprocal integration of activity between higher areas and V1, as envisaged by theories of ''re-entry'' (Edelman, 1989; Engel, Fries, Roelfsema, König, & Singer, 1999), are not necessary conditions for conscious awareness. Indeed it showed that activity in areas disconnected from V1 can have a conscious correlate, a finding instrumental if not unique in leading us to the supposition that activity at any given stage of a processing system can have a conscious correlate. We note that if the result had been what we had predicted from the published literature, namely, that such a patient would be able to ''see'' without having any conscious awareness of having seen, then our theory would not necessarily be wrong. But then, we may not have formulated it either.

The second set of unexpected results came from psychophysical experiments. Given the unity and wholeness of our vision, we had supposed that different attributes of the visual scene are processed at the same time or at least that an integrator area or process would bring the results of the processings undertaken by the different systems together to provide an integrated image, one in which all the attributes of vision are seen in perfect temporal and spatial registration. But the assumption had not been tested. When we did so, we were surprised to find that when two attributes of vision, for example color and motion, occur at the same time, they are not necessarily perceived at the same time (Moutoussis & Zeki, 1997a,b; Zeki & Moutoussis, 1997) and that color is in fact perceived before motion. This argued for an autonomy of

the two processing systems and, by extension, for an autonomy of other processing systems as well. Again, if our result had been of the expected variety and had shown that color and motion are perceived at the same time, one would not conclude that our theory is wrong. But, once again, we would have been less motivated to formulate it in the first place.

As it happens, these two cardinal results are mutually supportive and in turn receive support from other lines of evidence. The whole, taken together, have led us in the direction that we outline below. The two cardinal experiments alluded to above were thus mere catalysts in bringing together conceptually, and under the banner of consciousness, many different studies, including especially new psychophysical ones on relative perceptual times (Zeki & Bartels, 1998b). Each group of results, some old and some new, leads us to a proposition. Some of these are so well known that to repeat them is to appear trite and invite ridicule; others are more novel and radical. If we thus condense the results of experiments into propositions here, it is because these propositions form a mutually supportive and linked chain which leads us ineluctably to our current view. Our view differs, perhaps significantly, from other views about integration, binding, and consciousness. We take little pride in this but only plead that we were driven in our direction by the logic that links the findings that are at the basis of our formulation. We were of course impressed most by the experiments of which we have firsthand knowledge, namely our own, and by such reading as curiosity drove us to in the light of our results. In the process, we may have missed other important findings and perhaps even done an injustice to others. For this we apologize.

The present article represents a synthesis of views on different aspects of conscious vision which we have recently exposed elsewhere (Zeki & Bartels, 1998a,b). It incorporates our three theories of the asynchrony of consciousness, of multistage integration, and of relative perceptual sites and leads us toward a more general theory of visual consciousness.

I. THE PARALLEL PROCESSING SYSTEMS OF THE VISUAL BRAIN

We take it as accepted by most, if not all, workers that the visual brain consists of many different visual areas, each having a distinctive pattern of connections and each undertaking its task simultaneously and in parallel with the others (Zeki, 1975). The principle of the multiplicity of visual areas (Zeki, 1969a, 1971; Allman & Kaas, 1971, 1974) has now been established beyond doubt through the work of many, even though the actual number of distinct areas has increased and remains uncertain (Felleman & Van Essen, 1991). Each area has highly specific connections, with V1 and V2 on the one hand and with further areas in the temporal, parietal, and frontal lobes on the other. The connections between the blobs of V1, the thin stripes of V2 and V4, all of them involved with color, and the connections between layer 4B of V1, the thick stripes of V2 and V5, all involved with motion, are good representative examples and too well known to chart in detail here (Livingstone & Hubel, 1984a; Hubel & Livingstone, 1987; DeYoe & Van Essen, 1988; Zeki & Shipp, 1988). We therefore define a processing system as one which includes the specialized cells of V1 and V2 (housed in specialized compartments within these two areas) and the specialized areas to which they project; beyond that, we also include in the processing

system the further projections of a specialized area. Examples of the latter may be found in the motion-related cortex that surrounds area V5 and is reciprocally interconnected with it (Zeki, 1980; Wurtz, Yamasaki, Duffy, & Roy, 1990; Howard, Brammer, Wright, Woodruff et al., 1996) or the areas to which V4 connects in the medial temporal lobe (Desimone, Fleming, & Gross, 1980; Zeki & Marini, 1998). We do not equate these parallel systems with the M and P pathways (e.g., Livingstone & Hubel, 1988). Too many studies have shown that systems previously thought to have been derived exclusively from the M pathway have a significant P input and vice versa (e.g., Saito, Tanaka, Isono, Yasuda, & Mikami, 1989; Maunsell, Nealey, & DePriest, 1990). Rather, we believe that a processing system will draw upon any input to undertake its function (Zeki & Shipp, 1988).

We further accept that different processing systems are specialized to undertake different tasks (Zeki, 1978a; DeYoe & Van Essen, 1988; Livingstone & Hubel, 1988; Zeki & Shipp, 1988). The evidence for this is derived from studies of anatomical connections, physiological properties, and clinical cases and is alluded to throughout this review. The contrary view, with which we do not agree, states that there is no specialization within V1 or V2 (Lennie, Krauskopf, & Sclar, 1990; Leventhal, Thompson, Liu, Zhou, & Ault, 1995; Gegenfurtner, Kiper, & Fenstemaker, 1996) or indeed in the visual cortex at large (Schiller, 1997).

This leads us to Proposition 1: The visual brain consists of parallel, distributed, and functionally specialized processing systems.

II. THE BASIC ANATOMY OF THE PARALLEL PROCESSING SYSTEMS

There is of course a wealth of detail concerning the connections of the processing systems and the stages that constitute them. We do not review these here but concentrate only on those aspects that are of special interest to us in outlining our theory.

(a) Each processing system consists of several stages which we refer to as *nodes* (Bartels & Zeki, 1998). As an example, we give the motion system consisting of layer 4B of V1, the thick stripes of V2, area V5 and other motion-related areas surrounding it. Each one of these constitutes a node of the motion processing system and the forward connections within this processing system are of the ''like-with-like'' variety. By this we mean that the directionally selective cells of layer 4B connect with area V5, which is also rich in directionally selective cells, either directly or through the thick stripes of V2, which also contain directionally selective cells (Zeki, 1969b; Lund, Lund, Hendrickson, Bunt, & Fuchs, 1975; DeYoe & Van Essen, 1988; Zeki & Shipp, 1988; Shipp & Zeki, 1989a,b). Equally, the color system extends from the blobs of V1 to the thin stripes of V2 and from there to V4, all containing wavelength selective cells; beyond V4, it extends to areas in the inferior temporal cortex. Each one of these stages in this processing system constitutes a node, the forward connections within the entire processing system being again of the like-with-like variety (Zeki & Shipp, 1989; Nakamura, Gattass, Desimone & Ungerleider, 1993). By ''node'' or ''stage'' we therefore refer to a whole area, such as V4 or V5, or to the functional subdivision of an area, such as the blobs and interblobs of V1 or the thin, thick, and interstripes of V2 (Bartels & Zeki, 1998). The consequence of a like-with-like forward connectivity is to enlarge receptive fields and to confer different, and often more complex, properties on cells in a given node, compared to the antecedent

one, in hierarchical fashion (e.g., Hubel & Wiesel, 1962). We emphasize here what we shall detail more generally below, namely, that not all of these nodes need be simultaneously or sequentially active for visual perception to occur.

Proposition 2: Forward connections within a processing system are of the like-with-like variety and lead to cells of increasing receptive field size and complexity in hierarchical fashion.

(b) There are many anatomical opportunities for the nodes comprising the different processing systems to communicate with each other. These connections, which we refer to collectively as lateral connections because they constitute links between different processing systems, can be conceptually subdivided into three varieties and share the common property that they differ from the like-with-like variety in that they also include ''like-with-unlike'' connections: (i) lateral connections between nodes, which can be of the like-with-like variety as in the ''blob-to-blob'' connections within V1 (Livingstone & Hubel, 1984b) or of the like-with-unlike variety, for example the lateral connections that link the thick and thin stripes of V2 (Rockland & Lund, 1983; Rockland, 1985; Lund, Yoshioka, & Levitt, 1993; Levitt, Yoshioka, & Lund, 1994); (ii) direct connections between the specialized areas, e.g., the direct link between V4 and V5 (see Felleman & Van Essen, 1991 for a review), which in our evidence are not especially strong; (iii) the return connections from the specialized areas, back to the areas feeding them (Shipp & Zeki, 1989a,b; Rockland, Saleem, & Tanaka, 1994; Rockland & Van Hoesen, 1994). All three could be categorized as being of the like-with-like and like-with-unlike varieties: the connections within V1 can be of the blob-to-blob or the blob-to interblob variety, and much the same is true of the connections between the stripes of V2 (Rockland, 1985). Moreover, in contrast to the forward connections from V1 and V2 to V4 and V5, the return connections from these specialized areas to V1 and V2 are diffuse. For example, while the output from V1 to V2 and from V2 to V5 obeys the like-with-like principle (see above), the return input from V5 to layer 4B of V1 is not restricted to the territory of directionally selective cells in that layer, but is much more diffusely spread and includes the territory of cells that project elsewhere (Shipp & Zeki, 1989a). Similarly, the return input from V5 to V2 is more diffuse and not restricted to the territory of the thick stripes but includes that of the thin stripes and interstripes as well (Shipp & Zeki, 1989b); a similar diffuse projection has been found from V4 to V2 (Zeki & Shipp, 1989). The motion and color systems thus have the anatomical opportunity of communicating with each other through these return projections as well as through direct lateral connections. What is surprising, given the wealth of these like-with-unlike anatomical opportunities, is how stable the properties of cells in the visual nodes are. Most orientation and direction selective cells are indifferent to the color of the stimulus and most wavelength selective cells are indifferent to the form or direction of motion, even after prolonged stimulation with an attribute to which they are not selective. Moreover, many motion selective neurons are indifferent to texture or form of the moving object (Albright, 1992). On the contrary, in spite of these lateral connections, the majority of cells within a given processing system continue to be concerned with a given attribute of the visual scene rather than with all attributes. If these lateral connections mediate any integrative role, then that role and its consequence have yet to be discovered, although it remains possible that they help to derive one attribute (e.g., motion) from another (e.g., form) (Zeki, 1993) or be important

in conflict conditions, as when, because of their small receptive fields, the direction cells in V1 signal component motion which does not represent the true direction of motion signaled by cells in V5 (Zeki, 1993). A central claim of the temporal binding hypothesis (von der Malsburg & Schneider, 1986; see also Engel et al., 1999) and of other integrative proposals (Gegenfurtner, 1997) is that the binding of different attributes is mediated by a special temporal relationship (e.g., synchronous firing) between two or more specialized areas. While this imaginative proposal may well turn out to be true, it is worth noting that, in spite of the many anatomical opportunities mentioned above, there is no present compelling evidence for it in terms of activity between visual areas that process different visual attributes of the visual scene. Thus, whereas the properties of the forward projecting anatomical system can be used to account for the properties of cells at successive stages within a processing system, there is no known common characteristic feature that emerges from the lateral connections. Moreover, though the forward like-with-like connections within a processing system are hierarchical, the lateral ones are not, by definition.

This leads us to Proposition 3: The lateral interconnections that anatomically link the different processing systems can be of the like-with-like, the like-with-unlike, or the diffuse variety and are not exclusively hierarchical; they do not appear to bring about cells that integrate different submodalities.

(c) Overall anatomical characteristics of nodes: There are other features of the anatomy of the multinode processing systems that are worth emphasizing. First, there is no node that constitutes a terminal stage in a processing system, since there is no known node that is a recipient only. Each node receives inputs and sends outputs (Zeki, 1993); indeed, each has multiple outputs, as if the result of the operations that each performs is of interest to several other areas. Anatomical evidence shows that there is no single area to which all the specialized visual areas connect, which would enable it to act as an integrator capable of binding signals coming from all the different visual sources. There are in fact common areas to which two different processing systems project. But when this happens each input appears to maintain largely its own territory within the common recipient area, with minimal convergence or overlap with other inputs, thus leading us to speak of a *juxtaconvergence* (Shipp & Zeki, 1995). Each node is therefore only part of a more extensive processing system, which includes, besides subcortical stations, areas in the temporal, parietal, and frontal cortices. The latter areas, too, constitute only parts of the processing system, since they all project to further areas and are reciprocally linked with the earlier visual areas from which they receive input.

This leads to Proposition 4: There is no ‘‘terminal’’ station in the cortex for a given processing system and no common ‘‘terminal’’ area to which the different processing systems connect.

III. INTEGRATION AND BINDING WITHIN AND BETWEEN PROCESSING SYSTEMS

Most discussions of integration and binding do not give adequate definitions of the terms, assuming them to give one at all. But the intended meaning is quite clear: it refers either to the ‘‘integration’’ or the ‘‘binding’’ of what is processed by the

different processing systems (that is, the binding of different attributes) or, more commonly, to the ''binding'' of the responses of cells within a single processing system. In the latter instance, it is supposed that this binding distinguishes the firing of the ''bound'' cells from that of all others and constitutes the neural basis for the kind of perceptual salience that is evident in figure–ground segregation (Engel, et al., 1999). A much studied example is whether two cells that are specific for the same orientation but with receptive fields located in different parts of the field of view will synchronize their responses (or have responses that will oscillate together) when a single long line of the appropriate orientation falls on the receptive fields of both, as opposed to a condition in which the line is discontinuous and more or less restricted to the individual receptive fields of the orientation selective cells (Engel, et al., 1999). Crick and Koch (1990) have distinguished three kinds of binding. The first two are based on the developmental processes and on experience; the third one is what they call ad hoc binding. For our purposes, we need a more extensive and inclusive definition of binding or integration. This is not a mere periphrastic exercise but is important for our theory of multiconsciousnesses. This supposes that activity at each node can become perceptually explicit (that is, require no further processing to create a conscious percept) and therefore have a conscious correlate and that visual consciousness therefore consists of many microconsciousnesses (Bartels & Zeki, 1998; Zeki & Bartels, 1998a). The information that is explicitly present at a node (in the form of neuronal activity) is rendered explicit not only by virtue of the input to it, but also because of its anatomical machinery and specialized physiological capacities, which can partially be thought of as memory and thereby as implicitly stored information. Because nodes within a single processing system are hierarchically connected (Proposition 2), the input to one node may implicitly contain part of the information rendered explicit in it. By implicit information we mean information that requires further processing at the same or at further sites to become perceptually explicit. We consider that the enlargement of receptive fields as one progresses from the lateral geniculate nucleus to V1, or from V1 to V2, or from V1 to V5 are examples of physiological integration which have perceptual and therefore conscious consequences, especially since this kind of binding is accompanied by a modification of receptive field properties. This enlargement therefore confers on cells at a node unique information which is not explicitly present at nodes above or below it; it generates new ''experiential'' cells (Zeki, 1993) whose responses are perceptually explicit, and what becomes perceptually explicit reflects the physiological capacities of the cells at that node. For example, direction selective cells in V1 and V5 signal the motion direction of the components of a moving complex pattern, whereas other V5 cells respond to the motion direction of the whole pattern (Movshon, Adelson, Gizzi, & Newsome, 1985; Rodman & Albright, 1989). The combination of physiological and psychophysical experiments in monkeys revealed that the reliability and sensitivity of neurons in V5 equal that of the behavioral response (Newsome, Britten, & Movshon, 1989; Britten, Shadlen, Newsome, & Movshon, 1992), suggesting that a conscious percept might be based on activity of only few cells in a node. A more compelling example is to be found in the color system. The enlargement of the receptive field properties of the wavelength selective cells as one progresses from V1 to V4 is accompanied by a qualitative jump—the responses of the cells in V1

correlate with wavelength composition alone, whereas the responses of some cells in V4 correlate with perceived color, irrespective of precise wavelength composition (Zeki, 1983a). This qualitative jump, we presume, is brought about because the enlargement of the receptive fields in V4, which often have large suppressive or excitatory surrounds (Zeki, 1983b; Desimone, Moran, Schein, & Mishkin, 1993), enables them to undertake the comparisons that are critical for generating constant colors (Land, 1986). But we are conscious both of the constancy of colors and of changes in wavelength composition coming from a given part or from the whole of the field of view. Each stage can therefore make a direct contribution to conscious perception, but of a different variety, as is also suggested by the clinical evidence (see below).

We therefore distinguish between two types of binding or integration (two terms which we use interchangeably); these differ from each other in physiological implementation and type of neuronal code used: (a) generative binding, which is always hierarchical and preconscious. It generates cells with new receptive field properties, is accompanied by receptive field enlargement, and is mediated by a ''like-with-like,'' ''bottom-up'' input. It combines the activity of two or more cells onto a third cell in a reliable and reproducible fashion, and the response of the third (receiving) cell depends entirely on the firing of the cells feeding it. The example of V4 cells given above is one among many. Other examples may be found in the generation of simple cells of V1 from center-surround cells (Hubel & Wiesel, 1962), disparity cells in V3 from the orientation selective cells of V1 (Zeki, 1978b; Poggio, Gonzalez, & Krause, 1988), and face selective cells (Perrett, Rolls, & Caan, 1982), which combine input from lower level cells. Generative binding thus results in new classes of experiential cells, whose activity has a conscious correlate. Since this conscious correlate is restricted to the visual domain for which that class of cell is specialized, we refer to it as a microconsciousness. We refer to this kind of binding as preconscious because it is the process of binding itself that generates a new class of cell, whose activity can have a conscious correlate. Since, within each system, enlargement of field size and consequent modification of response is strictly hierarchical (Proposition 2), it follows that generative integration is also strictly hierarchical. (b) Parallel binding: here we refer to the coupling of the activity of cells—e.g. through synchronous or oscillatory firing or any other form of communication—within a single area or across different areas. In view of our theory, which supposes that activity at each node has a conscious correlate, we consider this binding postconscious, since it is the microconsciousness generated at a given node of one processing system that is bound to the microconsciousness generated at a given node of another (or the same) processing system. We hypothesize that mere communication between areas will not result in a microconscious correlate. It is only the cellular activity at the nodes which does so. Therefore, ''binding'' must result in a change of the activity at the nodes involved so that altered microconsciousnesses are generated at each. The binding can also be between two groups of cells at a given node, whose activities have a conscious correlates. Unlike its generative counterpart, postconscious integration does not entail receptive field enlargement or modification, since it does not necessarily entail the bringing together of the responses of two cells onto a third cell. It may instead require a form of communication (e.g., synchronous or oscillatory firing) between two sets of cells

which are grouped together by a neural code that is different and independent of the one that codes for the feature specificity of cells. Preconscious binding need not affect the cells whose response is integrated because their output is integrated in a third, recipient cell. An example of parallel binding is to be found in the cells of the visual brain that have receptive fields at the midline and that are commonly thought to mediate the interhemispheric integration that links the separate representation of the two hemifields, giving us an unbroken view across the midline. Yet physiological studies show that many, if not most, of these cells, especially in areas V1 and V2, do not have larger receptive fields than cells which represent central, but not midline, parts of the field of view and most commonly have fields that do not cross the midline, or do so only marginally (Van Essen & Zeki, 1978).

As well, unlike generative binding, parallel (postconscious) binding is not hierarchical (Proposition 3); the micro-consciousness generated by the activity of cells at any given node of a processing system can be bound to the microconsciousness generated by the activity of cells at any given node of another processing system (see below under the theory of multistage integration). Nor is this restricted to integrating the activity of different nodes between different systems; it could equally apply to integrating the activity of cells within a specialized system. This can be exemplified by the type of binding reported to exist between cells within a given node, namely, the synchronization between the responses of two groups of orientation selective cells with receptive fields at different locations, when they are both responding to a continuous line of the same orientation (see Engel et al., 1999). Activity at each has a microconscious correlate (our hypothesis) and it is these microconsciousnesses, we believe, that would be brought together in such an example. Here we depart from the more common belief that it is the binding itself that brings about the conscious experience (e.g., Crick & Koch, 1990b; Engel et al., 1999).

The distinction between generative and parallel binding that we make here has not been made before, but it has been proposed that what we call parallel binding facilitates figure–ground separation (binding within a node) or brings different visual attributes such as color and motion together through the synchronous or oscillatory firing of cells in different nodes (von der Malsburg & Schneider, 1986; Engel et al., 1999) and that this is necessary for generating conscious perception (Crick & Koch, 1990b; Singer, 1998; Tononi & Edelman, 1998). But despite its theoretical attraction, there is no unanimity of view that synchronous or oscillatory firing in this context is of functional or perceptual relevance (Lamme & Spekreijse, 1998) nor is it known how the synchrony is generated, that is, whether it is of a top-down, bottom-up or thalamocortical nature (Llinás, Ribary, Contreras, & Pedroarena, 1998). The evidence we present below leads us to the view that parallel (or postconscious) binding, supposing it to occur, may not be necessary for the normal functioning of nodes, including the generation of a microconsciousness.

This leads to Proposition 5: Generative or preconscious integration is hierarchical and limited to a given processing system; it leads to a new class of receptive field properties and to

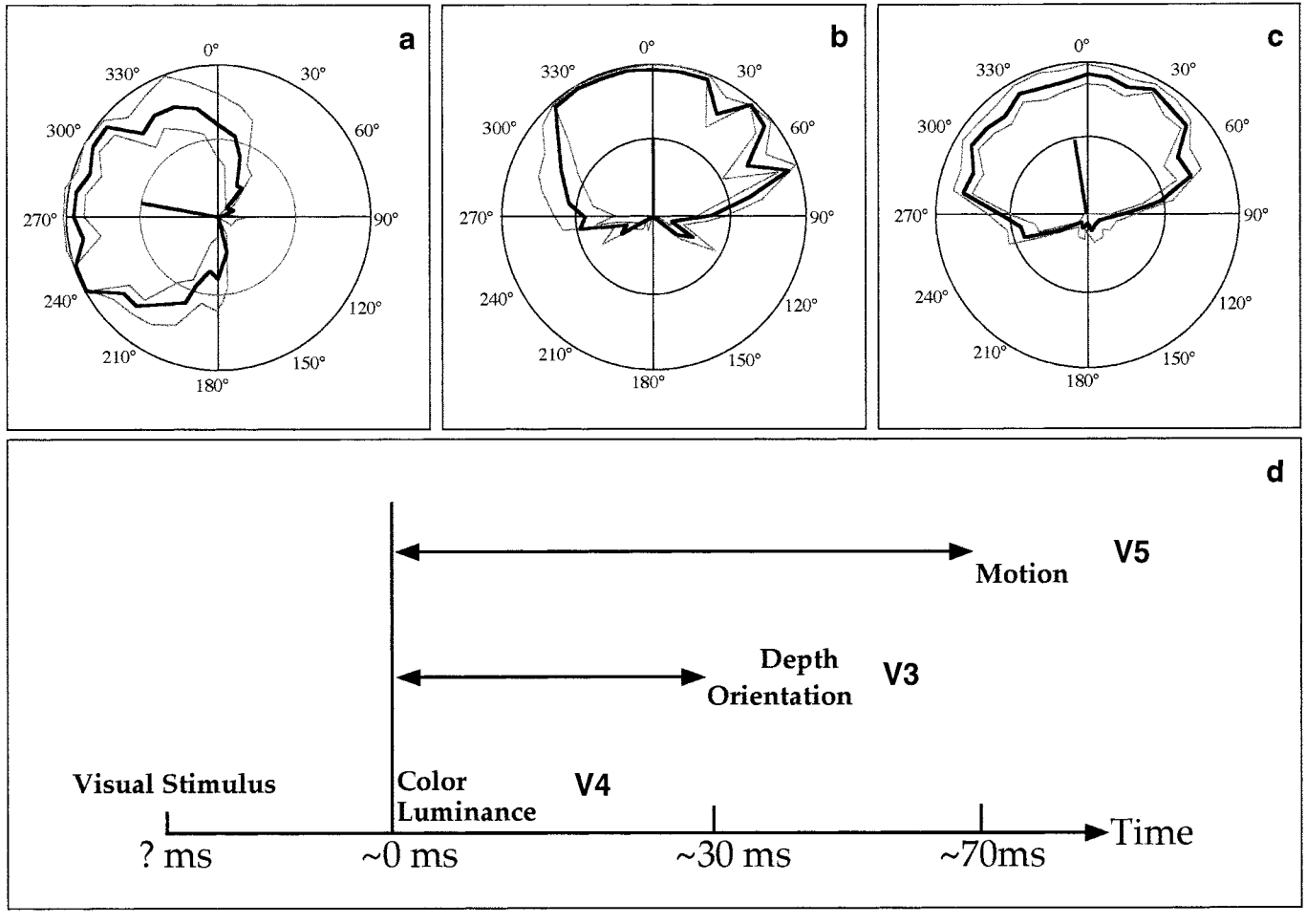
Proposition 6: Parallel or postconscious integration is not hierarchical and can occur between different nodes of different processing systems as well as within a single node.

The distinction between preconscious and postconscious binding is important for the theory of multistage integration.

IV. THE ASYNCHRONY OF CONSCIOUSNESS

We are conscious of what we perceive and are not conscious of what we do not perceive and do not perceive what we are not conscious of. The question that we address here is whether two visual events which occur together in real time are also perceived simultaneously, which amounts to asking whether we become conscious of the two events simultaneously. Intuitively one would suspect this to be so, since in our daily experience we perceive different modalities coherently (i.e., with precise spatiotemporal registration). But there is a large body of evidence which shows that different modalities (e.g., audition and vision) are perceived with different delays from the time of stimulus onset, in the sub-second range (Woodworth & Schlosberg, 1965). This is also true of attributes within a modality. An example in vision is when subjects are asked to pair two rapidly alternating states of two attributes, for example, a bar having one of two colors and one of two orientations. They are then found to consistently misbind attributes which occur at the same time, because the two attributes are perceived at different times. For example color is perceived before orientation, which is perceived before motion, with about 30- and 40-ms lag times, respectively (Moutoussis & Zeki, 1997b) (Fig. 1). This perceptual asynchrony within the visual system might presumably form the basis for the much studied illusory conjunctions (Treisman & Schmidt, 1982). It is a perceptual result that confirms psychophysically that neural processing of different visual attributes is segregated in the cortex; it is also one which those who have reported that all cells in visual cortex respond equally to all modalities (e.g., Gegenfurtner et al., 1996) should ponder and address in time. The result implies that the color processing system reaches its perceptual end point before the motion processing system. The consequence of this perceptual asynchrony is that, over brief periods of time, subjects misbind what happens in real time, attributing a color at time t to a direction of motion that occurs at time $t - 1$. The brain thus does not necessarily bind together what happens in real time but appears instead to bind the results of the operations undertaken by its different processing systems, which require different amounts of time to complete their tasks. In the sub-second window, the brain therefore misbinds in terms of real time (Moutoussis & Zeki, 1997a,b; Zeki & Moutoussis, 1997). What this result also implies is that there is no central perceptual integrator area that takes into account the different

FIG. 1. The theory of perceptual sites is based on evidence summarized in this figure. This theory supposes that attributes processed in the same node are perceived simultaneously, whereas attributes processed in different nodes are perceived asynchronously. The diagrams indicate the latencies with which one attribute is perceived with respect to another. The full 360° circle corresponds to 573 ms in time. If two attributes (e.g., left–right motion and up–down motion) are perceived at the same time, the vector will show no displacement from 0°. Any displacement of the vector will indicate that one of two attributes is perceived before the other. In the examples given above, color is perceived before motion (a) while left–right motion and up–down motion are perceived simultaneously (b). Depth and orientation are perceived at the same time (c). The relative perceptual times for different attributes are summarized in (d).


a
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180°
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240°
270°
300°
330°
Motion
V5
Depth
Orientation
V3
Visual Stimulus
Color
Luminance
V4
? ms
~0 ms
~30 ms
~70ms
Time

time lags of different systems with regard to real time, before binding their results together. It is important to emphasize that what subjects perceive consciously in these psychophysical experiments is the change in the two states, while they are pairing one with the other; they are not aware of what we measure, namely, the difference in relative perceptual times. Given that subjects have to pay equal attention to both attributes in order to pair them, the controversial phenomenon of ''prior entry'' (Cairney, 1975) is not relevant here. Collectively, this evidence supports the notion of a general asynchrony in perception, including visual perception.

This leads us to the following propositions:

Proposition 7: Another characteristic of the visual brain is a temporal asynchrony in vision and, reflecting the consequence of functional specialization in the time domain, visual perception is therefore also modular, and to

Proposition 8: When two visual events occur together, they do not have to be integrated for each to be perceived, and thus a mutual integration of activity between different processing nodes is not necessary for the creation of a conscious percept. Moreover, since perception is accompanied by conscious awareness, we are further led to

Proposition 9: Activity in each separate processing node generates a microconsciousness for the attribute for which that node is specialized. Consequently, there are several microconsciousnesses, corresponding to the activity of cells at different nodes within different processing systems.

But not all visual events that occur together in real time are perceived asynchronously. We can ask subjects to pair two events belonging to the same attribute and known to be processed by the same system. For example, we can ask subjects to pair left–right motion in one half of a TV screen with up–down motion in the other. We then find that they perceive the two events that occur together at the same time (Moutoussis & Zeki, 1997b) (Fig. 1b). We have used this evidence to enquire into the relative perceptual times of other attributes which the physiological evidence suggests are processed by the same system and even at the same node. A good example is to be found in area V3, in which cells are commonly selective for both orientation and depth (Zeki, 1978a, 1979; Poggio et al., 1988). Our experiments show that, when humans are asked to pair two different depths with two different orientations, they perceive the two at the same time (Fig. 1c). This finding, along with other ones, has led us to our theory of perceptual sites (Zeki & Bartels, 1998b), which states that attributes processed at the same site are perceived at the same time and those processed at different sites are perceived at different times. There are other interesting examples of this perceptual synchrony, which we have used to formulate our theory and which may in fact shed an interesting light on functional specialization in the visual brain in a round about way. It has been argued, for example, that the V4 complex is concerned with orientation, because of the presence of orientation selective cells in it (Zeki, 1975, 1983c; Desimone & Schein, 1987), although the orientation selective cells of V4 are more broadly tuned than their counterparts in V1, V2, and V3 (Zeki, 1997). Another view is that V4 is less concerned with orientation as such, but with form in relation to color (Zeki, 1997). If our theory of perceptual sites is correct, then we should be able to determine whether orientation and color are actually processed at the same or different sites by simply noting whether they are

perceived at the same time; in fact they are not (Moutoussis & Zeki, 1997b). Extending this yet further, we can ask whether orientations generated from random-dot motion are perceived at the same time as orientations generated from equiluminant colors. Now we find that when subjects are asked to pair one of two orientations generated from equiluminant color stimuli and presented in one half of a TV monitor with one of two orientations generated from random-dot motion and presented in the other half, the two are perceived at the same time, as if the perception of orientation is mediated by a single area, regardless of how it is generated (Zeki & Bartels, 1998b, and our unpublished results). We of course know that there are cells that are able to respond to their preferred orientation if the oriented line is generated from equiluminant stimuli (Gouras & Kruger, 1979; Thorell, De Valois, & Albrecht, 1984). So far such cells have been studied only in V1, but there is little reason to suppose that they may not be found elsewhere, nor any compelling reason to suppose that the V1 cells may not actually be at the basis of the perception. This leads us, as an aside, to suppose that V4 is not concerned with orientation as such. We have used many such pairings and our studies have led us to a general conclusion, which constitutes

Proposition 10: When two visual events that occur at the same time are perceived at the same time, it is because they are processed at the same site (node) and when they are perceived at different times, it is because they are processed at different sites (nodes).

V. MICRO-CONSCIOUSNESSES ARE FUNCTIONALLY SPECIALIZED

If activity at each node of a given, functionally specialized, processing system can have a conscious correlate, it is obvious that the microconsciousness generated at that node is functionally specialized because it relates to the specialization of the cells at that node. It would be difficult to conceive of the microconsciousness generated by the activity of cells in V5 to be related to color or that generated by the activity of cells in V4 to be related to motion. This leads us to

Proposition 11: The microconsciousnesses are functionally specialized, each microconsciousness being the reflection of activity in a particular, functionally specialized, processing node.

Our concept of microconsciousness perhaps requires some explanation. The concept has come as a surprise to many with whom we have discussed the issue because of the belief in the unity of consciousness, perhaps best enshrined in the famous dictum of Descrates ''Je pense, donc je suis,'' although he did not intend to imply a unity of experience by that phrase. But this unity is not at all apparent to us, nor it seems is it apparent to philosophers (e.g., Dennett, 1991). In general, we are not aware of being aware. Instead, we are aware of events, or more generally of that which we are attending to at a given time. It is generally thought that a good guide to being aware is communication through language. But during that communication, we are still not aware of being aware but only aware of our interlocutor or the subject matter. We only become aware that our interlocutor is conscious in an inferential sense, in that he or she is conscious because they are able to conduct this conversation with us and could not do so unless they were conscious. But that awareness of consciousness is elicited only when the question is framed. Even in common conversa-

tion, we are conscious of a few things only and commonly of one thing at a time only. Thus the term microconsciousness may itself be a misnomer, because it implicitly supposes that there is a higher unified and singular conscious entity, beyond all the microconsciousnesses.

VI. THE PROCESSING SYSTEMS ARE ALSO PERCEPTUAL SYSTEMS

We now address the supposition that we have made in the above discussion, that the processing systems are also perceptual systems and that activity at each node of a processing system can have a perceptual and therefore conscious correlate. The evidence for this comes from electrophysiological and anatomical studies discussed above, greatly fortified by clinical studies, which is what we expose below.

Implicit in the term ''processing'' is the supposition that it is preperceptual, a means of getting to the final percept, whatever that may be. This supposition itself makes implicit assumptions which are worth discussing. The most obvious of these is that there are separate processing and perceptual systems, or at any rate that the processing stages antedate the perceptual ones. In anatomical and physiological terms, this implies that the processing system feeds the results of its operation into the perceptual system. This may be true within an individual node. The results of Logothetis and his colleagues (Logothetis, 1998) have shown, for example, that at each node there are cells whose responses correlate with perception and others whose responses do not, and it is plausible to suppose that, physiologically, the latter are the precursors of the former. Imaging evidence suggests that activity in an area (node) must reach a certain height for a conscious correlate to be generated (Zeki & ffytche, 1998) but does not tell us whether that height is due to the intensity of response of cells, the number recruited, or their synaptic activity. The evidence for the supposition that some nodes are purely processing ones and that they feed the results of their operations into other, purely perceptual, nodes does not exist. It would indeed be a somewhat inefficient way of encoding everything twice, once at the processing site and then again at the perceptual site. There is better evidence for our rival suggestion that the processing systems are also perceptual systems (Zeki, 1998), which leads us to speak of *processing-perceptual systems.* We now extend this concept, and Proposition 9, by proposing that activity at each node of a processing-perceptual system has a perceptual and therefore conscious correlate (Bartels & Zeki, 1998). Evidence in favor of this is to be found in electrophysiological experiments which have shown that within each area, including area V1, there are cells whose responses correlate with percepts (Zeki, 1983a; Logothetis, 1998). Our supposition would gain weight if it can be shown that, with a lesion at a given node of a processing-perceptual system, a patient is not totally deprived of the capacity to experience the attribute for which that system is specialized, but instead has a residual perceptual capacity for that attribute which is a direct reflection of the physiological capacities of the nodes of the affected processing-perceptual system that are left intact by the lesion. It is now generally accepted that, because much of the visual input to the cerebral cortex passes through V1, lesions here usually, but not always (see below), result in total blindness. This is probably also true for V2 (Horton & Hoyt, 1991), which is interposed between V1 and the more specialized areas of the brain and which, like

V1, has all the attributes of vision represented in it (Hubel & Livingstone, 1987; DeYoe & Van Essen, 1988; Zeki & Shipp, 1988). What is perhaps more interesting is to look at the perceptual capacities of patients in whom V1 and V2 are not totally destroyed but who have lesions in the areas of the prestriate cortex to which they project. If there is any substance to our supposition that activity at each node of a processing-perceptual system has a perceptual, and therefore conscious, correlate then we should find that such patients are capable of a more elementary perceptual experience of the relevant attributes than normals but are nevertheless able to experience something of the relevant attribute.

Clinical Evidence for a Piecemeal Understanding of the Visual World Following Damage to the Prestriate Component of the Processing Systems

The visual fields are topographically represented in areas V1 (Henschen, 1893; Holmes, 1945) and V2 (Cragg, 1969; Zeki, 1969a), as if both areas are undertaking a ''piecemeal'' analysis of the visual world (Hubel & Wiesel, 1977). Receptive field sizes of cells also become larger as one proceeds from V1 to V2 to the more specialized areas (Van Essen & Zeki, 1978), an enlargement that is coupled to the emergence of new physiological properties (Hubel & Wiesel, 1962; Zeki, 1974; Desimone, Schein, Moran, & Ungerleider, 1985). It could therefore be expected that (1) a person with a relatively large lesion in the prestriate cortex but one which spares area V1, either partially or completely, should be capable of a piecemeal analysis of his visual world and (2) that a person with a lesion restricted to the prestriate component of a given processing-perceptual system should be able to experience all attributes of the visual scene, save the one processed by the compromised system; moreover, they should be able to experience something about the attribute processed by the damaged system, and that something must be related to the physiological capacities of the undamaged nodes of that processing-perceptual system, meaning the physiological capacities of area V1 and possibly V2. Below we consider examples from different patients to illustrate these two points, by showing that the well-known syndromes of achromatopsia, object agnosia, prosopagnosia and akinetopsia—all caused by specific lesions of the prestriate cortex—share a property in common. That common property is the ability to see and experience details of a given attribute without being able to combine the details into a whole and thus experience the whole. They are, in brief, able to see and understand what the intact nodes of their processing-perceptual systems allow them to see and understand.

Object Agnosia

The ponderous speculations of Lissauer (1890) coupled to the anatomicopathological discoveries of Henschen (1893, 1910) and the myelogenetic studies of Flechsig (1901) led to a general view that we ''see'' with V1 and ''understand'' what we see with the visual ''association,'' or prestriate, cortex, a notion that divided seeing from understanding and assigned a separate cortical seat to each (for a general historical review see Zeki, 1993). Since that time, belief in the concept of a global agnosia has been apparently supported by the finding that such patients can commonly draw even complex figures, though without being able to make any sense of the figure they

have drawn or to understand it. Yet how is it that these patients draw? There is good agreement that the drawing is piecemeal, small segments of the picture or of its outline—segments that the patient can see and understand—being drawn, one after another. Once they are drawn, the patient can still only recognize and understand small segments of his drawing and not its entirety. The patients' report of the process itself is more or less uniform. One patient stated that when he copied a complex figure, ''all he saw was a complex pattern of lines, which did not correspond to a particular object.'' This is well reflected in his description of the difficulty of recognizing common objects: ''I have come to cope with recognizing many common objects, if they are standing alone. . . When objects are placed together, though, I have more difficulties'' (Humphreys & Riddoch, 1987), the latter possibly an example of what has been called simultagnosia, or an inability to perceive more than one object in the field of view at a time. It is the simple components of a figure that the patients are able to see and to understand because the integrative mechanisms necessary to construct simple forms, such as lines, are intact, while those needed for more complex forms are compromised. Indeed the authors of this fascinating report state that the patient ''has intact registration of form elements (single lines and edges), but . . . his ability to integrate these elements into 'perceptual wholes' is in some way impaired. The intact information about the local form elements enables him to make accurate copies of stimuli he cannot identify'' (Humphreys & Riddoch, 1987).

The consequences of a large lesion are also illustrated by the famous patient of Adler (1950), who suffered from carbon monoxide poisoning at a Boston nightclub during the Second World War. She has also described her experiences in piecemeal terms. Shown a green battleship, she mistook it first for a fountain pen, then for a green knife, before identifying as ''a boat.'' She explained: ''At first I saw the front part. It looked like a fountain pen because it was shaped like a fountain pen. Then it looked like a knife because it was so sharp, but I thought it could not be a knife because it was green. Then I saw the spokes and that it was shaped like a boat, like in a movie where I had seen boats. It had too many spokes to be a knife or a fountain pen.'' Another patient, ''When looking at a picture . . . could identify individual detail but could not appreciate the significance of the entire scene'' (Gomori & Hawryluk, 1984). These descriptions are so representative that they apply to many agnosic patients.

In principle, one should be able to account for some of the characteristics of the syndrome, namely a capacity to see the details but not the whole, by appealing to the physiology of the visual pathways and in particular the capacities of the nodes that are left undamaged by the lesion. This is not an easy task, because the visual areas which are involved in the recognition of even simple objects, as well as the details of the integrative processes, are not known, especially in man. But there are clues to suggest that the residual capacity of a patient to see the details, the lines in particular, is related to the physiological capacities of areas V1 and V2, partially spared by the lesion. We suppose that the orientation selective cells, a conspicuous feature of the physiology of V1 and V2, are largely intact and that activity of cells at these nodes can become perceptually explicit, that is, have a conscious and perceptual correlate. This, to us, is a far more satisfactory explanation than vague references to uncoupling between ''seeing'' and ''understanding.''

This incapacity to combine simple elements is also evident in another example, which we interpret to be due to a lesion in V2, although the actual pathology is not available and our interpretation may turn out to be wrong. The case is that of an artist who became agnosic after a cerebral vascular accident and whose agnosia was accompanied by a mental deterioration and, more significantly, a restricted scotoma (Wapner, Judd, & Gardner, 1978). Since the patient was able to perceive details, we are inclined to attribute the scotoma to a lesion of V2, which is also known to cause scotomas (Horton & Hoyt, 1991) although it may have been more extensive. One of the interesting features of this artist was his failure to see illusory contours, for example Kanizsa triangles. When shown such a triangle he described it as ''a three cornered thing . . . I see three edges and three circles.'' The authors explain that the patient's ''descriptions and drawings focused on the individual elements physically present, omitting, despite probing, any reference to the subjective occluding figure'' (Wapner et al., 1978). The patient, in brief, was not able to ''fill in'' perceptually the gaps in the Kanizsa triangle. This failure is similar to the agnosic patient described above, in whom the failure of integrative mechanisms was such that he commonly failed to ''fill in'' or complete. Physiological evidence shows that cells which are capable of responding to the illusory borders which are characteristic of the Kanizsa figures are present in V1 and V2 (Von der Heydt, 1987; Grosof, Shapley, & Hawken, 1993). We thus conjecture that the artist is capable of seeing and understanding what the intact cells of his V1 and V2 are capable of signaling. Such an explanation is as plausible as, or even more so than, one which postulates a mysterious breakdown in ''understanding'' what was seen.

The above examples share the similarity that the pathological vision described is piecemeal but that subjects can both see and understand the elements in their field of view without being able to combine the details together to form a coherent whole; they thus neither see nor understand the larger picture created by the elements. One of the difficulties of interpreting the syndromes we discuss above in the way that we would like to is that the lesions are not really adequately characterized except for the patient of Humphreys and Riddoch (Humphreys & Riddoch, 1987). Carbon monoxide poisoning in particular results in diffuse damage which almost certainly involves many areas, but there are reasons to believe that it may spare parts of V1, especially the parts concerned with color (see below). We are on surer anatomical and pathological grounds when we look at the consequences of more specific damage, to the prestriate component of a given processing-perceptual system.

Prosopagnosia, or the Inability to Recognize (Familiar) Faces

The remarkable feature of prosopagnosia is that subjects commonly know that they are looking at a face but cannot recognize it. A somewhat frightening example is the record of the dissolution of facial recognition, while it happened. It is the experience of a man who, while talking to his physiotherapist, suddenly exclaimed, ''But Mademoiselle, what is happening is that I can no longer recognize you,'' although he knew who she was and knew that he was talking to her (Lhermitte, Chain, Escourolle, Ducarne, & Pillon, 1972). They do not seem to be able to combine the many individual features, which they are able to perceive, into a whole. One prosopagnosic patient

related how ''I can see the eyes, nose and mouth quite clearly but they just don't add up'' (Pallis, 1955). The point that we emphasize here is the residual ability of such patients, their capacity to see much but not to combine everything into a whole. If we suppose that the area in the fusiform gyrus implicated in the perception of familiar faces, and damage to which leads to prosopagnosia, is a distinct node, then the clear implication of the above is that the patient is able to experience what the antecedent nodes have processed, namely, the details of the face. More simply stated, the capacity of the patient is related to the physiological capacities of the intact parts of the processing system.

Cerebral Achromatopsia or the Inability to See the World in Color

This is in fact a somewhat complex syndrome, with different consequences (Zeki, 1990a; Rizzo, Smith, Pokorny & Damasio, 1993) and different degrees and time courses of recovery, presumably depending upon the extent of the lesion in the fusiform gyrus (Damasio, 1985) (see below). Achromatopsia provides even more compelling grounds for supposing that the processing and perceptual systems are one and the same. The syndrome is one in which patients either cannot see colors at all, describing the world in shades of ''dirty'' gray or one in which they can see some colors, more often reds, but not others, more commonly greens and blues (Zeki, 1990a), a condition which we refer to as dyschromatopsia. In the latter condition, a patient's ability to ''discount the illuminant'' (Helmholtz) is much impaired, with the consequence that they are not able to construct constant colors and hence cannot see colors in a stable way, like normals (Kennard, Lawden, Moreland, & Ruddock, 1995). This is also true of monkeys, where lesions to the V4 complex have been found to result in an incapacity to construct constant colors (Walsh, Carden, Butler, & Kulikowski, 1993). In humans, the damage that causes achromatopsia invariably involves area V4, which actually turns out to be a complex of at least two areas which we term V4 and V4α (Bartels & Zeki, 1999; Zeki & Bartels, 1999) (Fig. 2). But V4 is only one node in an extensive color processing-perceptual system that extends from V1 to the inferior temporal cortex (Zeki & Marini, 1998). Damage to the V4 complex may, and often does, leave the antecedent parts intact. The consequence is interesting and can be related directly to the physiological capacities of the nodes that are left intact by the lesion. It has been found, for example, that achromatopsic patients are able to discriminate remarkably well, and consciously, between lights of different wavelengths, even if they are not able to ascribe colors to them (Victor, Maiese, Shapley, Sidtis, & Gazzaniga, 1989; Vaina, 1994). Like humans, monkeys with V4 lesions can also discriminate between light of different wavelengths though with raised thresholds (Heywood, Gadotti, & Cowey, 1992). This, we believe, reflects the physiological capacities of the wavelength selective cells in V1 and probably V2, which respond when light of the appropriate wavelength is flashed into their receptive fields, without being concerned with the color of the stimulus in their fields (Zeki, 1983b).

An interesting insight is provided by an achromatopsic patient who had retained the ability to detect the border between two equiluminant colors, without being able to distinguish the colors on either side of the border (Heywood, Cowey, & Newcombe, 1991). The authors of this study seek a complicated explanation for this residual capacity, by supposing that there are two specialized prestriate areas, one special-

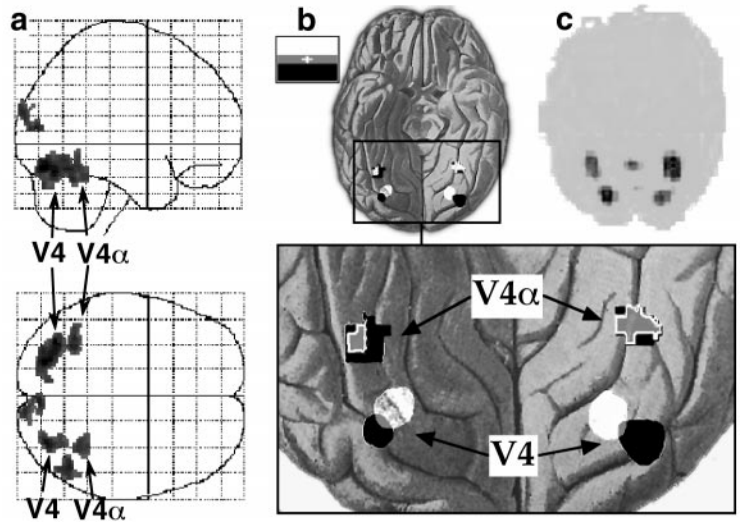

FIG. 2. Equating the processing system with the perceptual system: The human color center (the V4 complex) (a) is selectively activated when subjects view a multicolored abstract Mondrian scene in which the wavelength composition of the light coming from every point changes continually, without changing the perceived color of the patches, thus mimicking what happens when subjects view a multicolored scene under different illuminants. This color center is composed of two areas, the posterior, retinotopically organized area V4 and the anterior area V4α (b and c). (b) Projection of the activity obtained by either upper field (in red) or lower field (in green) stimulation with color vs. stimulation with black and white onto a ventral view of a human brain (group of four subjects; thresholds: V4; $Z = 4.81$, $p < .05$ corrected; V4α, $Z = 3.09$, $p < .001$ uncorrected); this shows that whereas V4 is topographically organized, V4α is not. (c) An independent component analysis separates independent maps of brain activity without *a priori* knowledge about the stimulus conditions. The isolation of the complete V4 complex from all other brain activity (shown here in the glass-brain view of a single subject's brain) indicates that V4 and V4α act independently of other areas as a functional unit. Because it is damage to this very area that leads to the syndrome of cerebral achromatopsia, when subjects are no longer able to see the world in color but only in shades of gray, we are led to equate the processing system with the perceptual system.

ized for the conscious perception of color and the other for extracting contours from color. But a simpler explanation might lie in the physiology of orientation cells in V1. These cells, though responsive to lines of particular orientation, will respond to a border between two equiluminant stimuli of the same orientation, without caring much about the color on either side of the border (Gouras & Kruger, 1979; Thorell, et al., 1984). This is precisely what the patient could discriminate, without being able at the same time to detect the difference between the two stimuli, just like a V1 interblob cell. V1 constitutes a node of the color processing system and, in the absence of V4, there is at present no good reason to suppose that the activity of such cells in it does not have a perceptually explicit correlate. And it is interesting to note in this context that cells such as the one described above, located in the interblobs of V1, project to V4, either directly or through the thin stripes of V2 (Livingstone & Hubel, 1984b; Zeki & Shipp, 1989; Nakamura et al., 1993). In brief, the knowledge of these achromatopsic patients reflects such physiological capacities as the wavelength selective cells of V1 and V2 have.

Akinetopsia, or Motion Imperception

Akinetopsia (Zeki, 1991) is a syndrome of motion imperception following cortical damage, more specifically to area V5, the motion center in the cerebral cortex (Zeki, Watson, Lueck, Friston et al., 1991; Watson, Myers, Frackowiak, Hajnal et al., 1993; Tootell & Taylor, 1995). Perhaps the best example is provided by the patient of Zihl (Zihl, Von Cramon, & Mai, 1983; Zihl, Von Cramon, Mai, & Schmid, 1991). Because of a bilateral lesion involving area V5, his patient is unable to see objects when in motion but only when they are stationary. This does not mean that she is unable to detect the presence of motion per se. Indeed, one study of this patient concludes that ''the overall deficit . . . is characterized by a large discrepancy between detection [of motion], which is relatively unimpaired, and discrimination, which can be severely impaired'' (Hess, Baker, & Zihl, 1989). This same study, as well as more recent ones (Shipp, de Jong, Zihl, Frackowiak, & Zeki, 1994), has shown that the patient is able to detect certain kinds of simple, slow, motion, leading the authors to say, ''Thus, the overall results suggest that the local component information necessary for the derivation of motion is intact and thus that the anomaly occurs at a later stage, where a more global analysis takes place'' (Hess et al., 1989). We take this to be a reflection of the capacity of the direction plus orientation selective cells of her intact area V1 and possibly those in areas V2 and V3 as well.

Because V1 is not directly connected to the frontal lobes, it has been suggested that we are not aware of what happens in V1, thus excluding V1 from direct involvement in conscious experience (Crick & Koch, 1995). This may well turn out to be so, and we have no compelling evidence that it is not. On the other hand, the evidence reviewed above could be interpreted to imply that activity in V1 itself has a direct conscious correlate. This is emphasized further by recent experiments in which a patient blinded as a consequence of a severe heart attack nevertheless retained the ability to see colors (Humphrey, Goodale, Corbetta, & Aglioti, 1995). But our further examination of him revealed that his color vision (which is completely divorced from form vision in that he is not able to perceive the form of the colors which he describes correctly) is wavelength based. In other words, he is not able to construct constant colors. Imaging experiments show that, when he discriminates colors according to wavelength, the activity in his brain is located in area V1 (Zeki, Aglioti, McKeefry, & Berlucchi, 1998). Even in spite of this, we are diffident about saying that activity in V1 has a conscious correlate, because we have no means of knowing what residual, and undetected, activity may have occurred elsewhere.

Thus, each of these syndromes provides evidence that activity in the intact part of the processing systems can have a perceptual correlate. There is much in the evidence that is incomplete. We do not have an adequate account of the total extent of the lesions in most cases, and in some none at all. Even if we did have such an account, there is no knowing at present how much of the system is compromised by damage to the output fibers. In spite of these difficulties, collectively the evidence is compelling in showing that there is always a residual visual capacity with lesions of the prestriate cortex and that this residual capacity is at present best explained by the physiological properties of the cells that are left intact by the lesion.

This leads us to Proposition 12: Damage to the prestriate component of a specific

processing system does not lead to the total loss of the capacity to see and understand the relevant attribute. Instead, the patient is left with a residual capacity to see and experience the elements of that attribute.

Clinical Evidence for Conscious Experience of an Attribute Not Processed by V1

If processing is separate from conscious perception and antedates it, as is implicit in the term ''processing,'' and if processing and perceptual sites were spatially separated in the brain, then one would expect that removal of a cardinal processing stage would lead to a condition in which neither the percept nor its conscious experience would be possible.

But what if the contrary is true, and activity at each node of a processing system does have a conscious perceptual correlate, even if it is disconnected from an antecedent cortical node which would be expected to process the signals in readiness for its perceptual experience? Such evidence does exist and comes from a study of the Riddoch Syndrome (Zeki & ffytche, 1998). This results from lesions of area V1 and was first described by George Riddoch during the Great War (Riddoch, 1917). So improbable were his conclusions that they were immediately dismissed by Holmes (1918) and relegated to oblivion for about 70 years (Zeki, 1991).

Riddoch had been studying British soldiers hit by enemy fire and blinded by lesions to their occipital cortex, and more particularly area V1. His perimetric studies had shown that, though blind when tested with static perimetry, they were not so when tested with dynamic perimetry. Crucially, he repeatedly describes his patients as being ''conscious'' of the motion, but not of much else besides (Riddoch, 1917). He explains, for example, that ''patients with restricted visual fields from occipital wounds . . . were immediately conscious of 'something' moving'' but he also writes that conscious awareness was restricted to the perception of visual motion, the subjects being ''. . . quite sure that neither shape nor colour could be atttributed to [the movement],' the nature of the movement being ''vague and shadowy'' (Riddoch, 1917). His explanation for this phenomenon was improbable: he supposed that the mechanisms of visual motion within V1 were spared, which is why his work was so easy to dismiss. But more recent studies confirm his observations. For example, our study of patient GY (Barbur, Watson, Frackowiak, & Zeki, 1993; Zeki, 1997), blinded by a lesion to his occipital lobe during childhood, showed that the patient could not only discriminate accurately the direction of fast-moving, high-contrast objects but that he was conscious of the direction of motion, in that he could describe it verbally. Interestingly, he first told us that the movement he saw was that of shadows, similar to the perception of motion when a normal individual, with eyes closed, can perceive the shadow when a hand moves against daylight. Later, he described this percept as that of a dark shadow against a dark background and a ''feeling'' that something was moving. An examination of the ''blindsight'' literature for other patients blinded by lesions in V1 shows similarly that they are commonly conscious of the visual stimuli presented to their blind fields (Zeki & ffytche, 1998). Sometimes subjects have a ''feeling'' but are ''absolutely sure of it'' (Weiskrantz, 1986), sometimes they see ''shadows'' or ''pinpoints'' of light (Weiskrantz, 1986; see Zeki & ffytche, 1998, for a review). Imaging studies show that area V5 is active when GY

is shown fast-moving stimuli which he can experience consciously (Zeki & ffytche, 1998). It would thus seem that preprocessing by area V1 is not a necessary precondition for the conscious experience of motion and that the notion that ''conscious vision is not possible without V1'' (Stoerig & Cowey, 1995; Stoerig, 1996) receives little support from these studies.

The ability of GY to experience consciously fast-moving visual stimuli presented to his blind field is almost certainly the consequence of a direct input from the pulvinar to V5, an input that bypasses V1 (Cragg, 1969; Benevento & Rezak, 1976). It is because of this alternative input to V5, curiously described as one ''which may not reach consciousness'' (Bullier, Girard, & Salin, 1994), that one can still obtain specific directionally selective responses from the cells of V5 in monkeys with lesions of V1 (Rodman, Gross, & Albright, 1989; Girard, Salin & Bullier, 1992). In fact, the imaging and psychophysical experiments on GY show that the transfer of signals along an equivalent alternative pathway in the human not only activates V5 but that that activity has a conscious correlate (Zeki & ffytche, 1998). Electroencephalographic experiments coupled with imaging ones have shown that this alternative pathway delivers signals from fast moving ($>5°$ sec^{-1}) objects to V5, in GY just as in normals, whereas signals from slowly moving ($<5°$ sec^{-1}) objects are delivered to V5 through V1 (Beckers & Zeki, 1995; ffytche, Guy, & Zeki, 1995, 1996). It is not surprising to find therefore that GY is able to experience consciously fast, but not slowly, moving stimuli. We do not suppose that only activity in V5 generates this conscious correlate. Imaging studies show that there are critical sites in the brain stem which are more active during conscious experience (Zeki & ffytche, 1998) and these may act as enabling systems—for example, through neuromodulation. The point we are making here is that to perceive fast motion and have a conscious experience of it does not require the mobilization of nodes antecedent to V5 in the V1–V2–V5 pathway.

The above results lead us to the conclusion that activity at a single node of a processing system, in this case the motion processing system, can have a conscious correlate, without necessarily involving antecedent stages of the visual pathways. This conclusion is supported by further experiments; for example, the fast circular motion that is perceived by humans when viewing the static work of Leviant entitled *Enigma* correlates with the selective activation of one node of the motion processing system, area V5 (Zeki, Watson & Frackowiak, 1993), while the motion aftereffect and the mental imagery of motion correlate mainly with activation of V5 (Tootell, Reppas, Dale, Look et al., 1995; Goebel, Khorram Sefat, Muckli, Hacker, & Singer, 1998). Similarly, the perception of afterimages induced by color correlates with the selective activation of one node of the color processing system, the V4 complex (Sakai, Watanabe, Onodera, Uchida et al., 1995). Hallucinations constitute another condition that lends itself to isolating neural processing directly responsible for specific visual experiences. Patients suffering from the Charles Bonnet syndrome have visual hallucinations that can be rather restricted—e.g. to the perception of objects, faces, colors, or textures. The brain activities during such hallucinations have been located to the regions in the ventral occipital cortex that are specialized for the corresponding attributes (ffytche, Howard, Brammer, David et al., 1998) without involving V1.

This leads us to Proposition 13, which is an extension of Proposition 9: Activity at a node of a processing system which is deafferented from antecedent nodes can have a conscious correlate, provided there is an input to it.

Imaging Evidence to Equate the Processing and Perceptual Systems

Perhaps the most convincing experimental evidence to date for equating the processing with the perceptual system comes from studies of color vision. Color is the end result of a complex series of operations; these depend on the physical properties of light and the surfaces reflecting it on the one hand and on the operations evolved by the brain to compare the relative efficiency of different surfaces for reflecting lights of different wave bands on the other. The brain evidently computes the ratio of light of any given waveband reflected from one surface and from surrounding surfaces. Computational theories have proposed different implementations for these operations which allow the brain to ''discount the illuminant,'' as Helmholtz (1911) called it, in order to construct constant color, which is independent of the wavelength composition coming from a single surface alone. The critical step in a color generating operation is thus ratio-taking (Land, 1974; Courtney, Finkel, & Buchsbaum, 1995), which enables the nervous system to compare the amount of light of a given wave band reflected from a given surface, with the amount of light of the same wave band reflected from surrounding surfaces. In spite of changes in the wavelength composition of the illuminant, which entails a change in the absolute amounts of light of different wave bands coming from every part of the scene, the ratios always remain the same; it is this operation that allows the nervous system to ''discount the illuminant'' and assign a constant color to a surface. The details of the neurophysiological implementation are not clear but the color pathways are relatively well understood and include the specialized compartments of areas V1 and V2, area V4, and further areas within the medial temporal cortex of both monkey and man (Zeki & Marini, 1998). Damage to the human color center, and more specifically to the V4 complex, results in the syndrome of achromatopsia if the lesion is bilateral and hemiachromatopsia if it is unilateral (see above). Interestingly, hemiachromatopsic patients are commonly unaware of their loss (e.g. Paulson, Galetta, Grossman, & Alavi, 1994, and our unpublished results), thus suggesting that the V4 complex itself is the conscious center for color. The V4 complex in fact consists of two areas, V4 and V4α. Of these, V4 has a distinct topographic organization, whereas V4α does not (McKeefry & Zeki, 1997; Bartels & Zeki, 1999; Zeki & Bartels, 1999)[1] and both

[1] The recently ''discovered'' ''new'' color center in the brain, termed V8 (Hadjikhani, Liu, Dale, Cavanagh, & Tootell, 1998) is in fact nothing more than a rediscovery of V4 as described in detail by McKeefry and Zeki (1997). This is accepted by Tootell and Hadjikhani, who have written that ''We assume that the cortical area described by McKeefry and Zeki is equivalent to our area V8, . . .'' (Tootell & Hadjikhani, personal communication) with which it shares the identical brain (Talairach) coordinates and hence does not provide any new insights into the conscious basis of color vision, as assumed by Heywood and Cowey (1998). This ''new'' color center does not include V4α, which evidently was beyond the resolution of the techniques employed by Hadjikhani et al. (1998). On the other hand, we and others have found it difficult to confirm the existence of area ''V4v'' of Tootell and his colleagues (Tootell, Dale, Sereno, & Malach, 1996) reputedly representing upper visual fields only (see also Kastner, DeWeerd, Desimone, & Ungerleider, 1998).

appear to be involved in the processing of colors, be they of abstract or naturalistic scenes. Indeed, sophisticated new techniques, such as independent component analysis applied to fMRI data, show that the two areas can act as a functional unit (Zeki & Bartels, 1999). But it is possible that the variation in the degree of severity resulting from lesions in the V4 complex (Damasio, 1985) is the consequence of unequal damage to this zone in different patients. If V1 and V2 were merely the processing stages and the V4 complex a mere perceptual one, then one would expect humans with a total damage to V4 not to be able to experience color at all, which is in fact what does happen. But supposing one were to try to mimic the real world, by asking humans to view a multicolored scene in which the wavelength composition of the light reflected from every part changes continuously, without changing the perceived colors (color constancy), because the ratio of light of any given wave band reflected from one part and from adjacent parts remains constant. Comparing brain activity evoked by this highly demanding task for the color system with the activity evoked when subjects view a static (and therefore computationally less demanding) multicolored scene, where would one expect the maximal activity to occur, which would identify the critical site of the operations undertaken to generate constant colors? Recent experiments (Zeki & Bartels, 1999) have shown that the ratio-taking operation is localized to the V4 complex and involves areas V1 and V2 only minimally (this is in contrast to experiments that compare the brain activity produced by viewing colored and black and white stimuli, when both V1 and V4 become strongly active (McKeefry & Zeki, 1997). This then adds to the clinical evidence reviewed above to suggest strongly that the processing site that is necessary for the generation of colors is the very site which, when damaged, leads to the syndrome of achromatopsia. It suggests, in summary, that the processing and perceptual site are one and the same (Fig. 2).

The evidence presented in the above section leads us to Proposition 14, which is an extension of Propositions 9 and 13: The processing sites and the perceptual sites are one and the same.

VII. THE AUTONOMY OF THE PROCESSING SYSTEMS

Implicit in the above discussion, and especially those of Sections III and IV, is the supposition that the different processing-perceptual systems are fairly autonomous of one another, that one can execute its functions more or less satisfactorily without the participation of the others. The admittedly incomplete clinical evidence does in fact suggest that the different processing systems operate with a fair degree of autonomy (Zeki, 1998). It has unfailingly and routinely shown that a lesion affecting the prestriate component of one processing system can lead to a specific perceptual incapacity, without affecting perception globally. This is implicit in all clinical evidence which shows a specificity of defect. Good examples are those of achromatopsia, akinetopsia, prosopagnosia, and what we shall term kinetic and akinetic object agnosia, conditions in which patients may only be able to perceive forms when they are in motion (Botez & Şeqebranescu, 1967; Bender & Feldman, 1972; Kertesz, 1979; Humphreys & Riddoch, 1987) or ones in which they are only able to see objects generated from luminance, not from motion (Regan, Giaschi, Sharpe, & Hong, 1992). The pedants would argue that in many examples of lesions in the prestriate cortex, the incapacity is not limited to one attribute, and the pedants are actually quite right, as their habit

usually is. Achromatopsia, for example, is commonly accompanied by prosopagnosia. This is a consequence of the opportunistic nature of lesions which are commonly not restricted to the territory of a given area. There is nevertheless a sufficient number of cases of achromatopsia unaccompanied by prosopagnosia, and vice versa, to render the pedantic argument nothing more than a tiresome distraction requiring a patient explanation.

All the evidence shows that area V4 of the human brain is critical for the perception of colors. Consistent with the nature of visual field representation in V4, the achromatopsia can even be quadrantic. It is noteworthy that, in the pure state, such patients can recognize and name objects, directions of motion, and depths; they can read and write and, to all intents and purposes, their general vision is good, apart from the achromatopsia. Moreover, even though achromatopsia is commonly accompanied by prosopagnosia, given the proximity of the cortical sites involved in processing color and familiar faces within the fusiform gyrus, there are cases of prosopagnosia unaccompanied by achromatopsia (Michel, Perenin, & Sieroff, 1986) and vice versa (Duvelleroy Hommet, Gillet, Cottier, de Toffol et al., 1997).

The same specificity can accompany lesions that include the territory of human V5 but exclude other areas such as the fusiform gyrus. Here one finds that the resultant akinetopsia is not accompanied by an achromatopsia, prosopagnosia, or object agnosia (Zihl, Von Cramon, & Mai, 1983; Zihl et al., 1991). Again, such patients can read, write, and detect depths and colors correctly, thus adding to the evidence of the autonomy of these areas. It needs to be added that the full gamut of defects that patients with specific lesions in the cortex suffer from is not necessarily known; patients are obviously more intensively studied for those defects which they spontaneously complain of. It is therefore possible that when such patients are studied in greater detail, and when more of them become available, the full extent of the disabilities will be better charted. But to a good first approximation, the syndromes described above are remarkably specific.

It was Wechsler who, in 1933, described a remarkable case of carbon monoxide poisoning that had left its victim substantially blind without affecting his color vision, or at any rate affecting it much less (Wechsler, 1933). This relative sparing of color vision in patients blinded by hypoxia has been accounted for (Zeki, 1993) by supposing that the richer vasculature of the blobs of V1 (where wavelength selective cells are concentrated) protects them (Zheng, LaMantia, & Purves, 1991). Whatever the ultimate explanation may turn out to be, Wechsler's observation has been repeated several times (see Zeki, 1993, for a review) and there is little reason to doubt that hypoxic episodes, or cardiovascular attacks, can result in severe damage to the visual brain, while sparing color vision to a greater or lesser extent. This sparing of a given attribute when all others are compromised is further testament to the autonomy of the individual processing-perceptual systems.

This leads us to Proposition 15: The processing systems are fairly autonomous of one another.

VIII. INTEGRATION IS A MULTISTAGE PROCESS

There are several lines of evidence which suggest that integration must be a multistage process. Perhaps the most suggestive is to be found in the facts of anatomy.

Given that each processing-perceptual system is multinodal (Proposition 14), it is worth asking why the connections between the nodes constituting the different processing-perceptual systems occurs right from the start, at the level of V1 and V2 (Proposition 3), and why they are not deferred until after some terminal stage. The answer is simple: there is no terminal stage in the cortex (Proposition 4). Instead, activity at each node can have a perceptually explicit correlate (Proposition 9) and generate its own microconsciousness which is functionally specialized (Proposition 11)—if this were not so, the unique information present at each node would be lost for conscious perception. Given that in our ordinary daily life we see all attributes in perfect registration, it seems natural to suppose that the activity in the different processing systems is integrated. One would suppose that the perceptually explicit correlate generated by the functionally specialized cells at a given node must be capable of being integrated with perceptually explicit correlates generated by the activity of cells at other nodes. And hence the nodes are connected with each other, according to both the ''like-with-like'' and the ''like-with-unlike'' principles (Proposition 3). Because the like-with-like pathway is strictly hierarchical and the like-with-unlike pathway is not (Propositions 2 and 3), integration itself can be hierarchical or not, depending on whether it is of the preconscious or postconscious variety (Propositions 5 and 6). It seems important to emphasize that the connections between different nodes of different processing systems simply allow for communication and integration between them, but that each node can function rather autonomously, and this includes the generation of a microconsciousness. It remains to be investigated when and to which degree such parallel binding between nodes actually occurs in normal subjects in the daily life.

For activity at each node of a processing-perceptual system to have a perceptual (and therefore conscious) correlate (Proposition 11) confers advantages in that it increases the number of perceptual repertoires. This would be reduced if the processing systems had to report to a ''terminal'' station—either a common one or individual ones—for integration to occur. Such an hypothetical integration area would have to code in a perceptually explicit way the results of the processing at each node separately as well as in the required combinations. A more economical way would be to render the activity at each processing site perceptually explicit, which can then be bound. The number of pairwise connections between N nodes equals $N * (N - 1)/2$. Even given the constraints of cortical connectivity, this would still create a vast repertoire which would not be possible if integration could occur only between ''terminal'' points or ''final'' stages. Our conjecture that each node corresponds to a perceptual site (Proposition 13) means effectively that there are far more such sites in the cortex than would be possible if there were only a terminal perceptual site for each processing system. Moreover, if the result of processing at a given node is not made perceptually explicit, it would be lost in later processing stages and no longer be perceptually accessible. The function of many nodes in a processing system is to discard some information in order to extract more global information. For example, a picture of a face composed of small dots will activate areas whose cells respond to dots and other areas whose cells respond to faces. Neither of the two stages explicitly codes information that the other stage explicitly codes for. The only way to preserve both types of information—both the dots and the face—is to make activity in

both areas perceptually explicit. It would be wasteful for the brain to make only the information of the anthropomorphically defined ''final stage''–the face area—perceptually explicit. Another example is color vision. The cells in V1 and V2 which are sensitive to wavelength composition (Zeki, 1983a,b) cannot code for color, which, by definition, remains stable despite changes in wavelength composition (Hering, 1877; Helmholtz, 1911; Land, 1974). It is the activity of cells in V4 that correlates with color (Zeki, 1983a). The information that the cells in V1 and V2 code for excludes them from coding simultaneously for the information coded for by cells in V4 (and vice versa). Nevertheless we are aware of what each set of cells codes for—the color of a surface and changes in the illumination condition.

If different and often mutually exclusive types of information are made explicit at different processing stages, it becomes tempting to suppose that percepts created at each stage of a processing-perceptual system can be bound to other percepts created by the activity at other stages within a given processing-perceptual system. This is especially so when activity in a single area is important for registering an attribute, no matter how that attribute is derived. For example, recent experiments show that the same area in the fusiform gyrus is activated when humans view objects generated from luminance and from motion (Bork & Zeki, 1998; Grill Spector, Kushnir, Edelman, Itzchak, & Malach, 1998). But once generated from motion, for example, one would suppose that the form has to be reintegrated with an earlier stage of the motion pathway to bind the form to the direction of motion. However, the degree to which such binding of percepts is really necessary remains an open question, especially since our perception may not be as unified as it is commonly believed to be.

Each processing-perceptual system has a certain hierarchical structure, by which we mean that the visual attribute is processed at a more complex level at a given stage than at the antecedent one (Proposition 2). The theory of multistage integration (Zeki, 1990b, 1993; Bartels & Zeki, 1998) nevertheless supposes that there is no perceptual hierarchy in binding since the perceptually explicit activity of cells at a relatively ''low'' level in one processing-perceptual system can be bound with the perceptually explicit activity of cells at a relatively ''high'' level of another, or the same, processing-perceptual system. A good example is provided by a green bus as it emerges from the shade into sunlight. The bus remains a bus and its color remains green, but the intensity of the light and even its shade change. The recognition of the bus as a bus requires the activity of cells in an area at a high level in the visual pathways (the fusiform gyrus) but the recognition of a change in the shade of green, and in both the intensity and wavelength composition of the illuminating light, depends upon the activity of cells in V1, and possibly V2. A functional corollary of this is that at any given time, many functional units—consisting of stages at different levels of different processing systems—are formed dynamically, with the same stages constituting different units with other stages at another time. The functional units that are formed therefore criss-cross between different stages of different processing-perceptual systems (Zeki & ffytche, 1998). It remains open whether these functional units are defined by binding or whether the mere activity in an area is sufficient to make the generated percept part of our seemingly unified perception. They are in a dynamic state and the pattern of functional units formed between different stages of different processing-perceptual systems at any given time should be amenable to

capture by imaging methods. The functional units formed will be further dynamically shaped by attentional and mnemonic factors.

TOWARD A THEORY OF VISUAL CONSCIOUSNESS

The propositions that we have given above form a chain which leads us towards our theory of visual consciousness. We have more confidence in some than in others. We are, for example, very confident of Propositions 1–4, 7, 12–13, and 15. Although the remaining ones do not carry the same levels of confidence on their own, they are so consistent with each other and with the known facts that, when considered as a whole, they are able to lead us toward a theory of visual consciousness, which we outline below, and which might be applicable to other parts of the brain:

We suppose that visual consciousness consists of many, functionally specialized, microconsciousnesses which are spatially and temporally distributed if they are the result of activity at spatially distributed sites (as in the case of color and motion). This we believe to be the direct consequence of the fact that the several, parallel, multinodal, functionally specialized, and autonomous processing systems are also perceptual ones and that activity at each node of each processing-perceptual system can become perceptually explicit. Activity at each node therefore has a microconscious correlate which is functionally specialized and asynchronous with the microconscious correlate generated by that at other nodes. If integration occurs between different nodes, the communication between them must influence the micro-consciousness that each creates in a consistent way, leading to consistent, integrated percepts. The communication itself does not create the bound percept. Since activity at each node can become perceptually explicit, it is imperative that the integration that may occur must be multistage and not hierarchical, leading us to the view that perceptual integration itself is multistage (indeed, our theory of multistage integration can be equally well called a theory of perceptual integration). It is therefore not surprising that there is no terminal station in the cortex, since activity at each node represents, in a sense, a terminal stage of its own specialized process, when it becomes perceptually explicit and acquires a conscious correlate. It is, we believe, the communication between nodes that changes the nature of the microconsciousnesses such that they generate a mutually consistent and integrated image in the brain. This leaves us with the grand problem of how, in physiological terms, the microconsciousnesses are bound together. Indeed, it raises the question of whether they are bound at all, given what appears to be the nonunitary nature of conscious experience.

Like Ramon y Cajal, one of our greatest, we do not wish to give our theory a dogmatic character. Like him, we are too well aware that neurology has been a graveyard for interesting ideas which can be replaced from one day to the next by unforeseen facts. But like him, too, we hope that something of the principles on which we base our theory will remain.

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