When is information represented explicitly in blindsight and cerebral achromatopsia?

R. W. Kentridge

Psychology Department, University of Durham, Durham DH1 3LE, United Kingdom. **robert.kentridge@dur.ac.uk www.dur.ac.uk/~dps1rwk**

Abstract: Discrimination of forms defined solely by color and discrimination of hue are dissociated in cerebral achromatopsia. Both must be based on potentially explicit information derived from differentially color-sensitive photoreceptors, yet only one gives rise to phenomenal experience of color. By analogy, visual information may be used to form explicit representations for action without giving rise to any phenomenal experience other than that of making the action.

I am largely sympathetic to O'Brien & Opie's (O&O's) position that consciousness might be built from many components and that consciousness of each of these components depends on their explicit representation. I do not, however, see that this model is necessarily inconsistent with neuropsychological dissociations between consciousness and performance.

O&O refer to a number of neuropsychological conditions in their article. In particular, they cast doubt on the dissociation between consciousness and performance in blindsight, based on the arguments of Campion et al. (1983) and they refer to the independence of processing modules as revealed by neuropsychological deficits of motion or color perception (Sacks 1985; Zeki 1993; also see Meadows 1974 and Zihl et al. 1983 as primary sources). The criticisms of the phenomenon of blindsight raised by Campion et al. (1983) were well dealt with at the time in commentaries, but since then further evidence has amassed that residual visual function in blindsight cannot be explained by scattered light (King et al. 1996), changes in subjects' decision criteria (Azzopardi & Cowey 1997; Kentridge et al., in press) or islands of spared visual cortex (Kentridge et al. 1997; Stoerig et al. 1998). O&O characterize blindsight as a subcortical phenomenon; they may not be right in this. Anatomically, it is possible for visual information to reach the cortex via projections that bypass striate cortex (see, e.g., Stoerig & Cowey 1995). Evidence from a recent functional magnetic resonance imaging study suggests that although visual stimuli do not elicit any activation in the damaged striate cortex of a blindsight subject, they do produce activation in extrastriate cortical areas (Stoerig et al. 1998).

This need not affect O&O's position if they are right in their suggestion that although blindsight subjects do not have normal visual experience in their blind fields, they do have other types of phenomenal experience associated with visual stimuli.

The extent to which residual visual ability and consciousness are dissociable after lesions to striate cortex is, therefore, still an issue crucial to O&O's position and worthy of further discussion. O&O are right that subjects with residual vision in scotomata caused by striate cortex lesions often report some awareness of events in their blind regions. If, however, subjects are asked to report, on a trial by trial basis, whenever they have any such experience whatsoever, there is a clear dissociation between their residual visual abilities and their awareness (see, e.g., Weiskrantz et al. 1995; Kentridge et al. 1997; Zeki & ffytche 1998). In other words, there are conditions in which these subjects do not report any phenom-

enal experience associated with a stimulus, yet are still able to make a correct explicit judgment about its nature. Does this inevitably lead us to some form of executive or process theory of consciousness or can a vehicle theory survive?

O&O's model implies that any cortical stimulus representation that is explicit, in that it supports stimulus specific behaviors, must also give rise to consciousness. I have noted that this does not appear to be the case in blindsight. There are many other neuropsychological examples. In cortical color blindness subjects not only deny conscious experience of the hue of stimuli, they are incapable of discriminating hues in forced-choice tasks (Heywood et al. 1998). Nevertheless, when presented with a stimulus consisting of a figure and background that differ from each other only in color (i.e., they are equiluminant), they can effortlessly (and consciously) see the figure (Barbur et al. 1994; Heywood et al. 1994). Color information that is present but presumably not explicitly represented in the subjects' undamaged brain regions could give rise to an explicit and conscious representation of form without giving rise to conscious experience of color.

O&O note that different brain regions subserve different functions (for example, motion perception) and that these functions can be computed on the basis of a variety of different types of stimuli (motion might be perceived on visual or auditory stimuli, for example). In my cortical color blindness example we might suppose that the module capable of extracting form from color differences (or, more specifically, from differences in the activation of cells responsive to specific wavelengths of light at different points in space), luminance differences, texture differences, and so on, is intact, whereas the module in which hue is extracted from ratios of wavelength specific activations at common points in space (a quite different calculation) is damaged. In other words, the second module depends on cells that are selectively activated by light with specific spectral content, while the first may utilize differences in the responses of color sensitive (but not color selective) cells across a color border: these cells signal color variation without coding the nature of the hues which comprise the border. Hue may be potentially explicit in both of these modules, but the structure of the network only makes it explicit in the second.

I hope O&O will have no problem with this distinction between potentially and actually explicit representations in cerebral achromatopsia. Can blindsight be dealt with in a similar manner? The activities of cells driven by pathways originating in the eye depend on the way in which cells in the retina respond to the number and wavelength of the photons which fall upon them. Extracting the surface properties of objects like brightness or texture from this signal requires computation just as computations have to be made to extract hue from a number of different wavelength sensitive activations. Is there any reason to believe that all the modules that process this visual signal give rise to visual experience? The extraction of form from color does not give rise to a color experience. The dorsal stream leaving the primary visual cortex and passing up to the parietal cortex is strongly associated with action based on visual stimuli (Milner & Goodale 1996). Is it unreasonable to suggest that components of the visual signal that are potentially explicit elsewhere are extracted in this stream and give rise to action (and an awareness of action) without giving rise to a visual experience?