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The Case of Blindsight

LAWRENCE WEISKRANTZ

Chambers Dictionary, among others, provides a concise definition: Blindsight – a condition caused by brain damage in which a person is able to respond to visual stimuli without consciously perceiving them. It is associated with damage to human primary visual cortex (otherwise known as striate cortex or area V1, which causes blindness in parts of the affected visual fields, with a size and shape to be expected from the classical retino-cortical maps (Holmes 1918). If, however, subjects are required to guess about stimuli presented to their blind fields, they may be able to locate them in space or to discriminate them from each other, despite saying that they do not see them and have no awareness of them (Pöppel, Held, & Frost 1973; Weiskrantz et al. 1974; Weiskrantz 1998). (It is worth nothing that in clinical cases damage to V1 is rarely complete. Typically it is confined to one cerebral hemisphere and therefore the region of blindness in most patients is restricted to one half of the visual field [hemianopia] or less, located contralateral to the damaged hemisphere. In everyday life the normal half-field is sufficient for most visual negotiations, and a hemianopic human or monkey would appear to be quite normal to the casual observer.)

The historical origin of the oxymoron "blindsight" stems from animal research. The primate retina, including that of humans, sends its major nerve tract (after a relay in the lateral geniculate of the thalamus) to the visual cortex (striate cortex, V1). When this cortex is blocked or removed (with histological confirmation of the completeness of the V1 removal) in monkeys, the animals can still carry out visual discriminations, albeit with certain changes. Such a residual capacity is, in itself, not surprising because the optic nerve leaving the retina also traverses a number of routes, reaching other targets in the brain located mainly in the midbrain and thalamus (Cowey & Stoerig 1991). These targets, in turn, provide relays that project widely to a number of other regions in the brain (Cowey & Stoerig 1991). These routes remain intact even if V1 is removed or damaged. The extra-striate tracts from the eye contain fewer nerve fibers than those in the pathway that normally reaches V1, only about one sixth as many, but this smaller number is not trivial. For example, the pathway from the retina to the superior colliculus in the midbrain contains about five times as many fibers as there are in the whole auditory nerve. Animal research also demonstrates that residual capacity can be improved by repeated practice with stimuli in specific regions of the blind field. And so there is no mystery in the fact that animals can make some visual discriminations in the absence of V1: the mystery is that human subjects are blind.

Blindsight first emerged when human subjects were tested in the manner with which one is forced to study visual capacity in animals (Pöppel, Held, & Frost 1973; Weiskrantz et al. 1974), leading to the realization that the methodology is typically deeply different in humans than in other animals. Humans are usually asked to give verbal descriptions or to comment on visual appearances and differences, whereas animals are trained to reach for the location of visual events or to make alternative choices for which they are differentially rewarded, necessarily devoid of any commentary. When a human subject is asked to make a discrimination between, say, two colors, there is typically an explicit verbal instruction about the color as such, and more importantly there is an important implicit assumption that the subject will be aware of that attribute and make a report about it accordingly. But an alternative is to test a human subject in a manner that is closer to animal methodology, for example, to be instructed simply to reach toward the stimulus or to make a forced-choice "guess" about the visual stimuli. Forced-choice guessing entails asking whether a visual event is, say, located at position A or B, whether its color is X or Y, whether it falls in the first or second of two temporal intervals, or whether its shape or brightness is different in the first or second interval. When tested in such a forced-choice discriminative way, independently of verbal responses or commentaries, human subjects can sometimes match the performance of monkeys with visual cortex damage even though they may lack any acknowledged awareness of visual stimuli that they nevertheless can tell apart. Hence, the term "blindsight."

Blindsight is but one example of a number of dissociations in brain-damaged patients between an intact capacity and absence or altered awareness (Weiskrantz 1991, 1997). For example, good storage can be demonstrated in amnesic patients with medial temporal lobe damage for events that they say they do not "remember," visual responsiveness can be demonstrated in a "neglected" field by patients with unilateral visual neglect, good differential sensitivity can be found to unfamiliar faces vs. familiar faces by prosopagnosic patients who have no recognition of the faces as such, and "blind touch" or "numbsense" can be demonstrated in parietal lobe patients with loss of touch sensitivity, which appears to be a homolog of blindsight in the tactile mode.

The visual parameters that blindsight subjects have been reported to be able to discriminate include color, orientation of lines or gratings, simple shapes, motion, onset and offset of visual events (for reviews, see Stoerig & Cowey 1997; Weiskrantz 1998; Weiskrantz 2003). Attention can also be controlled by unseen cues in the blind field controlling the responses to loci of unseen targets (Kentridge, Heywood, & Weiskrantz 1999). Recent research also has found that the emotional expression of unseen faces in the blind field can be guessed at better than chance levels (deGelder et al. 1999). In connection with possible extra-striate routes in the absence of V1, it is of interest that fearful and fear-conditioned faces generate differential amygdala responses in blindsight subject GY, and that the amygdala responses covary with neural activity in the posterior thalamus and superior colliculus (Morris et al. 2001).

Blindsight, however, is altered compared to the capacity of normal vision. By varying the spatial frequency of a sine-wave grating until it can no longer be discriminated from a homogenous patch, it is possible to measure the subject's acuity. It is reduced by about two octaves in spatial frequency, relative to the normal seeing hemifield, but is still creditable (Weiskrantz 1998). Contrast sensitivity is also reduced. There is good motion sensitivity for the detection of simple displacement of bars or solid spots, and there can be sensitive judgments about the direction of movement of a bar or a spot (Weiskrantz, Barbur, & Sahraie

1995). More complex patterns of motion, however ("third order motion"), seem to be seriously affected (Azzopardi & Cowey 2001). Color discrimination remains (Barbur et al. 1994), again in the absence of any experience of color per se, although there is a relative shift in sensitivity toward the long wavelengths (red) and a decrease in sensitivity to middle wavelengths (green). Otherwise the shape of the spectral sensitivity curve is qualitatively normal, although with reduced sensitivity. Also, the change in spectral sensitivity under dark adaptation is preserved (Stoerig & Cowey 1992).

Damage absolutely restricted to the visual cortex occurs relatively rarely in clinical patients. Animal work with monkeys has made it clear that if the damage extends outside the striate cortex, the residual visual capacity is reduced (Pasik & Pasik 1982). Therefore, most human blindsight research has concentrated on a small number of well-chosen subjects (e.g., DB, GY, FS, CS), with appropriately restricted pathology (and who are willing to endure the long testing sessions). This self-imposed restriction, however, may be too conservative. Current research suggests that residual visual function occurs in the majority of cases of visual cortical damage if additional brain damage is only moderate and if a common metrical range of spatio-temporal parameters (Sahraie et al. 2002) is used in each case.

Given the counter-intuitive nature of blindsight, early skepticism naturally led to questions about its validity (just as was true for earlier examples of implicit processing in neuropsychology, e.g., intact memory in the absence of "remembering" in amnesia). It has been suggested by Campion, Latto, and Smith (1983), for example, that there may be stray light falling in the intact visual field, or that the cortical lesion in particular cases may be incomplete and patchy (Wessinger, Fendrich, & Gazzaniga 1997) or that subjects really see but deny this, perhaps because of a very conservative criterion, or that their vision is really essentially qualitatively normal but the percepts are rendered very faint because of the brain damage. All of these alternatives are important, but have been directly addressed in various focused reviews, experimental analyses, and by MRI and ERP analyses of the lesions of blindsight patients (cf. Weiskrantz 1998, 2001, 2003; Azzopardi & Cowey 1997; Kentridge, Heywood, & Weiskrantz 1997). In particular, stray light has been stringently controlled, especially by the use of stimuli equiluminant with the background, and the use of the optic disc (blind spot) as a control region. Regarding incomplete lesions, subjects such as GY have been extensively and repeatedly mapped in MRI and the lesion is found to be complete except for the most posterior region, corresponding to the small area of macular sparing in the visual field (which, of course, is not used for testing blindsight), and islands of intact vision are not found in him (Kentridge, Heywood, & Weiskrantz 1997). Of course, in the monkey (in whom there is "blindsight"; see below) the completeness of the V1 lesion can be confirmed histologically. Regarding criteria in signal detection terms, the use of criterion-independent two-alternative forced-choice psychophysical methods still reveal blindsight. Blindsight as studied in subjects such as DB cannot be simulated by weak normal vision (Azzopardi & Cowey 1997), and in any event there are aspects of the subject's commentaries that are not touched by signal detection theory (Weiskrantz 2001). This subject continues to provoke lively discussion not only among neuroscientists but also among philosophers and others interested in the nature of conscious awareness and its putative neural basis.

Under certain conditions, blindsight subjects may say that they are aware that something is happening, they may feel it. This is especially the case when a stimulus contains rapid transient onsets and offsets, or moves very rapidly. This has been labeled "Blindsight Type 2" in contrast to "Blindsight Type 1," when discriminations occur in the total

absence of any acknowledged awareness (Weiskrantz 1998). The distinction between Blindsight Type 1 and Type 2 has allowed one to carry out functional brain imaging contrasting states *with* awareness and *without* awareness, in both conditions using simple movement discriminations, which can be carried out with a high level of success – 90 percent or better (Weiskrantz, Barbur, & Sahraie 1995). In the unaware condition, but not the aware condition, activity is seen in the superior colliculus of the midbrain (Sahraie et al. 1997). This structure also was active in a blindsight subject in response to red stimuli but not to equiluminant green (Barbur et al. 1998), in line with the greater sensitivity of blindsight subjects toward the red end of the spectrum. In contrast, in the aware Type 2 state, dorsal cortical areas, especially foci in the right prefrontal cortex, are active. Such research reflects one of the strong interests of neuroscientists in blindsight and related phenomena in seeking routes to unravelling the neural mechanisms that underlie conscious awareness. The distinction between Type 1 and Type 2 is not assumed to be absolute and binary – it is possible, in principle, that there may be gradations; the distinction between its two extreme states has heuristic value.

The range of stimuli that can be discriminated by blindsight subjects – from simple form to facial expression to color to location of spatial locus by reaching or saccading – is such as to discount any simplistic relationship between them and the differential capacities of the ventral and dorsal cortical streams (Milner & Goodale 1995). Also, fMRI evidence exists that robust activation in either the dorsal and ventral streams occurs given the appropriate visual stimuli (e.g., movement vs. colored objects), leading to the conclusion that neither dorsal nor ventral cortical activity is sufficient in blindsight to generate conscious vision, nor that there is an imbalance between the two streams in blindsight (Goebel et al. 2001).

To be asked to discriminate stimuli that they cannot see is a patently strange request, and some subjects balk at it (as, indeed, do some experimenters in issuing the instruction). Therefore, other counter-intuitive methods of assessing residual function commend themselves, especially for screening of brain-damaged subjects for possible rehabilitation (with repeated practice of stimuli in the blind field). Some of these methods depend upon asking the subjects to discriminate stimuli lying entirely in their intact, seeing hemifields, but with the experimental demonstration that their performance can be altered by the presentation of stimuli in their blind fields, which can enhance or interfere with intact perception (see review by Weiskrantz 1990). Visual reflexes can be used: the most quantitatively sensitive method depends upon changes in the diameter of the pupil, which constricts not only to increase in light energy, but to a wide variety of stimuli without any energy change, including color, movement, and spatial frequency of sine-wave gratings. By varying spatial frequency of sine-wave gratings, the contrast sensitivity and acuity of the blind field can be accurately measured by pupillometry (Barbur & Thomson 1987), with results that mirror the psychophysical capacity as measured by forced-choice guessing. From these one can identify a narrowly-tuned spatiotemporal visual channel that remains in the absence of V1, a peak sensitivity in the region of 1–3 cycles per degree (Barbur, Harlow, & Weiskrantz 1994). Also, sensitivity to color and complementary color after-images can be detected (Barbur, Weiskrantz, & Harlow 1999). The pupil can also be used to measure similar capacities in animals, where verbal report of course is impossible (Weiskrantz, Cowey, & Le Mare 1998).

Finally, given that the existence of residual visual capacity was first definitively demonstrated in animals with visual cortex lesions, the question arises as to whether they too show blindsight for the discriminations they can perform. Recent experiments yield a positive

answer (Cowey & Stoerig 1997; Stoerig & Cowey 1997). Monkeys with unilateral removal of V1 can detect and locate light stimuli with impressive sensitivity in their affected hemifield. They can also readily be trained, of course, to make differential responses in their normal visual hemifields for lights vs. non-lights (blanks). But when the same lights are projected into their affected field, the monkeys reliably treat them as blanks. That is, the very stimuli that they can detect with impressive sensitivity are classified by them as being blanks, as non-lights – just as a human blindsight subject does. Thus, the contribution made by the primary visual cortex to visual awareness appears to be similar in humans and other primates, and brings into common perspective and framework both the historical animal research and the more recent human blindsight research. Blindsight has made us aware that there is more to vision than seeing, and more to seeing than vision.

See also 14 Split-brain cases; 16 Coming together: the unity of conscious experience; 40 Preconscious processing; 42 Consciousness of action; 48 Duplex vision: separate cortical pathways for conscious perception and the control of action.

Further Readings

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Split-Brain Cases

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After the first callosotomy surgeries were performed, the general consensus among the medical community was that severing the corpus callosum had relatively little, if any, effect on an individual's behavior (Akelaitis 1941). Nearly twenty years later, it was quite a shock to discover that under experimental conditions, the two hemispheres could simultaneously maintain very different interpretations of the same stimulus. These findings immediately called into question the unity of subjective experience, a fundamental characteristic of human consciousness. How could the split-brain patient not experience any disruption in their experience as a unified self when the two hemispheres are physically and functionally disconnected? In this chapter, we review the research that has led to a conceptualization of the split brain as two minds within one body and the implications of this research for the scientific study of consciousness. We argue that consciousness is a neural function that emerges from the integration of information across available functional modules.

Characterizing Consciousness in the Split-Brain Patient

Since the first reports of hemispheric differences in information processing and impaired interhemispheric transfer in split-brain patients, there has been a great deal of interest in the subjective experiences of these patients. Despite the substantial literature documenting split-brain patients' reports of no alterations in senses of self following callosotomy surgery, the common interpretation of the split-brain condition is that disconnection of the two hemispheres results in a "splitting of the self." Given the prevalence of such misconceptions, we would like to take this opportunity to review what is known about the subjective experiences of split-brain patients and how this information shapes our understanding of neural bases of consciousness.

Bilateral representation of fundamental sensory information

Both hemispheres of the split-brain patient receive ascending projections from a common brainstem, enabling duplicate representation of a great deal of basic sensory information. Both hemispheres receive proprioceptive information, automatically coding the