Roots of Blindsight

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Abstract: The paper reviews the historical background to demonstrations that there is residual visual function in the total absence of striate cortex (V1) in monkey and humans. The late 19th century evidence by Munk and others, as reviewed by William James, was that this was not possible in humans, and doubtful at best in monkeys. It has gradually become realized, starting in the middle of the 20th century, that even total bilateral removal of striate cortex in monkeys does not abolish all visual capacity, including spatial and pattern vision. The situation regarding unilateral or incomplete bilateral lesions in the monkey did not become clarified until Cowey’s doctoral work in the 1960s, demonstrating that field defects were not absolute, that sensitivity continued to improve over several months of postoperative testing, that the size of the field defect gradually shrunk, that the sensitivity was poorest at the centre of the field defect, and that recovery was not spontaneous but depended on sustained practice. In human subjects with unilateral lesions from the 1970s onwards, using forced-choice methodology parallel to animal studies, a wide range of visual discriminations was demonstrated but with alterations or complete absence of acknowledged awareness by subjects (blindsight). Various varieties of skepticism are discussed and rebutted. The gap between humans and animals was diminished by the demonstration by Cowey and Stoerig that monkeys, like humans, classify responses to blind-field stimuli as being “unseen”. Further recent degrees of closure and developments in human blindsight research are discussed.

Background of Monkey Research

It is always risky trying to identify the very first seeds from which a plant’s roots have sprung. It is not clear when the concept first emerged, but “blindsight” as a word in the English Language (“a condition in which the sufferer responds to visual stimuli without consciously perceiving them.” Oxford Concise Dictionary) first surfaced in 1973. One nurturing source, however, was the long history of animal research on occipital cortex, which had demonstrated that primates could carry out visual discriminations in the absence of visual cortex. (see Weiskrantz, 1972, 1998). Admittedly there was a wide range of views, even extreme disagreements, and the answer was slow to emerge. Luciani (1884, p.153), for example, had concluded that "some time ....after their extirpation, [monkeys] visual sensations becomes perfect again; they are able to see minute objects, what they want is the discernment of things.....; they are deficient, in a word, of visual perception," but his conclusions regarding the lesion were doubted by William James, without citing his evidence. James was a great believer in foreign sources, and so he referred (1886) to Luciani’s work only in the German, failing strangely to refer to an excellent English translation (by one of the editors of the Brain) of the original Italian, which had appeared in that journal in 1884. James thereby ensured, unfortunately, that most of his English readers would take the Lucianis prescient view no further. Ferrier (1886) had concluded that the occipital lobes were completely dispensable for visual function (did not cause "the slightest appreciable impairment"), but it is almost certain that the lesions were incomplete. William James, on the other hand, cited Munk to the effect that bilateral occipital lesions in the monkey caused permanent and total blindness. In fact, Munk allowed that "very gradually [the monkey’s] vision slightly improved so that he will not bump into things" (1881, translation by von Bonin, 1960, p. 106) and, depending on the limits of the lesion, recovery could be much more complete. James also quotes Schäfer’s work (1888), at the time correctly, that blindness was permanent. But later Schäfer (1900) concluded "the blindness was not permanent, unless the lesion extended somewhat in advance of what is generally taken to be the limit of the lobe, on the inner and lower surface." (1900, p. 753). James ended up hedging his bets; he suggested that occipital removal "makes the animal almost blind......a crude sensitivity to light.....may remain," (my italics), adding, if so, that "nothing exact is known about its nature or its seat. (1890, p. 63).”

But work in the first half of the 20th century established clearly that monkeys without striate cortex could respond to light. Thus, Marquiss and Hilgard (1936) demonstrated that a conditioned
eyelid response to light could be demonstrated in monkeys with complete striate cortex removal (and with histological confirmation of the lesion and complete degeneration of the lateral geniculate nucleus). Heinrich Klüver's work (1927, 1942) was widely accepted as providing the best empirically-based description of the nature of the "crude sensitivity" that James allowed as a possibility. Klüver concluded that in the absence of striate cortex monkeys (with rather extensive lesions exceeding the limits of striate cortex) could only discriminate the total amount of luminous energy and not its pattern or distribution. Weiskrantz's study (1972) suggested an extension to an integration of all retinal ganglionic activity, not just for luminous flux, so as to include total contour length and movement. Subsequent work, however, made it clear that the capacity was richer than could be accounted for by mere summation of activity. For example, in their seminal research the Pasiks and colleagues, Pasik and Pasik, 1971, 1982; Schilder et al., 1972) demonstrated pattern discrimination and brightness discrimination, both with total flux equated, in monkeys with total removal. They also measured the visual acuity and orientation discrimination thresholds, demonstrating good, but reduced capacity. Humphrey (1970, 1974; Humphrey and Weiskrantz, 1967) taught monkeys to respond to a variety of "salient" visual events, demonstrating simply and elegantly by requiring the animal merely to reach out and touch the source of a visual event. He found that the animals could respond to small objects and to their location. It could not have been explained by random eye movements to intact regions (if any) of the visual field because it was later demonstrated that monkeys with striate cortex removal can locate stimuli randomly located in space, and do so with durations too brief to allow an eye movement to be executed before the stimulus disappeared (Weiskrantz et al., 1970. Humphrey's and other's evidence, in turn, could be linked to the original "two-visual system theory," in which cortex was postulated to mediate visual identity, and the subcortex to mediate the detection of visual events (Ingle, 1967; Schneider, 1967; Trevarthen, 1968). It was influential at the time, and still deserves to be.

If the situation regarding total striate lesions in the monkey historically was slow to gel, it was even more uncertain concerning subtotal lesions - which are the directly important comparison with human clinical cases, on which the evidence for human blindsight eventually was based. Bilateral and total lesions in clinical patients in the absence of lesions well outside of visual cortex are rare. Human subjects with unilateral damage, both in clinical examination and in everyday life, demonstrate a region of blindness in the visual field, or at least some part of it, that is rendered defective by a V1 lesion, and well-known maps in textbooks plot the correspondence between lesion and field defect. The measurement of the field defect clinically, visual perimetry, is based on the subject's responses to the presence of a light presented systematically over the whole visual field. The instruction is simply "tell me (or press a key) when (or whether) you see a light." Whether it yields a fair representation of the human visual defect is another matter to which I will return later. But if monkeys, in seeming contrast, possess some residual function even with total removal, it is not clear what would, or should, result from a subtotal lesion. If there was a corresponding defect in a specific region of the monkey's visual field, would it be an absolute hole, would it be fuzzy at the edges, or indeed would there be any hole at all (Weiskrantz, 1961)? The answer is difficult to reach just by looking at the animals because, aside from some transitory misreaching for food, the animals appeared to be quite normal (Cowey and Weiskrantz, 1963; Weiskrantz, 1972).

**Cowey's Monkey Perimetry Results**

To have a definite answer one must have good evidence as to where the animal is looking at the crucial moment when visual capacity is measured objectively. The bare minimum one wants for perimetric charts in the monkey is reliable evidence about the detection of a visual event as a function of its position in the animal's visual field. This had never been obtained for the monkey before 1960. Indeed, the eminent physiologist, John Fulton, declared in his influential neurophysiology textbook (1949, p. 345) that perimetry in the animal is "virtually impossible." Earlier, Settlage (1939, p. 106) had commented that "there is no known method of perimetric examination of the monkey's visual field."

That is how matters stood when Alan Cowey started his doctoral research in the late 1950s. In fact, the effort started even earlier, while he was still an undergraduate. At Cambridge we had a kind of research project auction in which ideas where thrown out to the assembled Part II Psychology students for their undergraduate research projects. Richard Gregory and I had earlier discussed the eye position issue together in relation to visual cortical lesions, and he made the clever suggestion of using highlights on the eye to assess eye position - the highlights have very useful optical properties. And so I tossed out this idea for grabs to the student meeting, and Alan took it up and perfected it. He proceeded to demonstrate that
photographs of highlights could be used to assess eye position in human subjects reliably and with an error of no more than 2 degrees, and even lateral head movements (of about 1° eye diameters) could be tolerated. And so the necessary groundwork was done.

In his subsequent doctoral research, completed in 1961, Cowey applied the measurement technique to monkeys with visual cortex lesions. The animals’ differential behavioral responses to random sequences of lights or blanks - differential key presses - were recorded. The method allowed him to tell where the animal was looking (typically, gazing fixedly at a small mirror in which the animal could see the reflection of its own eye), and hence performance could be related to eye position and thus to the position of the stimulus in the visual field. It was no mean task - it was, in fact, heroic. Training and analysis took up to 100,000 trials per animal, but the results were clear (Cowey, 1962; 1963; 1967; Cowey and Weiskrantz, 1963): local impairments were found approximately where they would be predicted from known primate anatomy and electrophysiology, for example that of Talbot and Marshall (1941), and Daniel and Witteridge (1961). And so on this point Ferrier and others were wrong. But the main conclusion had to be qualified in several very important ways. The defect was by no means an absolute one; in fact, it was only when the intensity was reduced by several log units below the training level that the defect could be defined reliably. Secondly, the ability of every animal improved markedly and radically during several months of postoperative testing. In fact, it was necessary to continue to reduce the flash intensity further and successively in order for the defect to be revealed. Thirdly, there was a gradual shrinkage in the size of the defective region over the same period. And so on these two points Munk was right and James was wrong. Fourthly, thresholds were not distributed uniformly, but were highest in the centre of the defective region of visual field and lowest at the edges. Fifthly, the gradual improvement probably did not occur spontaneously, because an animal who was not tested for two years postoperatively showed an unchanged picture on retesting, although it did then show the typical gradual improvement with subsequent retesting. Two tests conclusively and painstakingly ruled out that the results were due to artifacts based on stray light (Cowey, 1962, 1963). (For reviews, see Weiskrantz and Cowey, 1970, Weiskrantz, 1972).)

The landmark findings of Cowey’s still stand unchallenged. They proved that the monkey’s visual defect is not absolute, and that its size and sensitivity need not be permanent. Clear and reliable quantitative determinations of the detection thresholds within and across the field defect were obtained, both before and after intervals of time and after practice. The benefits of practice were shown in an elegant study by Mohler and Wurtz (1977), who demonstrated that practice in a portion of the monkey’s field defect can lead to a differential improvement in sensitivity in the practiced region relative to the unpracticed region.

Given that the monkey's field defects are not absolute, and human perimetry charts show regions of absolute blindness, it is not surprising that it continued to be widely assumed that there is a major evolutionary separation between the visual systems of these two species of primates, even though the neuroanatomy seems closely similar: In both species there are parallel pathways originating in the retina, projecting to non-cortical targets that might allow visual function to be sustained in the absence of the cortical target. Thus Marquis (1935) and Monokow (1914), among others, appealed to a doctrine of encephalisation of function, such that the higher the phylogenetic status of the animal, the more vision is dependent upon the visual cortex and its integrity, i.e., visual processes that must be presumed to be mediated subcortically in the monkey are elevated to an absolute dependence on the cortex in the human. That position, in some ways, carries some curious implications (see Weiskrantz, 1961, 1977), but I just note here that such an eccentric view has not altogether disappeared today.

**Human Research Background**

But, leaving perimetry aside for the moment, in other respects, just as with the monkey, opinions regarding the effects of V1 damage in humans were widely divided well into the middle of the 20th century. William James was in no doubt. “The literature is tedious ad libitum....The oc- cipital lobes are indispensable for vision in man. Hemispheric disturbances come from lesion of either one of them, and total blindness, sensorial as well as psychic, from destruction of both.” (1890, p. 47). A similar conclusion was advanced by Gordon Holmes, the doyen of British neurologists during World War I, who held the view that “severe lesions of the visual cortex produce complete blindness,” although he added that if the lesions were “incomplete” an amblyopia could result in which objects appeared indistinct, although moving objects might “excite sensations” (1918, p. 384) - (in other words, parenthetically, he suggested that Riddoch’s claim that some patients could see moving stimuli but not stationary ones was based on incomplete lesions). On the other hand, in striking contrast, Holmes’ neurological counterpart with the
German forces, Walter Poppelreuter (1917; see 1990 translation), declared that he could never find an absolutely blind scotoma with occipital damage; some rudimentary function was always present, which was also the conclusion of Wilbrand and Saenger in 1917 in their study of several hundred cases. A similar conclusion was drawn by Teuber et al. in their influential 1960 monograph of World War II and Korean War brain-damaged soldiers. They reported that there was no permanent visual blindness in the field defects in any of their brain-damaged group of more than 46 patients, even though their perimetry charts showed absolute blind regions. They especially advocated the use of adjunct methods based on dark adaptation, t-scope presentation of forms, recognition of hidden figures, perception of apparent and real motion, and so forth as methods of increasing sensitivity. Teuber, however, later was more cautious about his colleague’s evidence - and also of Poppelreuter’s - because of the problem of diffusion of light into intact visual fields. And, of course, the issues were and are always complicated by the vagaries of the limits and complexity of lesion sites in clinical cases.

And so, surprisingly, about the largest, and the most intensely studied system in the brain, the visual system, by the mid-20th century there was still controversy regarding the limits and permanence of field defects caused by cortical damage in human subjects. Fundamentally, the situation had not advanced that much beyond the position at the end of the previous century. In some ways we were further ahead in characterizing the field defects of monkeys, in relation to the site of damage, than we were for human subjects.

Whatever the ultimate answer is about permanence and absoluteness, the point is that during clinical examination, and in everyday life, human subjects who have sustained a V1 lesion resolutely insist that they do not see the stimulus in a region of the visual field. But given the earlier results showing that monkeys with V1 lesions can respond to visual events and discriminate them, and given that the human visual system has a similar anatomical organization to that of the monkey, why is the human field defect blind? Are we forced to conclude that there is a qualitatively distinct quantal evolutionary gap?

There are two important differences in the comparison between human and monkey. Human perimetry charting is typically based on the subject reporting “yes”, (or pressing the “yes” key) when a stimulus detected. The monkey perimetry carried out by Cowey used two alternatives, essentially, “yes, I detect”, and “no, I do not.” Secondly, of course, the human subject is instructed and reports verbally (even if there is a response key, he or she would have been instructed as to its use verbally). And the verbal instruction would inevitably be of the form, “press”, or “say yes”, when you see a light.” It would be possible to do monkey perimetry using only a single “yes” key, but at the expense of large numbers of false positives and shifts in response criteria. But it is not possible to instruct the monkey verbally, and certainly not to instruct it to report its actual visual experience.

**Human Blindsight Research with Animal Methodology**

If one is comparing monkey and human, therefore, one must try to compare like with like methodologically. That means, first, dispensing with the luxury of asking human subjects what they see, but, second, to require a discrimination along the lines of animal testing, using forced-choice methods. (An obsessional investigator would even dispense peanut rewards if the research grant would rise to such lavishness.)

The first group, as far as I am aware, who deliberately dispensed with a dependence on subjects’ verbal reports were Pöppel et al. in 1973. Animal evidence by Denny-Brown and Chambers (1962) and Humphrey (1974) had noted that monkeys with total visual cortical removal would direct their eyes to novel visual events. Pöppel et al flashed a light briefly in different locations of the field defects caused by gun-shot wounds in war veterans, and encouraged them to look in the direction in which the flash had just occurred. The point of departure in their study was not that they recorded visual reflexes, in fact, they did not, but that they asked their subjects voluntarily to look the direction in which the flash occurred. In other words, they engaged an instrumental response, an operant response, if you will. The instruction caused some considerable puzzlement in their subjects, because they could not actually “see” the flash, but with encouragement they “played the game”. There was a weak but significant positive correlation between the original target position and the position taken up by the eye, at least for eccentricities out to about 25 degrees.

Shortly afterwards a patient, DB, was seen at the National Hospital who had undergone surgical removal of a tumour from the right calcarine fissure, and who appeared to be able to locate events in his blind hemifield better than one might have expected from clinical perimetry. Elizabeth Warrington and I, and colleagues, confirmed the eye movement result of Pöppel et al., but then we carried out a range of tests based on monkey
testing, as for example opened up by Nick Humphrey’s work and the Pasiks (Weiskrantz et al., 1974). We followed it up in this way for a further 10 years, including detection and visual acuity, motion directional discrimination, orientation discrimination, spatial localization, summarized in book form (Weiskrantz, 1986, 1998). DB often reported being completely unaware of the stimuli he could detect or discriminate. Soon afterwards work on another hemianopic subject, GY, was started up by Keith Ruddock, John Barbur and colleagues (Barbur et al., 1980) and this expanded into a world-wide enterprise with this subject. Cowey some years later also pursued work on human blindsight research, actively combining it with his animal work. This is not the place for a review (several are available, Cowey and Stoerig, 1991; Stoerig and Cowey, 1997; Weiskrantz, 1998; 2001b) - the important point was that subjects seemed to be able to make visual discriminations in the clinically blind fields even though they acknowledged no awareness of them. Blindsight as a term got attached almost by happenchance, when I quickly had to find a title for a seminar to the Oxford neurologists, (“blindsight and blindsight”). The response requirements for the visual discriminations were forced-choice, typically with two verbal responses (red/green; present/absent, grating/uniform field; moving/not moving; moving left/moving right, etc.) or preferably, two response keys. To avoid response bias, some tests employed a two-interval forced-choice paradigm, (was the specified target stimulus in the first or second temporal interval?), usually with essentially the same result as with two forced-choice alternatives in a single interval.

Thus, it would appear that, in terms of objective performance, the subject with a restricted V1 lesion can respond to visual stimuli even in the areas of the visual field that for the human are defined as absolutely blind. In neither the monkey nor the human is there an absolutely functionless visual field. In both species two (or more) alternative choice methodology is used to demonstrate this. But there still is an incomplete gap in this comparison between monkey and human. That is, we generally would assume, as a matter almost of definition if not of preconception, that the monkey must be aware of the stimulus when it presses the “present” key, whereas even when the human subject correctly signals the difference between present and absent (or a variety of other binary choices along other dimensions) he nevertheless asserts that he does not perceive it, but is doing it by guessing as insisted upon by the experimenter. DB would frequently judge, after a block of trials, that he was just performing at chance (“pure guesswork”) even when his performance was better than 90%, and express puzzlement when shown the results. In one memorable experiment on GY using a Posner attention paradigm, he commented to Kentridge, the experimenter that he might as well stop because as far as he was concerned nothing was happening. (Kentridge et al., 1999). (Fortunately the experiment went on!). Another kind of response of subjects, if they do not deny having any experience whatever, is that they “know” or “feel” that something is happening, especially with rapidly moving or sudden transient stimuli, and yet do not “see” anything. We will return to the apparent monkey-human gap later in this essay.

Commentaries and Points of Controversy

The relationship between the strength of the discriminatory performance and the subject’s awareness of the stimuli can be studied more directly by providing keys not only for the discrimination itself but also, on separate keys, for the subject’s reports of presence or absence, or some degree, of awareness, which we have termed “a commentary key paradigm” (Weiskrantz, 1986; Weiskrantz et al., 1995; Sahraie et al., 1997). Thus, there are two scales: one for discrimination, the other for awareness. Blindsight, as is the case with all implicit residual phenomena in neuropsychology, is essentially a disjunction between the two psychophysical scales in contrast to their close bonding in normal, intact function. I have argued that this is a more satisfactory and direct route to interpretation than trying to appeal to signal detection theory to derive presence or absence of acknowledged awareness from the discrimination data themselves. Like ning the difference between the blindness revealed in clinical perimetry and its absence in blindsight psychophysics to a difference between a “yes/no” response requirement in clinical testing, which allows for a response-criterion shift, and forced-choice guessing in blindsight, which is criterion independent, Azzopardi and Cowey (1997) arranged for both the “yes/no” and two alternative forced-choice discriminations to be independent of response bias. The result was there still was a difference between the two modes of discrimination for the blindsight subject but not for the normal subjects, leading to the conclusion that “blindsight is unlike normal, near-threshold vision” (1997, p. 14190). However, and this is the critical point, even if there had been no such difference, the subject’s reported commentary would still be that there was no awareness, and this would be the case whether it was forced-choice yes/no or 2AFC (Weiskrantz, 1995, 2001a). Nor can awareness be
finessed by *fiat* from an implicit assumption, taken by some to be almost a matter of definition, that a significant d’ *necessarily* entails awareness of the discriminative stimuli. An off-line commentary is not equivalent to a bias in the on-line discrimination task.

As this is a paper about roots, it is worth mentioning that almost at the very outset somehow the qualifier “controversial” got attached, barnacle-like, onto “blindsight” in the same way that “red” is attached to “pillar box” or “deep” is to “sea” (no pun intended.). Perhaps it was just part of the same background characterized by William James:

“....the quarrel about the function of occipital cortex is very acrimonious; indeed the subject of localisation of functions in the brain seems to have a peculiar effect on the temper of those who cultivate it experimentally.” (1890, p.46).

Aside from the continuation of such a vigorous tradition, another reason for controversiality is that blindsight - among the whole set of neuropsychological implicit syndromes - is the most deeply counter-intuitive. One no longer finds evidence of long-term retention by priming in amnesic subjects so surprising (at least, not now - although it was vigorously doubted at the outset) but to be able to *discriminate* without some degree of conscious *seeing* - impossible! It naturally attracts skepticism. Soon after our 1974 paper with Warrington and other colleagues at the National, critiques were offered (see Campion et al., 1983): the phenomena could have been based on an artifact due to stray light into the intact visual field, or inadvertent eye movements to bring the stimuli into the intact field, or blindsight might be weak but normal vision, together with a change in criterion; or the striate cortex damage might be incomplete, and, finally, why so few cases? Such concerns deserve to be taken seriously, and they have been. The issues have been well ventilated and I do not propose to review them in detail here, but I will focus on one of them a little later. Briefly, stray light and inadvertent eye movements were already put to rest, or should have been, I believe, already by 1979 and were reviewed in my Bartlett Lecture (Weiskrantz, 1980), and later in the blindsight book (1986), at least for DB. The claim of weak normal vision, similarly, does not convince unless near-perfect performance in the blind field is taken to be weak vision. Azzopardi and Cowey’s conclusion, based on a signal detection analysis, countering the view that blindsight is like normal near-threshold vision, has already been alluded to above. Other suggestions (Gazzaniga et al., 1994; Fendrich et al., 1992) had to wait for better brain imaging techniques to demonstrate completeness of striate cortex lesions, at least within the limits of high resolution MRIs (Barbur et al., 1993; Baseler et al., 1999; Morland, personal communication; see also Kentridge et al., 1997; Weiskrantz, 1995 regarding visual “islands”). (Unfortunately MRI imaging is not possible in DB because of metal clips implanted during surgery.). Incomplete lesions are, of course, always a possibility given the vagaries of the clinical case material, but they can be ruled out as a general explanation. Of course, residual function in the monkey cannot be due to islands of intact striate cortex because there is histological confirmation, but some of the critics will not accept the animal primate evidence as having validity for the human visual system (Gazzaniga et al., 1994; see also Weiskrantz, 1995, 1998, and Weiskrantz et al., 1998 demonstrating a very close correspondence between monkey and human pupillometry and human blindsight psychophysics.). In sum, I believe there is now direct evidence concerning all of the issues, at least in critically studied subjects, except the question of small numbers of subjects. But I do wonder sometimes why the term “controversial” perseverates. The term has enduring adhesive and somewhat disengaging properties. Aside from the territorial imperative that is commonly a root of attack in science, I believe one reason for continuing debate stems from a positive attribute, because it is an example in cognitive neuroscience that entails the conjoining of specific evidence with theoretical and philosophical as well as scientific implications at both the neurological and the perceptual and conceptual psychological levels. It occupies a very large arena, and there is a lot to tempt a skeptic. Critiques can and have come at it from several different angles. It is not just whether there is implicit perception, with all the issues entailed there, but whether it specifically applies to hemianopia and specifically and uniquely to V1 lesions, whether there is subcortical mediation of unconscious perceptual discrimination, what might be the role of visual reflexes versus commentaries, whether blindsight might be a route to the neural correlates of conscious perception, what are the evolutionary and phylogenetic differences, if any, in neural organization between human and non-human primates, is there a comparison with putative examples of blindsight in normal subjects, and a host of issues raised by philosophers regarding the relevance of the empirical evidence to the philosophy of mind, and especially of consciousness. I take such a very rich, tempting target as a large plus sign for cognitive neuroscience and hope it is a sign of the future.
Closing some Ellipses

Some major apparent gaps still remain to be discussed. The first is the matter of whether blindsight is rare, which is commonly assumed. Until recently only a small number of suitable subjects have been tested intensively, which has been taken by some to imply that blindsight is a rare condition. Even Azzopardi and Cowey start their paper (1997, p. 14190) as follows: “Blindsight is a rare and paradoxical ability of some human subjects with occipital lobe brain damage.” But we simply do not know how rare it is. In my Bartlett lecture I reported that I had found evidence of residual visual function in 14 out of 22 National Hospital patients with occipital damage in scotomata judged by clinical assessment to be absolutely blind; and I could not rule it out in the other 8 cases (Weiskrantz, 1980). But this was carried out under far from ideal conditions, on acute cases, and with no possibility of thorough psychophysical investigation or follow-ups. More recently Arash Sahraie and his colleagues in Aberdeen have started to tackle the question head-on by testing a population of hemianopic patients with a variety of aetiologies. But it is necessary to introduce two further matters in order to deal with his study, which I take to be the first really intensive survey to be initiated. The first is that until now there has been an absence of a common spatiotemporal metric to compare all hemianopes. Arbitrary choice of a particular and narrow set of stimulus parameters can lead one badly astray, as Weiskrantz et al (1991) showed in a repetition of a study by Hess and Pointer (1989) that had reported negative results for blindsight. These were confirmed using the same parameters, but with a slight change in temporal parameters the results were strongly positive. Sahraie’s basic paradigm (see Sahraie et al., 2002) is detection of sine wave gratings over a range of spatial frequencies with a range of temporal frequencies. Secondly, the question is how to characterize the mode in which patients say they “know” that something has happened, even though they do not “see” it, a commentary frequently uttered by DB and GY. When it occurs, it is generally for higher contrast and higher temporal frequencies than for those discriminations that can be carried out in the complete absence of any awareness, by sheer guess-work according to the subjects. I have called the latter, pure form, Type 1 and the other Blindsight Type 2. I recognize that the distinction may be a smooth and shaded one, rather than binary, and not everyone likes it, but one needs some sort of term. Petra Stoerig prefers “amblyopia” instead of Type 2, and indeed back in 1972 I also suggested that same term for the residual function in the monkey. But in our original 1974 human blindsight paper in *Brain*, we state why we do consider amblyopia to be inappropriate, at least for DB. I still adhere to this view that amblyopia, “fuzzy vision” is not an adequate or appropriate term. In any case, whatever one calls it, Arash Sahraie uses a commentary key paradigm to plot the thresholds of both Type 1 and Type 2 for all patients over a wide range of spatial frequencies. So far, in the first 10 patients tested thoroughly, 8 out of 10 show blindsight (personal communication). (The two that do not have deep lesions extending into the thalamus). I do not believe any Scottish hemianope will escape his attention, and so the issue of rarity is on the way to be solved. There certainly is no other way to do it, in my mind.

Another gap concerns the use of meaningful, especially emotional stimuli in the blind field, and their relation to the amygdala via an extrastriate route. Marcel (1998) reported that unseen meaningful words in the blind field could prime the meaning of ambiguous words in the intact field. DeGelder and her colleagues (1999, 2001, 2002) have published evidence showing that discrimination of facial expressions and also of evocative stimuli - e.g., puppy dogs versus spiders - is possible by GY and DB in their blind fields, calling the phenomena “affective blindsight.” fMRI evidence demonstrates activation of the amygdala by emotional visual stimuli, correlated with that in the superior colliculus and pulvinar, in the absence of striate cortex (Morris et al., 2001). (The link of blindsight with the amygdala in this way closes a personal ellipse for me, given that I started my research with the amygdala before embarking on visual research.). Cowey had reported in his original thesis research that monkeys with V1 lesions do not react behaviorally to emotional stimuli, such as snakes, or tempting food, but we do not know if there are reflexive autonomic changes or activation via the subcortical route implicated in humans, nor whether discriminations would be possible by formal testing.

Another elliptical path started in a somewhat misleading way, based on the possibility of a reflex response to visual stimuli. When the results of the monkey field defects were published, a suggestion was put to us that the monkeys may have been responding to their pupillary changes. Cowey and I showed that the behavioral results were unchanged when the pupils were paralysed with atropine. Of course the animals might still have been responding to the command signal controlling the reflex circuitry even though the final effector was blocked - an unsatisfiable possibility. But rather than dismissing the pupil, it has turned out to be a very important adjunct to the assessment of blindsight. Pupillary
changes occur to sine-wave gratings, movement, color, that are closely correlated with the forced-choice psychophysical findings of blindsight (Barbur and Forsyth, 1986; Barbur and Thomson, 1987). As such they provide a very useful screening method for its occurrence (Weiskrantz et al., 1999). In fact, pupillometry can actually be more sensitive than psychophysics. For example, it reveals the complementary colored after-effect of a colored patch presented to the blind field (Barbur et al., 1999), demonstrating the integrity of successive color contrast. As this is an “unseen” after-effect of an “unseen” stimulus, it would present a challenge for psychophysical demonstration.

After-effects connect with yet another ellipse. DB reports that he consciously perceives after-images following his fixation of “unseen” stimuli in his blind field. They are produced by a range of achromatic and chromatic stimuli, and approximate to Emmert’s Law (Weiskrantz et al., 2002). Several years ago it was reported that after-effects can be generated by invisible stimuli “fixed by imagination” (Weiskrantz, 1950; Oswald, 1956), which also conform to Emmert’s Law. To date no other blindsight subject has reported after-images, but the demonstration of after-effects by pupillometry suggest that they might occur but are below threshold for other subjects. DB, the longest-standing and practised blindsight subject, may well have recovered sensitivity which is only potential in other subjects. A rather more likely explanation for DB, however, stems from his history of migraine. There is evidence that migraineurs have an increased duration of visual adaptation effects (Shepherd, 2001; and increased amplitudes and defective habituation of evoked potentials; (Afra et al., 1998; Connolly et al., 1997). In any event, the presence of after-images in DB offers a unique opportunity to study conscious and unconscious attributes of precisely the same stimulus presented to the same position in the same visual field. Because of the surgical metal clips this cannot be done in DB with fMRI, but Cowey and I and colleagues are actively pursuing the matter with event-related potentials.

There is one final ellipse to discuss, and it is a major one. Alan’s seminal work on monkeys with V1 lesions, as we have seen, stood in contrast to the evidence on human subjects but it was also a backdrop against which the human subjects, like the monkeys, could be shown to possess to residual function. One essential route for the human research was the background work with monkeys, demonstrating that their field defects, as Cowey showed so clearly, are not absolutely blind nor static. Nor, it seems, are they in humans. But this is clearly demonstrable in humans only if the discrimination is not based on verbal judgments of “awareness” or “seeing”, but on forced-choice methodology, akin to those used in animal research. When commentary keys are provided for the human subjects they can report their awareness or lack of it in parallel with their psychophysical choices. But if the monkey had a commentary key, how would it judge the appearance of the stimuli? Would the monkey have blindsight?

No one has succeeded in training monkeys on a commentary key paradigm. But Cowey, together with Stoerig, has closed the gap in a logically related way. The commentary key is, essentially, an off-line judgment of a parallel discrimination. Cowey and Stoerig confirmed, firstly, that the animals can detect and localize stimuli very sensitively in their blind fields: when a light is randomly and briefly presented in the blind field, the monkeys detect it very well. But in a subsequent experiment the animals were trained to discriminate between “lights” or “blanks”, by rewarding them appropriately for differential touching either of two loci, in the intact hemifield, one for lights and one for blanks, in a random series of lights and blanks. They were rewarded for correct performance for both keys. The crucial question was how the animals would respond to lights in the blind field, the same lights for which the animals earlier had shown such exquisite sensitivity for detection and localization. The answer was that the monkeys consistently pressed the “blank” panel for lights in the blind field. The result was reliable and robust, even with increases in luminance and even when the stimuli were moving. (Cowey and Stoerig, 1995, 1997; Stoerig et al., 2002). And so the monkey performs just as a human blindsight subject does - good discrimination of stimuli which are classified as being non-visual. The monkey has blindsight. (Or at least one form of blindsight - we still do not know whether it is Type 1 or Type 2.). This is further evidence for the similarity in organisation between the primate and human visual systems.

It is time to draw things to a close. I started with Cowey’s definitive work on V1 in the monkey. Blindsight in human subjects, as a concept and as a empirical pursuit occurred more than 15 years afterwards, and the closure of the ellipse back to monkeys via blindsight, from Cowey and Stoerig’s work, much more recently. But from whence did any idea itself arise about the possibility of unconscious perception? Well, here is an extract from a paper by Alan and me, published in 1963.

“Discussions which attempt a comparison of vision in man and in other animals usually omit to say what we
mean by two of the terms most frequently used, namely see and blind. When we say that a human being sees a visual stimulus we mean that he had experienced something....if he is incapable of this experience we say he is totally blind; if visual stimuli fail to elicit the experience....we say the subject has an absolute scotoma. If the subject can respond to a light only because it makes him blink or because it can be used as a conditioned stimulus for blinking, we nevertheless say he is blind for he can tell us that he did not see the stimulus......Is it not conceivable that the monkey is much better equipped than man to utilize the effects of a visual stimulus as cues but that seeing a stimulus is organized in a very similar manner in the two species?” (Cowey and Weiskrantz, 1963, p.113)

The paper from which I quote was jointly authored, but I know that Alan wrote that section because I can remember discussing it with him quite intensely in draft form. In my own case I think the distinction between performance and awareness derived mainly from my being primed from the years of work with Elizabeth Warrington on implicit memory and amnesia, which accu-

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