

Continuing Commentary

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Commentary on John Champion, Richard Latto, and Y. M. Smith (1983) Is blindsight an effect of scattered light, spared cortex, and near-threshold vision? *BBS* 6:423–486.

Abstract of the original article: Blindsight is the term commonly used to describe visually guided behaviour elicited by a stimulus falling within the scotoma (blind area) caused by a lesion of the striate cortex. Such “vision” is normally held to be unconscious and to be mediated by subcortical pathways involving the superior colliculus. Blindsight is of considerable theoretical importance since it suggests that destriate man is more like destriate monkey than had been previously believed and also because it supports the classical notion of two visual systems. It is also of potential clinical importance, since it has been claimed recently that systematic practice in blindsight can lead to the recovery of normal visual function in patients with cortical lesions. From a review of the literature it is concluded that all of the phenomena of blindsight can be attributed either to light scatter into unimpaired parts of the visual field or to residual vision resulting from spared striate cortex. The possible contribution of other factors is also considered. It is concluded that blindsight studies have generally failed to control for such nonblindsight interpretations partly because of poor methodology and partly because of difficulties in defining the term “blindsight.”

Experiments were carried out to investigate the extent to which subjects can exhibit performance similar to blindsight when they are using scattered light as a cue. This was done both with hemianopic subjects (by manipulating the amount of scattered and direct light coming from a stimulus) and with normal subjects (by presenting targets within their blind spots). Good blindsight performance was observed when only scattered light was available as a cue to the subjects. It is therefore concluded that an adequate case for blindsight has not been made. It is probably impossible to demonstrate the existence of blindsight on purely behavioural grounds. What is required is the establishment of relationships between visual function and independent anatomical evidence.

On inferring blindsight from normal vision

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Champion, Latto & Smith (1983) have certainly done a fine job in clarifying the conceptual and methodological framework of blindsight research. Yet, their conclusion that it is impossible to demonstrate the existence of blindsight on purely behavioral grounds seems too pessimistic to us. In this commentary we would like to describe a simple method that avoids one of the major sources of artifacts correctly deemed by Champion et al. to plague blindsight research, namely, shifts in response criteria. In addition, our method employs light stimuli that can easily be shown not to be scattered into the intact field of hemianopic patients.

Before briefly describing our experiments we would like to devote a few words to an important point that has perhaps been overlooked in both the target article and the first round of commentaries, namely, the site and the extent of extrastriate damage. Various theories of the neural substrates of visual perception view the primary visual cortex as providing a basis for high-resolution vision whereas attentional and high-order aspects of perception are likely to depend on extrastriate areas (see Marzi 1983). It seems reasonable, therefore, to assume lack of blindsight in a hemianopic patient suffering from concomitant

damage in a putative attentional system which orients visual attention to a given portion of the visual field in and around the scotoma. Posner and his associates (see Posner 1980) have shown that visual attention can be allocated to various portions of space without overt movements of the head and eyes, and that this implicit visual orienting is very important for subsequent overt visually guided behavior. Now, part of the variability observed in blindsight experiments might stem from variations in the patient's capacity to shift attention toward his island of blindness. Such an inability might result either as a consequence of the brain lesions involving “attentional” areas or as a consequence of the paradigm used to test residual vision, for example, blocked versus randomized stimulus presentation. In patients with long-lasting field defects this might lead to a “disuse” of the hemianopic side and thus to irreversible total blindness. This might explain why some patients recover part of their vision following extensive training in which they are forced to pay attention to the damaged side (Zihl 1980).

As mentioned above, in our laboratory we are trying to verify the existence of true blindsight phenomena by using stimuli and paradigms that prevent light from scattering into the normal hemifield and decisional changes in response from occurring. Subjects are tested in a simple reaction time (RT) paradigm which employs as stimuli very brief (5 msec) light flashes of low suprathreshold intensity generated by small LEDs (light-emitting diodes). Under such conditions, light scattering, as assessed by measuring the blind spot (see Schmielau & Marzi 1983) is

absent. Following testing with normal subjects we were able to ascertain with great reliability (the effect shows up in every subject and usually in every session) that RTs are faster for responses to two flashes presented simultaneously than for responses to one flash alone. The stimuli were presented at 10° and 30° along the horizontal meridian in the same or in opposite hemifields. On each trial the subject was instructed to press a key as soon as possible following the onset of either one or two lights, no matter which hemifield the light or lights appeared in. The various stimulus conditions were randomly alternated, and the subject's fixation on a central point was continuously monitored through a TV system. It is important to point out that the effect of spatial summation was found not only for stimuli presented in the same hemifield but also when the two flashes appeared across the vertical meridian.

There are at least two advantages in using such a paradigm to verify the presence of blindsight in hemianopic patients. First, since number and position of the stimuli are randomly alternated, on each trial there is only a one in four chance of not seeing the stimuli (i.e. when both flashes appear in the blind hemifield), and there is no reason for a change in response strategy. In fact, patients are told in advance that they may sometimes fail to see the stimuli.

Second, the site of stimulation that is closer to the boundary of the intact visual field is 10°, a distance that, considering the small size and the low intensity of the flashes (not to mention their short duration) makes light scattering into the normal side quite implausible.

Eleven patients with homonymous hemianopia caused by an occipital lobectomy or by vascular lesions restricted to the occipital lobe have been tested so far in the same apparatus and with the same procedure as normal individuals. The rationale is as follows: In normal individuals two flashes appearing simultaneously at different points in either the same or opposite hemifields yield faster RTs than a single flash presented in one hemifield. A hemianopic patient should provide the same result as normal individuals only for stimulation of the good hemifield, whereas, unlike what happens with normal individuals, the condition in which the two flashes are on opposite sides should yield the same RT as for single flashes. The presence of blindsight, however, should result in an increase in speed of response even for stimuli in opposite hemifields. Thus, as pointed out in Haber's (1983) commentary, a stimulus of which the patient is unaware should still be capable of influencing behavior in a measurable way.

The results so far obtained with our patients show that when the two stimuli fell onto the hemianopic side no responses were obtained, whereas when the double flash appeared on the normal side, RT was faster than for a single flash. No clear-cut evidence for blindsight could be obtained, since stimuli presented to opposite hemifields yielded average RTs not significantly different from those obtained for stimulation of the intact field alone. However, eight out of 11 patients showed a trend toward faster RTs for double contralateral stimulation as opposed to stimulation of the normal field alone. It is possible that using stimuli of higher intensity and duration might be necessary to reveal across-midline spatial summation in our hemianopic patients.

In conclusion, even though our efforts to demonstrate the presence of blindsight have so far been unsuccessful, we think that the above paradigm represents a simple, artifact-free approach to blindsight research. At variance with the work reviewed in the target article, which is usually aimed at testing the existence of blindsight in the damaged hemisphere directly, the main thrust of our approach is to infer the presence of blindsight by testing its influence on vision subserved by the normal hemisphere. Broadly similar to ours is the approach followed by Singer, Zihl, and Pöppel (1977) and by Marcel (1982 cited by Campion et al.). Singer et al. have reported that increment thresholds in the normal field can be influenced by stimulation in a contralateral scotomatous area. Marcel has

instead found semantic priming following stimulation of the hemianopic field. Finally, Pizzamiglio, Antonucci, and Francina (1984) have observed that in hemianopic patients stimulation of the entire visual field with a rotating visual background elicits a greater visual tilt effect than stimulation of the normal side only.

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Authors' Response

What is blindsight?

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Lutzemberger, Marzi, and Tassinari present us with a methodology intended to overcome major deficiencies in previous blindsight research. Let us first consider what this methodology is capable of achieving before we decide whether it has in fact done so. The striking deficiency of this commentary, as with *all* other papers on blindsight (with the exception of the target article), is that there is no explicit definition of blindsight. Without such a definition, we cannot know what the research set out to achieve and we cannot evaluate it. A scientific experiment is set up to achieve certain goals in relation to some theory about the external world. The theory might be well defined or it might be more in the nature of a hunch. The experiment might serve any one of a number of functions – a rigorous test of the whole theory or an extension or quantification of the theory. But, whatever its function, an experiment must have a defined goal.

Three different definitions of blindsight are implicit in the literature. Couched in terms of theoretical propositions, they are:

1. Destruction of striate cortex affects visual function. The way in which visual function is affected remains to be determined.
2. Some cortical damage leaves visual functions which are unconscious. The nature and extent of the necessary damage remain to be determined.
3. Destruction of striate cortex leaves visual functions which are unconscious. The range of unconscious functions remains to be determined.

In evaluating blindsight research we should consider the following questions:

1. Which proposition is it testing?
2. Is this a legitimate proposition to be testing?
3. Is the experimental paradigm appropriate to test the proposition?
4. Is the experimental procedure technically sound?
5. Is the proposition verified by the data?

We usually cannot get beyond the first question because it is unclear which of the three propositions is being tested. This is true of Lutzemberger et al.'s study and also of Zihl and Werth's (1984), published since our target article. In both of these studies the authors imply that they are

attempting to test the second proposition, that some as yet unspecified cortical damage leaves unconscious visual function intact, but in both cases, instead of addressing the higher-level analytical problems associated with deleting unconscious visual function, they concern themselves only with empirical problems of the fourth level.

In noting the reactions to our target article we feel that we should have distinguished more clearly these different levels of evaluation and that we ourselves were seduced into carrying out experiments prematurely. Of our three propositions, we regard only the first as legitimate, in that this alone is a human analogue of the monkey work; and deliberations since publication of our article in *BBS* confirm to us that consciousness, in this context at least, should not be considered. First, we have no grounds a priori for expecting striate cortex to abolish consciousness, and second, we cannot conceive of any methodology which would allow us to dissociate behaviour from consciousness without involving some operational definition of consciousness which was unacceptably arbitrary.

As we have indicated, the conceptual and methodological issues involved in such an enterprise are precisely those encountered in subliminal perception (Dixon 1971). [see also Holender: "Semantic Activation Without Conscious Identification" *BBS* 9(2) 1986.] The debate which raged over this should, alone, alert us to the empirical hazards and analytical complexities involved. This response does not allow us space to develop all the arguments and, indeed, such arguments would be redundant since they have been fully and adequately dealt with over the past twenty years in the psychological literature. An excellent, lucid review is provided by Diaper (1982). We can briefly outline the problem thus: The demonstration of subliminal perception requires that one have some measure of the subject's knowledge of a stimulus (we use the term "knowledge" in a neutral way to indicate stored information whether or not this is conscious) in the absence of phenomenal representation of the same stimulus. The debate in subliminal perception largely concerns the methodology necessary to demonstrate lack of phenomenal representation. The root problem is that, to our knowledge at least, there is no psychological model which would enable us to map phenomenal representation onto behaviour in a form precise enough to determine a threshold. Since phenomenal representation can be determined only through the medium of some behavioural metric, there is no way in which an objective threshold for phenomenal representation can be arrived at. This indicates to us that the analytical framework that allows phenomenal representation to be construed as a state with a threshold is inadequate. The view that proponents of blindsight appear to hold, that an appropriate behavioural metric can be found in the methodology of clinical perimetry or casual verbal report, seems to us so inadequate as not to warrant serious consideration. Until the blindsight community at least acknowledges the existence of a problem that is analytical rather than empirical, we fail to see how we can proceed further with the debate.

There is a legitimate question we can ask, however. Phrased in the most paradigm-free manner this is, "What knowledge of a visual stimulus does a subject without striate cortex possess?" Any experiment designed to answer this will involve some variant of what, in the subliminal perception context, has come to be called the

"two task paradigm." In this, one seeks to demonstrate above chance performance on one task and chance performance on another, where the two tasks are designed to tap different types of knowledge about a visual stimulus. In blindsight, for example, following the two visual systems model, one might seek to demonstrate that a subject possesses knowledge about the *location* of a stimulus but no knowledge about its *identity*. It is evident that if we are to be able to infer anything quantitative about the types of knowledge then the two tasks must be equivalently sensitive to the knowledge they purport to tap. Two tasks might be differently sensitive either because of some factor intrinsic to the nature of the task or because of some technical aspect of the paradigm such as the number of trials. For example, the argument put forward by Haber (1983) in his commentary, couched in the terminology used here, was that localisation tasks are sensitive indicators of knowledge (they require low energy) whereas identification tasks are relatively insensitive indicators of the relevant knowledge (they require high energy). We developed this point in our response to show how shifts in decision criteria serve to compound the problem and produce spurious performance dissociations (1983). Note that this is even before we begin to examine statistical issues or the particular measures employed.

To make this point more concrete, let us examine the standard blindsight proposition that "subjects are able to localise targets within perimetrically blind parts of the visual field." Stated operationally, this means "When a subject is asked if he can see a light target of luminance L1 and diameter D1 presented for duration T1 on one or a small number of occasions, he replies 'No.' This task usually requires little or no practice. When the same subject is forced to point to the guessed positions of a number of targets of luminance L2 and diameter D2 presented for duration T2 on a large number of occasions, there is a statistically reliable correlation between target position and response position. This task usually requires considerable practice." It must surely be obvious that without some framework allowing us to understand the relationship between task performance in the two paradigms, we cannot make any inference without the respective knowledge sources these tasks are supposed to tap.

Let us return to Lutzemberger et al.'s study. We wish to show how failure to acknowledge the issues we have raised above has led these commentators into a number of analytical difficulties although we do welcome their suggested paradigm as a useful addition to the battery of empirical devices available. We provide, in the form of a logical analysis, criticism of the argument presented in their introduction. It contains four sorts of entities:

<i>Structures</i>	S1	Striate cortex
	S2	Extrastriate cortex
<i>Behaviours</i>	B1	Voluntary attention shifts (orienting)
	B2	Visually guided behaviour
<i>States/Functions</i>	V1	Normal vision
	V2	Blindsight
	V3	Total blindness (no V2)
	V4	Perimetrical blindness (scotoma)
<i>Hypothetical processes</i>	P1	High resolution vision
	P2	Attentional vision

The argument:

- (i) Theories: S1 subserves P1; S2 subserves P2.
- (ii) Therefore: V4 plus P2 produces V2.
- (iii) Observations: B1 exists.
- (iv) B1 subserves B2.
- (v) Proposition: V4 without B1 produces V3.
- (vi) Because: V4 without S2 produces V3.

The following part is ambiguous:

Either (Option I)

(vii) Therefore: V4 without S2 produces V3 (permanent).

(viii) Thence: V4 without S2 plus B1 produces V1.

Or (Option II)

(vii) Therefore: V4 plus S2 without B1 produces V3 (permanent).

(viii) Thence: V4 plus S2 plus B1 produces V1.

Criticism of the argument:

1. P1 and P2 are not defined, nor is the relationship between them. We do not know how we could recognise either of them, whether they are alternative parallel functions or whether P2 subsumes P1. We do not know the conscious status of P1 or P2.

2. (ii) cannot be held to follow from (i) without the relationship between V4 and S1 and S2 being specified. For example, if (ii) were to follow from (i) this would imply (at least) that V4 was brought about by the absence of S1 so that S2 on its own produced V2 (blindsight). This would also imply that attentional vision and blindsight are equivalent. No evidence is offered to support any of these suppositions.

3. The logical status of (vi) is unclear. Is it an observation, theory, or mere assumption? (v) cannot follow from (vi) unless S2 subserves B1. If it does then this implies that P2 and B1 are equivalent. (ii) would therefore imply that V2 (blindsight) is due to B1 (voluntary attention shifts) in conjunction with V4 (scotoma). Blindsight, by definition, is not voluntary attention shifting. It is voluntary in the sense that subjects voluntarily make the movements, but because they are "guessed" movements it is difficult to see how they could be construed as attention shifting. If, by some semantic gymnastics, one did so construe them, then it would be impossible to distinguish between blindsight and normal vision.

4. *Option I*

If S2 subserves B1, then how can V4 without S2 plus B1 exist? If it did, why should it produce V1 and not V2? Such a proposition is incompatible with (ii).

Option II

Why should V4 plus S2 plus B1 produce V1 and not V2. Again this is incompatible with (ii).

The experimental paradigm. The reaction time paradigm described by Lutzemberger and his coauthors would be a useful way of indicating the knowledge of a stimulus possessed by a subject for two reasons. First, as they indicate, it is not prone to response strategy changes. Second, it is also not prone to the differential sensitivity problem. We expect that these commentators

are right about the minimal light scatter, but a better safeguard, as we indicated in the target article, would be to present stimuli bilaterally in the blind and sighted fields and compare stimuli presented in the blindspot and normal parts of the blind field.

Our only serious quibble is with the conclusions that could be drawn from data showing spatial summation across the hemianopic boundary. It cannot be claimed that this is an unconscious process for the reasons given above. Nor can it be claimed that it is an effect of nonstriate cortex without evidence of lesion localisation. With statistically nonsignificant results and a possible trend towards such summation in only some patients, the extent of the lesions becomes crucial. For example, did the patients showing evidence of summation have only striate damage whereas the others included extrastriate damage (impacts our Proposition 1) or, as is *equally* likely, given our knowledge of their lesions, did these patients have some residual striate cortex (impacts our Proposition 2). The existence of performance variation on its own could thus be taken to either support either of two theories depending on which particular theory one chooses to adopt and depending on the integrity or otherwise of a brain structure for which no evidence is offered.

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Commentary on Daniel C. Dennett (1983) Intentional systems in cognitive ethology: The "Panglossian paradigm" defended. *BBS* 6:343-390.

Abstract of the original article: Ethologists and others studying animal behavior in a "cognitive" spirit are in need of a descriptive language and method that are neither anachronistically bound by behaviorist scruples nor prematurely committed to *particular*