Does delay impair localisation in blindsight?

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ABSTRACT

The unconscious sensorimotor skills which survive compromise of the geniculostriate visual pathway have been linked with activity of the dorsal stream of extrastriate occipitoparietal cortex. These sensorimotor circuits are thought to operate in real time. Therefore, an introduction of a delay between visual stimulus presentation and the patient's subsequent motor response should severely compromise sensorimotor tasks such as localisation (moving hand or eye to the location of a previously presented visual target). We tested this hypothesis in patient DB, a well-studied case of blindsight whose localisation abilities were first documented in the 1970s. Using eye tracking and hand movement recording technologies, as well as stimuli that control for light scatter, we verified the original observations of DB's manual and saccadic localisation. Remarkably, the introduction of a 4 s delay did not compromise his ability to localise with either eye or hand. A control experiment reveals that this skill does not depend on an opportunity to make a decision at the time of stimulus presentation, circumventing the delay using memory. These data suggest that DB's manual and saccadic localisation skills do not depend on the circuits of the dorsal stream, or that delay, contrary to theory, does not severely compromise dorsal sensorimotor skills.

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1. Introduction

Lesions to the geniculostriate pathway in humans result in topographically predictable blindness in part or all of the contralateral visual field. Experimental lesions to the same pathway in monkeys produce what appears to be the same deficit, but remarkably the animals recover sensitivity of visual stimuli after a period of time, whereas humans do not (Weiskrantz, 1961). Weiskrantz, Warrington, Sanders and Marshall (1974) observed that the testing methods used for human and non-human primates were not equivalent, and discovered similar residual visual abilities in a patient with hemianopia when he was asked to look at or point to the position of "unseen" targets in a perimeter. This ability to localise unseen targets (and a host of other visual skills identified since) is referred to as blindsight (Weiskrantz, 2009).

Manual localisation and other residual visual skills in blindsight have been characterised in several cortically-blind patients and in people with hemidecortication for tumour (Perenin & Jeannerod, 1978; Pitito & Lee, 2007; Savina, Bergeron & Guitton, 2012) which convincingly rules out the possibility that this ability

must be mediated by spared striate or extrastriate cortex in the damaged hemisphere. In these hemidecorticates at least, some subcortical structures must have been responsible for these residual visual abilities (Ptito & Lee, 2007).

Subcortical contributions to localisation using saccadic eye movements are entirely consistent with nonhuman primate lesion studies (Mohler & Wurtz, 1977) and single unit neurophysiology (Marino, Rodgers, Levy & Munoz, 2008; Wurtz & Mohler, 1976). In fact, there is indirect evidence which links the visual midbrain to localisation using *arm* movements. For example, Hoffmann and colleagues have reported single unit activity related to arm movement in superior colliculus (SC), particularly in the deep layers (Stuphorn, Bauswein & Hoffmann, 2000; Werner, Dannenberg & Hoffmann, 1997).

Even if the SC has little to do with direct coding of arm movement per se, it may play a role in maintaining and updating eye- (and perhaps gaze) centred coding as part of several networks of cortical and subcortical regions that use such representations as a "common currency" for movements of eye, head and hand (e.g. Anderson & Buneo, 2002; also see Crawford, Henriques & Medendorp, 2011). Of course, except in unusual experimental situations, saccadic eye movements typically precede visually-guided reaching and grasping movements (Adam, Buetti & Kerzel, 2012; Biguer, Jeannerod & Prablanc, 1982) and are coupled in interesting functional ways including lead time of the eye (e.g. Fisk & Goodale, 1985), delaying saccades to new targets until

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reaching movements are completed (e.g. Neggers and Bekkering, 2000); or in pathological cases involuntary reaching for targets that are fixated (Buxbaum & Coslett, 1997; Carey, Coleman & Della Sala, 1997). Manual or saccadic variants of localisation in blindsight could depend upon the SC and associated structures in a bottom up, retinocentric first fashion, although there is little evidence to speak to this issue (although see Carey, Sahraie, Trevethan & Weiskrantz, 2008).

As far as we know, visual information to and from subcortical routes remain relatively intact after damage to the geniculostriate system in primates. In the monkey at least, even after chronic degeneration of many P beta retinal ganglions cells, the surviving retinal projections to subcortical visual structures appear to be unaffected (Cowey, Stoerig & Perry, 1989; Cowey, Stoerig & Bannister, 1994). Single cells in area V5 (MT; part of the occipito-parietal stream) with visual receptive fields continue to function after lesions of V1, demonstrating that they receive non-geniculostriate visual inputs (Azzopardi, Fallah, Gross & Rodman, 2003; Rodman, Gross & Albright, 1989). This visual activity is long lasting in V1-lesioned monkeys (Collins, Lyon & Kaas, 2003).

There are several ways for information to get to V5 in the absence of primary visual cortex. First, a pathway from the koniocellular layers of the LGN to V5 has been characterised in some detail (Sincich, Park, Wohlgemuth & Horton, 2004). Second, visual afferents to V5 as well as other areas of the dorsal stream of occipitoparietal cortex come from the superior colliculus (Gaymard et al., 2003) and the medial portions of the inferior pulvinar (Kaas & Lyon, 2007) which are also heavily interconnected with one another (Stepniewska, Qi & Kaas, 2000). These tecto-parietal pathways have been confirmed in humans by studies of Rushworth and colleagues using diffusion-weighted neuroimaging (Rushworth, Behrens & Johansen-Berg, 2006).

Although precise mechanisms are rarely specified, many scientists endorse this subcortical-dorsal stream account of localisation for saccades as well as for pointing/reaching movements of the hand (Baseler, Morland & Wandell, 1999; Brown, Kroliczak, Demonet & Goodale, 2008; Danckert & Rossetti, 2005; Glickstein, 2000; Milner & Goodale, 1995; Ritchie, Hunt & Sahraie, 2012; Ro, 2008; Ward, Danziger, Owen & Rafal, 2002; Van der Stigchel, Nijboer, Bergsma, Abegg & Barton, 2010; see also Bridge, Thomas, Jbabdi & Cowey, 2008). In neuropsychology, the link between non-geniculostriate visual structures and residual sensorimotor skills has been popularised by the two visual streams hypothesis of Milner and Goodale (1995). In their adaptation of the Ungerlieder and Mishkin (1982) hypothesis, the dorsal stream is responsible for the control of visually-guided actions. According to the model, for manual and saccadic localisation at least, the dorsal stream regions and their subcortical associates such as the superior colliculus and pulvinar are the most probable mediators of spared sensorimotor skills in the visual agnosic patient DF (Dijkerman, Milner & Carey, 1999; Goodale, Milner, Jakobson & Carey, 1991) as well as patients who can localise without awareness (see Goodale & Milner, 2010; Milner & Goodale, 2008, for recent reviews).

Testing this account of localisation in blindsight is difficult for a number of reasons. Cases of cortically-blind patients who have intact localisation with either eyes or hand remain relatively rare. Most of the studies that have been conducted have used targets defined by luminance (produced by small light bulbs or light emitting diodes) that were only varied in position along a horizontal or oblique meridian (reviewed in Carey et al., 2008). Under such circumstances light scatter, even if not consciously detected by the participant, could drive responses correlated with target position. The optic disc control for 2AFC detection has not been used in localisation tasks, which means that luminance artefact has yet to be ruled out for this particular

residual visual behaviour in the cortically blind. Although some attempts to control for or identify contributions of scattered light have been made in studies of manual (Danckert et al., 2003; Perenin & Jeannerod, 1975) and saccadic localisation (Zihl & Werth, 1984), these are the exception rather than the rule.

If localisation abilities are not an artefact of luminance cues, they provide a useful tool for examining the Milner & Goodale (1995) account of the most probable neuroanatomical substrate. Since original demonstrations by Goodale, Jakobson, & Keillor, 1994, substantial evidence has accumulated which suggests that the sensorimotor circuits of the dorsal stream operate in real time (c.f. Milner, Dijkerman, McIntosh, Rossetti & Pisella, 2003; Pettypiece, Culham & Goodale, 2009) and are severely compromised by any delay between visual target offset and initiation of the response. In the case of neurologically-intact participants, a stored representation of a target attribute can drive responses after a delay (in the same way that pantomiming grasping a pencil versus a pint of beer can be generated in the absence of an actual target or model). Generating a long lasting perceptual representation when novel targets are used requires participation of an intact geniculostriate—ventral stream pathway. For example, DF's sensorimotor skills are effectively wiped out by even very short delays of 2 s (Goodale et al., 1994). She has visual form agnosia following a ventral stream lesion (Goodale et al., 1991; James, Culham, Humphrey, Milner & Goodale, 2003; see also Karnath, Rüter, Mandler & Himmelbach, 2009). Goodale, Westwood & Milner (2004) have suggested that in neurologically-intact participants the transition to a memory-driven mode happens immediately after vision of the target is removed.

The development of a memory-based representation depends upon a participant's ability to consciously encode the relevant attribute such as the size or position of a stimulus. In the case of a cortically-blind patient this memory-driven mode for producing actions to targets after a delay cannot be used. Therefore, even if a patient can localise without awareness, removing the stimulus before allowing a reach or a saccade should completely attenuate the blindsight.

In a series of studies we have revisited patient DB's residual localisation abilities over 30 years after the original investigations (Weiskrantz et al., 1974). As part of these experiments we examined his ability to localise under immediate conditions and after a four second delay using both saccadic and manual localisation tasks.

2. Experiment 1: method

2.1. Participants

DB suffered a left homonymous hemianopia after surgery for removal of an arteriovenous malformation (AVM) in 1973. Surgical notes indicate removal of 6 cm of the medial calcarine cortex of the right hemisphere. Metal clips used in his surgery for the AVM prevent MRI verification of his lesion in the calcarine cortex; nevertheless one of us with colleagues has attempted a CT scan in 2002 (Weiskrantz, Cowey & Hodinott-Hill, 2002). This scan confirmed the damage to the upper bank, but information on the lower bank was limited due to distortion caused by the metal clips. In 1976 he experienced some return of vision in his upper visual field, however, when retested using Humphreys perimetry in 2003, the fields revealed a complete left homonymous hemianopia (Trevethan, Sahraie, & Weiskrantz, 2007a,b). All testing reported here was carried out in the lower left quadrant, a consistently blind area of visual field. Ratings of visual awareness on every trial confirmed that DB was performing without any self reported visual experience. We compared DB's performance to five age and sex matched control participants (manual localisation: mean age 65.4 years, SD 2.9, range 61–68 years; saccadic localisation: mean age 64.6 years, SD 3.0, range 61–69 years), all of whom could see the targets without visual correction (as is the case for DB in his intact visual field).

2.2. Apparatus and stimuli

Stimuli were generated by a PC incorporating a specialised graphics card (Cambridge Research Systems VSG 2.5) and presented on a 21″, 100 Hz monitor with a background luminance of 37 cd/m^2 at the x, y chromaticity of (0.309, 0.353).

Screen dimensions were 61° × 46° at a viewing distance of 350 mm. The monitor gamma corrections were carried out with a luminance meter (Optical, Cambridge Research Systems, UK) in 256 linear steps. A "Magic touch" touch-screen (grid resolution: 2048 × 2048) fixed on the computer monitor recorded manual endpoints (for calibration procedures, see Carey et al., 2008). During manual localisation, eye movements were monitored with a 50 Hz Picolo Eye Tracking Toolbox (CRS) with a spatial accuracy of +/−0.5°.

In addition, the analogue output of the eye tracker was displayed and was continuously monitored to ensure fixation accuracy. The eye tracker was also used to record eye position in the saccadic localisation blocks. Saccadic endpoints were estimated by calculating the average x and y position of the first five samples postsaccade, which were spatially distributed within 2° of one another (as in Carey et al., 2008).
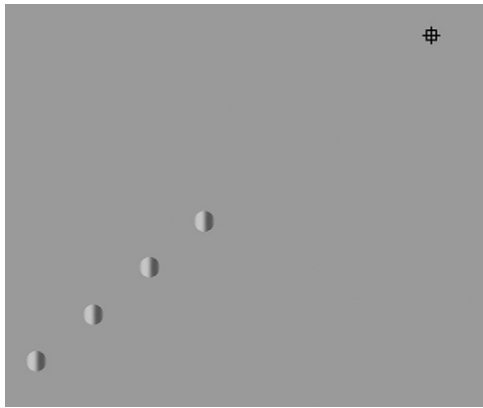

Fig. 1. Schematic representation of the target array. Note that the contrast displayed in this figure is not precise. Target sizes and positions are approximately to scale.

2.3. Procedure

The stimulus array was designed to essentially replicate the previous manual localisation data collected in DB (Weiskrantz et al., 1974). Participants fixated a high-contrast cross-hair (0.5°), then a 700 Hz tone (150 ms) signalled stimulus presentation (see Fig. 1); 2° diameter, 100 ms, 0.5c/° sinusoidal grating, with space-averaged luminance equal to the background) at one of four possible locations (28, 35, 42, 49° from fixation) on the 45° meridian (20 trials/position for DB, 10 trials/position for the matched controls).

An auditory tone (presented while participants fixated) indicated the 100 ms presence of one of the four extrafoveal targets, presented well into DB's visual field defect. On "immediate" trials, participants were allowed to move after the disappearance of the fixation cross, which happened immediately after target disappearance. For delayed trials, fixation was removed 4 s after stimulus presentation. Separate blocks required either a finger or a saccadic eye movement to the location. In manual blocks participants were encouraged to move their eyes as well as their hands to the target locations. We tested saccadic localisation in DB's sighted field in addition to his blind field, as we had some concerns about the abilities of any of our elderly participants to make saccades into the large "ganzfield" of our monitor after targets were removed (Carey et al., 2008).

3. Results

DB did not report any experience of the targets in these sessions, therefore his residual visual abilities are clearly Type 1 blindsight (Weiskrantz, 1998; see Sahraie, Hibbard, Trevethan, Ritchie & Weiskrantz, 2010 for discussion of the limits of DB's Type 1 and Type II abilities). DB's averaged reach and saccade endpoints as a function of target position appear in Fig. 2. (We have not added similar figures for the control participants as, unsurprisingly, their average endpoints typically overlap with or are adjacent to the target positions). These data show his
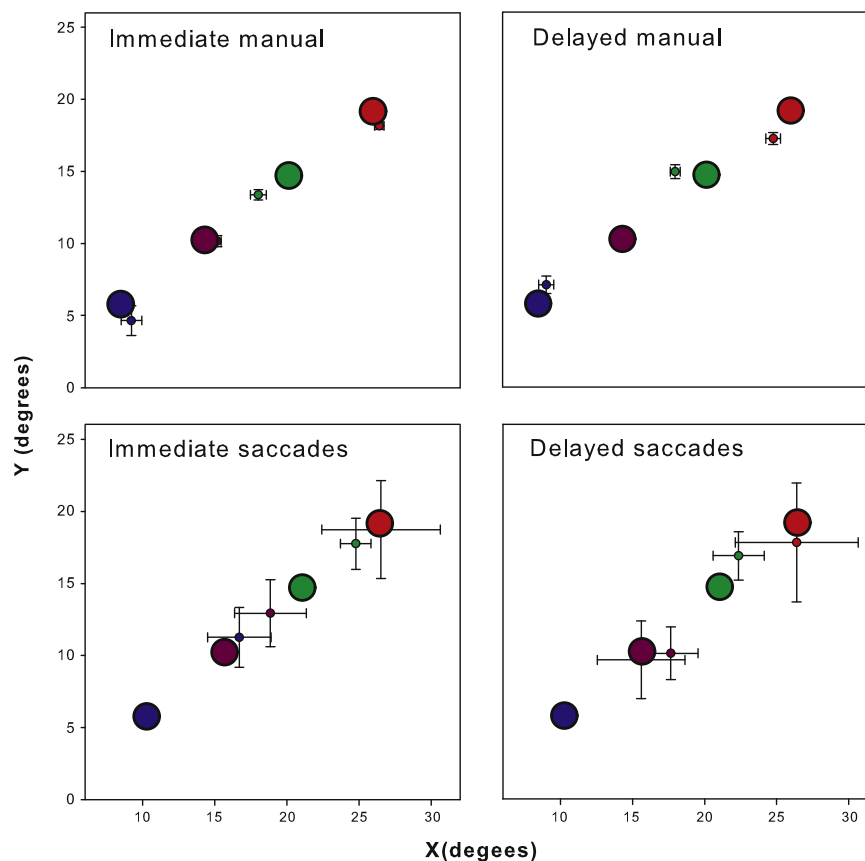

Fig. 2. Mean manual (top row) and saccadic (bottom row) endpoints as a function of target position of patient DB. Localisation in immediate conditions is depicted in the two left-sided panels, delayed conditions, the right. Error bars = 1 standard error, shown separately in X and in Y. When mean endpoints are not visible, they are obscured by the correct target, except in the delayed saccades condition (lower right) where mean endpoints for the blue target are obscured by the purple target (error bars remain visible). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)
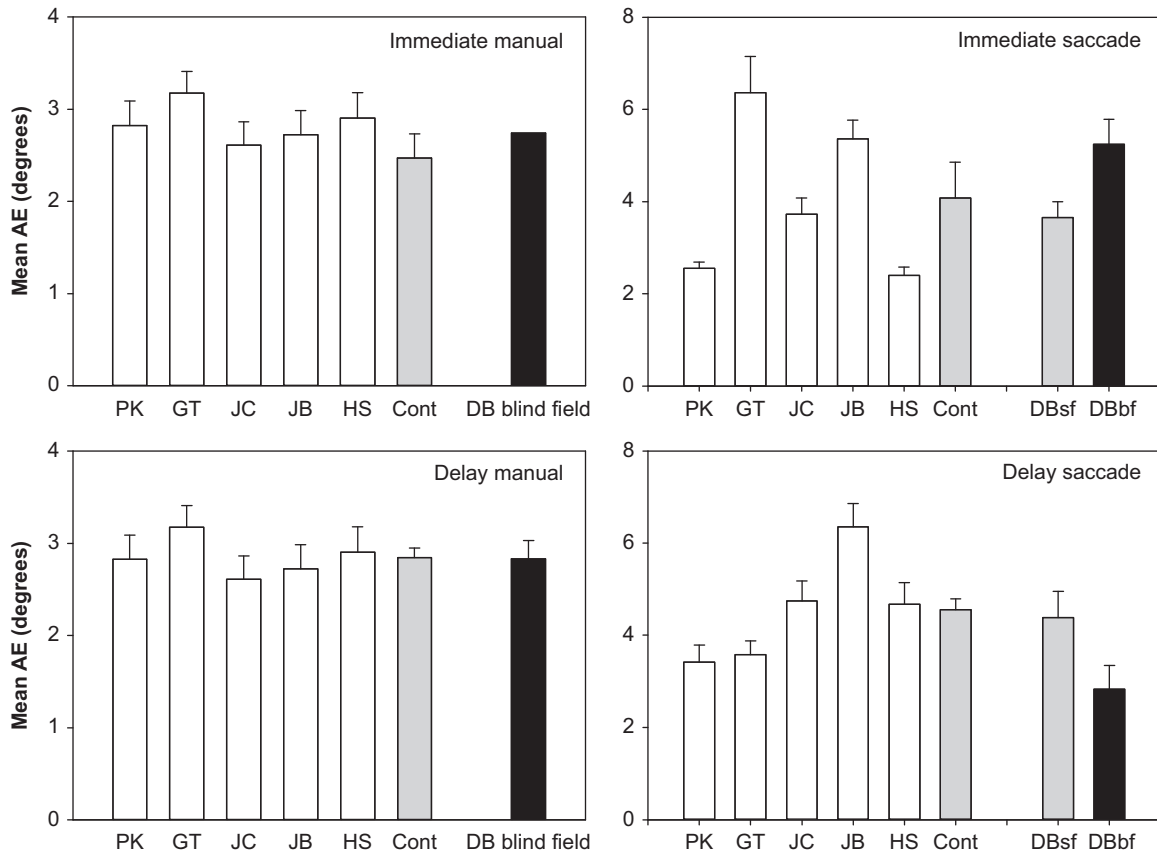
Fig. 3. Mean AEs of the DB and 5 controls for manual and saccadic localisation in immediate and delay conditions. AEs in the manual trials were calculated by computing the distance in degrees of the landing position of the finger on the touchscreen from the displayed target position. AEs in the saccadic condition were calculated in the same way, using saccadic endpoints defined as average x and y position of the first five samples post-saccade, which all were located within $2°$ of one another. Please note that the Y axis is enlarged for saccadic localisation. In both immediate and delayed conditions, DB is not significantly different from the controls. The small SEM in Immediate manual for DB (largely a reflection of the greater number of trials performed) is not visible on this scale. Given the difficulty in making a saccade into a empty screen, as an additional control condition we tested saccadic localisation in DB's good field. In these conditions, like the controls, he was always aware of the peripheral targets. sf=sighted field; bf=blind field.

average landing positions relative to the targets, and error bars represent variability (standard error of the mean) in the two corresponding axes. Our statistical analysis focusses on a derived measure, absolute error (AE), which is the unsigned distance between reach endpoint and target position for each trial. Mean AE for DB and controls, for manual and saccadic conditions (and the single variance estimate for this measure) appear in Fig. 3.

We used a robust procedure for testing the difference between individual's score and a control sample (Crawford & Garthwaite, 2002; Crawford & Howell, 1998). Manual localisation in DB was not significantly worse than the 5 age matched controls in immediate ($2.74°$ versus control $2.47°$, $t_{(5)}=0.40$, N.S.) or delayed conditions ($2.83°$ versus control $2.85°$, $t_{(5)}=-0.07$, N.S.). Similarly, in saccadic localisation DB was not significantly different than controls in immediate ($5.25°$ versus control $4.08°$, $t_{(5)}=0.61$, N.S.) or delayed conditions ($4.53°$ versus control $4.55°$, $t_{(5)}=-0.01$, N.S.). These data are comparable to independent tests of DB's ability to localise targets manually (AE=$1.51°$, versus $1.98°$ of the controls) and saccadically ($4.46°$ versus $3.32°$ of the controls; both from the 1D target arrays in Carey et al., 2008).

4. Experiment 1: discussion

These results demonstrate that luminance cues are not driving this blindsight behaviour in DB, as our stimuli had the same

average luminance as the background. Other experiments have demonstrated that luminance cues cannot drive some classes of residual vision such as 2 alternative forced choice detection (e.g. the optic disc control study), but to our knowledge all other demonstrations of residual localisation have utilised luminous targets (e.g. Blythe, Kennard & Ruddock, 1987; Danckert et al., 2003; Pöppel, Held & Frost, 1973; Weiskrantz et al., 1974). These data extend our previous findings using targets which control for luminance artefact (Carey et al., 2008).

These data also suggest that, in DB at least, localisation is uncompromised by delay. However, it is conceivable that on delay trials, DB made a decision after the tone which signalled target appearance, and maintained some sort of representation of that decision until the delay period was over. In other words, DB could decide on a response location (for example, coded in a conscious subvocal verbal way such as "upper left") and hold that response or location in memory. This type of encoding would be generated in a distinct way from the perceptual codes that neurologically-intact participants can use during delays, as these people have a conscious percept of the visual stimulus to represent. DB could have "guessed" a response location at time of target presentation (i.e. when he heard the auditory cue), and is as capable as any other participant of remembering it for the remaining 4 s before response was required by fixation offset. Although his reports of his performance during these blocks did not suggest he was aware of using any kind of strategy such as

this, we thought it prudent to control for it in a final experiment, by providing him with no auditory cue which time stamped target appearance in his bad field.

5. Experiment 2: method

5.1. Procedure

DB was tested with four blocks of 50 trials (with a rest at the half way point in each block) using the two dimensional target array from a previous report (Carey et al. 2008; see Fig. 4). Each trial lasted for approximately 6 s. After a random delay of 250–750 ms after experimenter initiation, on half the trials a 100 ms target was presented followed by a 3997 ms delay (target early trials); in the other half of the trials the 100 ms trial was preceded by a 3997 ms delay (target late trials). The response was called for, as in Experiment 1, by the offset of the fixation target. DB had a 2 s period within which the reach was to have been completed after fixation offset. The target early trials were equivalent to delay conditions in Experiment 1, and the target late trials were equivalent to immediate conditions of Experiment 1. Because DB was unaware where in the interval the target had appeared, he could not choose a particular response at the time the stimulus was presented.

5.2. Results

All of DB's responses in these blocks were unaware. DB's landing positions for early and late trials are plotted in the top and bottom panels of Fig. 4, respectively. These data mirror those for delay and immediate conditions of Fig. 1. His mean AE was 6.04° (S.E.M.=0.64) for the late trials and 6.51° (S.E.M.=0.60) for the early trials. As in our previous report (Carey et al., 2008), DB does not reliably distinguish between the two left sided targets when reaching to 2-dimensional target arrays. In these particular trials his landing positions cluster nearer to the upper left target, while in our earlier study his responses clustered near the lower target.

5.3. Experiment 2—discussion

In many studies of residual visual behaviour, methods have required that a visual event in the blind field be marked by some detectable stimulus, such as an auditory tone (c.f., Kentridge, Heywood & Weiskrantz, 1999; Moore, Rodman, Repp & Gross 1995) In this last experiment, by removing the auditory tone, we took a chance that localisation might have been completely disrupted in the absence of this sort of attentional marker of the unseen event. Nevertheless, as is clear from the results, whatever systems are responsible for localisation were clearly operable under these somewhat restricted conditions.

Fig. 4 shows that DB was as accurate localising targets which appeared early in the interval (equivalent to the delay trials of experiment 1) as he was for targets which appeared late in the interval (equivalent to the immediate trials of experiment 1). These results suggest that DB did not guess at the time of stimulus presentation as a strategy for localising in delay trials in our first study.
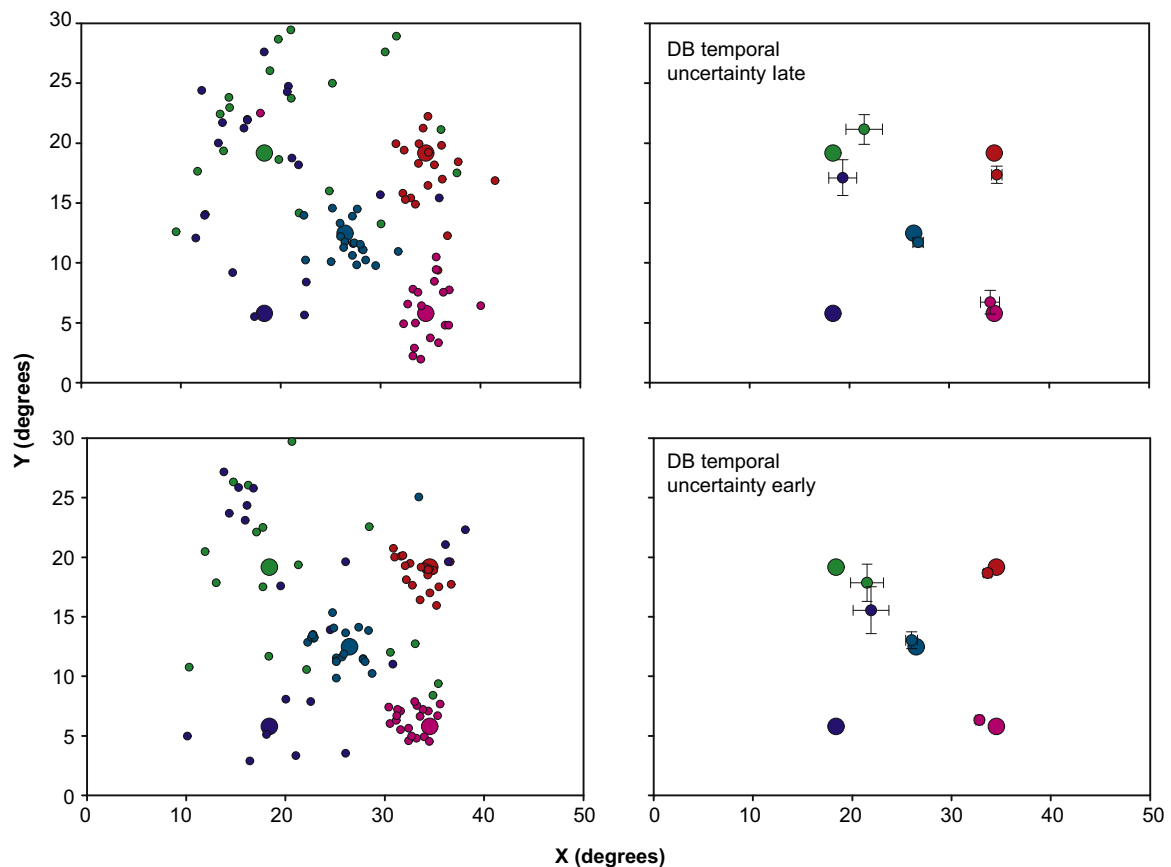

Fig. 4. DB's performance in conditions of temporal uncertainty of target presentation. The two left sided panels show his landing positions colour-coded according to the target that had been presented. The right sided panels show the X and Y mean landing coordinates for each target. In late trials (upper panels) the target was presented 3 ms before the go signal. In early trials, (lower panels), the target was presented early in the interval, 3997 ms before the go signal. In both conditions, his mean accuracy is remarkably good for three out of four target positions.

6. General discussion

In DB, manual and saccadic localisation ability is uncompromised by delay. Furthermore, his preserved performance in delay trials does not seem to depend on making a response choice at time of stimulus presentation and remembering that choice, as he remains able to localise under conditions where he did not know when, within a 4 s window, the target was presented.

These findings do not support the hypothesis that, in the absence of geniculostrate input, intact localisation depends upon subcortical-dorsal stream circuits that have to operate in real time. The extent of DB's lesion into occipito-parietal cortex is unknown; structural brain imaging cannot be carried out due to the implantation of a metal plate at time of surgery in 1970. However from the surgical notes it is probable that these regions (and their subcortical associates) are relatively intact, suggesting that they *could* be used for localisation. It may be that he uses a different brain circuit to mediate these behaviours and that other cases may show that delay eliminates blindsight, as predicted by the dorsal stream account (for example, see Streimer, Chapman & Goodale, 2009; Whitwell, Striemer, Nicolle & Goodale, 2011). For example, DB may make decisions about where to make his response to based on implicit information provided by brain structures which mediate other classes of residual visual behaviour; but these would not normally be associated with direct links to sensorimotor circuits.

Nevertheless, additional data from DB are inconsistent with the Milner and Goodale account. We have recently shown that his saccadic localisation abilities are eliminated if targets are embedded in a two-dimensional array, while his manual localisation remains relatively intact (Carey et al., 2008). This discrepancy is puzzling if posterior parietal regions and their subcortical associates mediate both of these behaviours. His superior manual skill in the original observations as well as the current data is curious in this respect, given the importance of eye-centred coordinate frames in the parietal reach region (Crawford et al., 2011; Medendorp, Goltz, Vilis & Crawford, 2003) and the usual retinal- to arm-centred hierarchies for reaching (see Carey, 2004). In these models eye centred codes are generated early in the transformations: hand eye-centred codes later. In fact, better performance in *saccadic* relative to manual localisation (rather than the other way around) could be more easily explained by current eye-to-hand coordinate transformation models.

Of course DB is a unique case in several ways. His residual visual abilities have extended to some rather remarkable feats of processing, including some abilities to identify form with contrast levels so low that normal observers cannot see them (Trevethan et al., 2007b). He also, under certain circumstances, can experience afterimages even though he cannot see the targets that produce them (Weiskrantz et al., 2002). We have also demonstrated performance levels in his impaired visual field which exceed those of normal observers by some distance, while in his sighted field, his performance is equivalent to the controls (Trevethan et al., 2007a). But DB *cannot see* in his bad field. As one of us has noted (Weiskrantz, 2009), anyone who has tested him is completely convinced that he is not simulating in any way.

DB's performance in experiment 2 suggests the intriguing possibility of dead zones within his bad visual field. In our previous report, we found some evidence using the same 2-dimensional target array that DB avoided the upper left portion and tended to respond to the lower left quadrant on those trials. In these trials he tended to respond to the upper left quadrant for those trials. Additional blocks of trials using the same target positions reveal that avoidance of the lower left target location was reliably obtained (but less dramatically) than in the temporal uncertainty experiment shown in Fig. 4. Unfortunately, we did not take the opportunity to explore the retinotopic or spatiotopic extent of this dead zone by systematic varying target position. Nevertheless, it may have some bearing on the likely neural substrates of his residual ability to localise with his hand. This observation establishes one limit to localisation abilities, in DB at least. Such limits, in the broader context of what stimulus parameters "ring fence" residual abilities in patients, may themselves guide future investigations of localisation in this patient and in others.

DB's remarkable accuracies allowed us to test rather stringently for residual localisation abilities, by comparing his average AEs to those of age-matched controls. We also have shown the variance in this measure and in endpoint position in X and Y coordinates. Of course, in order to demonstrate some sensitivity to target location, these detailed descriptions of behaviour are not absolutely necessary. Historically, in the few cases where localisation abilities with hand or eye have been found or claimed, measures tend to use mean eccentricity of response relative to target as their dependent measures (c.f. Blythe et al., 1987; Weiskrantz et al., 1974). These measures say little about variance in accuracy off the axis of target presentation, or they may, by constraining the responses to one dimension, inflate estimates of sensitivity to target location (discussed in more detail in Carey et al., 2008). Nevertheless, *any* sensitivity of mean response is demonstrating blindsight; trial to trial dispersion around a particular target is largely irrelevant.

In localisation without awareness, increased variability is rather unsurprising in these circumstances, although perhaps worthy of investigation in cortically-blind patients. For example, if some trials show particularly good sensitivity while many do not, experimenters would do well to ensure that fixation was maintained and consider level of awareness on those particular trials. Our rather conservative approach in these experiments (we ensured that all of DB's responses were obtained in Type I mode) demonstrate that this type of problem need not concern us here. If fixation and awareness are not at issue, and strong versus weak localisation patterns are obtained, variable signal strength in responsible brain structures could be implied (as opposed to a coarse coding account where variance in response positions is relative high throughout a session or across multiple sessions). In DB, the dispersion of his responses in these blocks, like those of our previous experiments (Carey et al., 2008) provide no evidence whatsoever that his mean response accuracy is being driven by a small number of extremely successful trials. DB is unusual in this respect, as we have noted here and elsewhere (Carey et al., 2008). Detailed data of the sort we have provided here are desperately needed in other patients who can localise in Type 1 mode, which could help narrow the search for which brain mechanisms maintain these behaviours. These may differ from patient to patient.

Additional details of patient behaviour, readily available with the measurement tools of today, may help researchers probe mechanisms underlying implicit performance of this sort. For example, detailed descriptions of visually-guided reaching responses in neurologically-intact participants can reveal independent coding of distance or direction of the arm movement for targets varied in a two-dimensional workspace (Desmurget, Pélisson, Rossetti & Prablanc, 1998; Gribble, Everling, Ford & Mattar, 2002). Data of his kind may speak to how the movements are controlled by feedforward and feedback-related mechanisms (e.g. van den Dobbelsteen, Brenner & Smeets, 2001).

Few investigators would disagree with the notion that there are numerous retinofugal targets in addition to the geniculostriate system which must be mediating subclasses of these behaviours (Cowey & Stoerig, 1991) *and may do so differently in different patients*. In patient GY, for example, dorsal extrastriate

regions are activated by visual stimuli without awareness (Baseler et al., 1999; see also Sahraie et al., 1997). Nevertheless, these activations were found while GY was detecting the presence or absence of targets; he was not localising them with his eye or his hand. A recent MRI analysis implicates pathways from the superior colliculus to visual cortex as well as dorsal stream and frontal areas in two of four patients showing blindsight in a criterion-free spatial summation task (Leh, Johansen-Berg & Ptito, 2006). Extending this type of fMRI work with manual localisation in the scanner is challenging, but no longer impossible with proper controls for body movements in the magnetic field, while requiring relatively small movements (e.g. Culham et al., 2003, Grafton, Fagg, Woods & Arbib, 1996; although the inertial challenges of more natural limb movements will be absent, see Carey, 2004).

Some data suggest that delay does impair sensorimotor skills in blindsight, unlike what is reported here. Danckert and Rossetti have argued (Danckert & Rossetti, 2003; Rossetti, 1998) that the scaling of maximal grip aperture in the blind field is eliminated by delay, but the actual data supporting this claim are difficult to evaluate as the original source was an oral presentation (Rossetti et al., 1995). Whitwell et al. (2011) revisit this question with patient SJ, who could grip scale under immediate conditions but was impaired after a delay of 2 s (although fixation compliance is challenging in a grasping experiment, as acknowledged by the authors). Striemer et al. (2009) have found that obstacle avoidance is compromised by delay in a cortically-blind patient. These data were, as one would expect, interpreted in terms of dorsal stream contributions to blindsight. Clearly differences in lesions size and location, age of onset and other factors are probably sources of variation which will only be appreciated fully when many more cases can be evaluated. Nevertheless, in case study neuropsychology, individual differences in patients with the same diagnosis can be theoretically problematic. Ideally, blindsight researchers will have more complete models which account for why only a subset of patients with cortical blindness have residual visual abilities. What we advocate here, based on discrepancies such as this one, is a more comprehensive approach to localisation in all cortically-blind patients.

Acknowledgements

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