

Bilateral deficits of transient visual attention in right parietal patients

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Summary

Vision tells us not only what and where objects are but also when they appear and disappear. We have found that patients with right parietal damage have an unusual loss in the ability to differentiate object onset from offset. Specifically, when a single target flickers at the same frequency as five distractors, but out of phase (it is light when they are dark and vice versa), right parietal patients require much slower rates than normals or left parietal patients to detect the target.

When we shifted the phase of the flicker in the target relative to the distractors so that the onsets and offsets were not simultaneous, the performance of the patients improved dramatically. Remarkably, the patients showed this loss equally in both visual fields. Since flicker detection was normal for the patients, we suggest that the deficit lies at a level where stimulus transients are interpreted as the appearance or disappearance of objects.

Keywords: parietal lobe; transient attention; hemispatial neglect

Introduction

Visual attention disorder is a common outcome after right parietal lesion (Vallar, 1998). Patients will often show visual neglect, a striking deficit in directing attention to the space contralateral to the lesion site (Posner *et al.*, 1984; Làdavas *et al.*, 1994). More often, extensive lesions to the right parietal lobe can cause visual extinction (Driver and Mattingley, 1998; Driver and Vuilleumier, 2001; Marzi *et al.*, 2001), which is the inability to detect a contralateral stimulus when two similar stimuli are presented to both hemifields at the same time while a single stimulus presented in either hemifield can easily be detected. Although most reports of neglect show deficits limited to the contralesional field, extinction can also occur when two identical stimuli are presented within the ipsilesional hemifield (Duncan *et al.*, 1999; Vuilleumier and Rafal, 2000). While the mechanisms of these effects are still unclear (Pashler, 1998), it has been suggested that parietal patients are impaired in the capacity to divide their attention between multiple objects in space (Vuilleumier and Rafal, 2000) and in time (Husain *et al.*, 1997; Shapiro *et al.*, 2002). We have recently found (Battelli *et al.*, 2001) a bilateral visual deficit in a simple apparent motion task. In that study, right parietal patients were severely impaired in discriminating dots that alternated in apparent motion from dots that flickered on and off in synchrony, and this deficit was seen in both the left and the right visual field. In contrast, these patients showed a normal

ability to perceive motion-defined shapes—a low-level motion task—in both fields. In addition, their ability to keep track of moving targets with attention—a high-level motion task—was impaired only in their contralesional, left field. These results suggested that loss of apparent motion was not a consequence of a deficit in underlying motion mechanisms: low-level motion was preserved in both fields for these patients and high-level motion was lost only in the contralesional field. An alternative explanation is that the deficit involves the accuracy in judging the relative timing of offsets and onsets. The unique aspect of apparent motion, compared with the other two tasks, is the discrete presentation of the dots at different times and locations. If the timing of the offset of one dot cannot be accurately judged, it may not be appropriately linked to the onset of the next dot as a single object in discontinuous motion. The motion percept would be lost and it would be difficult to distinguish dots flickering in synchrony from dots that are alternating (Verstraten *et al.*, 2000).

To determine if the apparent motion deficit was due to this timing problem, we have now tested right parietal patients on a judgement of synchrony of flicker in which the timing was very similar to that of the apparent motion experiment (Battelli *et al.*, 2001), except that no motion was involved. Two of the patients (J.L. and J.R.) tested in the present study were included in the previous research (Battelli *et al.*, 2001)

and our experiments reveal that their deficit is indeed related to the relative timing of onsets and offsets, but it is not a problem in the accuracy of timing as there was no evidence for a loss in temporal resolution. Rather, it is a loss of the ability to identify and discriminate onsets and offsets. Our results suggest that this loss is not occurring at a low level, where on and off transients appear to be registered at normal rates, but at a high level, where the transients are assigned to the appearances and disappearances of objects.

Visual search experiments have already indicated that object appearance and disappearance are critical events in a display that control the allocation of attention. For example, the sudden appearance or onset of an object is known to attract attention, leading search directly to the new object as the first item to be scrutinized (e.g. Yantis and Jonides, 1984). Enns *et al.* (2001) have demonstrated that this priority for a newly appearing object overrides the priority given to a large change or transient in the luminance of an existing object in the display, even when the new object has very low contrast. However, they reported an exception to this rule when an old item changed polarity, for example, from light to dark on an intermediate grey background. This change was as effective as the appearance of a new object, suggesting that the polarity reversal is interpreted as the replacement of one object with another.

Our results also suggest that the appearance and disappearance of objects are a central part of attentional processes. Our right parietal patients have a deficit in their ability to distinguish onsets and offsets of objects. However, they have no problem in detecting rapid transients, as long as they do not have to discriminate onset from offset. Since the patients showing this loss all have lesions in the right parietal lobe, we suggest that the loss is related to a disruption of attentional processes, in particular the determination of whether a transient signals the appearance or disappearance of an object.

Patients and methods

We tested three patients (J.L., W.H. and J.R.; Fig. 1, bottom three rows) affected by a right parietal lesion. They were all in the chronic stage of their neurological disorder, as we tested them no earlier than 1 year from lesion onset. They all presented from mild to severe signs of left visual neglect, which was assessed using line bisection, star cancellation and copying drawings from a neglect test battery (Black *et al.*, 1990). J.L. scored 40/100 and W.H. 50/100, both scores indicating severe neglect, while J.R. scored 10/110, which indicates mild neglect (the normal range is 0–5/100). We also tested three lesion control subjects with left parietal lesions (P.C., J.W. and J.S.; Fig. 1, top three rows) to determine whether the right parietal lobe is specialized in transient visual attention. Two (P.C. and J.S.) of the three left parietal patients showed signs of right visual neglect (P.C. scored 9/100, J.S. 40/100 and J.W. 1/100). Although the lesions of the left and right hemisphere patients were variable, they all

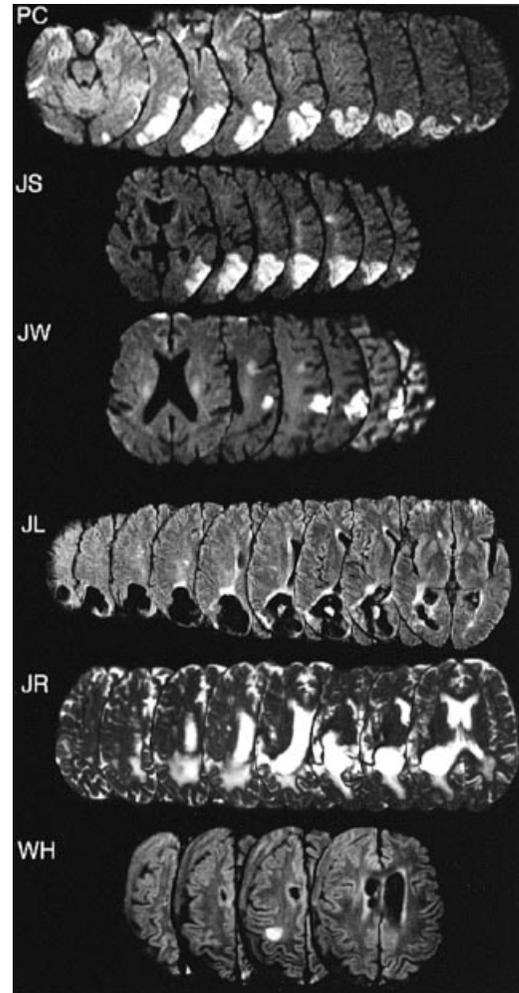


Fig. 1 Horizontal MRI sections through the cerebral hemispheres of six stroke patients with unilateral lesions. (*Top three rows*) Diffusion-weighted images of the patients with left-sided lesions (P.C., J.S. and J.W.). P.C. had a lesion extending through the left lateral occipital, angular and supramarginal gyri as well as an older white matter infarct in the anterior portion of the right superior frontal gyrus. J.S. had an infarct of the left angular and supramarginal gyri and J.W. had a smaller focal infarct of the left supramarginal gyrus. (*Bottom three rows*) Patients with right-sided lesions (J.L., J.R. and W.H.). Flair images for J.L. and W.H. and T₂-weighted images for J.R. are shown. J.L. and J.R. had extensive lesions of the right lateral occipital, supramarginal and angular gyri, as well as of the precuneus and the superior parietal lobule. Pre-existing white-matter lesions are also evident in J.R. W.H. had an infarct located in the right supramarginal gyrus and the right temporal pole, and a haemorrhage in the right external capsule. The slices shown depict the parietal lesion and a right frontal subdural haemorrhage.

had a parietal lesion in common and the location and size of pathological involvement were very similar between the two groups of patients (Fig. 1).

At the time of testing, patients J.W. and P.C. had intact visual fields, while J.R. presented with a left superior quadrantanopia, W.H. and J.L. had a partial left hemianopia and J.S. had a right hemianopia. During the testing sessions

all the stimuli were presented bilaterally in the left and right parafoveal fields, both of which were intact in all patients. Three subjects, two males and one female, with no history of neurological disease (mean age 65.6 years) served as age-matched normal controls. The study was approved by the Harvard Committee on the Use of Human Subjects and signed informed consent was obtained from all subjects.

Experimental procedure

The experiments were conducted on a G4 laptop computer connected to an Apple Studio Display. Software for the experiments. All software was written in Think C™ (Symantec Corporation, Cupertino, CA, USA), using the VisionShell™ routines (MicroML, St. Hyacinthe, Canada). The same basic equipment was used in all experiments.

On each trial in Experiments 1, 2 and 3, six squares subtending $1 \times 1^\circ$ of visual angle were presented centred 2° from the fixation point simultaneously in the two hemifields (three in each hemifield vertically aligned, separated centre-to-centre by 1° and slightly displaced from vertical on each trial) (Fig. 2).

In Experiment 4, two, four or eight squares were presented in three separate conditions. They were arrayed, as in Experiment 1, in two columns, one in each field with one, two or four squares in each column, spaced vertically by 1° when there was more than one. In Experiment 5, only one column of three squares was presented on each trial, on either the left or the right of fixation.

In all cases, the squares flickered sinusoidally at the same temporal frequency (varied from 9 to 1 Hz, which corresponds to a peak-to-peak asynchrony between the target and the distractors of $\sim 55\text{--}500$ ms at 180° phase shift). In Experiments 1 and 2, the background was black (2.56 cd/m²). In Experiment 1 the squares varied in brightness from the same black as the background to a maximum of 139 cd/m² (97% contrast). In Experiment 2, the flicker contrast was varied (see below). In Experiments 3, 4 and 5 the background was grey (82.5 cd/m²). The contrast of the sinusoidal flicker was fixed at 45% in Experiments 3, 4 and 5, the squares varying from 43.5 cd/m² at the darkest to 115 cd/m² at the brightest. In Experiments 3, 4 and 5, the mean value of the sinusoidal variation matched the background luminance.

In all cases, the target position was chosen randomly from the available locations. In Experiment 1, the target was 180° out of phase with the distractors so that it was light while the distractors were dark, and vice versa. In Experiment 3 the target was shifted out of phase by 45° , 90° , 135° and 180° with respect to the distractors in four different conditions, whereas in Experiment 4 and 5, only 90° and 180° phase shifts were used. The exposure time was unlimited for all experiments. In Experiment 2, only the target square flickered among five stationary squares. The contrast of the sinusoidal target flicker was adjusted to determine the threshold of visibility of the flicker at temporal frequencies of 8, 17 and 35 Hz, in separate conditions.

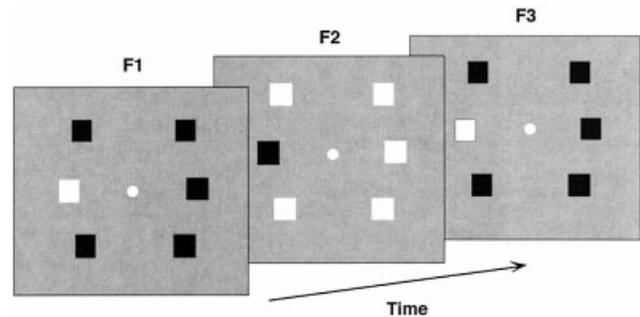


Fig. 2 An example is given for the basic paradigm of the tasks we used in the four experiments (this example is taken from Experiment 3). The trial began with the subject fixating the bull's eye in the centre. The six squares began to flicker at the fastest rate (here three frames are depicted: F1, F2 and F3). The temporal frequency was progressively decreased until the subject's vocal response occurred. The response (the target location) was entered by the experimenter and a new trial was started. The target (the middle left element in this sequence), randomly assigned across trials, flickered out of phase with the distractors.

The subjects were seated 57 cm from the monitor in a dimly lit room. During each trial, the experimenter, while monitoring the subject's eyes, progressively decreased the temporal frequency (in minimum steps of 0.02 Hz; Experiments 1, 3, 4 and 5) or raised the contrast (Experiment 2) until the subject responded verbally by giving the location of the target (e.g. upper right). The flicker (or contrast) was changed quite slowly, so that on average ~ 50 s passed before the subject responded. When the answer was correct, the next trial was started; when it was incorrect, the frequency was lowered further (or the contrast was raised further in Experiment 2) until the subject gave the correct response, indicating which square was the odd one (out of phase or flickering not steady). We emphasized to the observers that it was important to maintain fixation and we asked the subject at the beginning of each trial to fixate the bull's eye. The experimenter visually monitored the subject's eye position throughout the trial to detect any gross movements. We ran 20 trials for each hemifield. The average left and right temporal frequency thresholds (or contrast for each given temporal frequency in Experiment 2) were calculated. The subject's vocal response was entered by the experimenter on the keyboard.

Results

Experiment 1: discrimination of an out-of-phase target

We presented the subjects with six squares displayed vertically, three in each visual field and displaced 2° from central fixation. The target square flickered out of phase with the distractors (it was light when the distractors were dark and vice versa). At slower rates, the out-of-phase target is easily

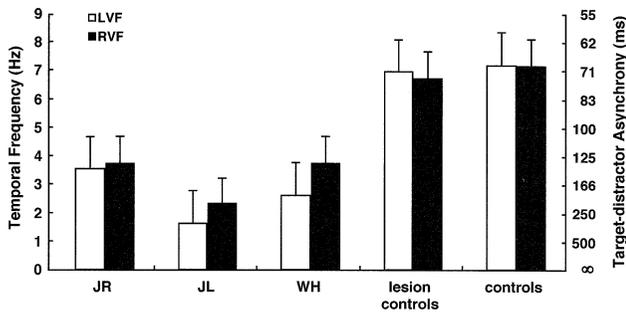


Fig. 3 Temporal frequency threshold for each unilateral parietal patient (J.R., J.L. and B.H.), for three lesion control subjects (left parietal lesion) and three age-matched normal controls. The threshold is expressed as the average temporal frequency at which the subject could just detect the out-of-phase target. The lower the threshold the worse the performance. Standard errors of the mean are shown as vertical bars. Asynchrony (ms) between the target and the distractors is reported on the right axis. LRVF = left visual field; RRVF = right visual field.

seen, but at the highest rates all the squares appear to be flickering identically.

The three right parietal patients all showed a severe loss in this task in both left and right visual fields, obtaining an average temporal frequency threshold of 2.66 and 3.31 Hz (corresponding to a target–distractor asynchrony of 188 and 151 ms) for the left and right visual fields, respectively (Fig. 3). In contrast, the age-matched controls obtained an average threshold of 7.2 Hz (69 ms asynchrony) in both visual fields. Performance of the three left parietal patients was similar to that of the controls (average threshold of 7 and 6.7 Hz, 71 and 75 ms for the left and right visual field, respectively).

Experiment 2: flicker detection

One could argue that the deficit in Experiment 1 was due to inability of the patients to detect rapid luminance changes, implying that their lesions also affected areas subserving basic sensory processing. For example, selective damage to neurons in the magnocellular pathway could degrade the ability to process flickering stimuli (Kaplan and Shapley, 1982). As a control, we tested the patients' flicker detection thresholds. The display was similar to that of the first experiment except that now only one of the six squares flickered while the distractors were steady. We increased the contrast of the flicker progressively from 0% contrast until the subject reported the target location correctly or until 100% contrast was reached. We obtained thresholds for flicker detection at temporal frequencies of 8, 17 and 35 Hz. Two patients (J.R. and J.L.) were tested on this task, and their performance was in the range of three age-matched normal controls (Fig. 4). This demonstrates that basic temporal processing is spared in right parietal patients (Spinelli and Zoccolotti, 1992). There is no loss in temporal resolution and,

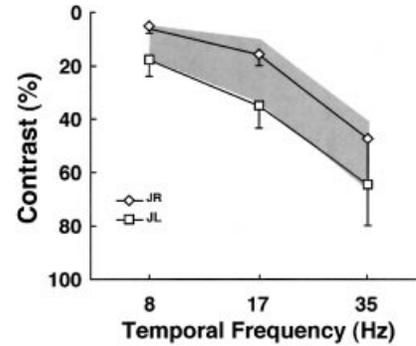


Fig. 4 Flicker detection threshold for temporal frequencies of 8, 17 and 35 Hz for two neglect patients (J.R. and J.L.). Standard errors of the mean are shown as vertical bars. The shaded area shows the range of thresholds for three age-matched normal controls. Data from the left and right visual fields have been collapsed because they did not differ.

consequently, the explanation of the deficit in Experiment 1 must lie elsewhere.

Experiment 3: testing different phase shifts

Detecting the rapid changes of flicker is not the same as experiencing each light and dark phase of the flicker as individual events, a percept referred to as Gestalt flicker (van de Grind *et al.*, 1973) and having a much lower threshold rate (7–8 Hz; Verstraten *et al.*, 2000). The parietal patients may have preserved the detection of flicker but suffered a loss in the rate at which they can individuate events with attention. In this case, a rapid stream of events would appear a confusing blur at speeds at which a normal observer would still experience a rapid succession of individually identifiable events. We assume that this individuation of the on and off phases of the flickering target is required to judge its timing relative to that of the distractors. This temporal limit of individuation is known to be the limiting factor for apparent motion in normals when the alternating dots are spaced far enough apart to prevent response from low-level motion-selective units (Verstraten *et al.*, 2000).

Alternatively, we considered that the patients might have no difficulty with rapid flicker or rapid individuation but are unable to distinguish the onsets from the offsets of the targets and distractors. All changes might appear simply as transients, unmarked in direction, so that the target, as it appeared, would be hard to distinguish from the distractors as they disappeared. Note that this loss in marking the direction of the transients could not be happening at an early stage, as it would affect the maximum rate at which flicker could be perceived, and this did not happen (Experiment 2). However, there is a higher level at which transients must be processed before being experienced. Any given onset, for example, might be either an appearance (of a light object on a grey background) or a disappearance (of a dark object on the same grey background). Damage at the level that sorts out these assignments would degrade the distinctiveness of the appear-

ances and disappearances of the targets and distractors. Clearly, the differences between appearances and disappearances cannot be completely lost, but if they were degraded the patients might require a slower rate to distinguish them. Our next experiment tested this hypothesis against the possibility of a lowered rate of individuation mentioned above.

In Experiment 1 the onsets and offsets of the target stimulus were always shifted 180° out of phase with those of the distractors. Therefore the appearance and disappearance of the target and the distractors were aligned in time. This will be a difficult task if onsets and offsets are hard to distinguish. In order to test this possibility, we can shift the onsets and offsets of the target 90° out of phase from the distractors rather than 180°, as was the case in Experiment 1. Onsets and offsets now no longer overlap (Fig. 6C), and if the problem had been distinguishing onsets from offsets the task should now be easier for the patients (although slightly harder for normals as the target–distractor difference is reduced). On the other hand, if the problem were the rate at which events can be resolved, the shift should not improve performance for the patients.

In this experiment, we tested four phase differences between target and distractors, 45°, 90°, 135° and 180°, each in a separate block. The results showed that normal subjects' performance improved progressively from 45° to 180°. In contrast, the performance of the two right parietal patients (J.R. and J.L.) was very different. First, at 90° phase shift the patients came close to normal levels of target detection. Their performance then dropped at 135° and 180°, returning to the level seen in Experiment 1 (Fig. 5).

These results demonstrate that the deficit is not primarily one of the limiting rate at which the patients can judge offsets and onsets. When the target and distractor transients are shifted maximally out of alignment (at 90°), the patients are able to make judgements at a higher, almost normal rate. The difficulty at 180° (and in Experiment 1) may therefore be related to the ability to differentiate transient onsets from transient offsets, as this is required to discriminate the target and the distractors when their transients are exactly aligned (Fig. 6E). This is a problem that normals also show for brief light pulses: the contrast required to detect a pulse is much lower than the contrast required to tell if the pulse was an increment or a decrement (Krauskopf, 1980). Recall again that if there is a problem discriminating onsets from offsets, it cannot be occurring at an early level because the patients show normal flicker detection for local variations of contrast.

In the data for the normals, the effect of phase on performance is consistent with the effect of phase on the difference between the target and distractor waveforms. As the phase shift increases, the difference between the two waveforms rises to a maximum at 180° shift and must then drop back to zero at 360° phase shift as the target shifts back into phase with the next cycle of the distractor waveform. Because the difference signal is strongest at 180°, the subjects can detect the target at higher frequencies at this phase than at others, producing an inverted U-shaped curve whose left half,

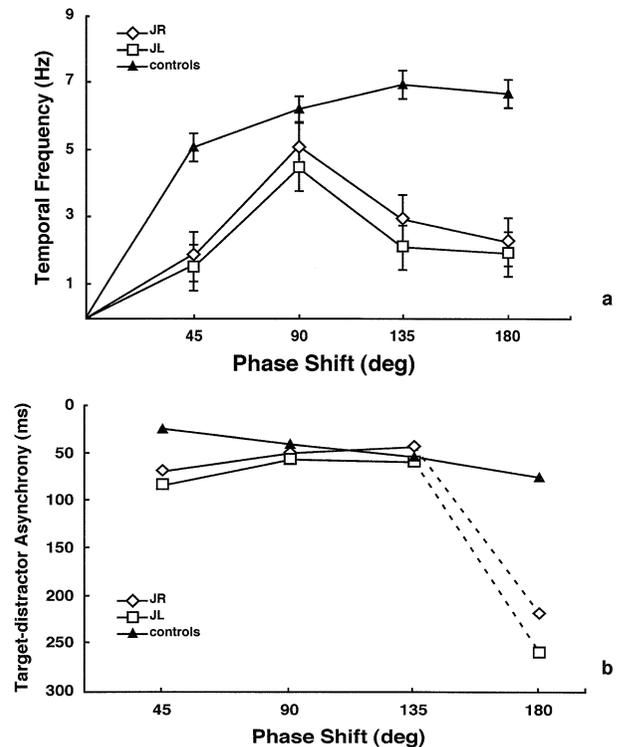


Fig. 5 (A) Temporal frequency thresholds for temporal phase shift of 45°, 90°, 135° and 180° are reported for two neglect patients (J.R. and J.L.) and two age-matched normal controls. Data from the left and right visual fields have been collapsed because there was no significant hemifield difference, either for the patients or for the control subjects. Standard errors of the mean are shown as vertical bars. (B) Asynchrony (ms) between the target and the distractors for the four temporal phase shifts that were used. Note that the peak-to-peak asynchrony of the two sine waves (target versus distractors) is a function of both temporal frequency and phase shift. For the patients, the asynchrony for phases 45–135° is based on the assumption that transient polarity is lost (see rightmost column of Fig. 6). The average data from the left and right visual fields are reported because there was no significant hemifield difference either for the patients or for the control subjects.

peaking at 180°, is seen in Fig. 5A for the normal subjects. If we had continued collecting data beyond 180°, the performance would drop again, mirroring the results between 0° and 180° (e.g. 270° is equivalent to 90° and 360° to 0°). This inverted U-shaped curve is the classic result for detecting a phase difference between two otherwise identical waveforms (White *et al.*, 2002).

Although the underlying variable used to detect the difference between the target and distractor waveforms is unknown, it may be temporal offset. When the threshold temporal frequency at each phase is converted to temporal asynchrony (Fig. 5B), the value for normals falls between 24 and 70 ms across the different phase settings.

The data for the right parietal patients are quite different. They peak at 90° rather than 180° and drop back down at 180° rather than 360°. This pattern changes at twice the rate of the

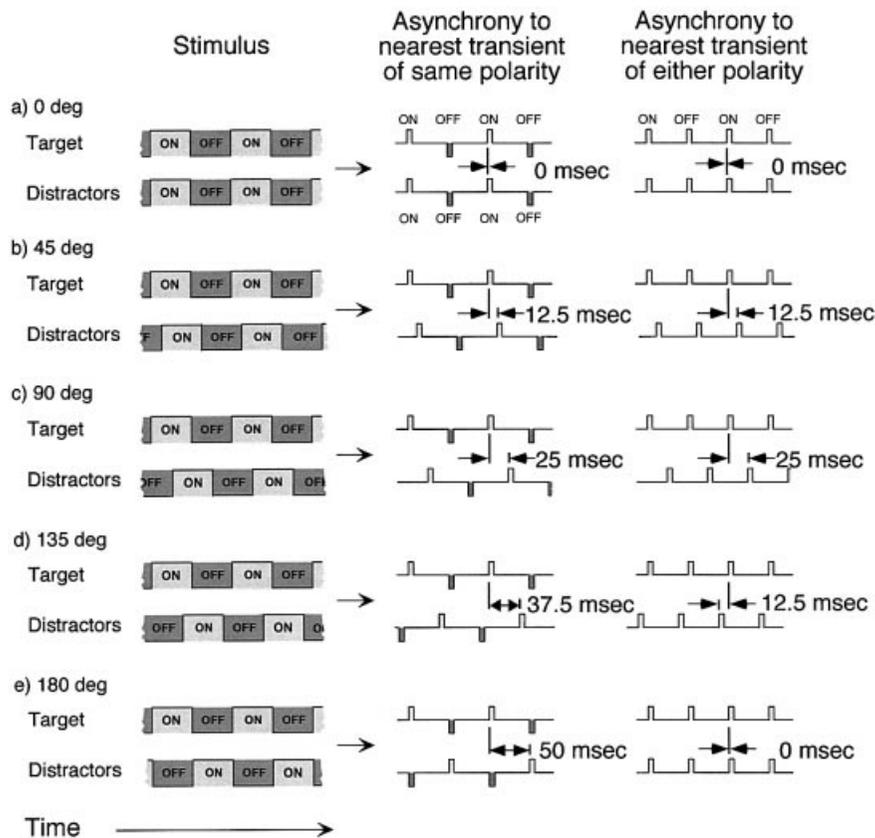


Fig. 6 The original waveforms (shown as square waves for convenience; sinusoidal waves were actually used) and their corresponding transients with polarity preserved and lost, for five different phase shifts. The peak-to-peak temporal shift between target and distractor waveforms is given in ms for each phase shift with the values shown for a 10 Hz alternation rate. For shifts up to 90°, the offset between the target peak and the nearest distractor peak is the same whether the transient polarity is preserved (middle column of waveforms) or lost (rightmost column). However, beyond 90° the target–distractor offset continues to increase if polarity is preserved but drops back to zero if transient polarity is lost. The temporal asynchrony for 45° and 135° will be equal in the rightmost column (12.5 ms) but it is three times as large at 135° than at 45° in the middle column (12.5 and 37.5 ms). Sensitivity to phase shifts of 45° and 135° should therefore be equal if the transient polarity is lost but should be three times better for 135° than for 45° if polarity is preserved. The normal data showed the latter pattern while the patients showed more equal sensitivity for the two conditions. For phase shifts other than 0° and 180°, the target transient occurs alone and so can be detected without identifying whether it is an onset or an offset. At 180°, however, the target can be detected only by discriminating the onsets from the offsets, and so detection becomes (rightmost column) impossible if polarity is totally lost.

normal data. This would be expected if the difference between positive and negative segments of the waveform were lost (for example, if both half cycles became positive, as happens for full-wave rectification, or if only transients were registered and not their direction, as shown in the rightmost set of waveforms in Fig. 6). Whatever the reason for the loss of difference between positive and negative segments, the outcome of such a loss would be that the two waveforms would no longer be maximally different at 180° phase shift. In fact, they should be indistinguishable in this condition. The two waveforms would now differ most for a 90° shift (Fig. 6C), where the target transient lies halfway between the two transients of the distractor waveform (one an off- and the other an on-transient, but these are no longer differentiable).

In addition to the inverted U-shape curve peak at 90° rather than 180° for the patients (Fig. 5A), there are two other features of importance. First, at 180°, the patients can respond, albeit only at low speeds. If the loss of difference between positive and negative segments were complete, the patients would be unable to detect the targets at the 180° phase shift no matter what the speed, even in static images in which there was simply a white target among black distractors. However, they can perform this discrimination, indicating that they have not lost the distinction between static black and white but perhaps only the distinction between the on- and off-transients. When the stimulus is slowed sufficiently, the patients may then rely on the sustained response to the white or black phase of the

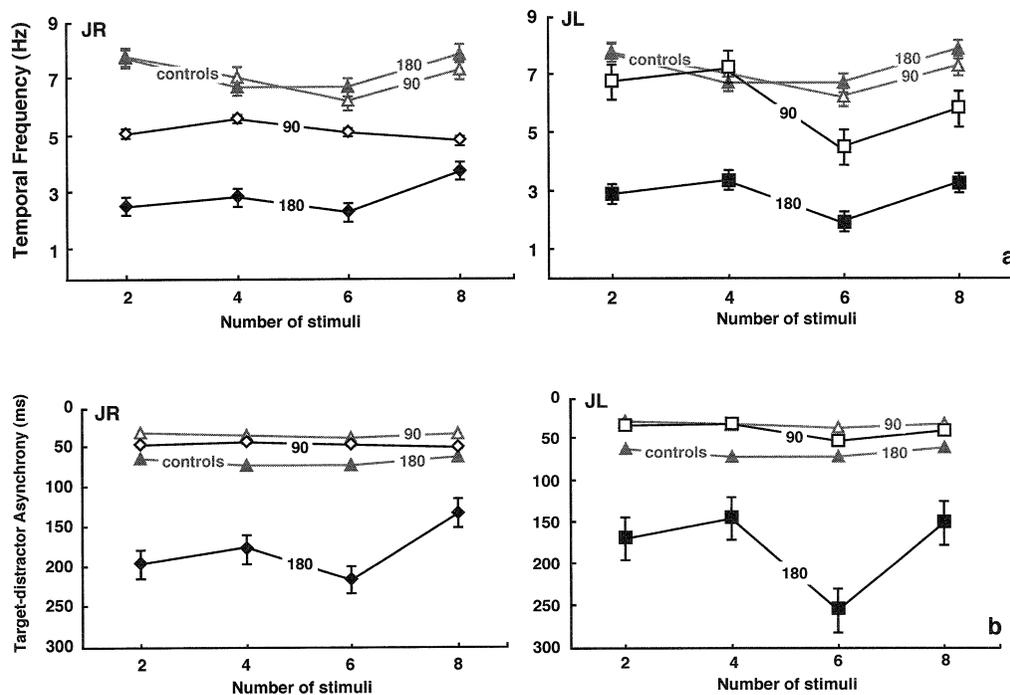


Fig. 7 (A) Temporal frequency thresholds for temporal phase shift of 90° (open symbols) and 180° (black symbols) are reported for two neglect patients (J.R. and J.L.) as a function of the number of stimuli (data for six stimuli are taken from Experiment 3). Data from two age-matched controls are shown for 90° (open triangles) and 180° (grey triangles) phase shifts. Data from the left and right visual fields did not differ and have been collapsed. Standard errors of the mean are shown as vertical bars when larger than the data symbols (± 1 SE). (B) Asynchrony (ms) between the target and the distractors for the two temporal phase shifts we used (90° and 180°). Data from the left and right visual fields have been collapsed because there was no significant hemifield difference either for the patients or for the control subjects.

waveform to perform the task rather than the response to the transitions between black and white.

Secondly, in Fig. 5B the patients' data for phase shifts of 45–135° are plotted in terms of temporal offset between target and distractors for the degraded waveforms (with on- and off-transients no longer distinguished). These offsets are computed on the basis of a waveform that is doubled in frequency (twice as many peaks), like those shown in the rightmost set of waveforms in Fig. 6. At a given temporal frequency, then, the maximum target–distractor offset between these doubled waveforms is at a 90° shift of the original waveforms, and the offsets for 45° and 135° are equal. Using these degraded waveforms to compute the offsets that correspond to the temporal frequency thresholds of Fig. 5A, we find that patients can detect delays in the range of 40 to 80 ms, not much different from the delays that the normals detect (with the possible exception of the 45° condition). The assumption that the temporal delay is the variable underlying detection is speculative but it produces an interesting match between the performance of the patients and the normals over the 45–135° conditions. In terms of temporal frequency, the patients' thresholds only approach that of the normals for the 90° condition. In terms of temporal delay, however, the patients match the normals over a wider range (45–135°). The temporal delay in the 180° condition cannot be based on the

distorted waveform (with transient polarity lost) because no detection should be possible here if on- and off-transients are indistinguishable. We must assume that the patients call on a different process to detect the target in the 180° condition, and the delay for the patients shown in Fig. 5B for 180° is based on the undistorted waveforms (transient polarity preserved).

Experiment 4: varying the distractors' numerosity

Visual masking provides another possible explanation of the deficit in our patients. The simultaneous transients of Experiment 1 (and in the 180° condition of Experiment 3) might be masking each other (Breitmeyer, 1984), whereas when the distractors are moved to 90° phase (Experiment 3) the target is the only transient in the field when it appears. We designed Experiment 4 to test the possibility of masking. The procedure was exactly the same as in the previous experiment but we varied the number of stimuli and tested only the critical 90 and 180° phase-shift conditions. We used two, four and eight test squares (six stimuli were used in Experiment 3), always presented bilaterally. If the concurrent transients are masking the target, masking should be reduced in the conditions with two and four stimuli and increased with

eight stimuli. Consequently, the performance at the 180° phase shift condition should improve when there are fewer stimuli.

The results in Fig. 7 indicate that the patients find the 180° condition equally difficult no matter how many distractors are changing simultaneously with the target. Furthermore, performance at 90° remains constant across conditions, confirming that the patients can clearly distinguish the transient events when onsets and offsets are not aligned in time. These results rule out a masking interpretation. Although the patients' data at 180° fall well below that of the controls, the patients again perform at or near the normal range for the 90° condition. When expressed as target–distractor asynchrony (Fig. 7B), the patients' thresholds in the 90° condition lie between the controls' thresholds for 90 and 180°. The results of this experiment also rule out visual crowding as a possible interpretation of the deficit. Like masking, crowding would require that the performance degrade as the number of distractors increases.

Experiment 5: unilateral presentation

Throughout every experiment the stimuli were always displayed simultaneously in both visual fields. To make sure the deficit was not due simply to a delay in inter-hemispheric transmission (Newman and Albino, 1979), we repeated Experiment 3 with unilateral presentation: on each trial the stimuli were presented in only one field, either to the left or to the right of fixation (just three of the six squares shown on each trial).

With unilateral presentation, the deficit was still present in both hemifields and the rate at which the patients could perform the task did not differ between hemifields. Averaging the patients' results across left and right fields gave temporal frequency thresholds of 2.8 and 2 Hz at 180° (178 and 250 ms target–distractor asynchrony) and 5.1 and 4.9 Hz (49 and 51 ms) at 90° for J.R. and J.L., respectively. Performance of an age-matched control was 7.2 and 6.7 Hz (69 and 37 ms) at 180° and 90°, respectively.

Discussion

In conclusion, right parietal patients experienced a severe reduction in the speed at which they could distinguish between onsets and offsets, and this loss occurred in both visual fields. The deficit was seen despite normal responses to flicker. We suggest that the deficit is a disruption of a high-level mechanism responsible for assigning on- and off-transients to the appearance or disappearance of objects.

Although ipsilesional visuospatial deficits have already been documented in neglect (Kim *et al.*, 1999), they are often associated with right frontal-subcortical lesions. In these rare cases, a deficit usually manifests itself on line bisection tasks only and is not accompanied by a contralesional deficit. Further cases of ipsilesional deficit have already been described in classic, contralesional neglect (Duncan *et al.*,

1999; Vuilleumier and Rafal, 2000). More specifically, in the Vuilleumier and Rafal study, right parietal patients were impaired in attending to concurrent identical stimuli within the ipsilesional visual field, while they performed normally when the two stimuli were different. They characterize this deficit as a type of extinction that occurs even when both stimuli are in the 'good' ipsilesional field. In our experiments, one concern is that the long exposure time would allow the patients to move their eyes. If the deficit is bilateral, any eye movements that do occur should not affect performance as there is no positioning possible that will improve detection. If the deficit is unilateral, however, we should expect that if the patients move their eyes, they would try to position the stimuli in the ipsilesional, 'good' side of their vision (Husain *et al.*, 2001) to overcome any deficit when the target was initially presented in their impaired field. This would predict that no impairment would be seen. This did not happen: the patients did poorly, and equally so, for targets on the left or right of initial fixation in the 180° conditions in Experiments 1, 3, 4 and 5. Thus, even if the patients did move their eyes on some trials, it did not eliminate the deficit, a result that is only expected for a bilateral deficit.

Could our result be due to extinction in which the distractors render the target inaccessible to awareness? In most cases of classical visual extinction (Baylis *et al.*, 1993; Marzi, 2001), as well as in visual extinction within the ipsilateral (Vuilleumier and Rafal, 2000) visual field, the deficit is more pronounced between identical items. A recent study (Gilchrist *et al.*, 1996) showed that visual extinction was significantly reduced in a bilateral parietal patient when a black and a white square were presented simultaneously, one in each hemifield. In the present study we demonstrate a bilateral deficit for rapid transient events that are quite dissimilar. When the target is a light object, the distractors are dark objects and vice versa. Despite the fact that the target in our task always differed from the distractors, the patients were unable to identify the target normally when the transient events were simultaneous in time. This result differs from classic extinction. Instead, we suggest that the deficit arises from an inability to distinguish the onsets from the offsets of the squares. Our right parietal patients can easily detect a flickering target at high frequencies of flicker (see Experiment 2), but they have difficulty identifying the target that is flickering out of phase with the distractors. The cause is not solely a loss in temporal resolution, as the patients' performance improves dramatically when the transients of the target are shifted out of synchrony with the transients of the distractors (as in the 90° phase shift in Experiment 3).

Our deficit does not appear to be related to simultanagnosia (Luria, 1959). If our patients were unable to attend to more than one object at a time, they would certainly have difficulty identifying the phase relation between different objects, such as the target and the distractors in our task. However, they should have just as much or more difficulty for the 90° condition as they did for the 180° condition, as here again the target is only defined by its relation to the distractors. This

was not the case—the patients performed well in the 90° condition. Moreover, there were no clinical indications of simultanagnosia for our patients. Simultanagnosic patients more often sustain bilateral parietal damage and only rarely present with inferior parietal damage (Kase *et al.*, 1977). However, a milder form of simultanagnosia (with impaired perception of overlapping figures presented at the same spatial location) can occur more commonly after right hemisphere lesion (De Renzi *et al.*, 1969). Simultanagnosia is also usually associated with Bálint's syndrome (Husain and Stein, 1988), manifesting a psychic paralysis of fixation, among other symptoms, whereas our patients did not show such symptoms.

The results demonstrate that the right parietal patients have difficulty discriminating offsets and onsets only when they occur simultaneously in our rapid test sequences. We showed that the deficit was not caused by masking between these simultaneous transients: performance did not decrease as the number of simultaneous transients was increased (Experiment 4). We also showed that the loss was not related to interhemispheric transmission delay brought on by the presence of simultaneous transients in both visual fields: the deficit was unchanged for stimuli presented unilaterally (Experiment 5). In contrast, we did find a dramatic improvement in performance at 90° shift condition in Experiment 3. In this condition, the transients of targets and distractors are no longer synchronous and we believe this is the key to understanding the deficit seen in the other conditions. With the 90° offset, the target and distractor transients do not have to be identified as onsets or offsets in order to detect the target. The target is now the only transient in the field and can be detected as a unique feature. The ease with which the right parietal patients identified this unique target is consistent with evidence showing that left neglect patients perform normally on a unique feature, pop-out tasks in *both* fields (Esterman *et al.*, 2000). Studies on normals show that transients like those in our displays are pop-out features, as is polarity itself (white targets in black distractors), either alone (Gilchrist *et al.*, 1997) or in conjunction with other features (Theeuwes and Kooi, 1994).

Several studies have reported delayed or slowed processing for right parietal patients. Judgements of simultaneity (Rorden *et al.*, 1997) show a delay in registering events in the left compared with the right visual field and studies of the attentional blink (Husain *et al.*, 1997) indicate that attention is disrupted over a longer period in these patients. Similar effects may also underlie the disorders in the orienting of attention in time that have been demonstrated recently in unilateral parietal patients without neglect (Shapiro *et al.*, 2002). In contrast, the deficits seen in our right parietal patients cannot be explained by a loss in temporal resolution or by delayed or slowed processing. If delayed or slowed processing were playing a role in the poor performance at 180° phase shifts, it would also have to degrade the performance for 90° phase shifts, but this did not happen. Performance in the 90° condition was almost at normal levels,

indicating that, at least for this task, loss in latency, speed or temporal resolution of response was not an important factor. The deficit, we suggest, is not the speed of the response but the identification of the response as onset or offset.

Why should parietal damage lead to difficulty in identifying whether a transient is an onset or an offset? This seems to be a very low-level loss for such a high-level cortical area. Recall, however, that the patients have no loss in the basic low-level task of detecting flicker at very high rates. Their loss is at more moderate rates, at which normals can see not only that a light is flickering but also that it is either on or off at each given moment. This ability to individuate the successive states of the light gives way to an experience of steady flicker at a frequency of 8 Hz or so in normals, and this threshold rate is called the Gestalt flicker fusion rate (van de Grind *et al.*, 1973). Above this limit of 7–10 Hz, subjects cannot perform attentive tracking, report the direction of apparent motion, or make phase judgements for widely spaced items (Forte *et al.*, 1999; Verstraten *et al.*, 2000; Victor and Conte, 2002). Below this rate, the transient attention system can individuate each successive state, whereas above this rate the only experience is one of flicker and no scrutiny of individual states is possible. It has been suggested that this limit is associated with higher-level attentional processes (Forte *et al.*, 1999; Verstraten *et al.*, 2000). We found in Experiments 1, 3 and 4 that the control subjects have a threshold that is never above 8 Hz, indicating that our task probably also relies on this rapid process of attentive individuation.

We suggest that the right parietal lesions have damaged this aspect of the transient attention system. In particular, one step in interpreting a sequence of transients as individual states is to determine whether each on- and off-transient in the sequence corresponds to the appearance or the disappearance of an object. This is not trivial: an on-transient may be the appearance of a light object or the disappearance of a dark object. Specifically, appearance or disappearance is not simply a matter of brightening or dimming. The object appearing can be white on a dark background (thus producing brightening in the display) or a dark object on a light background (dimming in the display). We claim that the reason the parietal area plays a role is because transients must be interpreted in terms of object polarity; in other words, whether the object itself is light or dark. If this interpretation process is degraded, the patients can detect the transients but they cannot identify them (experience them as appearances or disappearances) except at slower rates. We propose that this is the higher-level function that is impaired in the right parietal patients.

In agreement with our results, recent neurophysiological findings (White *et al.*, 2002) suggest that temporal phase discrimination occurs at a cortical level. Concurrently, functional MRI studies show a strong right-hemisphere dominance in regions of the right parietal cortex that are transiently active when attention shifts between spatial locations (Yantis *et al.*, 2002) or during tasks where targets

are presented at unexpected locations (Corbetta *et al.*, 2000). The same regions are involved in all our right parietal patients' lesion. The fact that the deficit is not present in our left parietal patients with comparatively similar lesions strongly suggests that the right parietal lobe is specialized in determining *when* objects appear and disappear. This finding reveals a new component in the visual deficit of neglect patients. Specifically, our results suggest that there is a broad deficit in both visual fields that would limit the patients' ability to process simultaneous onsets and offsets. Any information displays, computer interfaces or natural environments that require the patient to monitor rapid changes would appear confusing and disorganized even in their good field. However, it is important to know that this ability is not lost at slower speeds. Our results, together with previous data (Robertson *et al.*, 1998; Husain and Rorden, 2003), suggest new and more appropriate approaches to rehabilitation.

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