Neglected Time: Impaired Temporal Perception of Multisecond Intervals in Unilateral Neglect

James Danckert[1], Susanne Ferber[2], Carson Pun[2], Carol Broderick[1], Christopher Striemer[1], Sherry Rock[3], and Dwight Stewart[1,4]

Abstract

■ Recent neuroimaging and neuropsychological studies have suggested that the right hemisphere, particularly frontal regions, is important for the perception of the passage of time. We examined the ability to estimate durations of up to 60 sec in a group of eight patients with unilateral neglect. When estimating multisecond intervals, neglect patients grossly underestimated all durations. On average, healthy controls (HC) demonstrated reasonably accurate estimates of all durations tested. Although the right hemisphere lesioned control patients without neglect also tended to underestimate durations, these underestimations were significantly better than the performance of the neglect group. These findings suggest a pivotal role for a right hemisphere fronto-parietal network in the accurate perception of multisecond durations. Furthermore, these findings add to a growing body of literature suggesting that neglect cannot be understood simply in terms of a bias in orienting attention to one side of space. Additional deficits of the kind demonstrated here are likely to be crucial in determining the nature and extent of the loss of conscious awareness for contralesional events. ■

INTRODUCTION

Lesions of the right posterior parietal or superior temporal cortex often lead to the disorder of unilateral neglect in which patients fail to attend to or respond to stimuli in the contralesional—in this case left—side of space (Danckert & Ferber, 2006; Mort et al., 2003; Karnath, Ferber, & Himmelbach, 2001). Patients may fail to eat food from the left half of a plate, fail to groom the left side of their body or face, and will bump into objects on the left side of space (see Danckert & Ferber, 2006 for review). Neglect is not typically considered a disorder of primary perceptual or motor capacities, but is instead thought to be due to deficient orienting of attention toward contralesional space (Husain & Rorden, 2003; Driver & Mattingley, 1998). More recent findings have suggested that, in addition to impaired attentional orienting—which may manifest itself as a severe bias toward processing *ipsilesional* stimuli—neglect patients suffer from impairments to spatial working memory and saccadic or spatial remapping (Ferber & Danckert, 2006; Malhotra et al., 2005; Malhotra, Mannan, Driver, & Husain, 2004; Pisella, Berbevoric, & Mattingley, 2004; Pisella & Mattingley, 2004; Wojciulik, Rorden, Clarke, Husain, & Driver, 2004; Husain et al., 2001). This strongly suggests that a simple attentional bias toward ipsilesional space is unlikely to be the only factor needed to produce the cardinal symptom of neglect—a loss of awareness for contralesional events.

Furthermore, several authors have demonstrated impairments in temporal processing in neglect. One example of a spatio-temporal deficit in neglect comes from the so-called temporal order judgment (TOJ) task in which subjects must determine which of two lateralized stimuli was presented first (Berberovic, Pisella, Morris, & Mattingley, 2004; Robertson, Mattingley, Rorden, & Driver, 1998; Rorden, Mattingley, Karnath, & Driver, 1997; note: Rorden et al., 1997 investigated patients with extinction). In this task, the left stimulus can precede the right (or vice versa) by varying amounts of time, and the two targets can also be presented simultaneously. For healthy individuals, the subjective point of simultaneity, in which they respond ''left first'' (or ''right first'') on around 50% of trials, coincides nicely with the objective point of simultaneity (Stelmach & Herdman, 1991). In contrast, for neglect patients, a bias is seen in TOJs such that the left stimulus must precede the right by more than 250 msec before the patient accurately reports that it was presented first (Berberovic et al., 2004; Robertson et al., 1998). However, the delayed processing of the left stimulus may be due to various sources of error, including a difficulty in disengaging attention from the ipsilesional side in a timely manner.

Demonstrations of nonspatial impairments in neglect patients—impairments that could be taken to reflect to some extent impaired temporal processing—have

[1]University of Waterloo, Canada, [2]University of Toronto, Canada, [3]Freeport Rehabilitation Hospital, Kitchener, ON, Canada, [4]Grand River Hospital, Kitchener, ON, Canada

come from studies examining auditory attention (e.g., sustained auditory attention, frequency and pitch discrimination; Cusack, Carlyon, & Robertson, 2000; Robertson et al., 1997) or the allocation of attention in the temporal domain (e.g., using the attentional blink task; Hillstrom, Husain, Shapiro, & Rorden, 2004; Snyder & Chatterjee, 2004; Shapiro, Hillstrom, & Husain, 2002; Husain, Shapiro, Martin, & Kennard, 1997; Chun & Potter, 1995; Raymond, Shapiro, & Arnell, 1992; see Schneider, 1999 for review; see also Rizzo, Akutsu, & Dawson, 2001 for a demonstration of an altered attentional blink in patients with discrete cortical lesions without neglect). It is important to emphasize here that there is a difference both in behavioral and neural terms between attention *to* time, the allocation of attention over time, and the perception of time per se (Buhusi & Meck, 2005; Coull, 2004). Although the first two capacities will undoubtedly rely on and interact with mechanisms of attentional selection and temporal perception, the perception of the passage of time itself is also likely to depend on distinct behavioral and neural properties from those networks and mechanisms involved in attention (see Buhusi & Meck, 2005, for a review). For example, although an increased attentional blink may not entail any specific spatial biases, it may, nevertheless, be indicative of a more fundamental problem of disengaging attention evident in neglect patients, rather than reflecting any impairment of the perception of the passage of time itself. Finally, in a recent review, Husain and Rorden (2003) suggested that deficits in the temporal allocation of attention (evident in such tasks as the attentional blink) merely *exacerbated* the spatial deficits evident in neglect. This point has also been made by Robertson et al. (1997) who demonstrated a strong correlation between nonlateralized deficits of sustained auditory attention and spatial biases, as demonstrated on clinical tests of neglect. Another related possibility is that the temporal deficits in attention are related to reduced levels of tonic arousal in neglect patients. This hypothesis gains some support from the finding that a phasic alerting cue (a loud, spatially noninformative tone) can improve TOJs in neglect patients (Robertson et al., 1998).

As mentioned above, exploration of the temporal dynamics of the allocation of attention in and of itself does not explicitly address the capacity to perceive the passage of time. Although we appreciate that the perception of the passage of time is necessarily distinct from perceiving basic physical properties of events in our environment (i.e., given that we do not perceive time itself per se), there are, nevertheless, commonly used tasks to assess the perception of the duration of physically perceivable events. More specifically, one task involves the discrimination of various stimulus durations with reference to a standard duration (see Buhusi & Meck, 2005; Wearden, 1999, for a review). For example, the subject may be presented with a pair of tones separated by a standard temporal duration (usually in the millisecond range) followed some time later by a second pair of tones that may be separated by a longer, shorter, or identical temporal duration. The task then is to determine whether the duration between the two tones in the second pair was longer or shorter than the standard duration. Typically, poorer discrimination performance is seen for durations that are longer than the standard when contrasted with durations that are shorter than the standard (see Wearden, 1999, for a review). Using a procedure similar to this, Harrington, Haaland, and Knight (1998) found that patients with right hemisphere brain damage were more impaired than patients with left hemisphere damage. The nature of the impairment was such that difference thresholds were elevated in the patients, indicating that durations longer than the standard were more often confused with it than durations that were shorter than the standard. In other words, the right hemisphere patients demonstrated an *exaggeration* of the normal tendency observed in this kind of interval timing task. Lesion overlay analysis demonstrated that the prefrontal and premotor cortices were commonly involved in a group with anterior right hemisphere lesions, whereas the inferior parietal cortex was always involved in the group with posterior right hemisphere lesions (Harrington et al., 1998). These findings are in accordance with single neuron recordings in nonhuman primates using similar duration discrimination tasks (Oshio, Chiba, & Inase, 2006; Leon & Shadlen, 2003). That is, neurons on the lateral intraparietal region demonstrate an increase in firing rate during encoding of the standard duration and throughout the delay period prior to making a comparison with the test duration (Leon & Shadlen, 2003). Prefrontal neurons show modulation of firing rates depending on the presentation order of short and long durations, indicating a role for this region in the implementation of a strategic set related to temporal processing (Oshio et al., 2006). Interestingly, of the human patients with right hemisphere lesions studied by Harrington et al. (1998), only 2 of the 18 demonstrated neglect. The authors concluded that a prefrontal–parietal network in the right hemisphere is critical for temporal perception and went further to suggest that the inferior parietal involvement may be explicitly linked to covert shifts of attention within the temporal domain (Harrington et al., 1998). This latter hypothesis is somewhat akin to the possibility we raised above that impaired TOJs and a prolonged attentional blink in neglect patients may be related to a more general deficit in disengaging attention.

Only one single case study to our knowledge has explicitly examined temporal perception in neglect (Basso, Nichelli, Frassinetti, & di Pellegrino, 1996). In one experiment, the patient was asked to discriminate between short (300 msec) and long (700 msec) stimulus durations presented at various locations within the right visual field (the patient had already demonstrated neglect

for the leftmost stimuli presented within the right visual field). Stimuli in the leftmost positions were judged by the patient to be longer than stimuli in the rightmost positions (Basso et al., 1996). That is, the patient made substantially more discrimination errors when presented with a short duration in leftmost positions and, conversely, made more discrimination errors for long durations presented in the rightmost positions. In a subsequent experiment, the patient was asked to estimate 1-sec durations by pressing a space bar to extinguish a visual stimulus presented on a computer screen. After training with stimuli presented directly above fixation, the patient's performance was similar to that of controls. However, when stimuli were now presented throughout the right visual field (as in the first experiment), the patient again tended to overestimate the 1-sec duration for the leftmost stimulus positions (Basso et al., 1996).

Both the single case study (Basso et al., 1996) and the investigation of a larger group of right hemisphere lesioned patients discussed above (Harrington et al., 1998) examined temporal perception at the subsecond level (or at most a single second level; Basso et al., 1996) and found a general tendency toward overestimation of durations as a consequence of right hemisphere damage. In a recent study using repetitive pulse transcranial magnetic stimulation (rTMS), results showed that reaction times (RTs) to make subsecond temporal discriminations were slowed only after rTMS was applied to the right inferior parietal cortex and not the left (Alexander, Cowey, & Walsh, 2005). The effects were not only site-specific but also task-specific, given that pitch discriminations were unaffected by rTMS over either the right or left parietal cortex. Taken together, these results make a strong case for a role for the right parietal cortex in temporal perception at the subsecond level. One interpretation of this role would suggest that such fine-grained temporal distinctions are crucial for the control of goal-directed actions (Alexander et al., 2005; Buhusi & Meck, 2005; Walsh, 2003a). The inferior parietal cortex is ideally poised to integrate processing from the dorsal ''action'' stream—from V1 to more superior regions of the posterior parietal cortex—with processing in the ventral ''perception'' stream—from V1 to the inferotemporal cortex (Goodale & Milner, 1992). Recent research has also suggested that the right parietal cortex is involved in comparing forward models of intended actions with actual sensory outcomes in order to modify goal-directed actions on-line (e.g., Danckert et al., 2002; Sirigu et al., 1996). For these and other motor functions, subsecond timing will obviously be critical. One final suggestion based on neurophysiological studies of nonhuman primates and functional neuroimaging in humans would be that there are two distinct systems for processing subsecond and multisecond intervals (the latter is often referred to simply as ''interval timing''; Buhusi & Meck, 2005; Lewis & Miall, 2003a, 2003b). This model would suggest that millisecond timing depends most heavily on the cerebellum and is more automatic in nature, whereas flexible control of multisecond temporal perception is more likely to depend on the basal ganglia and its connections with parietal and prefrontal cortices (Buhusi & Meck, 2005; Lewis & Miall, 2003a).

We wanted to investigate temporal perception in neglect patients at the multisecond level to determine whether neglect leads to a more fundamental and pervasive impairment in the ability to perceive the passage of time. In other words, would neglect patients show impaired temporal perception for durations longer than those thought to be involved in accurate motor control (e.g., Alexander et al., 2005)? A second goal of our study was to develop a test for the perception of the passage of time that does not involve comparisons to a standard interval. We felt that this was important given that neglect patients seem to have difficulty disengaging attention in a timely manner even from nonspatial stimuli and that their spatial working memory capacity has been demonstrated to be severely compromised (Ferber & Danckert, 2006; Malhotra et al., 2004, 2005). To do this, we had a group of patients with neglect (NP) perform a temporal estimation task in which an illusory motion stimulus was presented centrally for various multisecond durations after which the patient gave a verbal estimate of the duration in seconds (Figure 1). A group of eight healthy older individuals (HC) and six patients with right brain damage (RBD) without neglect acted as controls.

METHODS

Participants

Clinical details for the patients with neglect (NP) are presented in Table 1.

Patients were defined as having neglect on the basis of line bisection, cancellation, and figure copying performance. A significant rightward bias on line bisection was defined as anything greater than 5% of the total line length, whereas impaired cancellation performance was defined as anything greater than 10% omissions of targets on the left side. Neglect was scored as present or absent on figure copying by visual inspection of the patient's performance. Patients were considered to have neglect if they met at least two of these three criteria (deviation in line bisection, more than 10% omissions of leftward targets in cancellation tasks, omissions in figure copying). Neglect patients were further characterized as having mild, moderate, or severe levels of impairment based on laterality scores for cancellation, line bisection, and figure copying (see Schindler, Clavagnier, Karnath, Derex, & Perenin, 2006). For line bisection, rightward deviations were converted to a percentage score based on total line length, whereas for cancellation performance the laterality score was derived from the raw

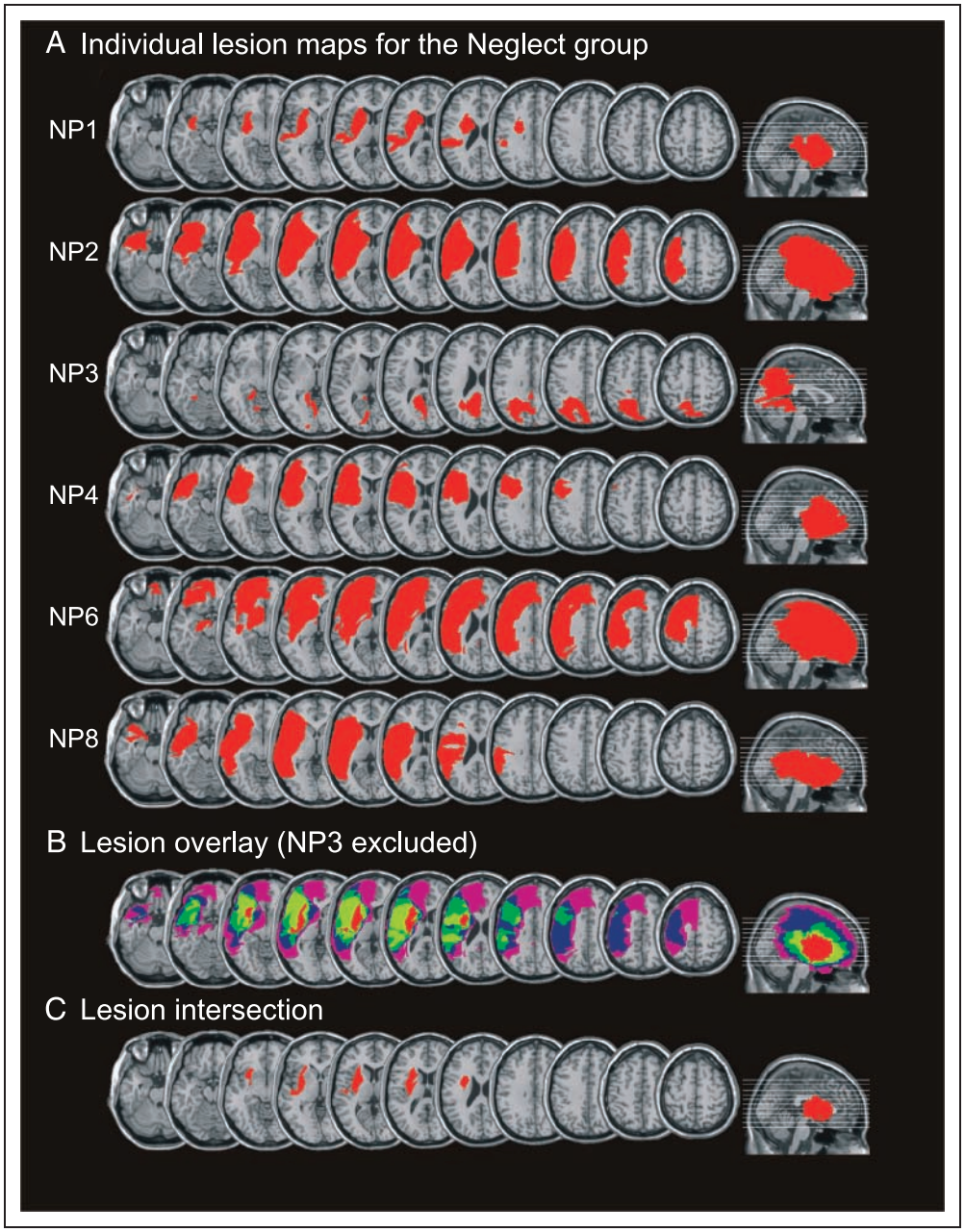

Figure 1. (A) Individual lesion maps for the six neglect patients with scan data available. (B) Lesion overlay analysis for five of the six patients (NP3 failed to show any overlap and so was not included in this analysis). Each patient's lesion is given a distinct color with the region of overlap for all five patients indicated in red. (C) Lesion intersection analysis. The common areas lesioned in five of the six patients are indicated in red (essentially, this is the same region highlighted in red in B presented here in isolation for clarity). We found overlap mainly in the insula, supramarginal gyrus, caudate, putamen, and the superior temporal gyrus.

number of omissions to the left side minus the right-sided omissions expressed as a percentage of the total number of omissions. For each task within each individual patient, laterality scores were assigned a level of severity in the following manner: 0–33% = mild, 34–66% = moderate, and 67–100% = severe. An overall severity rating for each individual patient was then obtained by taking the median severity score across all tasks for that particular patient (Schindler et al., 2006). Six patients with RBD (all men) also acted as controls. None of the RBD patients presented with neglect on any of the tests used. Mean age of the RBD group was 66 (±14.4 *SD*) years. On average, the patients were tested more than 24 months post onset of their stroke (patient RBD3 was the only patient tested within 1 month of stroke onset).

Individual lesions for the neglect patients are presented in Figure 1, whereas the RBD patients' lesions are presented in Figure 2.

Lesion overlay analysis was conducted for each patient group to control for possible effects of lesion sites on our behavioral data. For the neglect group, one patient's scans (NP7) showed no abnormality. This may have been related to the time at which the scan was taken as computed tomography (CT) images collected in the very earliest stages poststroke often fail to show abnormalities. Unfortunately, this patient does not reside locally, making it unfeasible to conduct a repeat scan. For a second patient (NP5), although we were able to obtain a radiologist's report for her CT scans, we could not obtain the scan data itself due to the fact that she

Table 1. Clinical Details of Patients

				Cancellation				
Patient	*Age*	*Sex*	*Time Post Stroke (Months)*	*L*	*R*	*Figure Copy*	*LB*	*Severity*
NP1	68	F	4	18.5	3.7	+	5.08	Mild (12.5%)
NP2	55	M	22	66.67	33.33	+	9.11	Mild (31.25%)
NP3	88	F	3	96.3	29.6	+	19.7	Mild (25%)
NP4	78	F	4	23.53	5.9	+	3.98	Moderate (60%)
NP5	76	F	6	24	9.8	+	4.9	Mild (25%)
NP6	55	M	11	14.8	7.4	+	5.9	Moderate (33.33%)
NP7	78	M	4	40.7	0	+	7.3	Severe (66.67%)
NP8	51	M	3	26.67	6.67	+	2.5	Mild (25%)
RBD1	73	M	12	3.2	0	−	1.14	n.a.
RBD2	71	M	4	0	0	−	−1.6	n.a.
RBD3	57	M	>1	0	0	−	−4.6	n.a.
RBD4	64	M	>1	0	0	−	−7.36	n.a.
RBD5	45	M	4	0	0	−	−5.7	n.a.
RBD6	86	M	55	0	0	−	−7.36	n.a.

LB = line bisection task. Performance on line bisection was calculated as the amount of deviation from true center and then converted to a percentage of the total line length (rightward deviations are scored as positive). Cancellation performance represents the percentage of omitted left (L) and right (R) sided targets. Note that for all patients ''cancellation'' performance was derived from the star cancellation task with the exception of patients NP4 and NP8, who completed the bells cancellation task, and NP5, who completed a letter cancellation task. Neglect on a figure copying task is indicated by the + symbol (absence of neglect on figure copying is indicated by −). Severity measures were derived from a formula in Schindler et al. (2006) (see Methods section).

had been seen initially at a regional hospital outside of our area. Lesion data were analyzed using the protocol outlined by Ferber and Danckert (2006). All lesions (defined as the hypointense or hypodense stroke compared to its surrounding parenchyma) were traced on CT scans on a slice-by-slice basis using ANALYZE 7.0 AVW Software (Biomedical Imaging Resource, Mayo Foundation, Rochester, MN; Robb, 2001; Robb & Barillot, 1989; Robb et al., 1989). All scans were then transferred to the ICBM152 template from the Montreal Neurological Institute (www.bic.mni.mcgill.ca/cgi/icbm_view), based on the average of 152 normal MRI scans approximately matched to Talairach space. This transformation was a two-step process using Automatic Image Registration version 5.2.5 software (AIR; http://bishopw.loni.ucla.edu/AIR5): the first step was a spatial normalization protocol including a linear 12-parameter affine transformation (including aligning scans to AC–PC aligned Talairach space). The second step was a nonlinear fourth-order parameter warping model to make scans fit best to the template. The resulting images had a voxel size of 1 mm × 1 mm × 1 mm. Using the transformed lesion maps, we estimated the proportion of each Brodmann's area or anatomical region involved in each patient's lesion, using the ''broadmann'' and ''AAL'' templates in MRIcro (http://people.cas.sc.edu/rorden/). To then superimpose the individual brain lesion maps, the template ''ch2'' in MRIcro was used (Rorden & Brett, 2000). We should point out here, however, that lesion overlay analysis on such a small sample size is problematic. Certainly, with such a small sample size, we are not able to address questions related to the critical lesion site for neglect or, for that matter, the critical lesion location for any observed temporal estimation deficits (see Karnath et al., 2001).

All participants gave informed consent prior to participating in the experiment and the experimental protocol received ethics approval from the Office of Research at the Universities of Waterloo and Toronto and the Tri-Hospital Research Ethics Board of Kitchener-Waterloo in accordance with the Helsinki declaration. Eight neurologically healthy older controls with normal or corrected-to-normal vision (4 men; mean age = 73 years; range = 60–74 years) also completed the temporal estimation task after giving written consent. Healthy controls were excluded prior to participation if they reported any history of neurological or psychiatric illness.

Apparatus and Procedure

Patients and controls were seated approximately 50 cm in front of a computer screen with a refresh rate of 75 Hz.

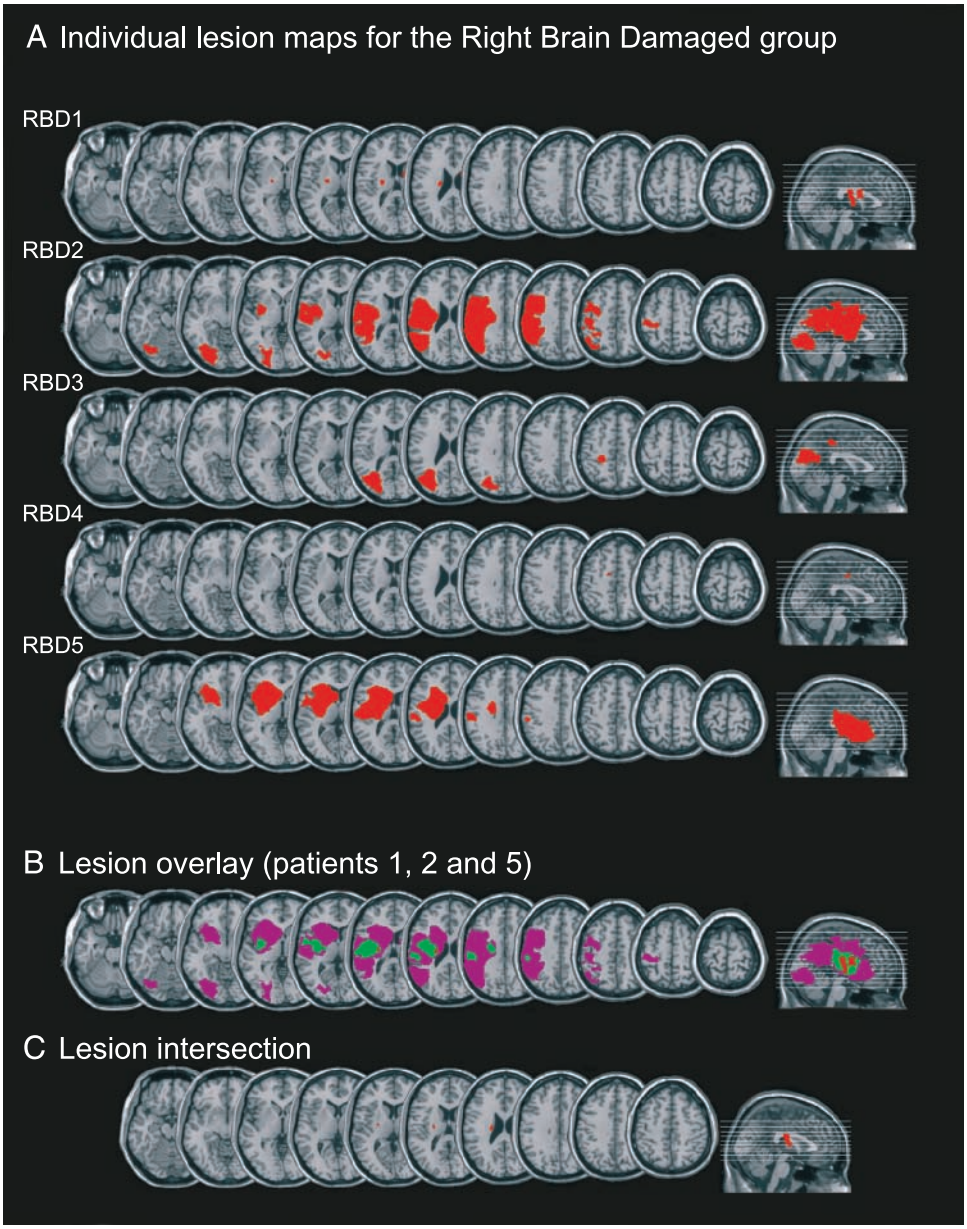

Figure 2. (A) Individual lesion maps for the five RBD patients with scan data available. (B) Lesion overlay analysis for three of the five patients (patients 1, 2, and 5). The remaining two RBD patients failed to show any overlap with any other patient. Each patient's lesion is given in a distinct color with the region of overlap displayed in red. (C) Lesion intersection analysis. This is essentially the same as the lesion overlay analysis but visually depicts only those areas commonly lesioned in the three RBD patients included in the analysis. Overlap was found in very discrete regions of the right basal ganglia including the lentiform nucleus and the thalamus.

Participants were free to move their head and eyes but were encouraged to maintain a stable posture throughout the experiment. E-Prime software (Psychology Software Tools) was used to present stimuli and record the participant's responses, which were made via a standard keyboard. An illusory motion stimulus was created as the stimulus event that required temporal estimation. The stimulus array consisted of eight open circles (each circle subtends 3.5° of visual angle) arranged in a larger circle around a central point on the screen. The radius of the larger circle around which the smaller circles were arranged subtended approximately 8° of visual angle. Each circle was filled-in in sequence in a clockwise direction (Figure 3). No fixation point was used; however, a secondary requirement to read aloud numbers (subtending 1° of visual angle horizontally and 1.5° vertically) presented in the center of the illusory motion stimulus (described in more detail below) effectively meant that participants fixated the center of the display.

Illusory motion was used as the stimulus duration to be estimated to ensure there was a salient event to capture and maintain the patient's attention throughout a trial. In previous work on temporal estimation in humans, the stimulus event to be estimated is often simply the presence of a geometrical shape (see Wearden, 1999, for a review). We felt that it was important to employ a more engaging stimulus to ensure full attention throughout a trial. In initial development, we also felt that modulating the speed of the illusory motion stimulus may prove to be a useful means of examining

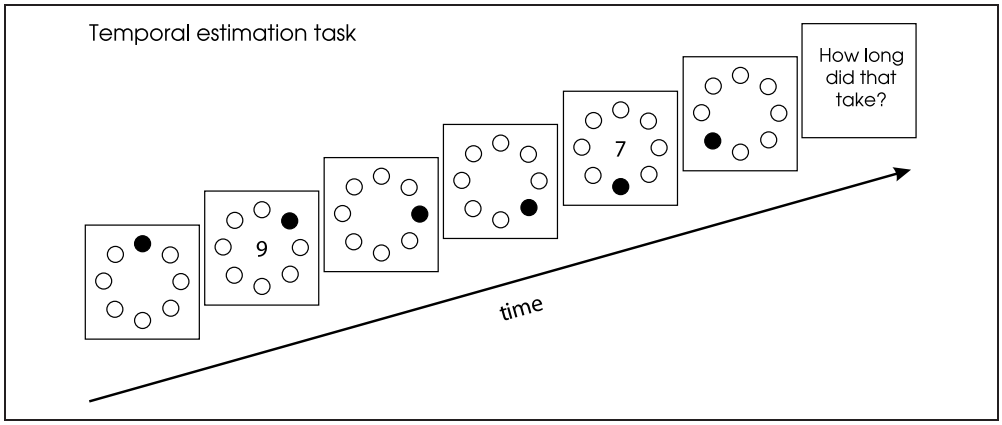

Figure 3. Schematic representation of the temporal estimation task. An illusory motion stimulus was presented centrally with numerals appearing in the center of the large circle at random temporal intervals. Participants read these numbers aloud to prevent them from counting durations in their head. A trial was started by the experimenter and, at the end of each duration, the participant was asked to indicate how long they felt that particular event had lasted, making their estimates to the nearest whole number in seconds.

potentially subtle differences in performance. As will become obvious later, modulating the speed of illusory motion for the patients turned out to be unnecessary. Pilot testing in healthy individuals demonstrated that the speed of the illusory motion stimulus had no influence on temporal estimations. That is, in six healthy younger individuals, we examined temporal estimations with illusory motion speeds of 2.5, 0.83, and 0.42 cycles per second (where a cycle is one full rotation of the illusory motion stimulus; the speeds used correspond to a single circle in the display being filled-in for 50, 150, and 300 msec, respectively). Temporal estimations did not differ at these different illusory speeds. Therefore, the illusory motion speed used for the patients and older controls was kept constant at 0.83 cycles per second (i.e., 150 msec duration for each circle being filled-in).

During the presentation of the illusory motion stimulus, participants were also asked to report verbally the identity of single numerals (numbers from 1 to 9 were used) presented centrally for 300 msec at pseudorandom temporal intervals throughout a trial (Figure 3). Certain conditions were placed on the timing of presentation for the centrally presented random numbers. First, no random numbers could appear within the first or last 500 msec of a trial to ensure that these critical time points did not involve any distraction from the secondary task. Second, the minimum duration between numbers was set at 500 msec to ensure sufficient processing time between numbers. Finally, the maximum duration between numbers was set at 1500 msec to ensure that more than one number was presented during the smallest duration trials (i.e., 5-sec trials). Within these last two parameters (i.e., min and max duration between numbers), the temporal duration between numbers to be reported varied randomly. Although pilot testing revealed that reporting the centrally presented numbers did not dramatically influence the temporal estimations in a group of younger healthy controls, we nevertheless felt that it was necessary to prevent participants from silently counting out the durations to be estimated. One further justification for including the requirement to report centrally presented numbers involves Scalar Timing Theory (STT; Figure 4; Wearden, 1999; Gibbon, 1977). This influential theory was originally developed to explain many of the results in animal timing experiments which typically involved the discrimination of subsecond durations (see Wearden, 1999; Gibbon, 1977, for reviews). The model includes a pacemaker that emits pulses possibly in the form of a Poisson distribution, characterized by an average rate of pulse emission that remains constant while individual durations between pulses are random (Gibbon, 1977, 1992). When estimating the duration of a stimulus event, the pulses emitted by the pacemaker are gated by a switch to an accumulator which presumably represents and stores these pulses accurately (some versions of STT suggest different contributions from the accumulator and short-term or working memory stores whereas others simply conflate the two). Information contained in the accumulator is then compared to some standard duration, perhaps maintained in a long-term memory store, and a decision is made regarding this comparison (i.e., is the just perceived duration longer or shorter than the standard? Figure 4).

Although this model has proven invaluable in accounting for the timing behavior of animals and humans at the subsecond level, there have been fewer examples of its application to the perception of multisecond intervals. Importantly, recent work suggests that for humans, the properties of scalar timing are only likely to apply to durations longer than a second if internal counting is prevented (Rakitin et al., 1998; Wearden, Denovan, Fakhri, & Haworth, 1997). Thus, we felt it was imperative to include the secondary task of identifying numbers in the temporal estimation task. The fact that our pilot tests failed to show any differences in estima-

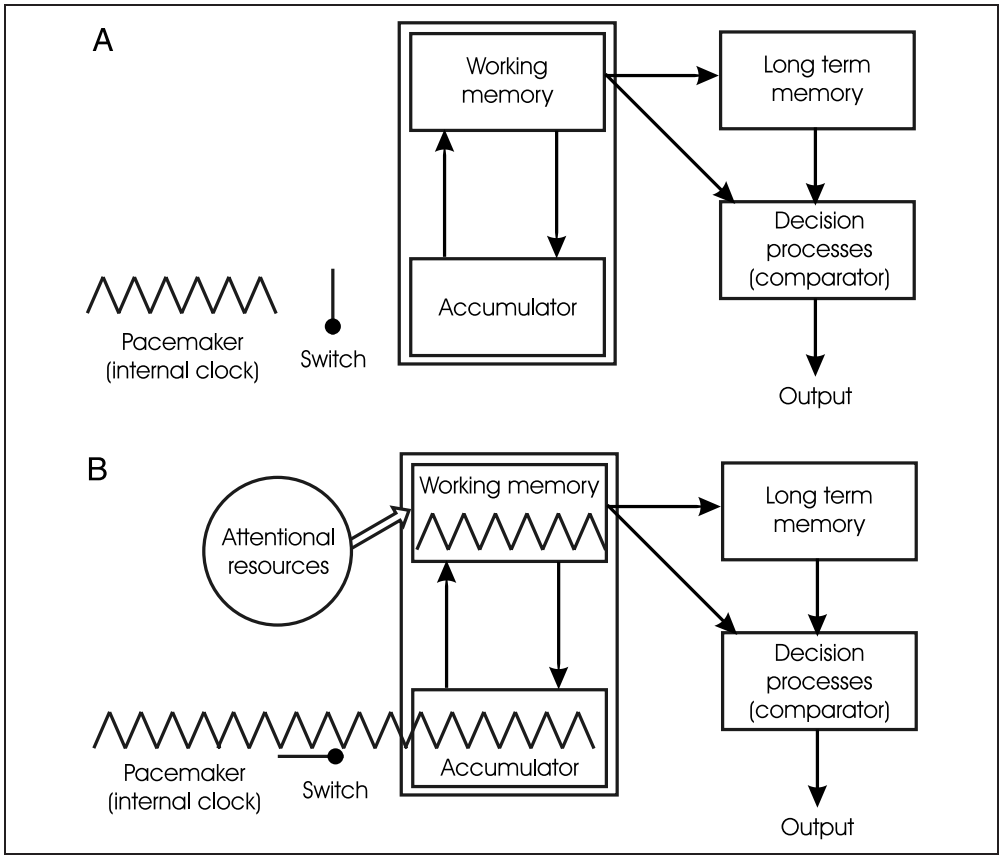

Figure 4. Schematic representation of the component processes hypothesized to be responsible for temporal perception in the Scalar Timing Theory (STT; see Wearden, 1999). A pacemaker or internal clock emits pulses in a Poisson distribution (A). These pulses are gated by a switch (B) to an accumulator that may also involve short-term or working memory functions. Pulses collected in the accumulator are compared to prior experience in some long-term memory store and a decision regarding the current time interval is made. Theoretically, attentional resources can be applied at any stage of the model, however, they are indicated here as having a direct impact on processing within the accumulator (B).

tions when the secondary task was included does not mean it is an ineffective manipulation. Indeed, all our participants reported that this requirement made it impossible for them to count durations in their head.

Each trial was started by the experimenter pressing the space bar on the keyboard once the patient had indicated they were ready. At the conclusion of the duration, the patient was asked to estimate to the nearest second how long the stimulus had been present on the computer screen. Durations of 5, 15, 30, and 60 sec were used. Durations shorter than 5 sec were not chosen as pilot work had suggested there was very little variance in responses made at these durations in either patients or healthy controls. Patients completed four trials at each duration with durations randomized throughout the task.

Data Analysis

Mean raw estimates at each duration were calculated for each individual participant. These data were then submitted to an analysis of variance (ANOVA) with group as the between-subject factor (HC, RBD, and NP) and duration as the within-subjects factor (5, 15, 30, and 60 msec). In addition, for each individual, a least squares regression analysis was used to determine the goodness of fit between an individual's estimates and the actual durations. From these equations, group mean r^2, slope, and intercept values were contrasted between the groups using independent-samples t tests. Finally, variance measures (i.e., standard deviation, *SD*) for each individual were calculated at each duration separately. The coefficient of variance (*SD*/mean) for each individual, for each duration, was then examined to determine how well the performance of each group conformed to expected properties of STT. That is, STT would predict that although variance increases as the duration to be estimated also increases, the coefficient of variance should remain stable (Buhusi & Meck, 2005; Wearden, 1999). Group means were calculated from individual coefficient measures and were assessed via an ANOVA with a between-subjects factor of group and a within-subjects factor of duration.

RESULTS

Temporal Estimations

ANOVA of the raw estimates made by each group indicated a significant Group by Duration interaction [$F(6, 57) = 7.93$; $p < .001$]. Independent-samples t tests at each of the durations tested indicated that controls made substantially higher estimates at all but the very first duration when contrasted directly with the neglect patients [5-sec duration: $t(9.67) = 1.62$, $p = .13$; 15-sec duration: $t(8.3) = 3.231$, $p < .01$; 30-sec duration:

Table 2. Mean Raw Estimates (*SD*) and Coefficient of Variance Measures for the Retrospective Estimation Task

	Duration (sec)			
	5	*15*	*30*	*60*
Raw Estimates				
Neglect	2.9 (0.77)	4.86 (1.7)	6.23 (2.01)	8.89 (3.18)
RBD	3.54 (1.7)	7.86 (4.35)	11.55 (6.01)	18.7 (11.28)
HC	3.82 (5.1)	11.1 (3.66)	28.06 (3.05)	49.26 (7.99)
Coefficient of Variance				
Neglect	0.23 (0.13)	0.24 (0.11)	0.24 (0.13)	0.18 (0.14)
RBD	0.34 (0.16)	0.19 (0.1)	0.16 (0.13)	0.17 (0.09)
HC	0.39 (0.21)	0.32 (0.18)	0.22 (0.12)	0.2 (0.15)

RBD = right brain damaged; HC = healthy controls.

$t(7.12) = 2.82$, $p < .05$; 60-sec duration: $t(7.17) = 3.39$, $p < .01$]. Note that Levene's test for equality of variance was significant at all four durations, indicating that variance was unequal across the two samples (hence, equal variances were not assumed and the appropriate degrees of freedom were chosen for the above *t* tests; see Table 2 and Figure 5).

The estimates made by the RBD controls fell, on average, somewhere in between that of the patients and healthy older controls (Figure 5). When contrasted directly to the healthy controls at each duration, the RBD group demonstrated significantly lower estimates at only the longest duration [5-sec duration: $t(12) = 0.59$, $p = .57$; 15-sec duration: $t(12) = 1.63$, $p = .14$; 30-sec duration: $t(12) = 1.92$, $p = .08$; 60-sec duration: $t(12) = 2.3$, $p = .04$]. Finally, when the RBD and neglect groups were contrasted directly, the neglect group showed significantly lower mean estimates for the 30- and 60-sec durations [5-sec duration: $t(12) = 1.02$, $p = .32$; 15-sec duration: $t(12) = 1.73$, $p = .11$; 30-sec duration: $t(12) = 2.27$, $p < .05$; 60-sec duration: $t(12) = 2.24$, $p < .05$].

For the neglect patients, despite the fact that they all massively underestimated even the longest durations, linear regression equations were significant for each individual patient's data (Table 3).

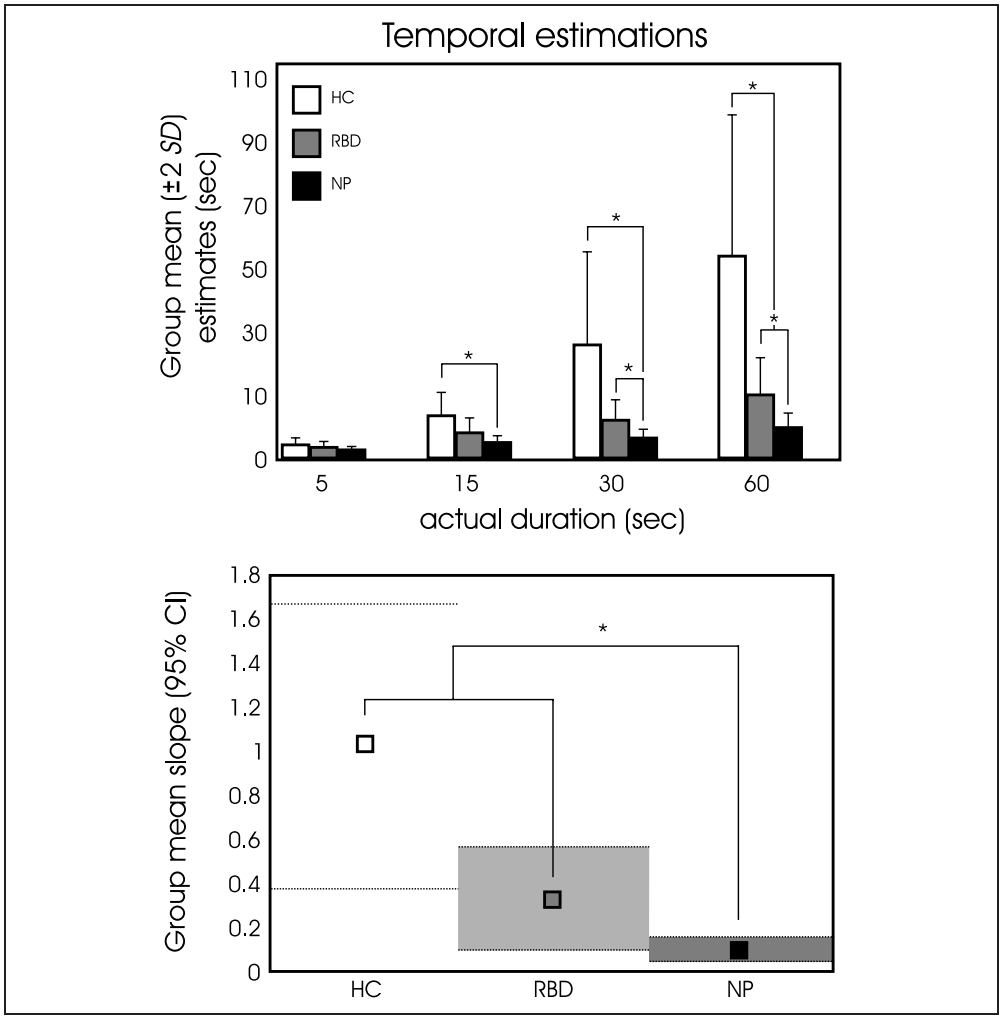

Figure 5. (Top) Group mean (±2 *SD*s) estimates in seconds for healthy older controls (HC; white bars), right brain damaged controls without neglect (RBD; gray bars), and the group of eight patients with neglect (NP; black bars). Significant differences between the groups are indicated by *. HC differed from RBD at only the longest duration while the HC group differed from neglect patients at all but the shortest duration. The RBD and neglect groups differed at both the 30- and 60-second durations. (Bottom) Group mean slope values for the HC (white squares), RBD (gray squares), and neglect groups (black squares). Horizontal dashed lines represent the 95% confidence intervals for each group (these areas are shaded for the RBD and NP groups). The slope for the neglect patients was significantly different from both the HC and RBD groups (indicated by *), whereas the latter two groups did not differ.

Table 3. Individual Linear Curves Fitted to Raw Temporal Estimations

	r^2	*Slope*	*Intercept*
NP1	.86	0.27	3.15
NP2	.71	0.1	3.8
NP3	.76	0.07	3.13
NP4	.57	0.09	2.54
NP5	.82	0.06	3.04
NP6	.85	0.08	0.82
NP7	.46	0.06	2.12
NP8	.73	0.12	4.24
Neglect Mean	**.72 (.14)**	**0.1 (0.07)**	**2.86 (1.05)**
RBD1	.88	0.33	3.02
RBD2	.85	0.5	1.55
RBD3	.87	0.6	0.21
RBD4	.85	0.05	0.85
RBD5	.33	0.08	5.96
RBD6	.92	0.42	6.5
RBD Mean	**.78 (.22)**	**0.33 (0.77)**	**3.02 (2.67)**
Controls Mean	**.88 (.11)**	**1.03 (0.22)**	**−0.74 (2.63)**

Group means (*SD*) are in **bold**.

That is, although the neglect patients underestimated the longest durations more so than either of the control groups, their estimations systematically increased with increases in the duration to be estimated. To compare the relationship between estimates made by each of the groups, we calculated linear regressions for each individual to obtain r^2, slope, and intercept values. Group means for each of these measures were then submitted to independent-samples t tests for each measure separately. When healthy controls were compared with the RBD group, only the intercept values were found to be significantly different [$t(12) = -2.63$, $p < .05$]. This difference is probably due to the nonsignificant difference in slope between the two groups (Figure 5B). That is, a shallower slope will lead to a higher intercept value. Put another way, the lower intercept value for the controls is indicative of their more accurate performance. When the healthy controls were contrasted with the neglect group, significant differences were observed on all measures [r^2: $t(14) = 2.49$, $p < .05$; slope: $t(14) = 3.38$, $p < .01$; intercept: $t(14) = -3.58$, $p < .01$; Figure 5). This indicates that, in contrast to the previous comparison between HC and RBD groups, the neglect group had a much shallower slope than the healthy controls. Although each individual neglect patient demonstrated a significant linear trend for their estimates, the strength of this trend at the group level was weaker than for the healthy controls as indicated by the lower r^2 value. Finally, direct comparison of the neglect group with the RBD controls found only a significant difference in the slope measure [$t(12) = 2.71$, $p < .05$; Figure 5].

Finally, we conducted an ANOVA on the coefficient of variance calculated for each individual at each time point. STT suggests that although variance measures should increase with increasing durations to be estimated, the coefficient of variance should remain constant. Our analysis found precisely that. For each group, there were no significant differences in the coefficient of variance across each duration (all ps $> .05$; Table 2). In other words, the estimates made by all groups conformed to expectations of STT (Wearden, 1999; Gibbon, 1977).

Lesion Overlay Analysis

Only seven of the eight neglect patients had scans available for lesion overlay analysis (NP5 was initially scanned at a regional hospital outside of our region with only the radiologist's report made available to us). Of those seven, one demonstrated no abnormality on CT (NP7), perhaps due to the early stage post stroke at which the scan was taken. Of the remaining six patients, one patient's lesion (NP3) failed to demonstrate any overlap with the remaining five patients. Therefore, lesion overlay and intersection analysis carried out on five of the eight neglect patients demonstrated that the common areas involved in these patients' lesions included the insula, the supramarginal gyrus, the putamen, the caudate, and the superior temporal sulcus (Figure 1). For the RBD group, scan data were only available for five of the six patients. Of those five, only three of the patients (Patients 1, 2, and 5) showed any overlap in their lesion location (Figure 2). For these patients then, the lesion overlay and intersection analysis highlighted a very small region of the right basal ganglia, including portions of the lentiform nucleus and the thalamus (Figure 2).

DISCUSSION

The results of the current study clearly indicate a deficit in processing the passage of time in patients with neglect. In all neglect patients, multisecond intervals were dramatically underestimated (Figure 5 and Tables 2 and 3). Although the patients with RBD also tended to underestimate durations, they were far less impaired than the neglect group, showing significant differences relative to the healthy controls at only the longest of durations. Despite the tendency toward underestimation of multisecond temporal intervals in all patients, significant linear trends were also observed for each individual such that shorter estimates were given for shorter durations (and vice versa; Table 3), implying that all patients

understood the task. Importantly, the slope describing this relationship was smallest for the neglect group, with no significant difference between the RBD group and healthy controls (Figure 5). This again underlines the observation that although right hemisphere lesions led to underestimations in both the RBD and neglect groups, this tendency was strongest in the neglect patients. In addition, as predicted by STT (Wearden, 1999; Gibbon, 1977), the coefficient of variance did not differ across all durations for any of the groups (Table 2). This indicates that the impaired performance in the neglect patients, nevertheless, conformed to crucial predictions that arise from STT and is therefore unlikely to have arisen due to any implementation of alternate strategies (i.e., strategies not normally employed by healthy individuals) to perform the task. Finally, analysis of the absolute error made by patients in the first half versus the last half of the task indicated no change in performance as the length of time spent performing temporal estimations increased.[1]

Previous research has already demonstrated that right hemisphere lesions lead to impaired temporal discrimination (Harrington et al., 1998). The current results suggest that, in neglect patients, any deficits of timing behavior are likely to be more severe than in patients with right hemisphere damage without neglect. This raises the question of whether impaired time perception/estimation plays a causative role in neglect or whether it merely exacerbates the spatial symptoms characteristic of the disorder (a suggestion made in a similar way by Husain & Rorden, 2003 when discussing nonspatial deficits in neglect). A third, related possibility might suggest that deficits in timing behavior are an epiphenomenon of the neglect syndrome. The latter two explanations would imply that there ought to be a strong correlation between neglect severity and the extent to which timing behavior is impaired. We found no evidence for such a correlation in our group of neglect patients who demonstrated a wide range of severity (Table 1; see Schindler et al., 2006 for the severity calculations). This failure to demonstrate a relationship between neglect severity and temporal estimation performance does not necessarily suggest a causative role for temporal perceptual deficits in the presence of neglect. Indeed, we would not suggest that impaired temporal estimations of the kind observed here *lead* to the neglect syndrome. The deficits we observed may be nonspatial in nature (although see Basso et al., 1996) and such a deficit alone cannot explain why neglect patients fail to represent stimuli and events from only one side of space. Instead, we would suggest that the full-blown neglect syndrome is due to combined deficits in a range of cognitive domains. We recently demonstrated that patients with neglect were severely impaired on a spatial working memory task (Ferber & Danckert, 2006; see also Malhotra et al., 2004, 2005). As is the case with the temporal deficits observed in the current study, a spatial working memory deficit in and of itself will not lead to neglect. Instead, these deficits must be combined with biases in spatial attention in order to produce the full neglect syndrome (Danckert & Ferber, 2006). It has long been demonstrated that patients with parietal lesions demonstrate a specific kind of impairment in the spatial allocation of attention—the so-called disengage deficit (Posner, Walker, Friedrich, & Rafal, 1984; see Losier & Klein, 2001 for review). The important point to emphasize here is that such a disengage deficit can be observed in patients with right parietal injury *without* spatial neglect (see also, Striemer & Danckert, 2007). Similarly, deficits in temporal estimations in and of themselves would be unlikely to lead to neglect. Indeed, disordered temporal discriminations are observed in patients with disorders or lesions affecting the basal ganglia and the cerebellum (see Ivry & Spencer, 2004 for review). These patients typically do not demonstrate neglect. Neither do some patients with frontal cortical lesions that lead to either impaired temporal perception (e.g., Harrington et al., 1998) or poor working memory capacity (Bor, Duncan, Lee, Parr, & Owen, 2006). Therefore, what we are suggesting is that a confluence of deficits including biases in spatial attention, poor spatial working memory, and disordered perception of the passage of time will lead to the loss of awareness for contralesional stimuli and events that is characteristic of neglect. Furthermore, we would not suggest that the areas commonly lesioned in neglect, including the temporo-parietal junction and the superior temporal gyrus (one of the regions commonly lesioned in our patients), are solely responsible for functions of temporal perception or spatial working memory (or for that matter, spatial attention). Instead, these regions of the association cortex in the parietal and temporal lobes are ideally placed to integrate different sources of information concerning the timing of external events, their spatial layout, and our ability to orient (either overtly or covertly) toward them. If a disruption to such an integrative functioning can be shown to have influenced not one but all of these component processes, then we would expect the patient to demonstrate unilateral neglect (see Danckert & Ferber, 2006 for review).

Turning now to a more detailed examination of what may lead to the impairment in the perception of the passage of time in our neglect patients, it is difficult to determine one specific cause for their behavior. In other words, despite the magnitude of underestimation exhibited by the neglect patients and the consistency of the effect across the whole group, STT does not allow for an unambiguous explanation of the result (see Wearden, 1999 for a discussion of this issue). As is often the case for timing experiments of the kind presented here, it is difficult to determine which component of timing behavior (e.g., the pacemaker, accumulator, memory stores, etc.; Figure 4) is at fault (Wearden, 1999). Potentially, underestimation could be due to alterations in the rate of pulses being emitted by the pacemaker (Figure 4), an explanation that receives some support from recent

work suggesting that the subcortical locus of neglect may be in the striatum, a region thought to be critical for the functioning of the pacemaker (Buhusi & Meck, 2005; Karnath, Himmelbach, & Rorden, 2002; Ivry, 1996). Indeed, lesion overlay analysis shows that both the caudate and the putamen were commonly involved in five of our neglect patients.[2] Nevertheless, the pacemaker would need to be slowed down (i.e., emitting fewer pulses per second) on an enormous scale to produce the underestimations seen here. In addition, the neural substrate most likely to be responsible for the pacemaker functions—the basal ganglia (Meck, 2005)—was not lesioned in all of the neglect patients. Indeed, in one of our neglect patients (NP3; Figure 1) in which there was no overlap with the other five patients, the basal ganglia were undamaged. In addition, one of our RBD control patients had a focal lesion of the right caudate and yet produced estimations within the range of the healthy controls. In other words, if a deficient (i.e., slowed) pacemaker was the *sole* cause of severe underestimation, one would expect this patient to look much like the neglect group, which was clearly not the case.

Although not all patients had their verbal working memory and long-term memory capacities tested here, previous work with four of the patients (Ferber & Danckert, 2006) showed unimpaired verbal working memory capacity on both experimental and standard clinical tests (i.e., Digit Span of the WAIS). This does not definitively rule out any possible contribution to the impaired temporal perception from deficient working memory processes. It may well be the case that maintaining a representation of the passage of time relies on distinct neural networks from those employed during verbal working memory tasks and may actually be more closely linked with processes of spatial working memory (which we have shown were severely impaired in four of the neglect patients tested here; Ferber & Danckert, 2006). In addition, although working and longer term memory functions are not likely to be responsible for the underestimations per se, it remains a possibility that diminished attentional resources, common among neglect patients, had a negative impact on these component processes of timing behavior (Figure 2B). It is important to re-emphasize here that, in all patients, performance was equivalent in the first and last half of the task. If sustained attention deficits could provide the sole explanation for the performance observed here, one would expect performance to become worse as the task wore on, which was obviously not the case. In addition, Robertson et al. (1997) suggest that sustained attention deficits are a ''marker'' of the neglect syndrome in that they correlate strongly with clinical measures of spatial impairment. Our patients demonstrated a range of severity of spatial impairment on those same clinical tasks (Table 1).[3] Given the extraordinary consistency of performance across the patient group in the temporal estimation task, this would suggest to us that neglect severity played little or no part in the observed impairment of temporal estimations. Nevertheless, further research exploring temporal estimations of multisecond intervals without a secondary task or with secondary tasks that alternately place demands on spatial versus verbal working memory capacity would be needed to address these issues thoroughly.

The final component of STT to consider involves ''comparator'' processes (Figure 2). The kind of comparator processes being invoked in this context involves relating the just experienced duration to some representation of durations in long-term memory that is based on prior experience (Figure 2).[4] Other types of comparator processes have been shown to be impaired in neglect patients (Danckert et al., 2002). For example, motor imagery performance, thought to involve the generation of internal motor plans normally used as efference copy for intended actions, was found to be disturbed in one neglect patient (Danckert et al., 2002; see also Sirigu et al., 1996). The efference copy of such intended actions is likely to be used to compare intended with actual movements—in other words, a comparator process (Blakemore & Frith, 2003). Although the current set of results may be suggestive of a disruption in comparator processes that relate the just experienced duration with representations of temporal durations in long-term memory, a great deal more research is required to determine whether this is the sole component process(es) of timing behavior that is disrupted in neglect. Indeed, deficient comparator processes may interact with deficits at other levels of the system to produce the results observed here.

One final aspect of the performance of the neglect patients warrants further consideration. Although previous research has demonstrated a role for the right parietal cortex in discriminating subsecond and suprasecond temporal intervals, this is the first demonstration that right parietal lesions also impair the ability to perceive the passage of time at much longer intervals (Alexander et al., 2005; Harrington et al., 1998; Basso et al., 1996). Several authors suggest that distinct neural networks are involved in subsecond and multisecond temporal discrimination and that the break between these two systems for temporal perception occurs at around 2–3 sec (Lewis & Miall, 2003a, 2006; Ivry & Spencer, 2004; Fraisse, 1963). Furthermore, temporal perception in the shorter range is considered by some to be ''automatic'' and more closely related to motor control, with the cerebellum forming the neural basis of such timing behavior (Lewis & Miall, 2003a, 2003b, 2006; Buhusi & Meck, 2005; Ivry & Spencer, 2004). In contrast, perception of longer temporal intervals is thought to rely more heavily on flexible cortical networks involving interactions between the basal ganglia and frontal and parietal cortices (Lewis & Miall, 2003a, 2006; Buhusi & Meck, 2005). Nevertheless, in several neuroimaging studies in which subjects are required to

produce responses aligned with various temporal intervals spanning subsecond, suprasecond, and multisecond ranges, virtually identical networks of activation are commonly observed (Macar et al., 2002; Rao, Mayer, & Harrington, 2001; Rubia et al., 1998). In addition, although cortical activations during temporal discrimination tasks can be observed bilaterally, one study found that only the right hemisphere activity in the parietal, caudate, and parahippocampal regions was significantly correlated with behavioral measures (Harrington et al., 2004). In an excellent review of neuroimaging studies of temporal perception, Lewis and Miall (2003a) demonstrate two distinct clusters of activations across studies depending on the nature of the task used, the involvement of motor responses to define temporal intervals, and the intervals tested. Their analysis clearly indicated that right hemisphere involvement including prefrontal and parietal cortices was most strongly evident in tasks in which motor responses were not necessary for defining temporal intervals and the intervals tested were supra- or multisecond durations (Lewis & Miall, 2003a).

One further possibility is that the involvement of the parietal cortex in a broad range of timing tasks is indicative of a role in ''magnitude'' judgments (Walsh, 2003b). Interestingly, this hypothesis has the potential to explain one apparent discrepancy between the current results and previous work. That is, previous work suggests that subsecond and suprasecond intervals tend to be *overestimated* by patients with parietal lesions, whereas the current results clearly demonstrate *underestimation* (Harrington et al., 1998; Basso et al., 1996). This apparent discrepancy can be explained with recourse to psychophysical functions applied to magnitude or intensity judgments of various kinds (e.g., brightness, weight, force, and spatial extent; Mennemeier et al., 2005; Ricci & Chatterjee, 2001; see also Hollingworth, 1909 for the seminal work on this ''magnitude'' function). That is, healthy individuals tend to overestimate the magnitude or intensity of smaller stimuli and underestimate the magnitude or intensity of larger stimuli across a range of different tasks and perceptual properties. Indeed, the classic ''crossover'' effect in line bisection, in which neglect patients bisect larger lines to the right of true center, whereas much shorter lines are bisected to the left of true center, may well be due to this same kind of psychophysical relationship applied to magnitude judgments of line length (Mennemeier et al., 2005). One suggestion in the context of this magnitude explanation of various neglect phenomena is that neglect patients demonstrate an *exaggeration* of the normal tendency to overestimate at the smaller end of a range of stimulus intensities while underestimating the longer range (Mennemeier et al., 2005; Ricci & Chatterjee, 2001). Further studies in which the tests used span both subsecond and multisecond duration discrimination within the same group of patients would provide insights into this potential explanation for the underestimation observed here.

It is becoming increasingly clear that models of neglect that simply refer to impaired mechanisms of attentional orienting are insufficient to explain the full range of symptoms and impairments that characterize the disorder (Danckert & Ferber, 2006; Pisella & Mattingley, 2004). Deficits of saccadic or spatial remapping, spatial working memory, and now temporal perception, will all need to be considered when constructing neurocognitive models of the disorder (Ferber & Danckert, 2006; Pisella & Mattingley, 2004; Wojciulik et al., 2004; Husain et al., 2001; Wojciulik, Husain, Clarke, & Driver, 2001; Heide, Blankenburg, Zimmermann, & Kömpf, 1995). As suggested above, neglect is likely to result from the confluence of these component deficits and concomitantly, the region of the parietal and temporal cortex commonly lesioned in neglect patients is likely to be crucial for *integrating* information processing related to these distinct functions. A loss of that integrative function, combined with the classic demonstration of biases in spatial attention, will lead to an extraordinary difficulty in consciously representing stimuli and events on the left side of space (Danckert & Ferber, 2006). In relation to time perception per se, the experience of the passage of time is intimately linked with the experience of change. Therefore, in order to create and maintain an accurate representation of an ever-changing environment, it would be necessary to accurately ''time-stamp'' behaviorally relevant or salient events in that environment. Inaccurate temporal perception of the kind demonstrated here would presumably make that process of ''time-stamping'' salient changes in stimuli or events difficult to perform. As already stated, this difficulty, in and of itself, would presumably not lead to a loss of awareness for *contralesional* events only. Instead, the temporal perception deficit would need to be coupled with a bias in orienting attention to one side of space. If one considers that all events in the environment compete for conscious representation (Desimone & Duncan, 1995), then such a combination of deficits would make it extremely difficult for contralesional events (or changes in the environment occurring in contralesional space) to compete with ipsilesional events for access to memory systems and, eventually, consciousness.

Acknowledgments

This work was supported by the Natural Sciences and Engineering Research Council (NSERC) of Canada Discovery Grants to JD and SF, an NSERC Canada Research Chair (Tier II) award to JD, NSERC graduate scholarships to CS and CB, Heart and Stroke Foundation (HSF) of Ontario Grants to JD, an HSF Masters Scholarship to CS, a Canadian Institutes of Health Research grant to SF, and the Leverhulme Trust. We thank Drs. F. Gao and S. Black for help with the lesion analysis.

Reprint requests should be sent to Dr. James Danckert, Canada Research Chair (Tier II) in Cognitive Neuroscience, Department of Psychology, University of Waterloo, 200 University Avenue West, Waterloo, Ontario, Canada N2L 3G1, or via e-mail: jdancker@uwaterloo.ca.

Notes

1. Absolute error was determined via a difference score calculated for each individual trial. That is, the patient's response was subtracted from the actual duration to be estimated with a positive score indicative of underestimation (there were no overestimations in any patient negating the need to deal with the sign of the difference score). *t* Tests comparing first half and last half performance for the group as a whole and for each patient individually were all nonsignificant.
2. The small number of both NP and RBD patients that we were able to include in our lesion overlay analyses makes any conclusions concerning the role of commonly lesioned areas in our data necessarily speculative. This is especially true for the RBD group in which only three patients were included in the analysis, which showed overlap in the thalamus and lentiform nucleus of the right hemisphere (Figure 2).
3. If severity was to be based solely on cancellation performance, we would then classify as ''mild'' neglect patients 1 and 6, with patients 4, 5, and 8 classified as ''moderate'' and patients 2, 3, and 7 classified as ''severe''. The point remains unchanged, as despite the range of severity in our patient group, their performance on the temporal estimation task was incredibly consistent across all patients.
4. In many cases, a ''standard'' duration is actually given to the subject to which all test durations must be compared. In our case, no such standard was used and patients were required to compare the test duration to some other stored representation, perhaps of how long a second or a minute ''should'' take to elapse.

REFERENCES

Alexander, I., Cowey, A., & Walsh, V. (2005). The right parietal cortex and time perception: Back to Critchley and the Zeitraffer phenomenon. *Cognitive Neuropsychology, 22,* 306–315.

Basso, G., Nichelli, P., Frassinetti, F., & di Pellegrino, G. (1996). Time perception in a neglected space. *NeuroReport, 7,* 2111–2114.

Berberovic, N., Pisella, L., Morris, A. P., & Mattingley, J. B. (2004). Prismatic adaptation reduces biased temporal order judgements in spatial neglect. *NeuroReport, 15,* 1199–1204.

Blakemore, S.-J., & Frith, C. D. (2003). Self-awareness and action. *Current Opinion in Neurobiology, 13,* 219–224.

Bor, D., Duncan, J., Lee, A. C. H., Parr, L., & Owen, A. M. (2006). Frontal lobe involvement in spatial span: Converging studies of normal and impaired function. *Neuropsychologia, 44,* 229–237.

Buhusi, C. V., & Meck, W. H. (2005). What makes us tick? Functional and neural mechanisms of interval timing. *Nature Reviews Neuroscience, 6,* 755–765.

Chun, M. M., & Potter, M. C. (1995). A two-stage model for multiple target detection in rapid serial visual presentation. *Journal of Experimental Psychology: Human Perception and Performance, 21,* 109–127.

Coull, J. T. (2004). fMRI studies of temporal attention: Allocating attention within, or towards time. *Cognitive Brain Research, 21,* 216–226.

Cusack, R., Carlyon, R. P., & Robertson, I. H. (2000). Neglect between but not within auditory objects. *Journal of Cognitive Neuroscience, 12,* 1056–1065.

Danckert, J., & Ferber, S. (2006). Revisiting unilateral neglect. *Neuropsychologia, 44,* 987–1006.

Danckert, J., Ferber, S., Doherty, T., Steinmetz, H., Nicolle, D., & Goodale, M. A. (2002). Selective, non-lateralised impairment of motor imagery following right parietal damage. *Neurocase, 8,* 194–204.

Desimone, R., & Duncan, J. (1995). Neural mechanisms of selective visual attention. *Annual Review of Neuroscience, 18,* 193–222.

Driver, J., & Mattingley, J. B. (1998). Parietal neglect and visual awareness. *Nature Neuroscience, 1,* 17–22.

Ferber, S., & Danckert, J. (2006). Lost in space—The fate of memory representations for non-neglected stimuli. *Neuropsychologia, 44,* 320–325.

Fraisse, P. (1963). *The psychology of time*. New York: Harper & Row.

Gibbon, J. (1977). Scalar expectancy theory and Weber's law in animal timing. *Psychological Review, 84,* 279–325.

Gibbon, J. (1992). Ubiquity of scalar timing with a Poisson clock. *Journal of Mathematical Psychology, 36,* 283–293.

Goodale, M. A., & Milner, A. D. (1992). Separate visual pathways for perception and action. *Trends in Neurosciences, 15,* 20–25.

Harrington, D. L., Boyd, L. A., Mayer, A. R., Sheltraw, D. M., Lee, R. R., Huang, M., et al. (2004). Neural representation of interval encoding and decision making. *Cognitive Brain Research, 21,* 193–205.

Harrington, D. L., Haaland, K. Y., & Knight, R. T. (1998). Cortical networks underlying mechanisms of time perception. *Journal of Neuroscience, 18,* 1085–1095.

Heide, W., Blankenburg, M., Zimmermann, E., & Kömpf, D. (1995). Cortical control of double-step saccades: Implications for spatial orientation. *Annals of Neurology, 38,* 739–748.

Hillstrom, A. P., Husain, M., Shapiro, K. L., & Rorden, C. (2004). Spatiotemporal dynamics of attention in visual neglect: A case study. *Cortex, 40,* 433–440.

Hollingworth, H. L. (1909). The indifference point. In H. L. Hollingworth (Ed.), *The inaccuracy of movement*. New York: The Science Press.

Husain, M., Mannan, S., Hodgson, T., Wojciulik, E., Driver, J., & Kennard, C. (2001). Impaired spatial working memory across saccades contributes to abnormal search in parietal neglect. *Brain, 124,* 941–952.

Husain, M., & Rorden, C. (2003). Non-spatially lateralised mechanisms in neglect. *Nature Neuroscience Reviews, 4,* 26–36.

Husain, M., Shapiro, K., Martin, J., & Kennard, C. (1997). Abnormal temporal dynamics of visual attention in spatial neglect patients. *Nature, 385,* 154–156.

Ivry, R. B. (1996). The representation of temporal information in perception and motor control. *Current Opinion in Neurobiology, 6,* 851–857.

Ivry, R. B., & Spencer, R. M. C. (2004). The neural representation of time. *Current Opinion in Neurobiology, 14,* 225–232.

Karnath, H.-O., Ferber, S., & Himmelbach, M. (2001). Spatial awareness is a function of the temporal not the posterior parietal lobe. *Nature, 411,* 950–953.

Karnath, H.-O., Himmelbach, M., & Rorden, C. (2002). The subcortical anatomy of human spatial neglect: Putamen, caudate nucleus and pulvinar. *Brain, 125,* 350–360.

Leon, M. I., & Shadlen, M. N. (2003). Representation of time by neurons in the posterior parietal cortex of the macaque. *Neuron, 38,* 317–327.

Lewis, P. A., & Miall, R. C. (2003a). Distinct systems for automatic and cognitively controlled time measurement: Evidence from neuroimaging. *Current Opinion in Neurobiology, 13,* 250–255.

Lewis, P. A., & Miall, R. C. (2003b). Brain activation patterns during measurement of sub- and supra-second intervals. *Neuropsychologia, 41,* 1583–1592.

Lewis, P. A., & Miall, R. C. (2006). Remembering the time: A continuous clock. *Trends in Cognitive Sciences, 10,* 401–406.
Losier, B. J., & Klein, R. M. (2001). A review of the evidence for a disengage deficit following parietal lobe damage. *Neuroscience and Biobehavioral Reviews, 25,* 1–13.
Macar, F., Lejeune, H., Bonnet, M., Ferrara, A., Poutha, V., Vidal, F., et al. (2002). Activation of the supplementary motor area and of attention networks during temporal processing. *Experimental Brain Research, 142,* 475–485.
Malhotra, P., Jager, H. R., Parton, A., Greenwood, R., Playford, E. D., Brown, M. M., et al. (2005). Spatial working memory capacity in unilateral neglect. *Brain, 128,* 424–435.
Malhotra, P., Mannan, S., Driver, J., & Husain, M. (2004). Impaired spatial working memory: One component of the visual neglect syndrome? *Cortex, 40,* 667–676.
Meck, W. H. (2005). Neuropsychology of timing and time perception. *Brain & Cognition, 58,* 1–8.
Mennemeier, M., Pierce, C. A., Chatterjee, A., Anderson, B., Jewell, G., Dowler, R., et al. (2005). Biases in attentional orientation and magnitude estimation explain crossover: Neglect is a disorder of both. *Journal of Cognitive Neuroscience, 17,* 1194–1211.
Mort, D. J., Malhotra, P., Mannan, S. K., Rorden, C., Pambakian, A., Kennard, C., et al. (2003). The anatomy of visual neglect. *Brain, 126,* 1986–1997.
Oshio, K., Chiba, A., & Inase, M. (2006). Delay period activity of monkey prefrontal neurons during duration-discrimination task. *European Journal of Neuroscience, 23,* 2779–2790.
Pisella, L., Berberovic, N., & Mattingley, J. B. (2004). Impaired working memory for location but not for colour or shape in visual neglect: A comparison of parietal and non-parietal lesions. *Cortex, 40,* 379–390.
Pisella, L., & Mattingley, J. B. (2004). The contribution of spatial remapping to unilateral visual neglect. *Neuroscience and Biobehavioral Reviews, 28,* 181–200.
Posner, M. I., Walker, J. A., Friedrich, F. J., & Rafal, R. D. (1984). Effects of parietal injury on covert orienting of attention. *Journal of Neuroscience, 4,* 1863–1874.
Rakitin, B. C., Gibbon, J., Penney, T. B., Malapani, C., Hinton, S. C., & Meck, W. H. (1998). Scalar expectancy theory and peak-interval timing in humans. *Journal of Experimental Psychology: Animal Behavior Processes, 24,* 15–33.
Rao, S. M., Mayer, A. R., & Harrington, D. L. (2001). The evolution of brain activation during temporal processing. *Nature Neuroscience, 4,* 317–323.
Raymond, J. E., Shapiro, K. L., & Arnell, K. M. (1992). Temporary suppression of visual processing in an RSVP task: An attentional blink? *Journal of Experimental Psychology, 18,* 849–860.
Ricci, R., & Chatterjee, A. (2001). Context and crossover in unilateral neglect. *Neuropsychologia, 39,* 1138–1143.
Rizzo, M., Akutsu, H., & Dawson, J. (2001). Increased attentional blink after focal cerebral lesions. *Neurology, 57,* 795–800.
Robb, R. A. (2001). The Biomedical Imaging Resource at Mayo Clinic. Guest editorial. *IEEE Transactions on Medical Imaging, 20,* 854–867.
Robb, R. A., & Barillot, C. (1989). Interactive display and analysis of 3-D medical images. *IEEE Transactions on Medical Imaging, 8,* 217–226.
Robb, R. A., Hanson, D. P., Karwoski, R. A., Larson, A. G., Workman, E. L., & Stacy, M. C. (1989). ANALYZE: A comprehensive, operator-interactive software package for multidimensional medical image display and analysis. *Computerized Medical Imaging and Graphics, 13,* 433–454.
Robertson, I. H., Manly, T., Beschin, N., Daini, R., Haeske-Dewick, H., Hömberg, V., et al. (1997). Auditory sustained attention is a marker of unilateral neglect. *Neuropsychologia, 35,* 1527–1532.
Robertson, I. H., Mattingley, J. B., Rorden, C., & Driver, J. (1998). Phasic alerting of neglect patients overcomes their spatial deficit in visual awareness. *Nature, 395,* 169–172.
Rorden, C., & Brett, M. (2000). Stereotaxic display of brain lesions. *Behavioural Neurology, 12,* 191–200.
Rorden, C., Mattingley, J. B., Karnath, H.-O., & Driver, J. (1997). Visual extinction and prior entry: Impaired perception of temporal order with intact motion perception after unilateral parietal damage. *Neuropsychologia, 35,* 421–433.
Rubia, K., Overmeyer, S., Taylor, E., Brammer, M., Williams, S., Simmons, A., et al. (1998). Prefrontal involvement in ''temporal bridging'' and timing movement. *Neuropsychologia, 36,* 1283–1293.
Schindler, I., Clavagnier, S., Karnath, H.-O., Derex, L., & Perenin, M.-T. (2006). A common basis for visual and tactile exploration deficits in spatial neglect? *Neuropsychologia, 44,* 1444–1451.
Schneider, W. (1999). Visual–spatial working memory, attention, and scene representation: A neuro-cognitive theory. *Psychological Research, 62,* 220–236.
Shapiro, K., Hillstrom, A. P., & Husain, M. (2002). Control of visuotemporal attention by inferior parietal and superior temporal cortex. *Current Biology, 12,* 1320–1325.
Sirigu, A., Duhamel, J.-R., Cohen, L., Pillon, B., Dubois, B., & Agid, Y. (1996). The mental representation of hand movements after parietal cortex damage. *Science, 273,* 1564–1568.
Snyder, J. J., & Chatterjee, A. (2004). Spatial–temporal anisometries following right parietal damage. *Neuropsychologia, 42,* 1703–1708.
Stelmach, L. B., & Herdman, C. M. (1991). Directed attention and perception of temporal order. *Journal of Experimental Psychology: Human Perception and Performance, 17,* 539–550.
Striemer, C., & Danckert, J. (2007). Prism adaptation reduces the disengage deficit in patients with right brain damage. *NeuroReport, 18,* 99–103.
Walsh, V. (2003a). Time: The back-door of perception. *Trends in Cognitive Sciences, 7,* 335–338.
Walsh, V. (2003b). A theory of magnitude: Common cortical metrics of time, space and quantity. *Trends in Cognitive Sciences, 7,* 483–488.
Wearden, J. H. (1999). ''Beyond the fields we know. . .'': Exploring and developing scalar timing theory. *Behavioral Processes, 45,* 3–21.
Wearden, J. H., Denovan, L., Fakhri, M., & Haworth, R. (1997). Scalar timing in temporal generalization in humans with longer stimulus durations. *Journal of Experimental Psychology: Animal Behavior Processes, 23,* 502–511.
Wojciulik, E., Husain, M., Clarke, K., & Driver, J. (2001). Spatial working memory deficit in unilateral neglect. *Neuropsychologia, 39,* 390–396.
Wojciulik, E., Rorden, C., Clarke, K., Husain, M., & Driver, J. (2004). Group study of an ''undercover'' test for visuospatial neglect: Invisible cancellation can reveal more neglect than standard cancellation. *Journal of Neurology, Neurosurgery, and Psychiatry, 75,* 1356–1358.