Visual attention capacity after right hemisphere lesions

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Abstract

Recently there has been a growing interest in visual short-term memory (VSTM) including the neural basis of the function. Processing speed, another main aspect of visual attention capacity, has received less investigation. For both cognitive functions human lesion studies are sparse. We used a whole report experiment for estimation of these two parameters in 22 patients with right side stroke. Psychophysical performance was analyzed using Bundesen's [Bundesen, C. (1990). A theory of visual attention. Psychological Review, 97, 523–547] Theory of Visual Attention (TVA) and compared statistically to lesion location and size measured by MRI. Visual processing speed was impaired in the contralesional hemifield for most patients, but typically preserved ipsilesionally, even after large cortico-subcortical lesions. When bilateral deficits in processing speed occurred, they were related to damage in the right middle frontal gyrus or leukoaraiosis. The storage capacity of VSTM was also normal for most patients, but deficits were found after severe leukoaraiosis or large strokes extending deep into white matter. Thus, the study demonstrated the importance of white-matter connectivity for both VSTM capacity and ipsilesional processing speed. The study also showed that lesions in a large region of the right hemisphere, including the putamen, insula, and inferior frontal cortex, do not lead to general deficits in the capacity of visual attention.

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1. Introduction

1.1. General background

The amount of information that can be reported from a single fixation seems to be limited by two factors (Shibuya & Bundesen, 1988). The first is the rate of visual information uptake (items per second) from the display. The second is the storage capacity of visual short-term memory (VSTM), which sets an upper limit for the number of objects that can be perceived simultaneously. Of the two functions, VSTM has received the largest research interest. In early studies Sperling (1960, 1967) showed that normal observers can report a maximum of about four unrelated items from a brief visual display. This limitation presumably reflects the maximum storage capacity of VSTM, a basic result that has been confirmed several times since (Shibuya & Bundesen, 1988; Vogel, Woodman, & Luck, 2001). Recently there has been a growing interest in various cognitive properties of the VSTM system (Alvarez & Cavanagh, 2004; Klaver, Smid, & Heinze, 1999; Lee & Chun, 2001; Luck & Vogel, 1997), and the first functional imaging studies of VSTM capacity have appeared (Todd & Marois, 2004; Vogel & Machizawa, 2004; Xu & Chun, 2006). These studies point to the posterior parietal cortex as critical for short-term retention of visual stimuli.

VSTM capacity is often estimated using change detection experiments (e.g., Luck & Vogel, 1997). However, in his classical studies Sperling used a whole report paradigm, in which a set of unrelated items (letters) were displayed at variable exposure durations. Besides more reliable estimation of VSTM capacity, this design has the advantage that it allows for simultaneous estimation of visual processing speed (Shibuya & Bundesen, 1988). Visual processing speed represents the total amount of information analyzed per second by the visual system. This functional parameter has been less investigated, perhaps because its effect on performance is difficult to separate from the VSTM limitation. However, Bundesen’s (1990) Theory of Visual Attention (TVA; see next section) provides a method to disentangle these two capacity limitations.
A number of recent studies have used the TVA model to investigate visual attention capacity after brain damage. Duncan et al. (1999) found that both VSTM capacity and visual processing speed were reduced bilaterally in a group of nine patients with neglect after right hemisphere damage. Duncan et al. (2003) showed that visual processing speed was severely reduced (but VSTM capacity only moderately) in two patients with simultanagnosia, and Habekost and Bundesen (2003) found bilateral reductions of VSTM capacity in a patient with a right frontal–subcortical lesion. Whereas these studies demonstrated the efficiency of TVA analysis for measuring visual attention capacity, the number of patients was not sufficient for a reliable mapping of critical regions. In a larger study Finke, Bublak, Dose, Müller, & Schneider (2006) investigated 18 patients with Huntington’s disease and found marked bilateral reductions in visual processing speed and VSTM capacity. Huntington’s disease is characterized by striatal atrophy, but also progressive cortical involvement. Since MR scans of the patients were not available in this study, Finke et al.’s results do not point clearly towards particular brain areas for visual attention capacity. A recent TVA based patient study by Peers et al. (2005) is more conclusive on this point. Peers et al. examined 25 patients with focal lesions in either the parietal or frontal cortex and found that deficits in visual processing speed or VSTM capacity occurred selectively after parietal lesions. For both functions there was a significant correlation in the parietal group between reduced capacity and relatively inferior lesions, in the region of the temporoparietal junction. However, the exact critical areas were unclear and Peers et al. suggested that damage in the underlying white matter could also be important. The present study provides a large new data set on this issue. We used TVA theory to derive estimates of VSTM capacity and visual processing speed in 22 patients with right side brain damage and compared these data to individual differences in lesion anatomy. Besides clarifying the importance of cortical structures for visual attention capacity, we were interested in testing whether damage to the underlying white matter is also critical. Influential theories claim that short-term memory (Fuster, 1997; Goldman-Rakic, 1995) and conscious recognition (Crick & Koch, 1995; Duncan, 1996) depend on integrated activity across widespread cortical areas. If this is also the case for the related functions of VSTM and visual processing speed, long-range cortico-cortical connections should be critical for both. Besides stroke in the white matter, the age-related condition of leukoaraiosis (diffuse abnormalities in the cerebral fibre tracts; Ward & Brown, 2002) should be relevant to this question. Our radiological examination therefore also included MR sequences that are sensitive to leukoaraiosis: fluid-attenuated inversion recovery (FLAIR) scans.

In the present study we focus on general reductions in visual attention capacity, which implies that perception is affected in both visual hemifields. Selective deficits in the contralateral visual field were common in our patient group, but these results are described in a parallel paper (Habekost & Rostrup, 2006). Together our two studies address the lesion anatomy of both nonlateralized and lateralized attention deficits after right hemisphere damage, an issue of great relevance for theories of the neglect syndrome. Though traditionally defined as a lateralized disturbance, neglect is now widely considered to include bilateral impairments as well. For example, Husain and Rorden (2003) have proposed that neglect patients are characterized by general working memory deficits (in addition to their bias for ipsilesional stimuli), and Robertson (1993) has pointed to reductions of arousal as a central part of the neglect syndrome.

1.2. Theory of visual attention (TVA)

The TVA theory forms a basic analytic frame for our study. The theory was presented by Bundesen (1990) and accounts for findings from a wide range of experimental paradigms such as single-stimulus recognition, whole report, partial report, detection, and visual search (for a recent review of TVA and the attention literature, see Bundesen & Habekost, 2005). The model has also been integrated with theories of memory, categorization, and executive function (Logan, 2002; Logan & Gordon, 2001). Whereas the original TVA model was framed at a cognitive description level, its principles have been shown to have a strong analogy at the single cell level (Bundesen, Habekost, & Kyllingsbaek, 2005). As mentioned above, TVA analysis is also being increasingly used for studies of attention deficits after brain damage.

TVA describes visual recognition and selection as a parallel processing race, where objects in the visual field compete for encoding into a limited number of VSTM slots. Encoding into VSTM implies conscious recognition. The total amount of processing capacity is limited, and distributed across objects according to their relative attentional weights. The exact properties of the processing race depend on individually variable parameter values, which are specified in a set of equations. We refer to earlier expositions (Bundesen, 1990; Duncan et al., 1999) for mathematical details. In relation to visual attention capacity two TVA parameters are important: (a) the visual processing speed, C: the total number of visual objects processed per second, and (b) the storage capacity of visual short-term memory (VSTM), K: the maximum number of objects that can be reported from a brief visual display.

The parameters are best understood in the context of the experimental design used to estimate them: whole report. In whole report tasks the subject must report as many items as possible from a briefly exposed array of simple unrelated stimuli (e.g., letters). The score (number of correctly reported items) is measured as a function of exposure duration and follows a characteristic pattern (Bundesen & Harms, 1999; Duncan et al., 1999; Habekost & Bundesen, 2003; Shibuya & Bundesen, 1988; see Fig. 1). Below a minimal exposure duration, t0, no items are reported. With postmasked alphabetic stimuli the perception threshold t0 is typically 15–20 ms in young healthy subjects (Bundesen & Harms, 1999; Shibuya & Bundesen, 1988). Perception thresholds are of secondary interest in the context of visual attention capacity and will not receive special attention in this study (they were normal for most patients; see Habekost & Rostrup, 2006, for details). Above the minimal effective exposure duration the curve rises sharply, but gradually flattens out over the course of a few hundred milliseconds. Given long enough exposure time performance approaches an asymptotic
value, usually interpreted as the maximum storage capacity of VSTM: $K$. The VSTM limit is typically estimated at three to four objects in healthy subjects (Sperling, 1967). Data fits of this parameter are improved by using noninteger values. For example, a $K$ value of 3.3 represents a probability mixture of VSTM capacity at three and four elements, occurring with 70% and 30% probability, respectively. The $C$ parameter is a measure of the total processing speed during visual recognition, and equals the slope of the whole report function at $t = t_0$. $C$ is highly dependent on the sensory properties and general discriminability of the stimuli. With high-contrast alphabetic stimuli, which are simple and highly familiar visual forms, $C$ typically varies between 15–50 elements/s in healthy subjects. If stimuli are presented unmasked, an afterimage of the stimulus is briefly sustained and the exposure duration effectively prolonged. The additional exposure duration is quantified by the $\mu$ parameter, which is necessary for model fitting but will not be considered in further detail.

$C$ can be reduced selectively to stimuli in the contralesional visual field after brain damage, whereas $K$ reductions seem to occur only bilaterally and probably reflect a general limitation.\(^1\)

Accordingly, for each participant we estimated a single $K$ value across the visual field, whereas $C$ was allowed to vary between sides.

2. Materials and methods

2.1. Participants

Medical records of all patients admitted to a brain injury rehabilitation centre (during a period of 3 years) and two university hospital stroke units (during a period of approximately 2 years) in Copenhagen were screened for radiological evidence (CT or MR) of stroke in the right side of the brain. To be selected for participation, a patient should also be at least 6 months postinjury and satisfy the following inclusion criteria: (a) normal visual acuity (Snellen score ≥ 9/10) and no field cuts, (b) no dementia (MMSE score ≥ 24), (c) no aphasia, (d) no history of major psychiatric or other neurologic disease, (e) no substance abuse, (f) age ≥ 70 years, (g) ability to fixate reliably, (h) auditory span of at least four elements, and (i) no additional damage in the left side of the brain.\(^3\) All patients who satisfied these criteria ($n = 34$) were invited to participate in the study. Twenty-two patients agreed, but 12 other patients did not respond to the invitation (the possible effects of selection bias are considered in the general discussion). All patients gave informed written consent according to the Helsinki Declaration, and approval was given by ethical committees in Copenhagen City and Copenhagen County (project no. KF 01-116/02). The mean age of the patients was 70 years,\(^2\) (g) ability to fixate reliably, (h) auditory span of at least four elements, and (i) no additional damage in the left side of the brain.\(^3\) All patients who satisfied these criteria ($n = 34$) were invited to participate in the study. Twenty-two patients agreed, but 12 other patients did not respond to the invitation (the possible effects of selection bias are considered in the general discussion). All patients gave informed written consent according to the Helsinki Declaration, and approval was given by ethical committees in Copenhagen City and Copenhagen County (project no. KF 01-116/02). The mean age of the patients was

\(^{1}\) There are both empirical and theoretical reasons to regard VSTM capacity as a general limitation in the visual system. In Duncan et al.’s (1999) TVA-based study of neglect patients no difference between $K$ estimates in the left and right visual fields were found. The control subjects also had $K$ values that were close to symmetrical. Besides this empirical finding there are theoretical reasons to assume side differences in processing speed, but not in VSTM capacity. The initial visual processing of stimuli is lateralized to a high degree, so it is plausible that its efficiency can be damaged unilaterally. In contrast, VSTM is conceived as the end-point of processing and probably depends on a more centrally localized system. In case of letter identification (as in whole report experiments) it can be assumed that stimuli do not reach VSTM until processing involves the left hemisphere (cf. hemi-alexia after callosal lesions; Molko et al., 2002). Further, the statistical ability to estimate $K$ is reduced when visual processing speed is low, as was the case in the left visual field for many patients in our study. Under these circumstances VSTM is rarely filled up during brief stimulus presentations, no clear ceiling effect in performance emerges, and $K$ estimation is therefore less reliable. More statistically robust estimates can be obtained by grounding the $K$ estimate in data from both visual fields (double as many data, clear ceiling effects in the right side). To check whether it would make a difference to analyze our data using a model with two $K$ values, we conducted an alternative TVA analysis

\(^{2}\) In the initial phase of the project two patients aged above 70 years were examined, because at this point it was unclear whether enough patients could be recruited. Independent of focal lesions, general processing capacity may be affected by non-specific factors related to aging, and the data from these two patients were not included in the analysis.

\(^{3}\) After the psychophysical examination had been conducted, the MR scan of three patients revealed strokes in the left side of the brain. The data from these patients were excluded from the analysis.
Table 1
Clinical and lesion characteristics

<table>
<thead>
<tr>
<th>Subj</th>
<th>Age/sex</th>
<th>Aetiology</th>
<th>Stroke (cm³)</th>
<th>Leukoaraiosis (cm³)</th>
<th>Postinjury (month)</th>
<th>Extinction</th>
<th>Bisection</th>
<th>Rey</th>
<th>Cancellation</th>
</tr>
</thead>
<tbody>
<tr>
<td>L1</td>
<td>70/M</td>
<td>Infarct</td>
<td>31.5</td>
<td>15.4</td>
<td>11</td>
<td>1/10</td>
<td>−6</td>
<td>0</td>
<td>0:0, 0:0</td>
</tr>
<tr>
<td>L2</td>
<td>66/M</td>
<td>Haemo</td>
<td>13.6</td>
<td>11.4</td>
<td>30</td>
<td>3/10</td>
<td>+2</td>
<td>0</td>
<td>2:2, 3:0²</td>
</tr>
<tr>
<td>L3</td>
<td>68/F</td>
<td>Haemo</td>
<td>2.0</td>
<td>12.9</td>
<td>14</td>
<td>0/10</td>
<td>+1</td>
<td>−</td>
<td>0:0, 0:1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L1</td>
<td>43/F</td>
<td>Haemo</td>
<td>47</td>
<td>0.0</td>
<td>12</td>
<td>2/10</td>
<td>−1</td>
<td>1</td>
<td>6:4, 3:0²</td>
</tr>
<tr>
<td>L2</td>
<td>58/F</td>
<td>Infarct</td>
<td>63.9</td>
<td>0.0</td>
<td>16</td>
<td>1/10</td>
<td>+9</td>
<td>−</td>
<td>1:0, 0:2</td>
</tr>
<tr>
<td>L3</td>
<td>45/M</td>
<td>Haemo</td>
<td>95.9</td>
<td>0.3</td>
<td>15</td>
<td>1/10</td>
<td>−8</td>
<td>0</td>
<td>0:0, 0:0</td>
</tr>
<tr>
<td>L4</td>
<td>53/M</td>
<td>Haemo</td>
<td>167.7</td>
<td>0.3</td>
<td>31</td>
<td>2/10</td>
<td>−15a</td>
<td>0</td>
<td>0:1, 1:0</td>
</tr>
<tr>
<td>L5</td>
<td>68/F</td>
<td>Infarct</td>
<td>137.5</td>
<td>0.4</td>
<td>28</td>
<td>9/10b</td>
<td>+7</td>
<td>0</td>
<td>6:2, 1:0</td>
</tr>
<tr>
<td>L6</td>
<td>44/F</td>
<td>Infarct</td>
<td>189.9</td>
<td>0.7</td>
<td>38</td>
<td>0/10</td>
<td>−1</td>
<td>0</td>
<td>3:1, 4:0a</td>
</tr>
<tr>
<td>L7</td>
<td>57/M</td>
<td>Infarct</td>
<td>142.7</td>
<td>0.0</td>
<td>18</td>
<td>−</td>
<td>−4</td>
<td>0</td>
<td>1:0, 1:0</td>
</tr>
<tr>
<td>L8</td>
<td>39/F</td>
<td>Infarct</td>
<td>–</td>
<td>–</td>
<td>16</td>
<td>0/10</td>
<td>+1</td>
<td>0</td>
<td>0:1, 0:0</td>
</tr>
<tr>
<td>L9</td>
<td>46/F</td>
<td>Infarct</td>
<td>153.3</td>
<td>0.0</td>
<td>41</td>
<td>3/10</td>
<td>+3</td>
<td>1</td>
<td>2:1, 1:0</td>
</tr>
<tr>
<td>L10</td>
<td>58/M</td>
<td>Infarct</td>
<td>35.1</td>
<td>0.0</td>
<td>13</td>
<td>8/10a</td>
<td>−2</td>
<td>1</td>
<td>3:1, 3:1</td>
</tr>
<tr>
<td>L11</td>
<td>54/F</td>
<td>Infarct</td>
<td>58.8</td>
<td>0.3</td>
<td>14</td>
<td>2/10</td>
<td>−1</td>
<td>−</td>
<td>1:0, 0:2</td>
</tr>
<tr>
<td>L12</td>
<td>47/F</td>
<td>Infarct</td>
<td>232.9</td>
<td>0.9</td>
<td>29</td>
<td>2/10</td>
<td>+2</td>
<td>0</td>
<td>3:0a, 0:0</td>
</tr>
<tr>
<td>L13</td>
<td>63/M</td>
<td>Infarct</td>
<td>214</td>
<td>1.6</td>
<td>26</td>
<td>1/10</td>
<td>−13a</td>
<td>0</td>
<td>1:1, 2:2</td>
</tr>
</tbody>
</table>

| S1   | 50/M    | Infarct   | 0.6          | 0.0                | 15                | 0/10       | +4        | 1   | 0:0, 1:0    |
| S2   | 50/M    | Infarct   | 0.6          | 1.6                | 30                | 0/10       | +1        | 0   | 1:1, 3:6    |
| S3   | 65/F    | Haemo     | 2.5          | 0.0                | 6                 | 0/10       | −2        | 0   | 0:0, 0:0    |
| S4   | 56/M    | Infarct   | 0.5          | 0.3                | 6                 | 0/10       | +1        | 0   | 0:0, 0:0    |
| S5   | 46/F    | Infarct   | 0.1          | 0.0                | 20                | 0/10       | −11a      | 0   | 0:0, 0:0    |
| S6   | 66/F    | Infarct   | –            | –                  | 10                | 1/10       | +5        | 0   | 4:0, 0:0    |

Subj.: subject; aetiology: haemorrhage or infarct; stroke: lesion volume of stroke-related brain damage (missing for CT scans). Leukoaraiosis: estimated volume of leukoaraiosis (missing for CT scans); postinjury: time between stroke onset and testing; extinction: frequency of left side omissions with bilateral stimulation (abnormal: >30%); bisection: average rightward deviation (mm) from centre on Wilson et al.’s (1987) line bisection test (abnormal: at least two deviations of more than 12.75 mm from the midpoint); Rey: number of left side omissions on Rey Figure Copying (abnormal: more than one left-side omission); cancel: number of left versus right side omissions on Weintraub and Mesulam’s (1985) cancellation test (figures and letters versions, respectively; abnormal: ≥3 omissions on the left relative to the right side).

a Abnormal performance.

55.1 years (SD = 9.5 years), and the group consisted of 10 men and 12 women. Postinjury time ranged from 6 to 41 months (mean: 20 months). All patients except two were right handed according to the Edinburgh Handedness Inventory. Twelve neurologically healthy participants formed an age-matched control group (5 men and 7 women; mean age: 56.6 years, SD = 5.4 years).4 The controls were recruited by local advertisements and paid for their participation, and also gave informed written consent. In addition to the psychophysical testing, participants were given a screening battery of neuropsychological tests: Snellen chart, MMSE (patients only), Weintraub and Mesulam’s (1985) cancellation test (letters and figures, unstructured versions), Wilson, Cockburn, & Halligan (1987) line bisection test, Rey Figure Copying, auditory span, Edinburgh Handedness Inventory, and a test for visual extinction (detection of finger movements unilaterally vs. bilaterally). Visual fields were assessed by confrontation (patients only). General deficits in visual attention capacity have been linked to the neglect syndrome (cf. Husain & Rorden, 2003), but clinical testing showed that neglect was generally weak or absent in our patients (see Table 1 for clinical and lesion characteristics).

2.2. Experimental procedure

The experiments were set up using E-prime software (version 1.1) and run on an IBM-compatible computer. Participants were seated with their eyes approximately 100 cm from the screen in a semidarkened room. Visual stimuli were shown on a 19 in. computer monitor capable of 200 screen refreshes/s (5 ms resolution). The refresh rate was checked using an oscilloscope. Five letters were selected randomly and without replacement from the set \{ABEFGHJKLMNPRSTWXYZ\} and flashed for 5–200 ms on a black background, followed by either a blank screen or a 500 ms bright pattern mask. The five letters were shown at the circumference of an imaginary half-circle centred at fixation, either in the left or right visual field (see Fig. 2). The radius of the circle was shown on a 19 in. computer monitor capable of 200 screen refreshes/s (5 ms resolution). The refresh rate was checked using an oscilloscope. Five letters were selected randomly and without replacement from the set \{ABEFGHJKLMNPRSTWXYZ\} and flashed for 5–200 ms on a black background, followed by either a blank screen or a 500 ms bright pattern mask. The five letters were shown at the circumference of an imaginary half-circle centred at fixation, either in the left or right visual field (see Fig. 2).
was approximately five visual degrees (viewing distance was not precisely controlled). The participants were also examined using two other experiments (partial and colour report), which targeted other psychophysical parameters than the general capacity of visual attention. These investigations are reported in a parallel article (Habekost & Rostrup, 2006), which focuses on a side differences in attentional performance. To match stimulus conditions in these other experiments, the letters used in the whole report experiment were either green or purple (with equal luminance: 36 cd/m²); the colour was selected randomly for each letter.

Participants were instructed to report as many letters as possible, but refrain from guessing. Report was unspeeded. The exposure duration was varied systematically, with six individually set exposure durations (based on performance in the practice trials). Four masked exposures were used, spanning an interval from the participant’s approximate threshold (20–40 ms) to 200 ms. To prolong the effective exposure duration, two unmasked displays (usually 100 and 200 ms) were also used. There were 25 repetitions for each of these 2 x (4 + 2) = 12 conditions, randomly intermixed within each testing block. The error rate was recorded throughout, and the percentage correct score was given as feedback to the participant after each testing block. A score of 80–90% was encouraged to prevent too liberal or too conservative reporting. Percentage correct was on average 84.3% (SD = 5.9%) and 87.1% (SD = 2.5%) in the control and patient groups, respectively. The total testing included 300 trials, organized in blocks of 60 trials, and all testing was completed within one or two sessions of maximally 1-h length, including breaks. In addition, participants were given 20–30 unspeeded warm-up trials at the beginning of each session.

To ensure central fixation before stimulus exposure in each trial, participants were instructed to look at a centrally placed cross and, after having signalled ready, to name a random digit that appeared for 300 ms at this position. Immediately afterwards the stimulus display was initiated by the experimenter. The instruction to fixate centrally was emphasized throughout testing. As an additional control the eye movements of all participants were recorded by a video camera, and the signal was mixed with a simultaneous camera recording of the computer display. The experimenter monitored the subject’s eye movements continuously on a TV screen during testing, and the mixed image was recorded on VHS tape. The VHS tapes were subsequently inspected for unwanted eye movements (i.e., away from the central cross before stimulus exposure) using 32 random samples for each participant. If an unwanted eye movement was detected in any of these 32 trials, the whole VHS tape was inspected and all invalid trials removed from the data set. This was done for one patient, who had 29 trials removed from his data set.

2.3. Data analysis

The best-fitting TVA parameter values to the observed data of each participant were estimated by a maximum likelihood fitting algorithm. The model fitting procedure used to analyze the results was basically the same as that employed in previous TVA studies, and we refer to Duncan et al. (1999) for mathematical details. Customized software for TVA analysis (Kyllingsbaek, 2006) was used, which also allowed for bootstrap analysis of the fits (Habekost & Bundesen, 2003; see also Efron, 1979; Efron & Tibshirani, 1993). The following parameters were estimated: $K$, $C_{th}$, $C_{light}$, $t_{th}$, $t_{light}$, and $\mu$. In order to make the model fitting more robust, $t_{th}$ values were constrained to be 15 ms at minimum. $K$ values were constrained not to be higher than the best score obtained by the participant. All observed data was included in the analysis (no exclusion of outlier trials). The reliability of each parameter estimate was evaluated by 1000 bootstrap repetitions. Each bootstrap sample was constrained to include at least one trial with the subject’s maximum score.

2.4. Lesion analysis

The lesions of all patients except two were identified by MRI. A total of 16 patients were examined in a 3 T scanner (Siemens Trio), and 4 patients were examined in a 1.5 T scanner (Siemens Vision). Two patients could not be MR scanned due to claustrophobia and a heart implant, respectively; instead CT scans from the acute stage were collected from their hospital records. For a high precision description of the structural anatomy, a 3D volumetric MP-RAGE sequence (1 mm³ resolution; 3 T: TR/TE/TI: 6.03/9.3/800 ms, flip angle: 8°; 1.5 T: TR/TE/TI: 13.5/7/100 ms, flip angle: 15°) covering the whole brain was performed. To characterize the lesions in further detail, patients were also examined using a FLAIR sequence (3 T: TR/TE/TI: 9000/102/2500 ms, flip angle: 150°; 1.5 T: 9000/110/2400 ms, flip angle: 180°). Using the combined information from these scans, the lesions were drawn on each individual’s MP-RAGE slices by an experienced neurologist who was blind to the psychophysical data. The MP-RAGE scans with traced lesions was normalized to a 1 mm isotropic T1 template using SPM2 (www.fil.ion.ucl.ac.uk/spm/software/spm2). Before normalization the lesion area was masked out from the intact part of the brain to prevent distortions (Brett, Leff, Rosden, & Ashburner, 2001). The volume of the (normalized) lesion was computed using the MRicro program (Rorden & Brett, 2001; www.mricro.com). Voxelwise statistical testing was performed to locate areas significantly related to abnormal performance. For each brain voxel patients were divided into two groups, either with or without damage in the voxel. The psychophysical scores of these two groups were compared using a t-test with significance threshold of $p = .01$ (not corrected for multiple comparisons). In addition to the analysis of stroke-related brain damage, white matter hyperintensities (leukoaraiosis) visible on the FLAIR images were traced by the first author. White matter hyperintensities are typically symmetrical in the two hemispheres, and each patient’s total volume was estimated by doubling the count from the left hemisphere, which was not affected by stroke. In case of the two patients who were not investigated by MR, their CT scans were not analyzed quantitatively, but the lesions were traced and a verbal description was given by the examining neurologist. Each patient was assigned to one of three subgroups: (a) strokes with severe leukoaraiosis ($n = 3$), (b) large (volume $> 30$ cm³) cortico-subcortical strokes with mild or no leukoaraiosis ($n = 13$), and (c) small (volume $< 3$ cm³) strokes with mild or no leukoaraiosis ($n = 6$). See Table 1 for lesion characteristics.

3. Results

In this section, we present test results (TVA parameter estimates) and relate this set of findings to lesion anatomy. The focus is on absolute levels of performance. For a discussion of intrindividudal side differences in the whole report experiment (and partial and colour report experiments) see Habekost and Rostrup (2006). The reliability of each TVA estimate was quantified by bootstrap analysis (Efron, 1979). Given a set of observations (e.g., the 300 trials in whole report) bootstrap analysis computes an estimate of the (standard) measurement error related to each test result. The bootstrap analysis showed that measurement error was generally low: On average 10.2% for $C$ and 2.7% for $K$, consistent with previous findings that TVA estimation of these parameters is very reliable (Habekost & Bundesen, 2003). This high test reliability should generally increase our statistical power to find significant relations between cognitive performance and lesion anatomy.

For each individual patient, we tested for pathological deviations from the control group using a method based on the t-distribution proposed by Crawford and Howell (1998) which is more robust than traditional testing based on z-scores (Crawford, Garthwaite, Azzalini, Howell, & Laws, 2006). The software program “singlims.exe” (www.abdn.ac.uk/~psy086/dept/SingleCaseMethodsComputerPrograms.HTM) was used. Scores deviating more than 1.87 SD from the control group mean reached significance on this test (i.e., were classified as pathological).

3.1. Whole report experiment

For a summary of the whole report estimates, see Table 2. The average $K$ value in the control group was $3.32$ (SD = 0.5), con-
The standard deviation of scores within each group is indicated in parentheses.

Table 2

<table>
<thead>
<tr>
<th>Group</th>
<th>(C_{\text{left}}) (s(^{-1}))</th>
<th>(C_{\text{right}}) (s(^{-1}))</th>
<th>(K)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control group (n = 12)</td>
<td>18.1 (5.0)</td>
<td>20.8 (5.2)</td>
<td>3.32 (0.5)</td>
</tr>
<tr>
<td>Leukoaraiosis group (n = 3)</td>
<td>8.6 (1.7)</td>
<td>12.3 (5.3)</td>
<td>2.15 (0.1)</td>
</tr>
<tr>
<td>Large lesion group (n = 13)</td>
<td>12.3 (5.4)</td>
<td>19.5 (7.4)</td>
<td>2.99 (0.8)</td>
</tr>
<tr>
<td>Small lesion group (n = 6)</td>
<td>14.7 (3.9)</td>
<td>18.8 (7.5)</td>
<td>2.90 (1.0)</td>
</tr>
</tbody>
</table>

\(\ast\) Significant \((p < .05\), Mann–Whitney\) deviation from the control group mean. The standard deviation of scores within each group is indicated in parentheses.

In the large lesion group, there was a clear split in the group: 10 patients had normal values above 3.0, but three patients had \(K\) values in the range of 1.15–2.12 (each of which was significantly abnormal by Crawford and Howell’s test). In the leukoaraiosis group, the average \(K\) value was 2.15 (SD = 0.1), which was a significantly different from the control mean \((p < .01\), Mann–Whitney\). All three patients in this group had a \(K\) value that was significantly abnormal by Crawford and Howell’s test. Among patients with small strokes, one patient with parietal damage and one basal ganglia patient had significant reductions of VSTM capacity, whereas the other four patients had \(K\) values in the normal range. Thus, out of the 22 patients, 8 had significant reductions in VSTM capacity: 3 with large lesions, 3 with leukoaraiosis, and 2 with small lesions. Besides these deficits, it is remarkable that most patients with large lesions had preserved VSTM capacity.

In the control group the average \(C_{\text{left}}\) value was 18.1 letters/s (SD = 5.0 s\(^{-1}\)) and the mean \(C_{\text{right}}\) value was 20.8 s\(^{-1}\) (SD = 5.2 s\(^{-1}\)). In the large lesion group, the average \(C_{\text{left}}\) value of 12.3 s\(^{-1}\) (SD = 5.4 s\(^{-1}\)) was much lower than the control group’s \((p < .05\), Mann–Whitney\), but the mean \(C_{\text{right}}\) value was close to normal: 19.5 s\(^{-1}\) (SD = 7.4 s\(^{-1}\); \(p = .73\), Mann–Whitney). Only two patients in the large lesion group had significantly reduced \(C\) values in both visual fields (by Crawford and Howell’s test), corresponding to general reductions of visual processing speed. In the leukoaraiosis group the mean \(C_{\text{left}}\) and \(C_{\text{right}}\) values were 8.6 s\(^{-1}\) (SD = 1.7 s\(^{-1}\)) and 12.3 s\(^{-1}\) (SD = 5.3 s\(^{-1}\)), respectively. The mean \(C\) values were significantly different from the control group mean in both sides (both: \(p < .05\), Mann–Whitney), but on the individual level only one leukoaraiosis patient had significantly reduced \(C\) values bilaterally. Finally, patients with small lesions had average \(C_{\text{left}}\) and \(C_{\text{right}}\) values at 14.7 s\(^{-1}\) (SD = 3.9 s\(^{-1}\)) and 18.8 s\(^{-1}\) (SD = 7.5 s\(^{-1}\)), respectively. The difference to the control mean did not reach significance in either visual field, but one patient with parietal damage was significantly below the control mean in both sides. In sum, visual processing speed was frequently reduced in the left side. This finding is considered in a parallel article dealing with side differences in attention (Habekost & Rostrup, 2006). In the present article we focus on general reductions of visual attention capacity, which implies that the ipsilesional hemisphere is also affected. Bilateral deficits in visual processing speed occurred much less frequently than selective contralesional reductions, but were found after severe leukoaraiosis and in two patients with large lesions, as well as a single patient with focal damage.

3.2. Lesion anatomy and psychophysical performance

In the large lesion group 10 out of 13 patients had \(K\) values in the normal range in spite of extensive damage to the right side of the brain. See Fig. 3 for a density plot of the normalized lesions of these patients. The lesions centred on the right putamen and medial insular region, where all patients but one was affected, and typically also involved more lateral segments of the insula and inferior frontal lobe. This is strong evidence that damage to these structures do not lead to chronic deficits in VSTM capacity.

Three patients in the large lesion group had significantly reduced \(K\) values. These patients also had the largest lesions in the group. Accordingly there was a significant negative correlation between \(K\) and lesion volume in the large lesion group as a whole: \(rs = -.70 (n = 12\), \(p < .05\)). However, among patients with normal \(K\) values there was no significant correlation: \(rs = -.33 (n = 9\), \(p = .39\) in spite of highly variable lesion size within this group (range: 35.1–167.7 cm\(^3\); SD = 51 cm\(^3\)). An explanation for the discrepancy is that very large lesions were systematically related to damage in particular regions and that damage here – rather than lesion volume per se – was critical. As illustrated in Fig. 3 patients with large lesions had a common damage focus (which was also shared by the three patients with reduced \(K\) values) in an anterior area that included the putamen and overlying frontal and insular cortex. Other cortical areas were affected to a more varying degree: the larger the lesions, the more they extended away (posteriorly, superiorly and laterally) from the common focus in the putamen and medial insula. For the three patients with VSTM deficits and very large lesions, this implied damage in remote areas not shared by the 10 patients without VSTM deficit. These unique areas of damage were all located in white matter tissue: Superiorly in the centrum semiovale, beneath the middle temporal gyrus, and near the temporal pole.

For a stringent analysis of critical brain areas for parameter \(K\), we performed a voxelwise test across the spatially normalized brains of all 17 patients in the study with available MR scans (but no leukoaraiosis). For each brain voxel, a \(t\)-test was used to assess the magnitude of difference in \(K\) values between subjects having a lesion at that location, and those that did not. The significant results corresponded closely to the unique areas of damage described for the three patients above. Three main foci emerged: The first was located in the superior white matter beneath parietal and frontal cortex, the second more posteriorly in temporal white matter, and the third in the anterior parts of the temporal lobe (see Fig. 4).

In each patient, the part of the lesion that covered these areas was often not completely infarcted, but rather partially damaged.

5 The lesion of one patient (LL8) in this group was not included in the overlap analysis, since only a CT scan was available. The lesion was located within the same area as the others.
(e.g., de-myelinated) white matter tissue. This is for example evident in the MR scan of the patient (LL13) with the most extreme reduction of VSTM capacity ($K = 1.15$; see Fig. 5).

In the patient group as a whole, the estimated volume of white matter hyperintensities (leukoaraiosis) had a strong negative correlation with $K$: $r_s = -0.68$ ($n = 20$, $p < .005$). This effect was due to three patients with severe leukoaraiosis (see Fig. 6), who all had marked reductions in VSTM capacity. The leukoaraiosis was close to symmetric in the two hemispheres, and periventricular as well as centrum semiovale regions were affected. Two of the patients had small strokes in areas (thalamus, putamen) that, judging from other patients, were not critical for VSTM capacity. The infarct of the third patient included periventricular white matter, which might have contributed to the observed deficit in $K$.

Two patients had significant $K$ reductions after focal lesions. The first of these patients had a small lesion in the intraparietal sulcus, which fits with two recent fMRI studies of VSTM capacity (Todd & Marois, 2004; Xu & Chun, 2006). However, only a CT scan was available for this patient, which is not optimal for detection of leukoaraiosis. Therefore, the influence of this factor cannot be ruled out. The second patient with $K$ reduction had a small lesion in the putamen and corona radiata. The reason for the poor performance of this patient is unclear, but it is conceivable that subcortical lesions may sometimes, due to their connectivity, profoundly affect the function of larger, structurally intact parts of the brain (Vallar, Cappa, & Wallesch, 1992).

Visual processing speed in the contralesional side, $C_{left}$, was often impaired (see Habekost & Rostrup, 2006), but reductions of $C_{right}$ occurred rarely. Eleven out of 13 patients in the large lesion group had a $C_{right}$ value within normal variability, which indicates that the anterior area commonly damaged in this group was not critical for ipsilesional processing speed. The two patients who did show significant deficits of $C_{right}$ had unique areas of damage around the temporo-parietal junction and in the middle frontal gyrus. As with parameter $K$, we checked these findings using a voxelwise test across the normalized brains of the 17 patients in the study without leukoaraiosis. The statistical analysis showed that $C_{right}$ reductions were significantly related to damage in the middle frontal gyrus, but the critical status of the temporo-parietal junction was not verified; instead a small focus was found in the middle part of the superior temporal gyrus (see Fig. 7).

$C_{right}$ and lesion volume were not reliably correlated in the patient group as a whole: $rs = -0.17$ ($n = 20$; n.s.). However, there
was a significant negative correlation between $C_{\text{right}}$ and the estimated leukoaraiosis volume: $r_s = -0.57$ ($n = 20; p < .01$).

4. Discussion

The information uptake from a brief visual display seems to depend on two factors: the rate of visual encoding, $C$, and the storage capacity of VSTM, $K$. The anatomical structures involved in these cognitive functions are still poorly charted. Recent functional imaging studies suggest that the storage component of VSTM is located in the posterior parietal lobe, but functional imaging show only regions that are (relatively) active during a given task, which is not equal to being functionally critical. Lesion studies provide a stronger design for finding critical regions, though localization is typically less precise. We investigated 22 patients with right side stroke and found that bilateral deficits in visual attention capacity were related to certain types of white matter damage, whereas lesions in a large, anterior part of the right hemisphere were not critical. We also found evidence relating damage in the right middle frontal gyrus to bilateral deficits in visual processing speed. In the following, these findings are integrated with results from the functional imaging literature for a more comprehensive account of the neural basis of visual attention capacity.

4.1. The patient sample

When evaluating the present results it is important to bear in mind that the patient group was not an unbiased sample of right
Fig. 5. MR scan of the patient (LL13) with the largest VSTM deficit. Note the widespread affection of white matter.

Fig. 6. FLAIR scans of three patients with severe leukoaraiosis and marked reduction of VSTM capacity. (Upper panels) Periventricular damage; (lower panels) centrum semiovale damage.
hemisphere strokes. First, since perception of left-side stimuli was necessary to complete the experimental testing, patients with visual field cuts were not included. Due to the location of the optic radiation this probably made selection of patients with posterior cortical damage less likely, which might explain why lesions were generally located quite anteriorly (centring on the putamen area). Specifically, the low number of patients with posterior parietal damage made it difficult to test the critical status of this region. Second, hospital records were screened retrospectively and individuals invited to participate on this basis. About a third of the contacted patients declined, and given the extensive testing program offered it is possible that more incapacitated patients tended not to participate. Together with the lack of posterior parietal lesions this might have lowered the statistical power of our lesion analysis to detect critical brain areas. Still, more anterior areas of the right hemisphere received good coverage and several important findings emerged in the study (see next sections). Third, all patients were in the stable phase of recovery (>6 months postinjury) and the deficits found were therefore of a chronic nature. Unlike studies of acute patients the confounding influence of diffuse metabolic disturbance or diaschisis should not be a serious problem for our lesion analysis. On the other hand, functional reorganization during the recovery period may have altered the neural localization of the cognitive functions targeted in our study. This potential confound is shared by many patient studies and ultimately, converging evidence obtained by other methods is needed to confirm the conclusions.
4.2. Noncritical areas for visual attention capacity

Ten out of 13 patients with large right hemisphere strokes had normal VSTM capacity, and 11 patients in this group had intact visual processing speed in the right visual field. This normal performance was found in spite of the fact that the whole report task featured demanding near-threshold stimulation and has previously been shown to be sensitive to small attentional disturbances (Habekost & Bundesen, 2003; Habekost & Rostrup, 2006; Habekost & Starrfelt, 2006; Peers et al., 2005). The lesions of these normally performing patients centred on structures in the right basal ganglia, insula, and inferior frontal cortex, whereas involvement of the posterior parietal cortex was minor or absent in most cases. VSTM capacity and ipsilesional visual processing speed was also normal in two patients with right thalamic lesions and two patients with focal damage in the right basal ganglia. The results show that a large, anterior part of the right hemisphere (as well as segments of the right thalamus) is not critical for the general capacity of visual attention, at least in terms of VSTM capacity and visual processing speed. The findings are consistent with another TVA based patient study by Peers et al. (2005), who found no relation between deficits in C or K and focal lesions in the left or right frontal cortex. The present results are perhaps more surprising since many of our patients had large cortico-subcortical lesions.

The findings may seem at odds with a case study by Habekost and Bundesen (2003), who found reduced VSTM capacity in a patient (GL) with damage in the right frontal lobe and underlying basal ganglia. The lesion was located in the general area pointed out as noncritical for K in the present study. The cause of patient GL’s low VSTM capacity is unclear. One possible explanation is that GL’s lesion included parts of the middle frontal gyrus, which might contain a relevant area for K (as suggested by a functional imaging study of Bundesen, Larsen, Kyllingsbaek, Paulson, & Law, 2002; see below). Few of our patients with normal VSTM capacity had lesions that extended into the middle frontal gyrus, and it is hard to say whether the exact same functional areas were damaged as in patient GL. Alternatively GL’s VSTM reduction might be caused by other, undetected factors than the structural brain damage, such as functional disturbance of structurally intact brain areas, perhaps combined with a low premorbid VSTM capacity (there is considerable normal variation in the distribution of K values). The case study did not allow firm conclusions to be drawn on this issue. Regardless, the evidence from this single patient does not affect the conclusion, based on 10 normally performing patients, that a large region centring on the right putamen and medial insula is not critical for VSTM capacity.

Our results bear on theories arguing that the right hemisphere contains a representation of both visual fields (Heilman & van den Abell, 1980; Mesulam, 1981) or that it plays a special role in general alertness (Heilman, Watson, & Valenstein, 2003; Posner & Petersen, 1990). Both theories imply that right side brain damage can lead to bilateral impairments of attention. This notion has been substantiated by studies of the neglect syndrome, which typically occurs after right hemisphere stroke. As reviewed by Husain and Rorden (2003) there is now much evidence that neglect patients often have bilateral attentional deficits in addition to their lateralized attention disturbance (i.e., the tendency to overlook contralesional stimuli), such as a protracted attentional blink (Husain, Shapiro, Martin, & Kennard, 1997), difficulties sustaining attention (Robertson et al., 1997), and poor spatial working memory (Wojciulik, Husain, Clarke, & Driver, 2001). However, it is not clear which part of the lesion anatomy of neglect patients that causes these bilateral deficits. Large patient studies have shown that the neglect syndrome as a whole typically occurs after large lesions in the posterior cortex that include the inferior parietal lobe (Mort et al., 2003) or the superior temporal gyrus (Karnath, Berger, Kükker, & Rorden, 2004). However, neglect has also been found after focal lesions in more anterior areas including the inferior frontal lobe (Husain & Kennard, 1996), insula (Manes, Paradiso, Springer, Lamberty, & Robinson, 1999), andputamen (Karnath, Himmelbach, & Rorden, 2002). Hence it is interesting that, according to our findings, these anterior parts of the right hemisphere are not critical for two main types of general attention capacity: (ipsilesional) visual processing speed and VSTM capacity. If neglect is an attention disturbance with both lateralized and nonlateralized elements, as commonly assumed (e.g., Robertson, 1993), our results suggest that anterior lesions are specifically related to the lateralized components. Consistent with this hypothesis, a large proportion of our patients with lesions in anterior areas showed asymmetrical perception (i.e., lower processing speed in the left visual field; see Habekost & Rostrup, 2006), but no general reduction in attentional capacity (i.e., normal C_right and K values). Accordingly, few had clinical signs of the full neglect syndrome. The previous findings of neglect in patients with focal lesions in the putamen, insula, or frontal lobe may be explained by diaschisis effects, where structural damage in anterior areas indirectly affects closely connected posterior areas such as the inferior parietal lobe or the superior temporal gyrus (Hillis et al., 2005). This hypothesis is supported by the fact that most of these studies (e.g., Karnath et al., 2002; Manes et al., 1999) have investigated patients within the first few weeks postinjury.

4.3. Cerebral connectivity and visual attention capacity

According to a standard view, short-term retention of visual information implies that the activation of neurons representing the information is sustained in a feedback loop (Hebb, 1949; Tallon-Baudry, Bertrand, & Fischer, 2001). The feedback process is usually assumed to depend on interactions between prefrontal and posterior cortical areas (Fuster, 1997; Goldman-Rakic, 1995) and possibly also cortico-thalamic networks (Bundesen et al., 2005). This way VSTM should involve information transfer between distant brain areas and depend on efficient long-range fibres. A related view holds that conscious recognition of visual objects (reflected in the C parameter) requires broadly distributed activity in parietal and frontal cortex (Beck, Rees, Frith, & Lavie, 2001; Crick & Koch, 1995; Duncan, 1996; Rees et al., 2000) besides visually specific areas. As with VSTM, successful integration of this complex activity should depend on fast reciprocal connections. On theoretical grounds one should therefore expect reductions in both K and C after...
damage in relevant parts of the white matter, specifically the long-range posterior–anterior or cortico-thalamic connections. Our data confirmed this general prediction.

Two lesion patterns in our study were systematically related to reductions in VSTM capacity: Severe leukoaraiosis and very large strokes in the right side of the brain. Both types of damage should compromise relevant cerebral connectivity. As mentioned in the Introduction, leukoaraiosis is a radiological term for diffuse abnormalities in the white matter, which can be seen as high intensity signals on fluid-attenuated inversion recovery (FLAIR) MR scans (Ward & Brown, 2002). These abnormalities usually result from subcortical arteriosclerosis, but similar changes may be caused by multiple sclerosis or metabolic disease. A common effect is de-myelination of fibre tracts, presumably leading to slower signal transmission (it should however be noted that the pathological significance of white matter hyperintensities on MR scans is not straightforward and may also reflect other types of white matter damage). Some degree of leukoaraiosis is a common finding in the elderly population, especially when examined by sensitive MR sequences. The clinical implications of the condition are controversial and it has been argued that leukoaraiosis is often asymptomatic (Bonanno et al., 2000). However, other studies have found associations with dementia (Inzitari et al., 1987), declines in general intelligence (Garde, Mortensen, Krabbe, Rostrup, & Larsson, 2000), and executive dysfunction (O’ Sullivan et al., 2004). To our knowledge the condition has not previously been associated with deficits in visual attention, but the present study suggests that severe leukoaraiosis is related to reductions in VSTM capacity and, less strongly, general visual processing speed. Severe leukoaraiosis typically affects both hemispheres diffusely at periventricular and centrum semiovale levels, as was also the case for the three patients in our study. The observed deficits in visual attention capacity could thus be related to several fibre systems and converging evidence from other findings should guide the interpretation.

Some of this evidence may be found in a different subgroup of our patients. Three patients with very large right side strokes also had reductions of VSTM capacity. Compared to other patients with large strokes, these patients had unique damage in the right centrum semiovale and temporal white matter. Statistical analysis confirmed that damage in these areas was significantly related to VSTM deficits. Both the centrum semiovale and the temporal white matter regions contain a large number of intrahemispheric and cortico-subcortical fibres that are potentially relevant for VSTM. For example lesions in the second branch of the superior longitudinal fasciculus (see Schmahmann & Pandya, 2006) have recently been related to neglect behaviour (Bartolomeo, 2006; Doricchi & Tomaiuolo, 2003; Thiebaut de Schotten et al., 2005). However, such lesions did not seem to be critical for VSTM capacity. Many of our patients with normal K values were damaged in this part of the white matter. Instead, the importance of more dorsally located fibres is suggested by the fact that both patients with very large strokes and patients with leukoaraiosis were strongly affected in the superior part of the centrum semiovale. On the other hand, the more inferiorly located damage in the temporal white matter corresponds to findings on critical areas for chronic neglect (Samuelsson, Jensen, Ekholm, Naver, & Blomstrand, 1997). As with the deficits after leukoaraiosis, the data do not point clearly towards a particular fibre system. Functional imaging studies of the cortical networks knit together by the fibres provide a broader context for interpretation (see next section).

Bilateral deficits in visual processing speed occurred rarely in our patient group, but correlated significantly with damage in the middle frontal gyrus and with leukoaraiosis. The critical status of the right middle frontal gyrus is consistent with several recent studies of fronto-parietal networks and visual perception, as discussed in the next section. Concerning the relation of $C_{right}$ to leukoaraiosis, rapid stimulus recognition should be impaired by diffuse damage to long-range fibres (e.g., between parietal and frontal cortex), similar to VSTM capacity. Damage in a small area in the middle part of the superior temporal gyrus also correlated significantly with low visual processing speed. However, it seems unlikely that this area is critical, as it was also damaged in other patients than the two who had significant bilateral deficits in visual processing speed.

4.4. The neural basis of visual attention capacity

The neural basis of visual attention capacity has been investigated by two main methods: functional imaging and studies of brain damage. Functional imaging studies have found bilateral fronto-parietal activity in visual working memory tasks (Corbetta, Kincade, & Shulman, 2002; Linden et al., 2003). Given this it may be surprising that we found no relation between damage in the right frontal cortex and VSTM capacity. One explanation is that lesions in our study were located relatively inferior in the frontal lobe, whereas VSTM may be associated with more superior areas, as suggested by a PET study of Bundesen et al. (2002). Another possibility is suggested by recent imaging studies that specifically targeted the storage limitation of VSTM. These studies indicate that the posterior node of the fronto-parietal networks is the most relevant for storage capacity (Owen, 2004). In an fMRI study Todd and Marois (2004) found that bilateral activity in the intraparietal sulcus and the intracopical sulcus correlated with the number of objects held in VSTM (see also Xu & Chun, 2006). Vogel and Machizawa (2004) reached a similar conclusion in an ERP study, in which activity at posterior parietal and lateral occipital electrode sites correlated with performance on a VSTM task. Rather than storage per se prefrontal areas may be critical for higher-order operations such as executive memory processes (Linden et al., 2003) or shielding VSTM representations from interference (Miller, Erickson, & Desimone, 1996).

Based on the evidence from functional imaging one should expect lesions in the posterior parietal lobe, perhaps specifically the intraparietal sulcus, to cause reductions in VSTM capacity. Strong evidence has been found in a group of neglect patients with damage involving the right parietal cortex (Duncan et al., 1999), but no systematic lesion analysis was attempted in this study. Both Peers et al. (2005) and the present study examined very few patients with damage in the intraparietal sulcus, which makes it
hard to assess the significance of this area. Instead the present study pointed to white matter located high in the centrum semiovale. This is compatible with the importance of the posterior parietal cortex, though not direct support for this hypothesis.

The second attentional capacity parameter, visual processing speed, should primarily depend on the efficiency of basic pattern recognition processes. Numerous studies have linked visual recognition to ventral occipito-temporal areas (Ungerleider & Mishkin, 1982; Milner & Goodale, 1995), which were generally intact in our patient group. In particular the left extrastriate cortex, which has been associated with letter recognition (Flowers et al., 2004; Polk et al., 2002), was not affected in any patients. However, in line with theories that multiple cortical areas are necessary for conscious recognition (e.g., Duncan, 1996; Rees, 2001), more dorsal regions should also be relevant for visual processing speed. Simultanagnosia, which typically occurs after bilateral parietal lesions, has been related to extreme reductions in visual processing speed (Duncan et al., 2003). In a visual change detection task, Beck et al. (2001) found that conscious perception correlated with activity in bilateral parietal and right dorsolateral prefrontal (DLPF) cortex. The critical role of the right DLPF was confirmed in a study by Turatto, Sandrini, & Miniussi (2004), who found that repetitive transcranial magnetic stimulation (rTMS) applied to this region impaired conscious perception of stimulus change, as compared to left DLPF rTMS and sham stimulation (a similar result for the right parietal cortex was reported by Beck, Muggleton, Walsh, & Lavie, 2006). Our finding, that damage in the right middle frontal gyrus is related to deficits in general visual processing speed, adds further support to the importance of the right DLPF for visual perception. The result may seem in conflict with the study of Peers et al. (2005), who found no general relation between frontal damage and C reductions. However, about half of Peers et al.’s frontal patients were damaged in the left hemisphere, which should be less important according to the studies of Beck et al. (2001) and Turatto et al. (2004). Of the seven patients with right frontal lesions in Peers et al.’s study, only two or three (“CG”, “PB”, and possibly “MS”) were damaged in the area pointed out by our analysis. For one of these patients (“CG”) C scores were not reported; the other two had the lowest C scores in the right frontal group. Thus, a closer inspection of Peers et al.’s data in fact shows support for our finding.

We also found a negative correlation between C\text{right} and leukoaraiosis. This may be related to disturbance of the connections between frontal and parietal areas, but this hypothesis awaits more data to be tested.

5. Conclusion

The neural basis for capacity limitations in visual attention is still a relatively unexplored issue. A main finding of the present study was that damage in a large anterior region of the right hemisphere, including the putamen, insula, and inferior frontal lobe, is not critical for VSTM capacity or ipsilesional visual processing speed. This finding has implications for theories of visual neglect, because it suggests that the bilateral attention deficits commonly found in this syndrome are caused by disturbed function in other, possibly more posterior structures. Instead our results pointed to the importance of white matter for attentional capacity, especially the connections between parietal and frontal cortex located superiorly in the centrum semiovale. Both stroke and leukoaraiosis (age-related changes in white matter) in these areas were related to bilateral deficits in visual attention capacity. The study also found evidence that the right middle frontal gyrus is critical for visual processing speed, supporting theories of frontal involvement in visual perception.

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