

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.

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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 temporo-parietal 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 contralesional 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 lateral-

ized 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, t_0 , no items are reported. With postmasked alphabetic stimuli the perception threshold t_0 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

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