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Cognition and Neurosciences

Visual attention capacity: A review of TVA-based patient studies

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Psychophysical studies have identified two distinct limitations of visual attention capacity: processing speed and apprehension span. Using a simple test, these cognitive factors can be analyzed by Bundesen's Theory of Visual Attention (TVA). The method has strong specificity and sensitivity, and measurements are highly reliable. As the method is theoretically founded, it also has high validity. TVA-based assessment has recently been used to investigate a broad range of neuropsychological and neurological conditions. We present the method, including the experimental paradigm and practical guidelines to patient testing, and review existing TVA-based patient studies organized by lesion anatomy. Lesions in three anatomical regions affect visual capacity: The parietal lobes, frontal cortex and basal ganglia, and extrastriate cortex. Visual capacity thus depends on large, bilaterally distributed anatomical networks that include several regions outside the visual system. The two visual capacity parameters are functionally separable, but seem to rely on largely overlapping brain areas.

Key words: Attention deficits, lesion anatomy, TVA based assessment, visual processing speed, visual span.

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INTRODUCTION

Despite the massive visual system of the human brain, our ability to consciously perceive information from a single fixation of the eyes is highly limited. Psychophysical studies have identified two distinct limitations of visual capacity. One concerns the maximum number of objects that can be perceived at the same time (*the visual span*). This cognitive factor, also referred to as the capacity of visual short-term memory (VSTM), has been widely studied (e.g., Cowan, 2001; Luck & Vogel, 1997; Sperling, 1960). The second capacity limitation, which has been studied to a much smaller degree, reflects the amount of visual information that can be processed per second (*visual processing speed*; e.g., Shibuya & Bundesen, 1988). A new line of research applies the conceptual framework of Bundesen's Theory of Visual Attention (TVA; Bundesen, 1990) to quantify attentional capacity. Within this framework both capacity factors can be simultaneously measured in a simple experimental task (whole report) when combined with data analysis by the equations of the TVA model. Since the introduction of this test method by Duncan *et al*. (1999) TVA-based assessment has been used in more than a dozen studies, mainly to investigate the effects of various types of brain damage on attentional function. In total, measurements of attentional capacity have now been collected from more than 300 individuals, both neurologically healthy and brain damaged. The present article is a review of this new research area. The review covers the theoretical background, the general method, and the main results obtained so far. It should be noted that TVA-based assessment can also be used to measure other aspects of visual attention, including distractibility and spatial bias, but these investigations are outside the scope of the present article (see instead Bundesen & Habekost, 2008).

Visual span

The human limitation in visual span has long been recognized. Already in the 19th century Cattell (1885) noted that subjects could not identify more than about four objects from a brief visual presentation. A series of studies by Sperling (1960, 1963, 1967) established this finding by more stringent experimental methods. The general task applied was *whole report*, in which a number of simple visual objects (letters) were flashed for a fraction of a second followed by a blank screen. The subjects were instructed to name as many letters as possible, but refrain from guessing. As the exposure time was made longer the number of correctly reported letters increased, but the performance curve flattened out when a score of about four correct was reached. This result indicated a clear upper limit for the number of items that could be perceived at the same time. The finding has been replicated many times, also using different experimental paradigms (e.g., change detection: Vogel, Woodman & Luck, 2001) and other stimulus types (e.g., digits: Starrfelt, Habekost & Gerlach, 2008a; Starrfelt, Habekost & Leff, 2008b; colours: Luck & Vogel, 1997) and seems to reflect a general limitation in the apprehension span for visual objects. Within the TVA-framework the visual apprehension span, the upper limit of the number of visual items that can be perceived simultaneously, is represented by the parameter K.

Visual processing speed

The limitation in visual processing speed is most easily recognized in a report task where only a single stimulus is presented. After a variable, brief time interval (typically between 10 and 100 ms) the stimulus is replaced by a pattern mask. The purpose of the

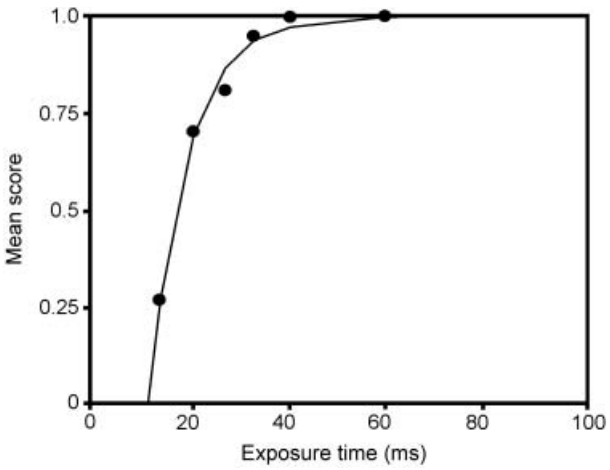

Fig. 1. Typical individual pattern of performance in an experiment where a single masked stimulus (a letter) must be reported. The mean number of correct reports (the score) is shown as a function of exposure time. Solid curves represent maximum likelihood fits to the observations based on TVA analysis. Below the visual threshold, t_0, the score is zero. The slope of the curve at the intercept with the *x*-axis equals the visual encoding rate for the stimulus, *C*. The performance curve follows an exponential function given by the following equation: Probability of correct report = $1 - \exp(-C * (t - t_0))$, where *t* is the exposure time.

mask is to erase the visual afterimage of the stimulus and thereby effectively control the time it is processed. In this task the mean score develops systematically as a function of the exposure duration; see Fig. 1 for an example. Below a specific, individually variable exposure duration, t_0, the score is zero. Under these conditions the visual system cannot extract any information about the identity of the stimulus, which therefore remains below the perception threshold. When the exposure duration is increased above the threshold, the score rises abruptly and continues to increase asymptotically towards 100% correct. As shown by Bundesen and Harms (1999) the probability of perceiving the stimulus (i.e., the mean score) as a function of exposure time follows a simple exponential function. An individual example of this function is illustrated by the solid line in Fig. 1. The exponential model implies that, above the perception threshold t_0, the probability of perceiving the stimulus as a function of time is determined entirely by a single constant factor: the visual processing speed *C*. Estimates of the visual processing speed for single letters or digits presented at fixation typically range from 50–200 elements per second in young healthy subjects.

TVA modelling of visual attention capacity

The TVA model (Bundesen, 1990) incorporates both of these capacity limitations in its general description of the visual perception process. According to TVA, consciously recognizing a visual object corresponds to encoding one or more of the object's properties into a visual short-term memory store. The memory store only has room for very few objects at the same time, hence the limitation in visual span (the *K* parameter in TVA). The encoding process that leads up to conscious recognition takes the form of a race between the elements in the visual field. See Fig. 2 for a schematic illustration of this process (for a detailed description of TVA theory, see Bundesen & Habekost, 2008). The visual system is assumed to process all objects in the visual field simultaneously (independently and in parallel), but at widely varying rates. The processing rate of each object determines its probability of being encoded into visual short-term memory, given that there are still available slots in the store. The sum of the processing rates for all objects equals the total processing capacity of the visual system (in the current stimulus situation), denoted by parameter *C*. The processing rate for each

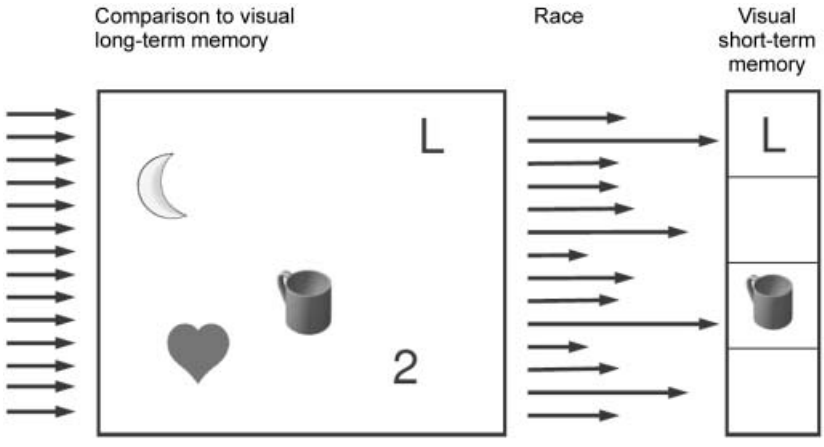

Fig. 2. Schematic illustration of the visual encoding process according to TVA. At the first, preconscious stage of processing each object in the visual field is compared implicitly against representations in visual long-term memory. This is done to determine how likely the object is to be important (and hence should be attended). The stronger the evidence that the object is important, the more processing capacity is devoted to it, so the faster the object can be processed in the ensuing race to become encoded in visual short-term memory and thereby be consciously recognized. The length of each arrow symbolizes the encoding rate for an object categorization. The combined length of all the arrows corresponds to the total processing speed in the visual system, parameter *C*. The number of slots in the visual-short term store corresponds to parameter *K*.

object reflects the proportion of the total processing capacity that has been allocated to it. This allocation (or weighting) of capacity is a defining property of attention, also modelled in TVA, but outside the scope of the present article.

The TVA model is formulated in mathematical terms, which makes it possible to derive exact predictions for the results of specific experimental situations. Bundesen (1990) used this quantitative precision to model a wide range of classical findings in the literature on normal visual attention. The empirical account by Bundesen covered such different experimental paradigms as whole report, partial report, cued detection, single stimulus recognition, and visual search. Recently it has been shown that the TVA model can also explain a wide range of attentional effects at the single-cell level (Bundesen, Habekost & Kyllingsbæk, 2005). The *C* and *K* parameters of the TVA model can thus be used to explain many central effects of visual attention and seem to represent very fundamental aspects of this cognitive function.

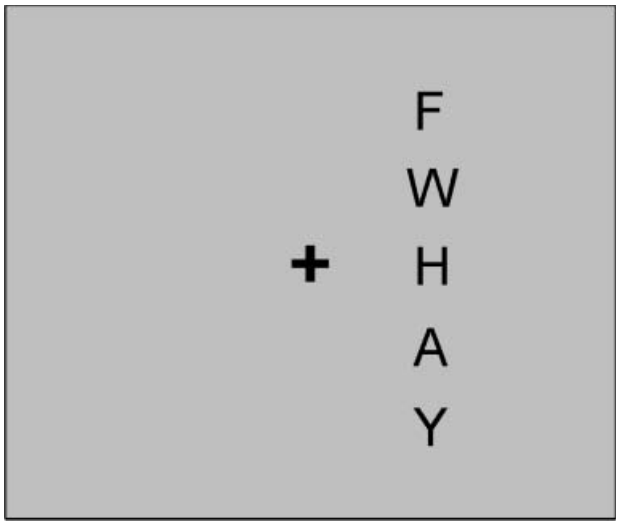

Fig. 3. Typical stimulus display used in TVA-based assessment.

The relation between visual span and processing speed

The two visual capacity parameters, *C* and *K*, are clearly separate: Limitations of visual speed are evident even in single-stimulus situations where visual span is irrelevant, and vice versa, the visual span can be demonstrated at long exposure durations where the limitation in processing speed does not constrain performance. Accordingly, *C* and *K* are defined and mathematically analyzed as independent parameters in the TVA model. However, as described in the present review, the two capacity parameters tend to co-vary empirically: In healthy individuals *C* and *K* values typically correlate about $r = 0.40$. In addition, many types of brain damage lead to deficits in both parameters. Thus, although *C* and *K* represent theoretically distinct capacity limitations, the empirical results point to some degree of relation between them. To anticipate one of the main conclusions of this paper, *C* and *K* probably depend on largely overlapping brain networks, which are often damaged simultaneously. Certain results point to brain areas dedicated specifically to *C* or *K*, but clear evidence on this issue is still lacking.

TVA-BASED ASSESSMENT OF ATTENTIONAL CAPACITY

The basic method

As mentioned, TVA can be used to analyze many different attentional paradigms, but the whole report task is the most direct way to estimate the visual processing speed, *C*, and the visual span, *K*. The exact details of the whole report procedure vary from study to study, but the basic design is this: An array of simple visual objects (typically five, but in some cases only one) is displayed on a computer screen; see Fig. 3 for an example. The stimuli of choice are typically letters, but digits and faces have also been used in some studies (e.g., Peers, Ludwig, Rorden *et al*., 2005; Starrfelt *et al*., 2008a,b). The presentation of the items is so brief that eye movements are prohibited (i.e., at most 200 ms) and the stimulus display is followed by either a set of pattern masks or a blank screen. If the blank screen is used an afterimage of the display persists for some time in the visual system (the "iconic memory" phenomenon, see Sperling, 1960), which prolongs the effective exposure duration. The task is to verbally report the identity of as many items as possible without guessing. For the best measurement of the TVA parameters, the exposure duration should be varied to cover the full range from the individual's perception threshold to near-ceiling performance. Also, to obtain reliable estimates each display condition must be repeated many times. The number of repetitions per condition depends on the purpose of the assessment (e.g., clinical or research) but is typically between 25 and 100; however, as few as 10 repetitions per condition may produce reasonably valid data (see Finke, Bublak, Krummenacher, Kyllingsbaek, Muller & Schneider, 2005, for a discussion).

Given this experimental procedure, the performance (mean number of items correctly reported) develops characteristically as a function of the exposure duration (see Fig. 4). When the exposure time is shorter than the tested person's perception threshold, t_0, the score is zero. After the threshold has been reached, the score rises abruptly. The slope of the curve at the point where the exposure time equals t_0 corresponds to the individual's visual processing speed, *C*. Over the course of a few hundred milliseconds the performance curve gradually levels off to approach an asymptotic value representing the maximum storage capacity of visual short-term memory, *K*. When testing subjects with low visual processing speed, unmasked displays can be used to prolong the effective exposure duration (due to the visual afterimage) for better estimation of *K*. This prolongation can be assumed constant across exposure durations and is denoted μ. This parameter is useful for curve fitting, but has not received much independent interest. In single item displays the *C* parameter is estimated in the same way as described above;

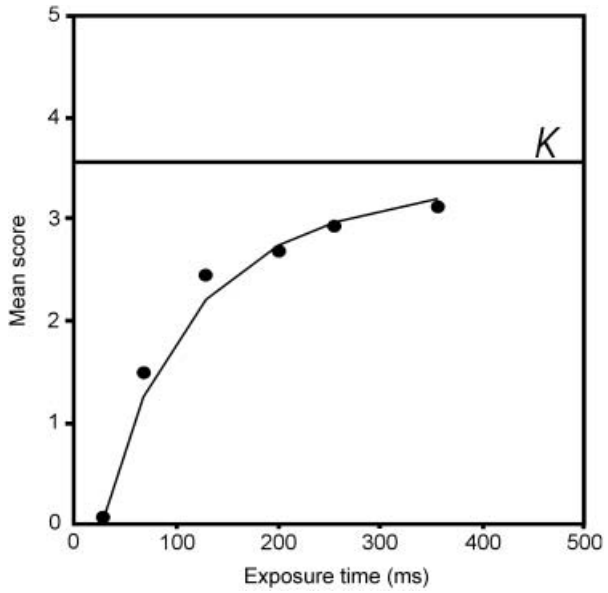

Fig. 4. Typical individual pattern of performance in an experiment where five stimuli (letters) must be reported. The mean number of correct reports (the score) is shown as a function of exposure time. Solid curves represent maximum likelihood fits to the observations based on TVA analysis. Below the visual threshold, t_0, the score is zero. The slope of the curve at the intercept with the *x*-axis equals the sum of the visual encoding rates for each of the five stimuli, *C*. The estimate of VSTM capacity, *K*, is marked by a horizontal line.

the curve levels off at the exposure duration where the subject can invariably perceive the stimulus (100% correct, see Fig. 1) and *K* estimation is not relevant in this paradigm.

Whole report experiments typically provide a large set of observations (e.g., 360 trials) for each participant. Given a psychometric model (such as TVA) of the probability distributions underlying these test data, the unknown parameters of the distributions (e.g., the individual's *K* and *C* values) can be inferred. The standard mathematical procedure for this estimation is the maximum likelihood method. Basically this is a computational algorithm that searches the parameter space for the set of values that maximizes the probability of obtaining the actual set of observations. Customized software for estimation of TVA parameters from whole report data has been developed (Kyllingsbæk, 2006) and can be downloaded from the internet (http://cvc.psy.ku.dk/). While the analysis itself is relatively easy to carry out, there are certain pitfalls for interpreting the results and the procedure should be supervised by a person who is familiar with TVA modelling (for a discussion of TVA-based data fitting, see Habekost, 2005).

Test qualities

A main quality of the TVA-based test method is its *specificity*. Because the estimates are delivered by mathematically independent fitting procedures (given an experimental design that produces adequate data), the *C* and *K* parameters are measured separately. TVA-based assessment is currently the only way to achieve this double estimation. Further, unlike most other cognitive and neuropsychological tests, TVA-based measurement is not significantly confounded by motor factors: Report is unspeeded and accuracy is the only important test variable. This implies that the measurements are directly specific to processes in the visual system. Another important quality of the method is *sensitivity*. By using near-threshold stimulation, the whole report task is so demanding that subtle impairments of visual function that are not evident on standard clinical tests have repeatedly been demonstrated in brain damaged participants (Habekost & Bundesen, 2003; Habekost & Rostrup, 2006; Habekost & Starrfelt, 2006; as explained in the introductory section on "the relation between visual span and processing speed"). A third test quality is *reliability*: TVA-based measurements of *C* and *K* generally have low measurement error (as measured by bootstrap statistics, see Efron & Tibshirani, 1993; Habekost & Bundesen, 2003). For example, in a typical whole report design with 25 repetitions per experimental condition used by Habekost and Rostrup (2007), the standard measurement error related to the *C* and *K* estimates were only about 10% and 3%, respectively. Finally, TVA-based assessment has high *validity* by being grounded in a theoretical framework that accounts for very large parts of the cognitive psychological literature on visual attention. Thus, the parameters relate directly to general theoretical constructs, rather than to particular aspects of the (whole report) test situation.

Normal variability

The TVA parameters represent separate aspects of visual processing and the model does not specify any dependence between their values; they are estimated individually from the data. However, there is evidence that the visual capacity parameters correlate to some extent. Finke *et al*. (2005) examined this issue in 38 healthy participants and found that *K* and *C* were moderately correlated ($r = 0.40$). Similar positive correlations between *K* and *C* have been found in the normal control groups of other TVA-based studies (e.g., Habekost & Rostrup, 2006). Thus, individuals tend to score relatively high or low on both capacity variables, which suggests that *K* and *C* to some extent depend on the efficiency of the same cognitive processes or brain areas. An interesting perspective on this issue comes from preliminary results in our lab, which suggest that both *C* and *K* scores are significantly correlated to IQ (Habekost, Starrfelt & Karstoft, 2008a; Habekost, Vogel, Rostrup, Bundesen, Kyllingsbaek & Waldemar, 2008b). Though more research is needed, one may speculate that the TVA capacity parameters are in some way related to the general efficiency of information processing in the brain.

Besides differences in general intelligence, another important source of individual variability is the participant's age. About 120 control participants have been tested in relation to the TVA-based patient studies reviewed in the next section. These individuals span a large age interval from about 20 to 70 years. The results of the studies are often not perfectly comparable because

different whole report designs and stimuli have been used, but an overall tendency towards reduced visual capacity with age seems evident. For example, a visual span above four items seems to be fairly common for people in their 20s (Habekost *et al*., 2008a), but occurs more sporadically when people are in their 50s and 60s. However, the currently available data does not allow for precise conclusions on the influence of age and more systematic investigations of visual attention capacity across the life span are needed. In the first study of this sort, which specifically targeted the development of visual capacity in old age, Habekost *et al*. (2008b) showed clear reductions of *C* and *K* as age progressed from about 70 to 85 years. The reductions were especially evident for visual processing speed, whereas marked reductions of the visual span occurred more sporadically and mainly for persons around 80 years or more. The decline in visual capacity was only weakly related to changes in the white matter or cerebral atrophy, so Habekost *et al.* suggested that other age-related processes, including biochemical changes, should be examined in future studies.

Practical issues

Various practical issues regarding testing are also important to consider when using the methods discussed here. In general, testing should be performed under constant lighting conditions and preferably in a semi-darkened room. To control exposure duration at the level of milliseconds, CRT- rather than LCD-screens (flat screens) should be used, as the physical refresh rate of LCD-screens is difficult to control. Because processing speed is different in central compared to peripheral vision, central fixation should be controlled either by direct observation, video recordings, or equipment for eye movement detection. In some instances forced choice responses may be preferred, but in most studies participants are instructed to respond only when "fairly certain" of their response and to refrain from guessing. The TVA modelling procedure can control for the influence of guessing to a large degree, but it is still desirable to keep this factor as constant as possible between subjects. It is not advisable to instruct subjects to respond only when "absolutely certain", as estimates of both the t_0 and the *C* parameters depend on processing at the lower limits of conscious perception. Thus, some degree of qualified guessing should be tolerated and even encouraged.

With regards to patient studies (including clinical investigations of single patients based on TVA) some further issues are worth considering. First, TVA testing is rarely applicable for assessment of patients in the acute phase, and it is by no means a bedside test. Rather, to obtain individually reliable TVA estimates based on whole report, at least 20–30 minutes of testing in appropriate surroundings (as described before) is usually necessary. Also, as the whole report paradigm relies on verbal report of the stimuli, aphasic patients can usually not be tested (though this problem may to some degree be resolved by using change detection paradigms). Frequent short breaks between testing blocks may be used when testing patients with fatigue, and in general the testing tempo should be driven by the patient. This again may increase the total time needed for testing. Overall, TVA-based assessment is clinically applicable, but should be considered a specialized testing procedure to be used for investigating specific questions regarding patients' visual attentional functions.

TVA-BASED PATIENT STUDIES

TVA-based assessment has already been used to study many different neuropsychological conditions: alexia, Alzheimer's disease, cerebral aging, Huntington's disease, neglect, simultanagnosia, and stroke in various parts of the brain. The list is growing each year, and ongoing work is currently using the method to describe ADHD and developmental dyslexia. Each study has provided evidence on a particular type of neurological disease or cognitive disturbance, but in this review we try to extract some more general conclusions from the research field. Most of the TVA-based patient studies concern damage in relatively circumscribed parts of the brain and therefore bear on the question of the anatomical basis for visual attention capacity. Lesions in three general brain regions have been repeatedly studied: (1) the parietal cortex, (2) the basal ganglia and overlying frontal cortex, and (3) the extrastriate cortex. In the following we summarize and discuss the findings from each anatomical area.

Parietal lesions

In the pioneer study that opened up the field of TVA-based assessment, Duncan *et al*. (1999) investigated nine patients with visual neglect following stroke in the right hemisphere. The strokes centred on the right parietal cortex, although adjacent regions were typically also affected to some extent. The patients were tested by a whole report task in which five letters were briefly flashed for variable exposure durations, either to the left or right of fixation. This way the *C* and *K* parameters could be estimated separately in each visual field. Compared to an age-matched control group the patients had significant reductions in the visual span, *K*, in both sides. They also had bilateral reductions of the visual processing speed, *C*, especially pronounced in the left hemifield. Given that visual neglect is traditionally considered a unilateral disturbance the bilateral reduction of the patients' capacity deficits was surprising, but later studies have confirmed that neglect is often associated with generalized impairments of attention (Husain & Rorden, 2003). Also, a TVA-based study by Bublak *et al.* (2005) reported a similar pattern of general visual capacity deficits in a single patient with right parietal damage. Bublak *et al*. used the same experimental design as Duncan *et al*., but fewer total trials to show that the TVA-based test method was clinically practical.

Duncan *et al*. (2003) used TVA-based assessment to study the attentional capacity of a patient with Balint's syndrome, or dorsal simultanagnosia, following bilateral parietal lesions, as well as a patient with ventral simultanagnosia (this second patient is discussed in the section 'Extrastriate lesions'). Two versions of whole report with variable exposure times were used, featuring

single and multiple stimuli, respectively. Contrary to the common notion that patients with simultanagnosia can only process one stimulus at a time, Duncan *et al.* found that the patient was quite consistently able to report two items from a multiple-item display. However, the patient's visual processing speed was severely reduced: The *C* value was about ten times smaller than that of the healthy controls, both when tested with single and multiple stimuli.

Peers *et al.* (2005) studied 13 patients with focal brain damage in the parietal cortex. Each patient's lesion was confined to either the left or the right hemisphere. Because no significant relation was found between lesion side and test performance, the data of the patients were pooled into one group. Peers *et al*.'s first set of experiments estimated parameter *C* by showing a single, masked stimulus at fixation for variable exposure durations. Compared to a matched control group the group of parietal patients had significantly reduced *C* values for both letter and face stimuli. Statistical analysis of the structural MRI scans indicated that lesions that were located relatively inferiorly in the parietal lobe were significantly associated with lower *C* values. Peers *et al*. concluded that the *C* deficits were probably related to damage around the temporoparietal junction, but could also be explained by lesions in the underlying white matter. Peers *et al.* went on to study the patients' visual span in a whole report experiment where six letters, three in each hemifield, were flashed for a fixed, relatively long exposure duration (200 ms followed by a blank screen). The group of parietal patients had significantly lower *K* values than the controls, and the statistical MRI analysis pointed to the same region in the inferior parietal lobe that seemed to be responsible for the *C* deficits.

In 19 patients with mild cognitive impairment (MCI) and nine patients with a diagnosis of probable Alzheimer's disease, Bublak *et al.* (2006) found general reductions of visual processing speed. The study did not include brain imaging and the anatomical background for the observed deficits is therefore unclear, but Alzheimer's disease in its initial stages is known to cause disturbed metabolism in the temporoparietal cortex (Jagust, Eberling, Reed, Mathis & Budinger, 1997). Bublak *et al*.'s findings on early Alzheimer's disease thus seem compatible with the results of Peers *et al.* (2005).

In conclusion, TVA-based patient studies have consistently found reductions of both visual processing speed and visual span following lesions in parietal cortex. The reductions often affect bilateral visual processing as well as the central visual field, even when the lesion is confined to one hemisphere, which suggests a general role of the parietal cortex for visual attention capacity. Lesions in the right hemisphere have been most studied, but the results of Peers *et al.* (2005) indicate that the left parietal cortex is also important. Indeed, bilateral parietal lesions can lead to very severe capacity reductions (Duncan *et al*., 2003). It is less clear which subregions in the parietal lobe are critical, and whether they differ for the two capacity variables. Only one of the mentioned studies (Peers *et al.*, 2005) conducted a systematic lesion analysis and due to the modest number of patients included, the conclusions were not very specific. Besides the importance of the temporoparietal region suggested by Peers *et al*., the results are compatible with the hypothesis that the white matter underlying the parietal cortex is critical for visual attention capacity. This interpretation is supported by another TVA-based study featuring systematic lesion analysis (Habekost & Rostrup, 2007), which is described in the next section. Another theoretically relevant area is the intraparietal sulcus, which has been linked to VSTM capacity in recent fMRI studies (Todd & Marois, 2004; Xu & Chun, 2006). However, as very few of the patients studied by TVA-based assessment so far have had lesions involving the intraparietal sulcus, reliable conclusions on this issue await future studies.

Frontal and basal ganglia lesions

Habekost and Bundesen (2003) provided the first demonstration of a deficit in parameter *K* following a lesion outside parietal cortex. They studied a patient with stroke in the right basal ganglia and overlying frontal cortex. The patient did not have neglect or other attentional disturbances as measured by standard clinical tests, but visual attention impairment was clearly evident in the TVA-based assessment. This way, the study of Habekost and Bundesen was an early demonstration of the sensitivity of the new test method. The same whole report procedure as in the study of Duncan *et al.* (1999) was used, and the results showed that the patient's visual span was reduced to about two items in both visual fields, markedly below the mean score of an age-matched control group. The patient's visual processing speed was also lower than normal in both visual fields, but the reduction from the control group mean on this parameter was only moderate.

The importance of the basal ganglia and frontal cortex for the capacity of visual attention was further explored in a large study by Habekost and Rostrup (2006, 2007), who investigated 26 patients with strokes affecting the right hemisphere. The strokes varied widely in size, but in most cases centred on the basal ganglia and the overlying frontal cortex; parts of the parietal and temporal cortices were also affected in some patients. A subgroup of four patients had focal lesions in the thalamus. This study used a standard whole report task, where five letters were presented randomly either in the left or right visual field. The results showed that reductions in visual processing speed for left-side stimuli were very common in this patient group (Habekost & Rostrup, 2006). As in the study of Habekost and Bundesen (2003) the patients typically had minor or no deficits on clinical tests of visual attention, so the high sensitivity of TVA-based assessment was confirmed. Statistical analysis of the patients' MRI scans showed that the frequently observed deficits in left-side visual processing speed were unrelated to lesion size, but instead depended on damage to the putamen nucleus of the basal ganglia. Surprisingly, most patients did not have reductions in visual span or processing speed for stimuli presented in the right hemifield, even after large strokes (Habekost & Rostrup, 2007). Habekost and Rostrup concluded that a large anterior part of the right hemisphere, including parts of the basal ganglia,

inferior frontal cortex, and insula, is *not* critical for these two aspects of visual attention capacity. General capacity deficits were found in the study, but only in patients where large parts of the cerebral white matter were damaged or after lesions that included relatively superior parts of the frontal lobe. The latter type of lesion was significantly related to bilateral reductions in visual processing speed.

The study by Peers *et al*. (2005) described in the previous section also included a group of 12 patients with focal damage in either the left or right frontal cortex. As before, the data of the patients were pooled into one group, because no significant relation was found between lesion side and test performance. Contrary to the parietal group, Peers *et al*.'s group of frontal patients did not differ significantly from the control group in terms of *C* or *K*.

Finke *et al.* (2006) studied 18 patients with Huntington's disease. Huntington's disease is a genetically based degenerative disease characterized by gradual degeneration of neurons in the basal ganglia, eventually disturbing cortical functions and leading to general dementia. Finke *et al*. used the same whole report procedure as Duncan *et al.* (1999) to show strong, bilateral reductions of both visual processing speed and visual span in the patient group. Further the severity of the deficits in *C* and *K* correlated significantly with the number of years since disease onset, and the TVA measures could therefore be viewed as cognitive markers for the progression of the disease. In a follow-up study of the same Huntington's patients, Finke *et al*. (2007) showed that the reductions in *C*, but not *K*, correlated with symptoms of simultanagnosia as measured by perception of overlapping figures, which parallels the findings in the case study by Duncan *et al*. (2003).

Unlike studies of the parietal cortex, findings of capacity deficits after damage in the basal ganglia and frontal cortex have been mixed. When damaged unilaterally, a substantial part of this region is probably not critical for the general capacity of visual attention ("general capacity" being defined as bilateral and/or foveal visual function). Even large unilateral lesions in the basal ganglia and inferior frontal lobe can leave general visual capacity intact (Habekost & Rostrup, 2007; Peers *et al*., 2005). There is, however, evidence that unilateral lesions in more superior parts of the frontal cortex can affect general visual capacity. Habekost and Rostrup (2007), controlling statistically for the influence of basal ganglia and inferior frontal damage, found a significant relation between general reductions of visual processing speed and damage in the right middle frontal gyrus. Reanalyzing lesions of individual patients in the study of Peers *et al*., Habekost and Rostrup also noted that patients with damage to the right middle frontal gyrus had the lowest *C* values in their group.

Finke *et al*. (2006) found strong general capacity reductions in Huntington's patients, but no imaging data were available for this study, so the anatomical significance of the result is hard to evaluate. Huntington's patients have bilateral damage in the basal ganglia, but also cortical dysfunction in later stages of the disease, where the strongest deficits were found. The cortical disturbance may explain the general reductions of capacity, but alternatively the results may be due to the fact that the patients' damage to the basal ganglia was bilateral. There is strong evidence that unilateral lesions in the basal ganglia lead to capacity reductions only in the contralesional visual field. A very consistent effect of right basal ganglia lesions, specifically in the putamen, is a slight but significant reduction of visual processing speed in the left visual field (Habekost & Rostrup, 2006). This effect might have been duplicated for both visual fields in Finke *et al*.'s Huntington patients.

Extrastriate lesions

Basic visual processes are of course critical for visual capacity and lesions to extrastriate areas may affect the representations of the stimuli to be perceived rather directly, by disturbing either sensory or early perceptual processes. Both visual field defects and impairments in shape perception may affect visual capacity, and deficits following extrastriate lesions may be selective for regions of the visual field or even for particular stimulus types. Visual field deficits may also require that testing is done in the preserved visual field only.

Duncan *et al*. (2003) were the first to investigate visual attentional deficits in a patient with a lesion in ventral visual areas in the left hemisphere. The patient's performance was compared to that of a patient with bilateral parietal lesions (see the section above on "Parietal lesions"). Duncan *et al.* found that the performance of the two patients was surprisingly similar: They both had reductions in *K*, but were consistently able to report more than one item, while their processing speeds were severely reduced. Thus their simultanagnosic deficits were interpreted as related primarily to reduced *C* rather than *K*. However, it should be noted that the patient with the ventral extrastriate lesion had *pure alexia* (also referred to as ventral simultanagnosia), a reading disorder characterized by slow but accurate word reading. As his letter identification skills were not perfect, the pattern of performance in this patient may reflect degraded visual representations of the particular stimuli used (namely, letters) rather than a general deficit in visual attentional capacity.

A complementary pattern of performance was reported in a patient with integrative visual agnosia (HE; Gerlach, Marstrand, Habekost & Gade, 2005), who had reduced visual span while her processing speed (as measured in the right visual field only) and perceptual threshold were normal. HE had a large lesion in ventral visual areas of the right hemisphere, including the middle and lateral occipito-temporal gyrus, and the inferior temporal gyrus. In addition, she had a clinically silent infarct of the right inferior parietal lobe, extending into the insula. Whereas HE's scores were normal with respect to visual processing speed, her performance was qualitatively different from controls. Controls spread their attentional resources over multiple stimulus positions, but HE mainly focused her attention on a single stimulus position.

Studying a patient with a subtle reading deficit following a lesion to extrastriate areas in the left hemisphere, Habekost and

Starrfelt (2006) used TVA-based methodology to reveal that the patient's deficit did not reflect a general reduction in visual capacity, but rather impaired shape perception (cerebral amblyopia) in parts of the visual field. The patient's processing speed and visual span were within the normal range both in central vision and in the left visual field, while his processing speed was severely reduced in the upper part of the right visual field. This sensory deficit had detrimental effects on reading, as fluent reading relies on fast and efficient letter identification and discrimination in this part of the visual field (Zihl, 1995).

Investigating visual processing deficits following occipito-temporal lesions in the left hemisphere in patients with pure alexia, Starrfelt *et al*. (2008a, 2008b) extended the standard whole report task by comparing patients' performance with two different stimulus types: letters and digits. In addition, processing speed was measured both for singly presented central stimuli and in the traditional five-item paradigm with peripheral presentation. One main finding emerging from these studies was that visual processing speed for single items presented centrally may be reduced while peripheral processing speed is within the normal range. This finding is important, as conclusions of normal processing speed have previously been made based on the basis of peripheral measurements only (e.g., Gerlach *et al*., 2005). In the five pure alexic patients reported by Starrfelt *et al*., central processing speed was consistently reduced for singly presented letters and digits, while reductions in peripheral processing speed were not consistently found. In addition, whereas central processing speed was reduced for both stimulus types in all patients, some patients showed an abnormal ratio between their central processing speeds for letters and digits (such that digits were perceived significantly faster). This suggests that, at least when assessing patients with extrastriate lesions, different stimulus types should be employed to fully characterize their visual attentional capacity. In addition to their reduced central processing speed the five pure alexic patients of Starrfelt *et al*. (2008a, 2008b), as well as the pure alexic patient reported by Duncan *et al*. (2003), had reduced visual apprehension span, indicating that ventral visual areas in the left hemisphere are of importance for visual span as well as processing speed.

As mentioned previously, visual field defects can prevent testing in both visual fields, which is often the case in patients with extrastriate lesions in whom the optic radiations or striate cortex are commonly also affected. One of the patients reported by Starrfelt *et al*. (2008b) had pure alexia without a field cut, and his performance could therefore be measured in both visual fields. His performance in the whole report experiment was quite similar in both sides with respect to both processing speed and visual apprehension span, suggesting that measurements based on left sided presentation only may be taken to represent the general visual processing capacity in patients with left-side extrastriate lesions.

In summary, deficits in both visual processing speed and visual apprehension span may be observed after extrastriate lesions. There is some indication that deficits may be restricted to either *K* or *C* in individual patients, but the evidence for such selectivity is sparse. In most patients, both parameters are reduced. However, other kinds of selective deficits may be observed: Deficits can be selective for regions of the visual field, for instance a single quadrant (Habekost & Starrfelt, 2006), or disproportionately affect processing speed in the centre of the visual field (Starrfelt *et al*., 2008a). Deficits may also differentially affect different stimulus types like letters and digits, at least to some degree (Starrfelt *et al*., 2008a, 2008b). TVA-based studies of visual capacity following extrastriate lesions have so far included only single (or multiple) case studies, mostly of patients with left hemisphere lesions. Larger group studies like those performed with frontal and parietal patients could greatly improve our understanding of the basic visual processes involved.

The lesion anatomy of visual attention capacity

Taken together, the TVA-based patient studies show that a normal level of visual attention capacity depends on intact function in many different parts of the brain, including several regions outside the visual system as traditionally defined. In the simplest case, damage in the primary visual pathways leads to blindness in parts of the visual field, which corresponds to a visual capacity of zero. In all other cases, reductions of visual capacity are relative rather than absolute: Conscious perception is still possible, but visual information is processed less efficiently than normal. Damage to extrastriate cortical areas typically degrades, but does not annihilate, the basic sensory representations of visual stimuli, which can lead to marked reductions of both the *C* and *K* parameter.

Lesions in parietal cortex, also unilateral ones, typically lead to generalized reductions in both the *C* and *K* parameter, and damage to certain parts of the frontal lobe and the basal ganglia seem to have similar effects. In our view the most plausible explanation for these findings is that fronto-parietal networks, in combination with the putamen and possibly other parts of the basal ganglia, are necessary to make activations in visual cortex reach consciousness (see, e.g., Dehaene, Sergent & Changeux, 2003; Rees, Wojciulik, Clarke, Husain, Frith & Driver, 2000). While patients with focal lesions in these areas (or in the white matter tissue connecting them; Habekost & Rostrup, 2007) do not completely lose the ability to perceive visual stimuli, the efficiency of the process is generally impaired. This inefficiency should be reflected both in visual processing speed, *C*, and the visual span, *K*, as found in many TVA-based patient studies. Generally speaking, effective perception of visual stimuli seems to require the coordinated activity of a very large brain network, which spans from sensory areas in the posterior cortex (and earlier in the visual system) to high-level regions in the parietal and frontal lobes as well as structures in the basal ganglia. This anatomical model is consistent with the recent neural interpretation of the TVA equations (Bundesen, Habekost & Kyllingsbæk, 2005), which also claims that processing of visual information is based on activity in these widely distributed brain structures. For instance, the strength of sensory evidence for particular visual categorizations (and hence, the

visual processing speed) should depend strongly on extrastriate areas, whereas the visual short-term memory system (i.e., the visual span) should depend on long-range feedback connections between sensory areas and the thalamus or frontal cortex.

By now, this general anatomical model of visual attention capacity has been firmly supported by TVA-based patient studies. However, a more detailed characterization of the brain systems that underlie visual attention capacity is still lacking. For example, it is unclear which parts of the parietal cortex are critical, or whether there are subtle differences depending on the side of the lesion. The anatomical resolution of the existing patient studies is not high enough to give precise answers to such questions. Brain damage typically affects several cortical and subcortical regions in a given individual, and it takes very large patient groups to statistically tease out the contribution of each theoretically relevant region (e.g., the intraparietal sulcus). Whereas a few TVA-based studies have begun to address lesion anatomy systematically (Habekost & Rostrup, 2006, 2007; Peers *et al*., 2005), more data are needed before detailed conclusions can be drawn. For example, one very specific hypothesis of the neural TVA model, that the feedback loop component of visual short-term memory depends on the reticular nucleus of the thalamus, has yet to be tested empirically.

The coarse anatomical resolution of the patient studies means that possible differences in the anatomical basis for the *C* and *K* parameters await further investigation. On the one hand, it is clear that visual speed and span draw on overlapping brain structures to a large extent, especially since both variables depend on the basic quality of sensory representations of the stimuli. Also, lesions in parietal, frontal, or basal ganglia regions often lead to reductions in both *C* and *K*, although one of the parameters is typically more affected than the other. On the other hand, as explained in the section above on 'The relation between visual span and processing speed', the two variables are clearly functionally separable. *C* and *K* correlate only moderately in the normal population, which also suggests that they rely on different processes to some extent. Given the present evidence it seems plausible that *C* and *K* depend on networks that are only partly overlapping. For example, the intraparietal sulcus may be particularly critical for *K* (as suggested by recent fMRI studies; Todd & Marois, 2004; Xu & Chun, 2006), whereas the putamen and the middle frontal gyrus may relate more strongly to visual processing speed (Habekost & Rostrup, 2006, 2007). Future studies of patients with very focal lesions in these areas, as well as studies of normal subjects using Transcranial Magnetic Stimulation (TMS), should prove very interesting.

CONCLUSION

We have presented a new line of research that applies the conceptual framework of Bundesen's Theory of Visual Attention (TVA) to quantify attentional capacity in the visual modality. Within this framework two distinct limitations of visual capacity, visual span and visual processing speed, can be measured independently based on whole report experiments and mathematical data modelling. In addition to this specificity, the method provides high sensitivity: Using this method, deficits in visual capacity have repeatedly been demonstrated in patients who showed no deficits on standard clinical tests. The method also has high reliability and validity. The number of studies using TVA for investigating deficits in visual capacity after brain damage is steadily growing. The effects of lesions in three general brain regions on visual capacity have been repeatedly studied: the parietal lobes, the frontal lobes and basal ganglia, and the extrastriate areas. We have reviewed these studies based on anatomical lesion location. In most of the reported patients with deficits in visual capacity, both parameters (speed and span) are affected. Thus, while the two visual capacity parameters are functionally separable, they seem to rely on cerebral networks that overlap to a large extent and are commonly damaged together. Future studies should address the degree to which either capacity parameter can be affected in isolation. Also, many regions of the brain (e.g., extrastriate areas in the right hemisphere) as well as many neurological conditions known to have attentional symptoms (e.g., Parkinson's disease) have not yet been studied using TVA-based methods. Thus there is ample room for future studies to inform anatomical models of visual capacity as well as further the theoretical understanding of visual attentional capacity.

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