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Visual masking & schizophrenia

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ABSTRACT

Visual masking is a frequently used tool in schizophrenia research. Visual masking has a very high sensitivity and specificity and masking paradigms have been proven to be endophenotypes. Whereas masking is a powerful technique to study schizophrenia, the underlying mechanisms are discussed controversially. For example, for more than 25 years, masking deficits of schizophrenia patients were mainly attributed to a deficient magno-cellular system (M-system). Here, we show that there is very little evidence that masking deficits are magno-cellular deficits. We will discuss the magno-cellular and other approaches in detail and highlight their pros and cons.

1. Introduction

The obvious symptoms of schizophrenia are personality and thought disorders. However, already Kraepelin (1893) noted that visual information processing is strongly deteriorated in schizophrenia too (called dementia praecox). For this reason, sensory deficits were often even proposed to be the primary causes of schizophrenia (e.g., Braff, 1981; McGhie and Chapman, 1961; Saccuzzo and Braff, 1981, 1986; Saccuzzo et al., 1974; Schwartz and Winstead, 1982; Slaghuis, 1998; Venables, 1964; Yates, 1966).

One of the most popular paradigms to investigate visual information processing is visual backward masking (monograph: Breitmeyer and Öğmen, 2006). In visual backward masking, a target is followed by a mask which deteriorates the visibility of the target and, hence, performance (Fig. 1A). Bachmann (1994; p. 11) estimated that masking is used as a tool in 14% of all articles in vision research and psychology. Enns and Di Lollo (2000) came to a similar conclusion.

Visual masking is a powerful tool in schizophrenia research. First, schizophrenia patients show clear and reproducible performance deficits compared to healthy controls in all studies on visual masking (except Luber et al., 2007). Second, masking deficits are potential endophenotypes of schizophrenia (e.g. Bredgaard and Glenthøj, 2000; Chkonia et al., 2010; Green and Nuechterlein, 1999; Keri et al., 2001a; Nuechterlein et al., 1994; Rund et al., 1993), i.e., deficits are stable over time (Chkonia et al., 2010; Lee et al., 2008; Rund et al., 1993), relatively independent of medication (e.g. Butler et al., 1996, 2002; medication improves performance: Brody et al., 1980; Butler et al., 1996; a trend for deterioration: Cadenhead et al., 1997), present in adolescents with psychosis, i.e., right from the beginning of the disease (Holzer et al., 2009; Perez et al., 2012; Rund et al., 1996; Saccuzzo and Schubert, 1981; Ueland et al., 2004; but see Lieb et al., 1996), present in healthy students scoring high on schizotypy (Cappe et al., 2012; Merritt et al., 1986) and, most importantly, present in first order relatives of patients (Chkonia et al., 2010; Green et al., 1997, 2006; Keri et al., 2001a). Indeed, genetic correlates have been reported (Bakanidze et al., 2013; Goghari and Sponheim, 2008). Third, masking deficits are not contaminated by differential aging effects (Green et al., 2003a), unaffected by learning (Rassovsky et al., 2004; Suslow and Arolt, 1998), independent of cognitive deficits such as working memory (Keri et al., 2001b), premorbid IQ, fluid IQ and intellectual decline (Koelkebeck et al., 2005), and personality aspects (Bogren and Bogren, 1999), and only slightly modulated by cognitive/emotional aspects such as reward (Rassovsky et al., 2005b). However, there is a correlation with social perception (Sergi and Green, 2002). Fourth, masking paradigms have a better specificity and sensitivity than cognitive tests such as the CPT (Chkonia et al., 2010).

Masking is sensitive to psychopathology showing strongest masking deficits for negative symptoms patients (Green and Walker, 1984, 1986; Slaghuis, 1998, 2004; Slaghuis and Curran, 1999; Weiner et al., 1990) and chronic schizophrenia patients (Rund, 1993).[1]

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[1] However, empirical evidence is mixed, possibly, depending on the method of assessing psychopathology (e.g. Chkonia et al., 2010; Rund et al., 2004).

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Here, we will show that masking is a powerful experimental tool for schizophrenia research but explanations about the underlying mechanisms need to be handled with care.

1.1. Definitions

In visual masking, a target is either preceded (forward masking) or followed by a mask (backward masking; Fig. 1A). If a mask does not spatially overlap with the target, it is called a paracontrast (in forward masking) or metacontrast (in backward masking) mask (Fig. 1C). If the mask spatially overlaps with the target, the mask is called a pattern mask (Fig. 1A).

In masking experiments usually the onset of the target versus the mask is varied. This onset difference is called the stimulus-onset-asynchrony (SOA). If strongest masking occurs for a simultaneous presentation of target and mask (SOA=0 ms), masking is said to be of A-type (not shown in Fig. 1). Interestingly, for some target–mask combinations, strongest *backward* masking occurs for SOAs greater than 0 ms. The non-monotonic masking function is called to be of B-type (Fig. 1B). Often the terms, integration and interruption masking are used synonymously for A- and B-type masking. However, these terms refer to potential mechanisms of masking and will not be used here. Often, instead of SOA, ISI is reported, which is the inter-stimulus interval between the target offset and mask onset.

An important issue in masking research is the energy ratio of target and mask. Stimulus energy is usually defined as stimulus luminance times duration (luminance is sometimes replaced by contrast). Often combinations of a high energy target with a low energy mask yield B-type masking (but see discussion). The spatial frequency of a masking grating (or Gabor) is the number of changes from black to white, usually determined in cycles per degree. Low spatial frequencies are in the range from 1 to 4 c/deg, high spatial frequencies from 8 to 16 c/deg.

1.2. M- vs. P-system

Physiologically, there are two major pathways in the visual brain, called the magno-cellular (M-) and the parvo-cellular (P-) system. The physiological characteristics of these systems are rather complementary. The M-system is particularly sensitive to high temporal frequencies such as abrupt onsets of stimuli and to low spatial frequencies. The M-system is "color blind". The P-system is sensitive to high spatial frequencies and color differences but it is less sensitive to high temporal frequencies. The M-system is often assumed to primarily process motion and localization information. The P-system is assumed to be related to the detailed processing of shape and color.

2. Mechanisms and models

2.1. Attention and iconic memory

Visual masking was introduced to schizophrenia research "as a measure of attention" (Saccuzzo et al., 1974). The basic idea is that a visual stimulus is stored in an iconic memory (Neisser, 1967). Targets are vulnerable to masking as long as they are in this buffer. Items are not erased when they are read out by attention into a more stable memory before the "mask arrives". In schizophrenia patients, this read out process is slower (or otherwise disturbed) and, hence, performance is more strongly deteriorated in visual masking (Braff, 1981; Braff and Saccuzzo, 1981, 1985; Merritt and Balogh, 1984; Patterson et al., 1986; Saccuzzo and Miller, 1977; Saccuzzo and Braff, 1981, 1986; Saccuzzo and Schubert, 1981; Saccuzzo et al., 1974, 1984; Schwartz et al., 1983; for

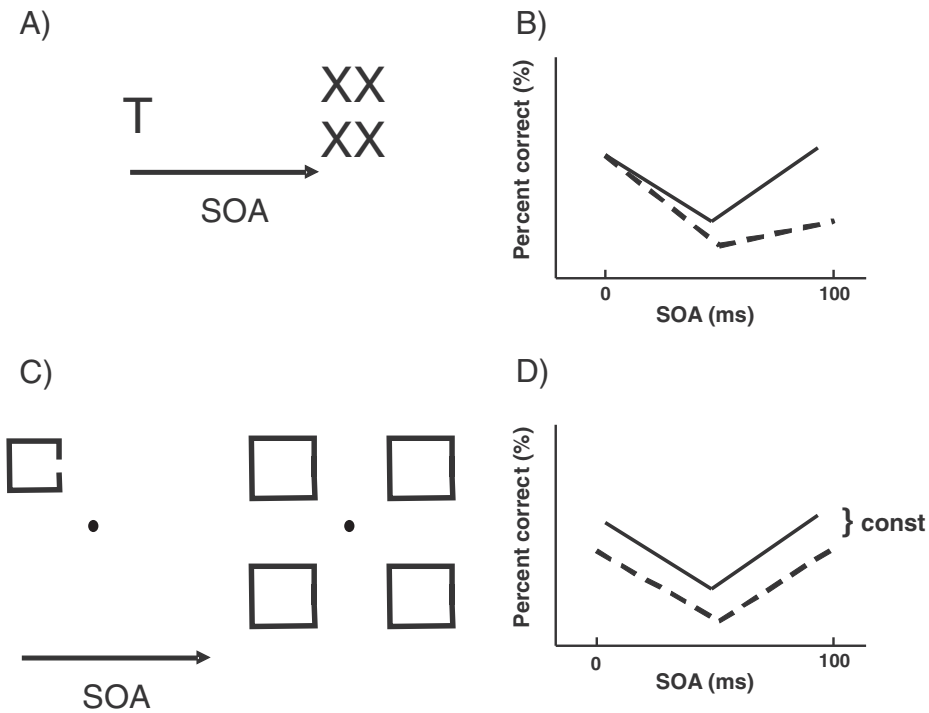

Fig. 1. A) Pattern masking. A target letter, e.g., a T, is followed by a mask, e.g., comprised of Xs after a variable SOA (backward masking). Pattern masks often produce A-type masking. **B)** B-type masking. Particularly, when the mask is a metacontrast mask or a pattern mask of weaker energy than the target, B-type masking occurs: good performance occurs for an SOA of 0 ms, i.e., simultaneous presentation of target and mask. Performance for medium SOAs, e.g., 50 ms, is worse than for shorter and longer SOAs. The solid line shows a typical B-type masking function for healthy controls, the dashed line a masking function for schizophrenia patients (e.g., Green et al., 1994a,1994b). B-type masking is stronger in the patients in accordance with a hypothetical hyper-active M-system of schizophrenia patients. **C)** Localization task. A target square is presented randomly at one out of four positions. After the square, four larger squares follow at all four potential target positions. Observers indicate the position of the target square. In addition, an identification task is performed where observers indicate which of the four sides of the target square contains a gap (here, a gap on the right side is shown). The masking squares are metacontrast masks because they do not spatially overlap with the target square. **D)** With these stimuli, B-type masking occurs in both patients and healthy controls. Performance of controls (solid line) is higher than for patients by a constant factor (dashed line) for *all* SOAs (e.g. Rassovsky et al., 2004).