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A comparison of masking by visual and transcranial magnetic stimulation: implications for the study of conscious and unconscious visual processing

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Abstract

Visual stimuli as well as transcranial magnetic stimulation (TMS) can be used: (1) to suppress the visibility of a target and (2) to recover the visibility of a target that has been suppressed by another mask. Both types of stimulation thus provide useful methods for studying the microgenesis of object perception. We first review evidence of similarities between the processes by which a TMS mask and a visual mask can either suppress the visibility of targets or recover such suppressed visibility. However, we then also point out a significant difference that has important implications for the study of the time course of unconscious and conscious visual information processing and for theoretical accounts of the processes involved. We present evidence and arguments showing: (a) that visual masking techniques, by revealing more detailed aspects of target masking and target recovery, support a theoretical approach to visual masking and visual perception that must take into account activities in two separate neural channels or processing streams and, as a corollary, (b) that at the current stage of methodological sophistication visual masks, by acting in more

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highly specifiable ways on these pathways, provide information about the microgenesis of form perception not available with TMS masks.

Keywords: Conscious and unconscious vision; Double dissociation; Target recovery; Target suppression; Transcranial magnetic stimulation; Visual masking

1. Introduction

Along with other visual scientists (e.g., Uttal, 1981), we define perception as the phenomenal registration or conscious awareness of sensory information. The temporally brief suppression of such conscious awareness by application of visual mask stimuli to the retinal surface or by application of a transcranial magnetic stimulation to the occipital pole, provide effective techniques for testing the relationship between brain events and conscious as well as unconscious information processing (Breitmeyer, Ogmen, & Chen, 2004; Breitmeyer, Ro, & Singhal, 2004; Dolan, 2002; Kamitani & Shimojo, 1999; Klotz & Wolff, 1995; Neumann & Klotz, 1994; Ohman, 2002; Wong & Root, 2003). Visual masking, a well-known phenomenon, refers to the partial to total suppression of the visibility of one briefly flashed stimulus, called the target, T, by a second brief stimulus, called the (primary) mask, M1. Henceforth, we refer to this reduction of target visibility by a primary mask as target suppression. Given that M1 produces target suppression, one can also introduce a secondary mask, M2, which by suppressing neural activity generated by M1, in turn produces a recovery of T's visibility. Henceforth, we refer to this phenomenon produced by M2 as target recovery. By noting how the visibility of the target changes during target suppression or during target recovery as one varies the time interval between the onsets (stimulus onset asynchrony or SOA) of T and M1 or of M1 and M2, one can infer the time course or microgenesis of visual object perception (Bachmann, 1994; Breitmeyer, 1984; Breitmeyer & Ogmen, 2000; Di Lollo, Enns, & Rensink, 2000; Francis, 1997; Herzog & Koch, 2001).

Although visual masking techniques have been around for over a century, more recently the new technique of transcranial magnetic stimulation (TMS) has also been used to study the microgenesis of object perception (Amassian et al., 1989, 1993; Corthout, Uttl, Walsh, Hallett, & Cowey, 1999; Corthout, Uttl, Ziemann, Cowey, & Hallett, 1999; Corthout, Uttl, Juan, Hallett, & Cowey, 2000; Corthout, Hallett, & Cowey, 2003; Kammer, Scharnowski, & Herzog, 2003). The rationale of the TMS-mask studies is basically similar to that use in visual-mask studies. The application of a brief TMS pulse disrupts neural activity in the neural structures of the underlying cortex. Specifically, a TMS pulse applied to the occipital pole briefly disrupts activity in retinotopically organized regions V1 or V2 of the visual cortex (Kamitani & Shimojo, 1999). By noting how target visibility changes as the SOA, either between T and the TMS pulse or between M1 and the TMS pulse, varies, important inferences can be drawn about the neural processes and their temporal properties underlying object perception. In both cases, we adopt the convention of using negative and positive SOA values to designate, respectively, the mask's onset preceding and following that of the target (see Fig. 1).

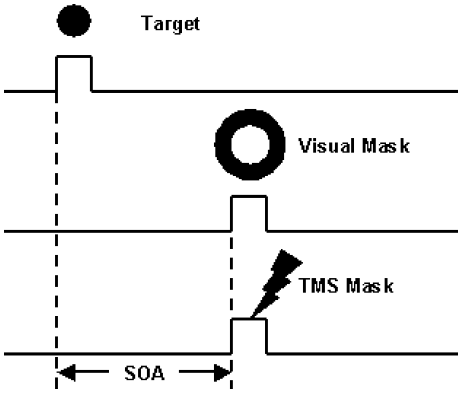

Fig. 1. Schematic diagram of a typical visual target consisting of a disk and a visual mask consisting of a surrounding ring or of a transcranial magnetic stimulus (TMS) pulse. The temporal sequence depicts target and masks with a positive stimulus onset asynchrony (SOA) separating the onsets of the target and the following masks.

2. Similarities between target suppression and target recovery by visual and TMS masking and their underlying mechanisms

2.1. Target suppression

Although there are a number of theoretical approaches to visual masking, they all agree on the major empirical regularities that define and distinguish forward masking, when the mask precedes the target, from backward masking, when the mask follows the target.[1] Particularly informative types of forward and backward masking are known as paracontrast and metacontrast, respectively. In these masking paradigms, as illustrated in Fig. 1, the target consists of, say, a disk, and the mask consists of a surrounding ring or annulus. Since the target and mask do not overlap spatially, the mask cannot retinotopically "overwrite" the information in the target-processing channels. One way of interpreting such results is that the mask reduces or eliminates the "signal" in the target-processing channels by a neural process of intra- or interchannel inhibition (Breitmeyer & Ogmen, 2000; Francis, 1997; Ogmen et al., 2003), while other theoretical interpretations rely on interactions either between fast, sensorily specific and slow, non-specific modulatory activities converging at cortical levels of processing (Bachmann,

[1] Many forms of visual masking exist and we cannot detail all of them (rev., Bachmann, 1994; Breitmeyer, 1984). However, one can broadly classify them as masking by integration, masking by interruption and masking by substitution. All three can be thought of as producing a reduction of the signal-to-noise ratio in the neural pathways responding to the target. In the first, typically obtained when target and mask overlap spatially, the neural response to the target and mask are integrated over short time intervals due to the limited temporal resolution of the visual system. This leads to an increase of noise in the target-responding pathways. In the second, due to neural inhibition produced by the mask, the signal in the target-responding pathways is actively suppressed. Integration can occur within magnocellular (M) and parvocellular (P) channels, as can inhibition (intrachannel inhibition). Additionally, M and P channels can inhibit each other (interchannel inhibition). For more details see Breitmeyer and Ogmen (2000). In the third, the neural representation of the mask replaces that of the target (Bachmann, 1994; Di Lollo et al., 2000).

1994) or among different levels of object representation (Di Lollo et al., 2000). Rather than proposing inhibitory effects of the mask activity on target activity, both of these theoretical approaches assume a selection or facilitation of the mask representation in place of that of the target. The depth of the suppression of the target's visibility in turn depends on the strength of the (inhibitory or other) process activated by the mask. Optimal masking strength leads to total loss of target visibility; somewhat less than optimal strength leads to nearly total—but not entire—suppression of target visibility, and so on. A characteristic that distinguishes para- and metacontrast masking effects from effects produced by spatially overlapping masks is that para- and metacontrast yield target visibilities that vary non-monotonically in a U-shaped manner with SOA, whereas masking with overlapping stimuli typically (Breitmeyer, 1984), though not invariably (Bachmann, 1994; Bachmann & Allik, 1976; Michaels & Turvey, 1979), yields target visibilities that vary monotonically with SOA. In Fig. 2A, we show typical results from para- and metacontrast studies conducted by us. In these studies, we presented an unmasked comparison disk along with the target disk that was preceded or followed by a mask ring. At a given SOA, observers used an adaptive tracking procedure to adjust the luminance of the comparison disk over a series of trials until the comparison disk's luminance contrast appeared to match that of the target. Lower (higher) comparison contrasts correspond to lower (higher) target visibility. Note that target visibility changes in a U-shaped manner as the paracontrast or metacontrast SOAs vary. For paracontrast, target visibility is highest when the mask ring precedes the target disk by several hundred milliseconds, decreases progressively to a minimum at a paracontrast SOA of −100 ms, then increases again as the paracontrast SOA approaches 0 ms. Turning to metacontrast, note that target visibility is high at an SOA of 0 ms, gradually decreases to a minimum at an SOA of 40 ms, and then gradually increases for further increases of SOA values. Note also that the depth of target suppression is greater for metacontrast than for paracontrast, a highly replicated finding (Breitmeyer, 1984).

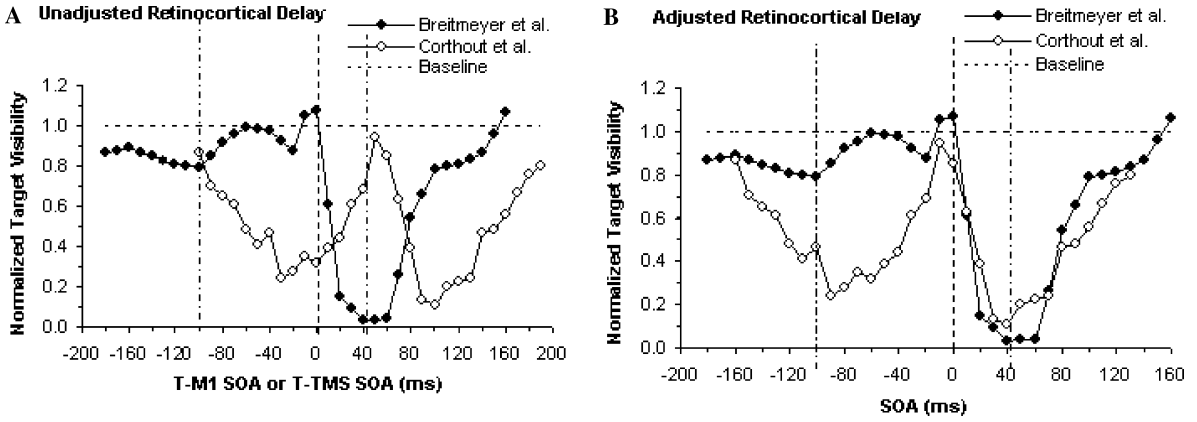

Fig. 2. (A) Comparison of a typical masking function obtained by Breitmeyer and Ogmen using a visual para- or metacontrast mask and a typical masking function obtained by Corthout, Uttl, Ziemann et al. (1999) using a TMS pulse as a mask. Negative and positive SOAs indicate that the masks were presented before and after the target, respectively. Results are not adjusted for retinocortical transmission delay. (B) Same as preceding but with results adjusted for a 60-ms delay of cortical M activity due to retinocortical transmission time (Baseler and Sutter, 1997).

Also in Fig. 2A, results taken from Corthout, Uttl, Ziemann et al. (1999)[2] show typical (average) changes of target visibility as a function of the SOA between a target and the TMS mask. In this experiment letters rather than disks served as targets, and after each trial observers were asked to identify the target letters. Here, the proportion of correct target identifications is taken as an index of target visibility. Note that in these results, as in our para- and metacontrast studies, two epochs of optimal suppression of target visibility are evident; an early one at an SOA of about −30 ms, and a later one at an SOA of about 100 ms (for a similar late epoch of TMS suppression see Amassian et al., 1989). As can be seen, the SOA epochs obtained with the visual-mask procedure do not coincide with two corresponding epochs obtained with the TMS-mask procedure. We attribute this to the fact that a visual mask, due to retino-cortical transmission time, has its suppressive effects on the target delayed by several tens of milliseconds relative to a TMS mask applied to occipital area of the skull. Estimates of earliest cortical response latency to visual stimulation, based on visually evoked potentials (VEPs) recorded by Baseler and Sutter (1997) and Vanni et al. (1997), indicate that the shortest VEP onset latency is on the order of 55–70 ms. Hence, inspection of Fig. 2B, with its adjustment of the TMS SOA axis so that its 60-ms value coincides with the visual mask SOA of 0 ms, reveals that the time courses of the two masking effects overlap more noticeably. Note especially that the two epochs of maximal suppression coincide much more closely, this despite the use of different observers, stimuli, and behavioral measures of target visibility. Additionally, the weaker target suppression produced by a prior TMS mask than a following one also coincides with the weaker and stronger para- and metacontrast masking effects shown in Fig. 2. We take both of these coincidences as strong evidence that a TMS mask produces paracontrast and a metacontrast masking effects akin to those produced by a visual mask, although definitive evidence awaits the direct within-observer comparisons of visual and TMS masking of identical visual targets.

Subject to further investigations, for now we support our conclusion by the following additional considerations on cortical mechanisms affected by visual and TMS masks. First, Corthout, Uttl, Walsu et al. (1999) and Corthout, Uttl, Ziemann et al. (1999) argued—correctly in our opinion—that the early and late epochs of target suppression by a TMS mask may correspond to suppression of, respectively, early feedforward response components and later feedback-dependent response components of V1 neural responses. Second, electrophysiological evidence (Andreassi, De Simone, & Mellers, 1975; Bridgeman, 1975, 1980, 1988; Macknik & Livingstone, 1998; Schiller & Chorover, 1966; Vaughn & Silverstein, 1968) indicates that it is not the primary, early component of V1 cortical neural activity that is suppressed by metacontrast but rather the secondary, late component. Moreover, recent work by Lamme, Super, Landman, Roelfsema, and Spekreijse (2000) indicates that these later neural components are percept-dependent, i.e., their strength varies in direct proportion to the behaviorally assessed visibility of a stimulus evoking them, whereas the earlier, primary components are stimulus dependent, in that their magnitude is affected by physical properties of the stimulus but does not vary with the behaviorally assessed perceptual state of the stimulus. According to Lamme et al. (2000) the later, percept-dependent components of V1 activation arise due to re-entrant feedback from higher cortical visual areas, whose own neural activities become progressively more percept-dependent the higher upstream they are

[2] We thank Erik Corthout for making the averaged masking data from Corthout, Uttl, Ziemann et al. (1999) available to us for incorporation in Fig. 2.

located in the cortical stream of processing (Leopold & Logothetis, 1996; Logothetis & Schall, 1989). Third, as shown by Macknik and Livingstone (1999), suppression of stimulus visibility by a paracontrast as compared to metacontrast mask correlates with activity level of the early V1 response components in the feedforward sweep of cortical activity. Since the magnitude of the later, feedback- or percept-dependent response components "feeds on" the magnitude of the earlier feedforward- or stimulus-dependent components, a reduction of the earlier components by paracontrast will result also in a reduction of the later components and hence to target suppression. Thus, given that Corthout, Uttl, Walsh et al.'s (1999, 1999) conclusion is correct, it follows that the two, early and late, epochs of target suppression result from reductions of cortical neural activities akin to the reductions produced by para- and metacontrast.

It should be noted here that our paracontrast masking functions actually contains two minima, one at an SOA of −100 ms; the other at an SOA of −20 s. Our ongoing investigations (Breitmeyer, Ogmen, & Mardon, in preparation) have confirmed the existence of two minima in the paracontrast masking function, an early one falling somewhere between −120 and −80 ms and a later one falling around −20 to −10 ms. As yet, we do not fully understand the existence of these separate paracontrast minima in the visual masking function, although we believe they relate to separate processing mechanisms for luminance contrast and for form. It should be noted that in more careful parametric analyses Corthout et al. (2000, 2003) also have found an additional early period of TMS-induced visual suppression. Whether the extra dip found in the TMS-masking functions corresponds to the local minimum at −20 ms in the paracontrast masking function and exactly what visual process it disrupts is yet to be determined.

2.2. Target recovery

Regarding target recovery, we note that there are many ways of producing it (see Breitmeyer, 1984, pp. 270–284); and, among these, a TMS mask can be just as effective as a secondary visual mask, M2, not only in terms of the result produced but also in terms of the process involved. For example, in the case of visual stimulation, if an M1 following a target, T, produces target suppression, then an M2, in turn following and suppressing the visibility of M1, can yield target recovery (Dember & Purcell, 1967; Purcell, Dember, & Hochberg, 1982; Robinson, 1971). Similarly, if a visual M1 suppresses the visibility of a preceding T, a TMS mask, in turn following and suppressing the visibility of M1, can produce target recovery (Amassian et al., 1993).[3] These two similar target recovery effects can be explained by correspondingly similar processes, namely, by the reduction of M1's visibility which otherwise would interfere with or suppress the visibility of T. This implies that, at least in these cases of target recovery, the neural process responsible for M1's visibility also is responsible for its mask effectiveness. This conclusion is supported by Amassian et al.'s (1993) finding that the recovery of T's visibility was directly proportional to the suppressive effects of the TMS pulse on the visibility of the prior M1. That is, M1's effectiveness in suppressing the visibility of T was directly related to its own visibility. This means that in this

[3] Besides applying TMS to the occipital pole, Amassian et al. (1993) also applied it to the temporo-occipital-parietal (T-P-O) area. The present discussion deals only with the results applied to the occipital pole, since only these results are directly comparable to those reported by Corthout, Uttl, Walsh et al. (1999), Corthout, Uttl, Ziemann et al. (1999), Corthout, Uttl, Juan et al. (2000), Corthout et al. (2003) and by Ro, Breitmeyer, Burton, Singhal, and Lane (2003).

TMS study [and in related studies in which M1 spatially overlaps T and a visual M2 follows M1 (Dember & Purcell, 1967; Purcell et al., 1982; Robinson, 1971)] the neural activity evoked by M1 and contributing to *its visibility is associated with* the neural activity evoked by M1 and contributing to *its mask effectiveness*, a strong indication that mask visibility and mask effectiveness derive from the same neural process.

3. Differences between target recovery by visual and TMS masking and their underlying mechanisms

Despite these similarities between the findings of target suppression and target recovery obtained with visual and TMS masks and between the effects of these masks on similar hypothesized underlying neural processes, significant differences become evident when one examines more closely the studies of target recovery obtained with metacontrast stimuli that do not overlap spatially.

3.1. In metacontrast, TMS (again) reveals an association between M1-evoked neural activities that contribute to its masking effectiveness and to its visibility

We first review such recovery, reported by Ro et al. (2003), produced by TMS pulses presented after the onset of M1. As shown in Fig. 3, the T-M1 SOA was always set at 42 ms, a value determined by prior experiment to yield optimal target suppression. The TMS pulse was presented at M1-TMS SOAs varying from 57 to 171 ms, a range that includes the value of 100 ms, optimal for suppressing the visibility of M1 (Corthout et al., 1999; Corthout, Uttl, Ziemann et al., 1999; Amassian et al., 1989). Like Amassian et al.'s (1993) results, Ro et al.'s (2003) results, shown in Fig. 4, revealed that the recovery of the disk target was optimal at those M1-TMS SOAs (100–143 ms) at which the suppression of M1's visibility by the TMS pulse also was optimal. By the argument outlined above, since the masking effectiveness of the spatially surrounding M1 was

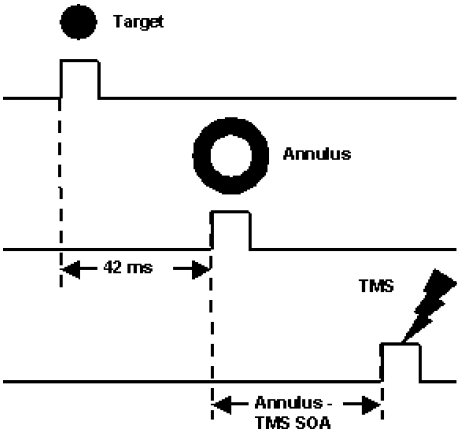

Fig. 3. Schematic diagram of stimuli used in Ro et al.'s (2003) experiment. The temporal sequences depict T (disk) and M1 (annulus) at a fixed T-M1 SOA of 42 ms. The SOAs separating the onset of M1 from that of the TMS vary from 51 to 171 ms.

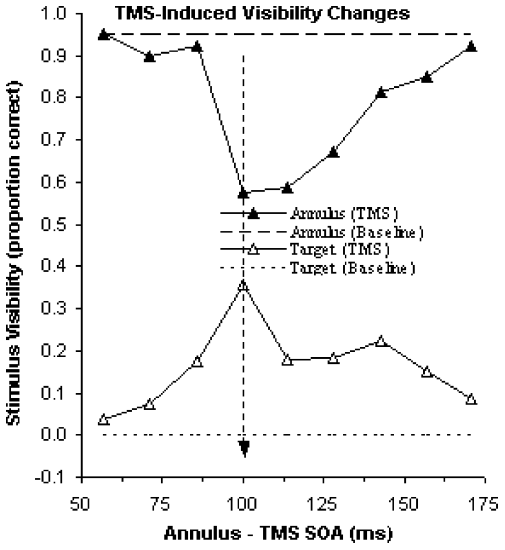

Fig. 4. Visibility, in proportion of perceived-stimulus reports, of the annulus (M1) and the preceding disk (T) as a function of the SOA separating the annulus from the following TMS pulse. Annulus baseline visibility (0.95) was obtained in the absence of the TMS pulse. The disk visibilities are TMS-induced *changes* of the proportion of disk reports relative to the baseline (no change) in the absence of the TMS pulse. Positive changes indicate TMS-induced recovery of the (otherwise suppressed) disk's visibility.

directly related to its visibility, one would again be inclined to conclude that the neural activity evoked by M1 and contributing to its own visibility is NOT dissociated from the neural activity evoked by M1 and contributing to its effectiveness as a mask. However, as shown below, this conclusion turns out to be wrong.

3.2. In metacontrast, a visual M2 reveals a double dissociation between the M1-evoked neural activities contributing to M1's masking effectiveness and to its visibility

Although the positive correlation between M1's visibility and its effectiveness as a mask is clearly evident when a TMS pulse is used to target recovery, the correlation can be shown to break down dramatically when a visual M2 serves to produce target recovery. This is evident from results of a study of target recovery reported by Breitmeyer, Rudd, and Dunn (1981) and recently replicated by Ogmen, Breitmeyer, Todd, and Mardon (2004). As illustrated in Fig. 5, Breitmeyer et al. (1981) investigated the effects of a secondary ring mask, M2, that spatially surrounded the primary ring mask, M1, on recovery of the visibility of a disk target, T, which in turn was spatially surrounded by M1. Prior experiment showed that the metacontrast SOA for M1's optimal suppression of T's visibility was 30–60 ms. An optimal T-M1 SOA value of 60 was used throughout the subsequent phase of the experiment that examined the effects of M2 on the visibility of T as well as that of M1 in order to establish the relationship, if any between M1's effectiveness as a mask and its visibility.

Unlike the studies using TMS pulses discussed above which only presented a TMS pulse after M1, the M2 in Breitmeyer et al.'s (1981) study was presented (for theoretical reasons that will

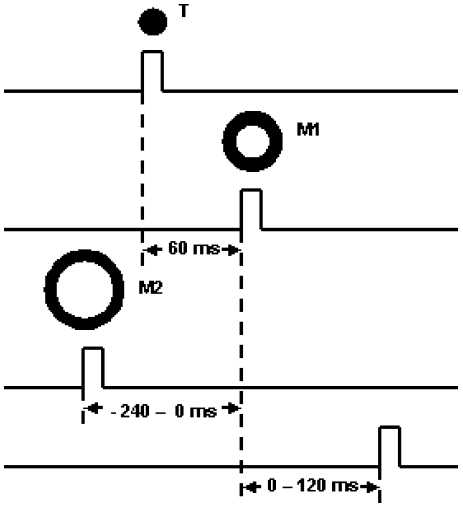

Fig. 5. Schematic diagram of stimuli used in Breitmeyer et al.'s (1981) experiment. The temporal sequences depict T (disk) and M1 (annulus) at a fixed T-M1 SOA of 60 ms. The SOAs separating the onset of M1 from M2 vary from −240 ms (M2 precedes M1) to 120 ms (M2 follows M1).

become evident later) at variable SOAs not only after M1 but also before M1. As shown in Fig. 5, the M2 could precede M1 at variable SOAs ranging from −240 to 0 ms or follow it at variable SOAs ranging from 0 to 120 ms. In this experiment, we determined the effect of M2 on the visibility of T in order to obtain a measure of target recovery and thus also of M1's masking effectiveness. Baseline target visibility of 0 (on a logarithmic scale) corresponded to the contrast visibility of the target when it was optimally suppressed by M1 at the T-M1 SOA of 60 ms. Since the degree of target recovery produced by M2 is related directly to the degree by which a M2 reduces M1's masking effectiveness, the inverse of target recovery gives a direct estimate of M2's effect on *M1's masking effectiveness*. We also measured the effect of M2 on *M1's visibility*. Here, baseline visibility of 0 corresponds to the contrast visibility of M1 when it was presented in the T-M1 sequence at an SOA of 60 ms, but without M2 being presented.

Fig. 6 shows how M1's masking effectiveness (open circles) and its visibility (closed circles) vary with respect to baseline as a function of M1-M2 SOA. The arrays of disk, small annulus, and large annulus, at the bottom of the figure are meant to portray approximately the contrast visibilities of all three of the stimuli, T, M1 and M2 at each M1–M2 SOA. Over the range of negative SOAs, note that T increases in visibility (target recovery) as the M1–M2 SOA shifts from −240 ms and reaches a maximum at M1–M2 SOAs between −90 and −30 ms; thereafter declining again. This trend shows that M1's masking effectiveness, the inverse of target visibility, decreases as M1–M2 SOA shifts from −240 ms, reaching a minimum at M1–M2 SOAs between −90 and −30 ms, and thereafter increasing. Note however, that M1's visibility does not change as M1–M2 SOA varies from −240 to 0 ms. Hence at negative M1–M2 SOAs, we obtain a *dissociation* between an unchanging, constant visibility of M1 and a nonmonotonic, U-shaped change M1's mask effectiveness. Turning now to the positive M1–M2 SOAs where M2 follows M1, we note that M1's masking effectiveness is not changed at all (i.e., there is no target recovery in that the black target disk remains invisible and the area occupied by it appears white at all positive M1–M2 SOAs).

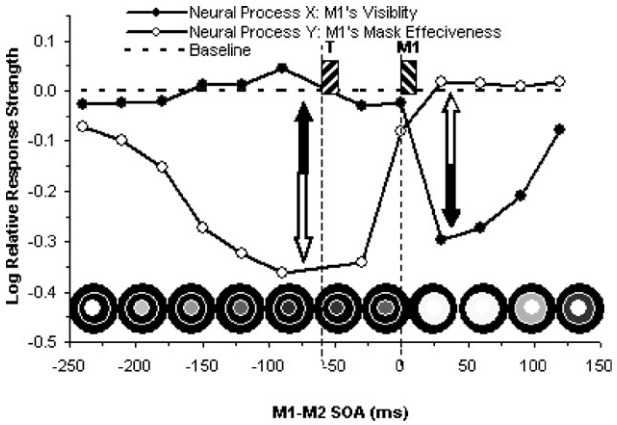

Fig. 6. Log relative response strength of neural activities contributing to M1's visibility (closed circles) and to M1's masking effectiveness (open circles) as a function of M1–M2 SOA. The array of disks and concentric annuli at the bottom of the figure render the (approximate) subjectively perceived contrasts of T, M1 and M2 as M1–M2 SOA is varied. [Adapted from Breitmeyer et al. (1981)].

However, if we look at M1's visibility, we note that it changes in a typical U-shaped manner found in metacontrast, with lowest visibility at an M1–M2 SOA of 30–60 ms, a result expected, since M2, coming after M1, acts as a strong metacontrast mask of M1's visibility. Hence at positive M1–M2 SOAs we again obtain a d*issociation*, this time between M1's nonmonotonic, U-shaped change of visibility and its nonchanging, high mask effectiveness.

4. Implications

4.1. Theoretical

In combination, these results are significant for theoretical as well as methodological reasons. As to theory, when considering the entire M1–M2 SOA range from −240 to 120 ms, we have established that the introduction of M2 into the T-M1 sequence effects *a double dissociation between M1's masking effectiveness and M1's visibility*. Similar to explanations of double-dissociations found in neuropsychological investigations (Teuber, 1955), we take this double dissociation as a strong indication for the existence and involvement of two separate neural mechanisms or processes in visual masking specifically, and in visual information processing more generally.

Before proceeding, it should be noted that both Francis (1997) and Bachmann (1994) can account for specific but not all aspects of target recovery that we shall discuss below. Francis (1997) notes that his model, as yet, cannot account for the fact that M2 blocks the inhibitory effect of M1 (on T) without suppressing M1's visibility. Bachmann (1988, 1994) would argue that M2 preceding the T-M1 sequence at optimal intervals of 60–90 ms, rather than blocking the masking effectiveness if M1, could have a *direct facilitatory* effect on T's visibility, which would counteract the suppressive effects of M1. While such an effect may be sufficient for obtaining target recovery,

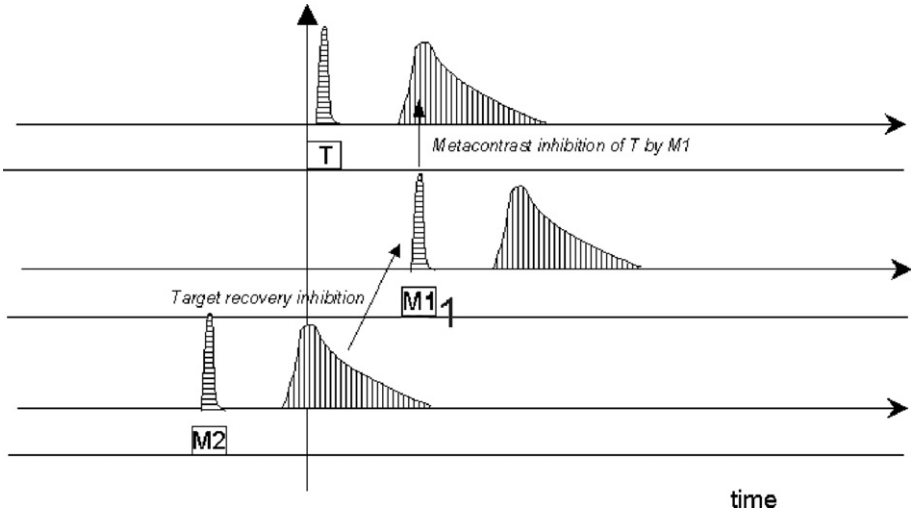

Fig. 7. Schematic depiction of inhibitory interactions leading to target recovery. For each-stimulus, the transient and sustained activities and their relative timings are shown. The mask M1's transient activity suppresses the sustained activity of the target, T, to produce metacontrast masking. The sustained activity of the secondary mask, M2, inhibits the transient activity of M1 thereby causing a release of M1's inhibition on T. This release leads to target recovery.

below we describe experimental results that rule out the necessity of direct facilitatory effects of M2 on T. For that reason, among others, we discuss the above findings in terms an updated dual-channel approach (Breitmeyer & Ogmen, 2000; Ogmen, Breitmeyer, & Melvin, 2003; Purushothaman, Ogmen, & Bedell, 2000), which, incorporating *mutual inhibition between separate parvocellular (P) sustained and magnocellular (M) transient neural channels*, can account for the main results. Within this specific dual-process context and for the sake of clarity, we give a more detailed account of the changes of M1's masking effectiveness and visibility across the range of M1–M2 SOAs.

Let us first consider the case of M2's influence on M1's masking effectiveness. Like prior dual-channel approaches (Breitmeyer, 1984; Breitmeyer & Ganz, 1976), we assume: (1) that the sustained-channel activity provides the contents for qualia-rich visual awareness of objects, (2) that, at some cortical level, there is approximately a 30–60 ms latency difference between the slower sustained activity and the faster transient activity generated by a stimulus onset, and (3) that there is mutual inhibition between the two types of cortical activity.[4] Given these assumptions, as depicted in Fig. 7, it follows that the slower sustained activity of M2 will inhibit the faster transient activity of M1, provided that M2 precedes M1. In particular, when M2 precedes M1 by about 60 ms, one should get maximal suppression of M1's transient activity and thus of its masking effectiveness. Hence, T's otherwise suppressed contrast visibility in turn ought to recover, i.e., be increased, maximally. In fact, as shown in Fig. 6, T's visibility recovers maximally at an M1–M2 SOA ranging from −90 to −30 ms (−60 ms was not tested). When M2 precedes M1 at

[4] Prior dual-channel approaches to masking were based on inhibitory interactions between feedforward activities in the M and P channels (e.g., Breitmeyer & Ganz, 1976). However, it is also conceivable that cortical M-channel activity could suppress the reentrant, feedback activity within cortical P channels.

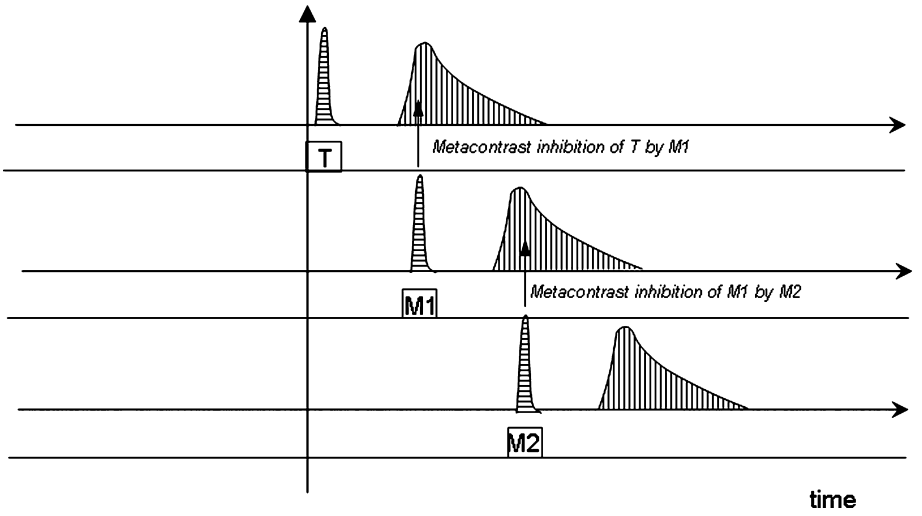

Fig. 8. Schematic depiction of how the secondary mask M2 affects the *visibility* of the primary mask M1. M2's transient activity inhibits the sustained activity of M1 reducing M1's visibility. However, M1's transient activity remains intact and therefore, M1's masking effectiveness on T remains unchanged.

progressively larger onset asynchronies, M1's masking effectiveness increases gradually to its maximum, and thus target recovery decreases gradually and approaches 0 at an M1–M2 SOA of −240 ms. This follows from the fact that the temporal overlap of mutual inhibitory interactions between M2's slower sustained activity and M1's faster transient activity decreases progressively as M2 precedes M1 by progressively larger onset asynchronies. For the same reason, a similar (but more precipitous) increase of M1's masking effectiveness, and thus a decline of target recovery, occurs when M2 precedes M1 at onset asynchronies progressively shorter than 30 ms. And continuing this trend, we see no noticeable decrease of M1's masking effectiveness, and thus no increase of target recovery, when M2 follows M1 at SOAs progressively larger than 0 ms.

Let us now turn to M2's influence on M1's visibility. At negative M1–M2 SOAs, there is little, if any, mutual inhibition between these activated neural channels because M2's fast transient activity always precedes M1's slow sustained activity. Hence M1's visibility remains at a high, unmasked level throughout the range of negative SOAs. However, at positive M1–M2 SOAs the conditions for metacontrast prevail (Fig. 8). Here, M2's faster transient activity can maximally inhibit M1's slower sustained activity at an SOA of approximately 30–60 ms. Hence *M2 can affect the responses contributing to M1's mask effectiveness separately from the responses contributing to M1's visibility.*

This theoretical interpretation is supported by data recently reported by Ogmen et al. (2004). They showed that target recovery produced when M2 precedes M1 increases linearly and significantly with the contrast of M2. However, M1's mask effectiveness increased little, if any, with M2's contrast. Since the response of P neurons increases linearly with contrast whereas that of M neurons saturates rapidly at low contrasts (Kaplan & Shapley, 1986), the contrast-dependent changes of target recovery and of M1's saturated mask effectiveness parallel strongly the contrast-dependent responses of P and of M neurons, respectively. In addition to the aforementioned double

dissociation, these findings also support our dual-channel approach to masking. Moreover, simulation studies showed a good quantitative agreement between our model and the psychophysical data (Ogmen et al., 2004). Finally, control experiments conducted by Ogmen et al. (2004), in which only M2 and T were presented, also showed that when M2 preceded T there was no change of T's visibility. This rules out direct facilitatory effects (e.g., due to attentional priming or perceptual retouch Bachmann, 1988, 1994) of M2 on the visibility of T. Hence, we conclude that the target recovery effects produced by M2 indeed are obtained indirectly by suppressing M1's mask effectiveness.

4.2. Methodological

With the theoretical significance of the double dissociation established, we can now turn to methodological considerations. Recall that, with a TMS pulse, the target recovery, and hence the decrease in the masking effectiveness of the mask, *is associated* with a reduction of the visibility of the mask (Amassian et al., 1993; Ro et al., 2003). In this sense, its effects are quite different from those of the secondary visual mask M2 discussed above. We believe this is due to the fact that the effects of a TMS pulse, although spatially local, are not pathway- or channel-specific. That is to say, *the TMS pulse does not selectively disrupt the separate P and M activities* in V1. With respect to M1, it suppresses not only the M1's cortical transient activity required for metacontrast masking of T, but also M1's cortical sustained activity required for M1's visibility. A careful "psychophysics" based on parametric variations TMS energy, which might reveal pathway-specific effects in V1, has yet to be established. Both forms of masking currently can reveal specific aspects of the temporal dynamics of conscious and unconscious processing in visual object perception (Corthout, Uttl, Walsh et al., 1999, 1999; Breitmeyer et al., 2004). However, until such time when a more systematic TMS psychophysics has been established, visual masking, with its precisely controllable parameters such as stimulus luminance, target-mask spatial separation, etc. (see Breitmeyer, 1984, pp. 103–120), can supplement TMS masking studies by providing additional information about the interactions and time differences between the responses of separate visual pathways.

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References

Amassian, V. E., Cracco, R. Q., Maccabee, P. J., Crocco, J. B., Rudell, A., & Eberle, L. (1989). Suppression of visual perception by magnetic coil stimulation of human occipital cortex. *Electroencephalography and Clinical Neurophysiology, 74*, 458–462.

Amassian, V. E., Cracco, R. Q., Maccabee, P. J., Crocco, J. B., Rudell, A. P., & Eberle, L. (1993). Unmasking human visual perception with the magnetic coil and its relationship to hemispheric asymmetry. *Brain Research, 605*, 312–316.

Andreassi, J. L., De Simone, J. J., & Mellers, B. W. (1975). Amplitude changes in the visual evoked potential with backward masking. *Electroencephalography and Clinical Neurophysiology, 41*, 387–398.

Bachmann, T. (1988). Time course of the subjective contrast enhancement for a second stimulus in successively paired above-threshold transient forms: perceptual retouch instead of forward masking. *Vision Research, 28*, 1255–1261.

Bachmann, T. (1994). *Psychophysiology of visual masking: the fine structure of conscious experience*. Commack, NY: Nova Science Publishers.

Bachmann, T., & Allik, J. (1976). Integration and interruption in the masking of form by form. *Perception, 5*, 79–97.

Baseler, H. A., & Sutter, E. E. (1997). M and P components of the VEP and their visual field distribution. *Vision Research, 37*, 675–690.

Breitmeyer, B. G. (1984). *Visual masking: an integrative approach*. New York: Oxford University Press.

Breitmeyer, B. G., & Ganz, L. (1976). Implications of sustained and transient channels for theories of visual pattern masking, saccadic suppression and information processing. *Psychological Review, 83*, 1–36.

Breitmeyer, B. G., & Ogmen, H. (2000). Recent models and findings in visual backward masking: a comparison, review, and update. *Perception & Psychophysics, 62*, 1572–1595.

Breitmeyer, B. G., Rudd, M., & Dunn, K. (1981). Metacontrast investigations of sustained-transient channel inhibitory interactions. *Journal of Experimental Psychology: Human Perception and Performance, 7*, 770–779.

Breitmeyer, B. G., Ogmen, H., & Chen, J. (2004). Unconscious priming by color and form: different processes and levels. *Consciousness and Cognition, 13*, 138–157.

Breitmeyer, B. G., Ogmen, H., & Mardon, L. (in preparation). Para- and metacontrast masking of the contour and the surface brightness of target stimuli.

Breitmeyer, B. G., Ro, T., & Singhal, N. S. (2004). Unconscious color priming occurs at stimulus- not percept-dependent levels of processing. *Psychological Science, 15*, 198–202.

Bridgeman, B. (1975). Correlates of metacontrast in single cells of the cat visual system. *Vision Research, 15*, 91–99.

Bridgeman, B. (1980). Temporal characteristics of cells in monkey striate cortex measured with metacontrast masking and brightness discrimination. *Brain Research, 196*, 347–364.

Bridgeman, B. (1988). Visual evoked potentials: concomitants of metacontrast in late components. *Perception & Psychophysics, 43*, 401–403.

Corthout, E., Hallett, M., & Cowey, A. (2003). Interference with vision by TMS over the occipital pole: a fourth period. *NeuroReport, 14*, 651–655.

Corthout, E., Uttl, B., Juan, C.-H., Hallett, M., & Cowey, A. (2000). Suppression of vision by transcranial magnetic stimulation; a third mechanism. *NeuroReport, 11*, 2345–2349.

Corthout, E., Uttl, B., Walsh, V., Hallett, M., & Cowey, A. (1999). Timing of activity in early visual cortex as revealed by transcranial magnetic stimulation. *NeuroReport, 10*, 2631–2634.

Corthout, E., Uttl, B., Ziemann, U., Cowey, A., & Hallett, M. (1999). Two periods of processing in the (circum)striate visual cortex as revealed by transcranial magnetic stimulation. *Neuropsychologia, 37*, 137–145.

Dember, W. N., & Purcell, D. G. (1967). Recovery of masked visual targets by inhibition of the masking stimulus. *Science, 157*, 1335–1336.

Di Lollo, V., Enns, J. T., & Rensink, R. A. (2000). Competition for consciousness among visual events: the psychophysics of reentrant visual processes. *Journal of Experimental Psychology: General, 129*, 481–507.

Dolan, R. J. (2002). Emotion, cognition, and behavior. *Science, 298*, 1191–1194.

Francis, G. (1997). Cortical dynamics of lateral inhibition: metacontrast masking. *Psychological Review, 104*, 572–594.

Herzog, M. H., & Koch, C. (2001). Seeing properties of an invisible object: feature inheritance and shine-through. *Proceedings of the National Academy of Sciences of the United States of America, 98*, 4271–4275.

Kamitani, Y., & Shimojo, S. (1999). Manifestation of scotomas created by transcranial magnetic stimulation of human visual cortex. *Nature Neuroscience, 2*, 767–771.

Kammer, T., Scharnowski, F., & Herzog, M. H. (2003). Combining backward masking and transcranial magnetic stimulation in human observers. *Neuroscience Letters, 343*, 171–174.

Kaplan, e., & Shapley, R. R. (1986). The primate retinal contains two types of ganglion cells, with high and low contrast sensitivity. *Proceedings of the National Academy of Science of the United States of America, 83*, 2755–2757.

Klotz, W., & Wolff, P. (1995). The effect of a masked stimulus on the response to the masking stimulus. *Psychological Research, 58*, 92–101.

Lamme, V. A. F., Super, H., Landman, R., Roelfsema, P. R., & Spekreijse, H. (2000). The role of primary visual cortex (V1) in visual awareness. *Vision Research, 40*, 1507–1521.

Leopold, D. A., & Logothetis, N. K. (1996). Activity changes in early visual cortex reflect monkeys percept during binocular rivalry. *Nature, 379*, 549–552.

Logothetis, N. K., & Schall, J. D. (1989). Neuronal correlates of subjective visual perception. *Science, 245*, 761–763.

Macknik, S. L., & Livingstone, M. S. (1998). Neuronal correlates of visibility and invisibility in the primate visual system. *Nature Neuroscience, 1*, 144–149.

Macknik, S. L., & Livingstone, M. S. (1999). Neuronal correlates of visibility and invisibility in the primate visual system. *Nature Neuroscience, 1*, 144–149.

Michaels, C. F., & Turvey, M. T. (1979). Central sources of visual masking: indexing structures supporting seeing at a single, brief glance. *Psychological Research, 41*, 1–61.

Neumann, O., & Klotz, W. (1994). Motor responses to nonreportable, masked stimuli: where is the limit of direct parameter specification. In C. Umilta & M. Moscovitch (Eds.), *Attention and performance XV* (pp. 123–150). Cambridge, MA: MIT Press.

Ogmen, H., Breitmeyer, B. G., & Melvin, R. (2003). The what and where in visual masking. *Vision Research, 43*, 1337–1350.

Ogmen, H., Breitmeyer, B. G., Todd, S., & Mardon, L. (2004). Double dissociation in target recovery: effect of contrast. In *Paper presented at the 4th annual meeting of the Vision Science Society*, Sarasota, FL, April 30–May 5.

Ohman, A. (2002). Automaticity and the amygdala: nonconscious responses to emotional faces. *Current Directions in Psychological Science, 11*, 62–66.

Purcell, D. G., Dember, W. N., & Hochberg, E. P. (1982). Recovery and nonmonotone masking effects. *Vision Research, 22*, 1087–1096.

Purushothaman, G., Ogmen, H., & Bedell, H. E. (2000). Gamma-range oscillations in backward masking functions and their putative correlates. *Psychological Review, 107*, 556–577.

Ro, T., Breitmeyer, B., Burton, P., Singhal, N. S., & Lane, D. (2003). Feedback contributions to visual awareness in human occipital cortex. *Current Biology, 11*, 1038–1041.

Robinson, D. N. (1971). Backward masking, disinhibition, and hypothesized neural networks. *Perception & Psychophysics, 10*, 33–35.

Schiller, P. H., & Chorover, S. L. (1966). Metacontrast: its relation to evoked potentials. *Science, 153*, 1398–1400.

Teuber, H.-L. (1955). Physiological psychology. *Annual Review of Psychology, 6*, 267–296.

Uttal, W. R. (1981). *A taxonomy of visual processes*. Hillsdale, NJ: Erlbaum.

Vanni, S., Tanskanen, T., Seppae, M., Uutela, K., & Hari, R. (1997). Coinciding early activation of the human primary visual cortex and anteromedial cuneus. *Proceedings of the National Academy of Sciences of the United States of America, 98*, 2776–2780.

Vaughn, H. G., Jr., & Silverstein, L. (1968). Metacontrast and evoked potentials: a reappraisal. *Science, 160*, 207–208.

Wong, P. S., & Root, J. C. (2003). Dynamic variations in affective priming. *Consciousness and Cognition, 12*, 147–168.