Perception & Psychophysics
2000, 62 (8), 1572-1595

Recent models and findings in visual backward masking: A comparison, review, and update

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Visual backward masking not only is an empirically rich and theoretically interesting phenomenon but also has found increasing application as a powerful methodological tool in studies of visual information processing and as a useful instrument for investigating visual function in a variety of specific subject populations. Since the dual-channel, sustained–transient approach to visual masking was introduced about two decades ago, several new models of backward masking and metacontrast have been proposed as alternative approaches to visual masking. In this article, we outline, review, and evaluate three such approaches: an extension of the dual-channel approach as realized in the neural network model of retino-cortical dynamics (Ogmen, 1993), the perceptual retouch theory (Bachmann, 1984, 1994), and the boundary contour system (Francis, 1997; Grossberg & Mingolla, 1985b). Recent psychophysical and electrophysiological findings relevant to backward masking are reviewed and, whenever possible, are related to the aforementioned models. Besides noting the positive aspects of these models, we also list their problems and suggest changes that may improve them and experiments that can empirically test them.

Visual masking occurs whenever the visibility of one stimulus, called the *target*, is reduced by the presence of another stimulus, designated as the *mask*. Visual masking has been, and continues to be, a powerful psychophysical tool for investigating the steady-state properties of spatial-processing mechanisms (Dakin & Hess, 1997; Foley & Chen, 1997; Glennerster & Parker, 1997; McKee, Bravo, Taylor, & Legge, 1994; Mussap & Levi, 1997; Stromeyer & Julesz, 1972). Although acknowledging the theoretical and practical importance of understanding the steady-state properties of spatial vision, in this article we focus on the dynamic properties of visual pattern processing, particularly those studied with the backward masking technique.

Backward visual masking is the reduction of a target's visibility by a mask presented after the target. Traditionally, four types of backward masking can be distinguished operationally. In backward masking by light, the target is masked by a significantly larger and spatially overlapping uniform flash of light (Crawford, 1947; Sperling, 1965). Metacontrast, another type of backward masking, occurs when the mask does not overlap the target spatially (Alpern, 1953; Stigler, 1910; Werner, 1935). In backward masking by structure, the mask overlaps the target, but rather than being uniform, it shares many of the structural features of the target (Turvey, 1973). Finally, in backward masking by noise, the mask typically consists of random-dot noise that overlaps the target (Kinsbourne & Warrington, 1962a, 1962b; Turvey, 1973). These masking methods can be distinguished from each other by the functions relating masking magnitude to the stimulus onset asynchrony (SOA) separating the target and the mask. In this review, we focus on metacontrast masking and masking by structure because they, unlike the other two types of masking, (1) can yield a nonmonotonic U-shaped (or Type B) masking function as SOA is increased and (2) can produce powerful masking effects when the target and the mask are presented to separate eyes (dichoptic viewing), as well as when they are presented to the same eye (monocular or binocular viewing; Breitmeyer, 1984; Michaels & Turvey, 1979; Turvey, 1973). Henceforth, our use of the term *backward masking* will apply only to metacontrast and to masking by structure.

The study of backward masking is informative for several reasons. First, the phenomenon is interesting in its own right, owing to the counterintuitive finding that the mask can impede the visibility of the target even though the target is presented first (Bachmann, 1994; Breitmeyer, 1984). Several formal quantitative models, as well as qualitative explanations, of this phenomenon have been proposed in the last two decades. We will review these models and explanations in subsequent sections of this article. Second, models of backward visual masking may be relevant to our understanding of a variety of spatiotemporal phenomena, such as motion perception, visual persistence, onset and offset reaction time (RT), and discrimination of temporal order (Breitmeyer, 1984). In particular,

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two neural network models (Francis, 1997; Ogmen, 1993), to be discussed below, can account for metacontrast, as well as for other spatiotemporal phenomena. Third, backward masking can be used as a tool to investigate the temporal sequencing and various levels of information processing in the visual system (for psychophysical approaches, see Bachmann, 1984; Bowen & Wilson, 1994; Michaels & Turvey, 1979; Muise, LeBlanc, Lavoie, & Arsenault, 1991; Turvey, 1973; for neurophysiological approaches, see Bridgeman, 1975, 1980; Kovács, Vogels, & Orban, 1995; Macknik & Livingstone, 1998; Rolls, Tovée, & Panzeri, 1999; Thompson & Schall, 1999). Psychophysical applications of these masking paradigms often simply assume that the after-coming mask acts to *erase* visual information or to *interrupt* its further processing. A clearer understanding of the underlying mechanisms by which such erasure or interruption can occur (Kahneman, 1968; Scheerer, 1973; Sperling, 1963) is required for the informed use of masking as a methodological tool. Fourth, higher level cognitive processes can modulate backward masking. For instance, the magnitude of backward masking is affected by perceptual grouping and segmentation (Caputo, 1998; Kurylo, 1997; Wolf, Chun, & Friedman-Hill, 1995) and by deployment of selective visual attention (Enns & Di Lollo, 1997; Havig, Breitmeyer, & Brown, 1998; Michaels & Turvey, 1979; Ramachandran & Cobb, 1995; Shelley-Tremblay & Mack, 1999). Closely related, backward masking recently has been used to study visual awareness (Bachmann, 1997; Dennett, 1991; Klotz & Neumann, 1999; Klotz & Wolff, 1995; Neumann & Klotz, 1994) and its implications for the controversial field of *subliminal* perception (Duncan, 1985; Holender, 1986; Kihlstrom, 1987; Marcel, 1983). Finally, backward masking has been used to study certain clinical anomalies related to vision and brain function, such as amblyopia (Tytla & Steinbach, 1984), closed head injury (Mattson, Levin, & Breitmeyer, 1994), developmental dyslexia (Williams, LeCluyse, & Bologna, 1990; Williams, Molinet, & LeCluyse, 1989), mania (Green, Nuechterlein, & Mintz, 1994a, 1994b), and schizophrenia (Green, Nuechterlein, & Breitmeyer, 1997; Green, Nuechterlein, Breitmeyer, & Mintz, 1999; Green et al., 1994a, 1994b; Merritt & Balogh, 1984; Saccuzzo & Schubert, 1981; Slaghuis & Bakker, 1995). Furthering our understanding of backward visual masking may, therefore, provide better clinical markers for these clinical conditions (Green et al., 1997; Williams et al., 1990; Williams et al., 1989).

In the following, we will outline and critically discuss several recent theoretical approaches to visual backward masking—most notably, three formal quantitative models—noting their positive features as well as their controversial aspects and shortcomings. Where possible, we also will note how the controversial aspects and shortcomings can be accommodated or corrected. These discussions will occur in the context of equally important recent empirical findings in visual masking.

RECENT THEORETICAL DEVELOPMENTS

A Neural Network Model of Retino-Cortical Dynamics

The dual-channel, sustained–transient approach to visual information processing has been very influential in the last three decades. Shortly after its introduction (Kulikowski & Tolhurst, 1973; Tolhurst, 1973), the sustained–transient approach was incorporated into theories of visual masking (Breitmeyer, 1980, 1984; Breitmeyer & Ganz, 1976; Matin, 1975; Weisstein, Ozog, & Szoc, 1975). A version of the sustained–transient theory of masking, proposed by Breitmeyer and Ganz, is illustrated in Figure 1. The theory originally made the following assumptions. (1) Sustained channels are involved in the relatively slow processing of object features, such as brightness, color, edges, and figural details. (2) Transient channels are involved in fast, coarse pattern processing and in signaling the spatial location or the change of spatial location (motion) of a stimulus. (3) Metacontrast masking and the related nonmonotonic, Type B backward masking is due to the interchannel inhibition of the slower responding, target-activated sustained channels by the faster responding, mask-activated transient channels. The assumption of interchannel inhibition had its neural analogue in the inhibitory interactions between X and Y neurons in the cat's lateral geniculate nucleus (LGN) and visual cortex (Hoffmann, Stone, & Sherman, 1972; Singer & Bedworth, 1973; Singer, Tretter, & Cynader, 1975; Stone & Dreher, 1973; Tsumoto & Suzuki, 1978). This interchannel inhibition has been interpreted as a neural analogue not only of metacontrast, but also of saccadic suppression (Breitmeyer & Ganz, 1976; Matin, 1974, 1975; Singer & Bedworth, 1973).

Even in unrevised form, the theory accounts for much of the masking data, as well as for a wide range of findings obtained from related areas of research (see chap. 7–10 in Breitmeyer, 1984). However, owing to developments in visual neurophysiology and neuroanatomy in the last two decades, as well as to the advent of new theories of cortical dynamics and competing theoretical accounts of masking, the sustained–transient approach itself is in need of revision and reevaluation. In the following, we present an updated sustained–transient approach to masking that incorporates recent findings in neuroscience and the dynamics of visual information processing.

One limitation of the early dual-channel models, as well as of the perceptual retouch (PR) model to be discussed below, is the use of discrete detectors as functional units. As a result, these models have difficulty in capturing distributed emergent properties of the nervous system that cannot be attributed to single neurons. In contrast, Bridgeman's (1971, 1978) neural network model, for example, explains the metacontrast phenomenon not by response patterns of single cells, but by analyzing collective distributed activities in the network. Likewise, the boundary contour system (BCS) model, to be discussed

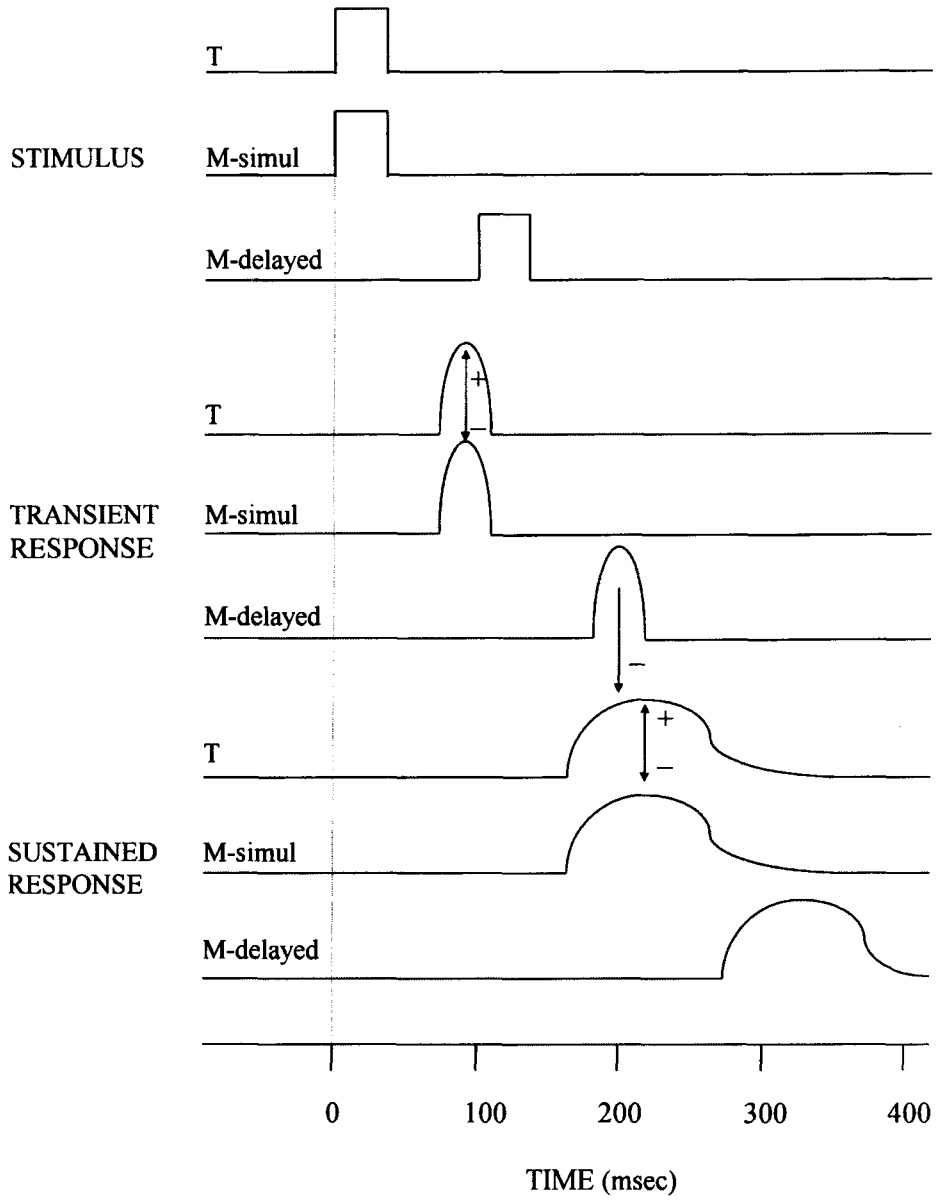

Figure 1. Neural interactions proposed by the sustained–transient theory. The top panel shows the timing of the target (T) and the mask (M) stimuli for zero (simultaneous target mask presentation) and positive (mask is delayed, metacontrast) stimulus onset asynchrony. The middle and lower panels illustrate neural responses generated at early stages (e.g., the retina) of transient and sustained pathways, respectively. The interactions proposed to occur between these signals at later stages of processing (see the text for a discussion of possible loci) are indicated by arrows. The + and − 2 signs indicate whether the interaction is excitatory or inhibitory, respectively. From "Implications of Sustained and Transient Channels for Theories of Visual Pattern Masking, Saccadic Suppression and Information Processing," by B. G. Breitmeyer and L. Ganz, 1976, *Psychological Review*, *83*, p. 17. Copyright 1976 by the American Psychological Association. Adapted with permission.

below, has a distributed neural network representation that was originally designed to explain boundary formation dynamics in the visual system. As was shown by Francis (1997), metacontrast is an emergent property of the model. Similarly, below we outline a neural network that not only incorporates the sustained–transient channel distinctions but also encompasses both the early and the late stages of visual processing and thus would correlate better with recent neurophysiological data.

A neural model of retino-cortical dynamics (RECOD) designed to incorporate dynamic, spatiotemporal properties of vision has been proposed recently by one of us

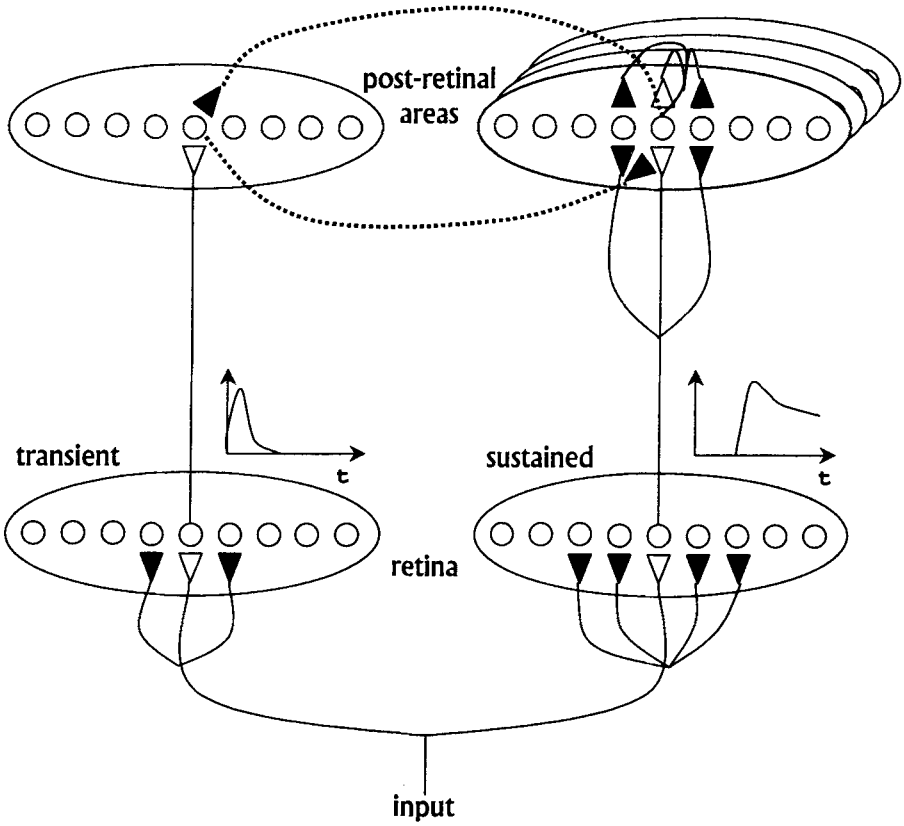

Figure 2. The retino-cortical dynamics model. The bottom two layers represent the sustained and the transient retinal ganglion cells, whose typical step-responses are illustrated next to each layer. The top layers represent their postretinal targets as lumped networks. The multiple copies of the sustained postretinal network represent spatial frequency channels, used to explain the perception and discrimination of blur over a broad range of physical stimulus blur profiles. The transient-on-sustained inhibition, shown by a dashed line, is used to implement the *reset phase of operation*. The reciprocal, sustained-on-transient inhibition, also shown by a dashed line, is added in order to take into account the effect of textured backgrounds. Open and filled synaptic symbols depict excitatory and inhibitory connections, respectively. From "A Neural Theory of Retino-Cortical Dynamics," by H. Ogmen, 1993, *Neural Networks*, *6*, p. 257. Copyright 1993 by Elsevier Science. Adapted with permission.

(Ogmen, 1993). The impetus behind the model was a study of neural mechanisms for spatial sharpening (deblurring) of contour information. Theoretical analysis showed that recurrent (feedback) inhibition is necessary to sharpen object boundaries and that the amount of sharpening depends critically on the relative strengths of afferent input signals and feedback signals. If the input signal to the recurrent network is relatively strong, as compared with the signal in the feedback pathway, the network does not generate any significant sharpening. Therefore, in order to achieve sharpening, the input signal should be much smaller than the signal in the feedback pathway. On the other hand, if the feedback signal is much stronger than the input signal, the cells in the recurrent network will be largely insensitive to changes in the input, because of the persistence of the signals in the recurrent loop. For example, if the stimulus moves, the recurrent signal at the new location will mainly override the feedforward signal generated at this location by the stimulus. In summary, theoretical analysis showed two conflicting tendencies in the recurrent network that need to be reconciled: (1) To avoid persistence in the feedback loop and to be able to read (accept and process) inputs, the input signal must be stronger than the feedback signal, and (2) to sharpen the activity, the feedback signal must be stronger than the input signal. A reconciliation to these conflicting tendencies was offered by the architecture shown in Figure 2.

The input is first encoded by transient and sustained retinal ganglion cells, shown as the bottom two layers in Figure 2, along with their typical responses to step inputs. The retinotopic inhibitory connections from the transient pathway to the sustained pathway, shown by dashed lines, are used to reset (i.e., curtail the persistence of) the feedback activity whenever the input changes at a given retinotopic location (reset phase). Sustained reti-

nal ganglion cells are used to send the input signal to the recurrent postretinal network. The initial overshoot of the response in the sustained cells (see the step response in Figure 2) puts the network in the feedforward dominant phase. As the step response of the sustained cells decays to its plateau, the strength of the afferent signal to sustained postretinal areas decreases, thereby allowing the network to enter the feedback-dominant phase, at which spatial sharpening occurs. Feedforward center–surround connections from sustained neurons to the postretinal network and feedback center–surround connections within this postretinal network are introduced to achieve spatial sharpening. A more detailed description of the postretinal network (not shown in Figure 2) takes into account some of the basic neurophysiology of the visual cortex—in particular, that concerned with the way cortical excitatory and inhibitory signals combine to give rise to oscillatory activity in the cortical sustained pathway (Azizi, Ogmen, & Jansen, 1996). These sustained oscillatory responses and their suppression by transient neural activation have been found in the cat visual cortex (Kruse & Eckhorn, 1996).

When presented with visual inputs, the model exhibits γ-range (40 Hz) oscillations with tight synchrony. Such γ-range oscillations with varying degrees of synchrony have been reported in cortical networks of several species (e.g., Livingstone, 1996; Maunsell & Gibson, 1992; Singer & Gray, 1995; Tallon-Baudry, Bertrand, Delpuech, & Pernier, 1996). Although several functional roles had been suggested for these oscillatory activities (Koch & Crick, 1994; Singer, 1994), no direct perceptual correlates were reported until the recent study of Fries, Roelfsema, Engel, Koenig, and Singer (1997) demonstrated that, in strabismic cats, the perception and the nonperception of monocular stimuli during binocular rivalry correlated, respectively, with increases and decreases in the strength and regularity of γ-range oscillations. Although controversial, recent human psychophysical results reported by Elliott and Mueller (1998) and Alais, Blake, and Lee (1998; see their Figure 1) indicate that pattern information presented at a 40-Hz flicker rate entrains and thus enhances the binding of visual features at preattentive levels of processing (Rensink & Enns, 1995; but see also Fahle & Koch, 1995, and Kiper, Gegenfurtner, & Movshon, 1996).

Analysis of the RECOD model shows that γ-range oscillations should occur in metacontrast masking. This prediction goes against the common acceptance in the literature. For example, the comparative analysis of metacontrast masking models published by Weisstein et al. (1975) noted oscillations in the masking functions generated by Bridgeman's (1971) model. However, this finding was used as one of the arguments for *rejecting* Bridgeman's model (Weisstein et al., 1975, p. 334). Subsequent implementations of this model by Bridgeman (1978) were based on a different statistical correlation measure (r^2 instead of r) that smoothed out oscillations predicted in Bridgeman's (1971) original model. That oscillations are not present in metacontrast masking functions has been echoed repeatedly in the literature, including recent reviews (e.g., Bachmann, 1994, p. 83). However, our own psychophysical experiments confirmed these model-based predictions of oscillating metacontrast functions (Purushothaman, Ogmen, & Bedell, 1997, 2000). The lack of systematic oscillations in prior metacontrast studies is most likely due to, along with the choice of stimulus parameters, the use of SOAs consisting of temporal sampling intervals that were too coarse. Thus, although rooted in a different theoretical question and analysis, the resulting model synthesizes two types of empirically based models proposed to explain the metacontrast phenomenon: the single-channel recurrent inhibition model, an adaptation and modification of the Hartline–Ratliff neural net (Ratliff, 1965), proposed by Bridgeman (1971, 1978), and the variety of dual-channel feedforward inhibition models proposed by Weisstein (1968), Weisstein et al. (1975), Matin (1975), and Breitmeyer and Ganz (1976). Theoretical analysis showed that these two types of models complement each other, rather than being competing alternatives (Ogmen, 1993).

The model makes a number of additional novel predictions, some of which have already been tested by psychophysical studies. For static targets, the predicted dependence of perceived blur and of blur discrimination on exposure duration agrees well with the data (Lacassagne, Bedell, & Ogmen, 1995). In addition, predicted blur discrimination thresholds as a function of base-blur follow a U-shaped curve, in agreement with the data (Lacassagne et al., 1995). For moving targets, the model predicts, in agreement with the psychophysical data (Chen, Bedell, & Ogmen, 1995), that an isolated target moving in front of a uniform background field should exhibit extensive smear, whereas its spatiotemporal flanking by other stimuli should reduce this smear through a transient-on-sustained inhibition (Purushothaman, Ogmen, Chen, & Bedell, 1998). This experimental paradigm is similar to the metacontrast paradigm, and the model's prediction of perceived smear is based on dual-channel mechanisms of previous metacontrast masking models (see Breitmeyer, 1984). Similarly, the RECOD model can be extended further by considering the effect of textured backgrounds. Inhibitory connections from sustained cells to transient cells, as was suggested by Breitmeyer (1984, 1986), can be used to prevent the suppression of a foveal sustained target during pursuit eye movements over a textured background.

Because the RECOD model lumps together postretinal areas, it cannot predict the physiological loci at which transient-on-sustained inhibition occurs. However, the partially unlumped version that generates oscillatory activities can be used to make some predictions, as we will discuss after reviewing the relevant neurophysiological data. The model also needs to be extended from boundary synthesis to figural processing. The proposed three-

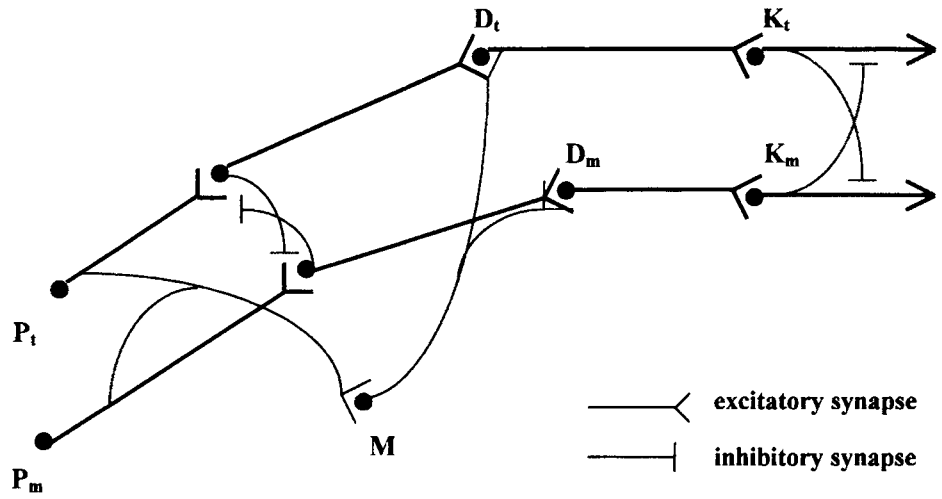

Figure 3. The perceptual retouch (PR) model. The specific pathway consists of receptors (P), detectors (D), and command neurons (K). The nonspecific pathway consists of the modulatory neuron (M), which pools its inputs from receptors and which projects diffusely to detector neurons. The subscripts t and m denote the cells activated by the target and the mask, respectively. From *Psychophysiology of Visual Masking: The Fine Structure of Conscious Experience* (p. 181), by T. Bachmann, 1994, Commack, NY: Nova Science Publishers. Copyright 1994 by Nova Science Publishers. Adapted with permission.

phase operation in the model can serve as the basis for such an extension, and its implications will be discussed below in the context of the neurophysiological data.

The Perceptual Retouch Theory of Visual Masking

A model of visual masking based on PR was introduced by Bachmann (1984) and elaborated more recently in his monograph on the psychophysiology of visual masking (Bachmann, 1994). Since interactions between activities of two anatomically distinct pathways assume a key role, one can consider it a two-process model (Bachmann, 1997). Although the model includes recurrent lateral inhibition at early stages in the afferent visual pathway, such inhibition plays a secondary role in backward masking by merely serving to preprocess target and mask representations that subsequently interact in a more crucial way, at later cortical levels, to yield the typically observed backward masking effects. Adhering as closely as possible to Bachmann's (1994) terminology and abbreviation scheme, we list the main assumptions and consequences of the model, as is illustrated in Figure 3. (1) A stimulus briefly activates both specific (SP) retinogeniculo-striate pathways and, via collaterals, nonspecific (NSP) retino-reticulo-cortical pathways. (2) Whereas the SP activity determines the contents of consciousness, the NSP activity generated in subcortical modulatory structures, M, is necessary for subjective awareness per se and arrives at the cortical locus containing the detector unit, D, with a latency that is about 50 msec longer than the SP activity. (3) For a stimulus to be consciously perceived, both SP impulses and NSP impulses must converge and overlap temporally at the same retinotopic cortical locus of D. Given these assumptions, a Type B backward masking function is generated by the following process of PR. Since the target and the mask stimuli, respectively, generate not only short-latency SP_t and SP_m activities, but also long-latency NSP_t and NSP_m activities, the temporal convergences of the SP and NSP impulses on the respective D loci are equal but nonoptimal for both the target and the mask when the two stimuli are presented simultaneously or else clearly successively (SOA = 0 msec, or else SOA > 150 msec). Thus, in either case, at D, the cortical loci of convergence of SP and NSP impulses, the signal-to-noise ratios representing the target and the mask stimuli are at equal but nonoptimal values. The outputs of the D_t and D_m units, respectively, activate the command units, K_t and K_m, through direct feedforward excitation but inhibit K_m and K_t through crossed feedforward inhibition. Since the excitatory and inhibitory inputs to K_t and K_m are equal, their outputs, correlating with the visibility of the target and the mask stimuli, in turn should be equal. However, owing to the latency difference of 50 msec between the SP and the NSP responses, an SOA of 50 msec produces an optimal temporal convergence of NSP_t and SP_m impulses at the retinotopic cortical locus D_m. Here, the signal-to-noise ratio of the cortical activity representing the mask attains its maximal value. Consequently, the excitatory activation of K_m will be significantly larger than that of K_t. Moreover, because of the feedforward inhibition, the output of K_t will be strongly inhibited, whereas that of K_m will not. The target's visibility, therefore, will be strongly suppressed while the mask's is enhanced.

Bachmann's PR approach to backward masking introduces several positive features. On the basis of a long history of well-known empirical findings, the existence of SP retino-geniculo-cortical processing pathways in vision is beyond dispute (for reviews, see Breitmeyer, 1992; Schiller, 1986; Shapley, 1992). Less well known but, nonetheless, equally indisputable from an empirical standpoint is the existence of NSP afferents that project to visual thalamic and cortical areas from subcortical reticular areas of the brainstem and midbrain (Frizzi, 1979; Hartveit, Ramberg, & Heggelund, 1993; Hassler, 1978; D. P. Purpura, 1970; Singer, 1977, 1979; Singer, Tretter, & Cynader, 1976; Steriade & McCarley, 1990). The latter, reticular complex has played an increasingly prominent role in recent formulations regarding its role in visual selective attention (Crick, 1984; LaBerge, 1995; LaBerge & Brown, 1989; Singer, 1994). Of relevance here is Berson and McIlwain's (1982) demonstration that direct (and indirect) inputs from transient Y ganglion cells of the cat retina can affect the responses of the deep-layer cells of the cat's superior colliculus. Moreover, S. B. Edwards, Ginsburgh, Henkel, and Stein (1979) have argued, on the basis of physiological and cytological criteria, that the cells in the deep layers of the superior colliculus ought to be classified as reticular, instead of collicular. If this scheme applies to humans, one has available, as is required by Bachmann's model, the anatomical, as well as physiological, bases for the activation of retino-reticulo-cortical (NSP) as well as retino-geniculo-cortical (SP) pathways when a brief target or mask stimulus is presented.

The role of brainstem and midbrain reticular activation in visual masking and information processing has been discussed extensively by one of us (Breitmeyer, 1984, chap. 10; 1986) as an addendum to the sustained–transient channel theory of visual masking introduced by Breitmeyer and Ganz (1976). The additions were introduced to account for the roles of selective attention and of visual exploratory behavior characterized by stimulus-guided fixation–saccade sequences in visual information processing (Breitmeyer, 1980). These considerations may be particularly relevant to recent findings showing that the magnitude of metacontrast masking is reduced by selectively attending to the target–mask area or configuration in the visual display (Enns & Di Lollo, 1997; Havig et al., 1998; Ramachandran & Cobb, 1995; Shelley-Tremblay & Mack, 1999). In relation to these findings, Bachmann's approach is consistent with a number of other relevant findings, including results predicted when pharmacological and neurosurgical changes to the sensitivity of the NSP activating system are introduced (Bachmann, 1994).

Furthermore, the PR model also accords well with the enhancement of the visibility of a stimulus when it is the second in a sequence of two (e.g., a mask following the target), relative to its being presented in isolation (Bachmann, 1988, 1994; Michaels & Turvey, 1979). These enhancement effects also may be related to contrast reversals of the first (target) stimulus when it is followed by a second (mask) stimulus (Brussell, Stober, & Favreau, 1978; Heckenmueller & Dember, 1965; Purcell & Dember, 1968). For example, under suitable conditions, a black circular target followed by a black annular surround may, at optimal masking SOAs, actually appear brighter than the white background on which the target–mask sequence is flashed. In Bachmann's PR model, both the enhancement of the second stimulus and the contrast reversal of the first stimulus could be explained by the enhanced output of D_m neurons, relative to D_t neurons, and consequently, a strong inhibition of K_t neurons, relative to a strong excitation of K_m neurons. This interpretation of the PR model assumes that it is the activity levels of the K neurons that determine the perceptual salience (brightness, contrast, clarity) of the stimuli.

In light of this interpretation, an easily correctable shortcoming in Bachmann's (1994) discussion of the PR model is a lack of specificity as to which of the two sets of neurons, the D (detection) or the K (command) neurons, is to be associated with the perceived aspects of the stimuli. At one point, Bachmann (1994, pp. 180, 183–184) claims that the perceptual efficiency and subjective clarity of a stimulus is directly proportional to the level of activity produced by the stimulus in the corresponding D neuron. If so, it is hard to understand why, as in metacontrast and other forms of backward pattern masking, the target stimulus is phenomenally absent or, at times, even contrast reversed (Brussell et al., 1978; Heckenmueller & Dember, 1965; Purcell & Dember, 1968). On the basis of the NSP-enhanced activities of the D neurons activated by their SP inputs only, one would expect that the mask would be seen more vividly than the target, but not that the target would be phenomenally invisible or contrast reversed. Alternately, Bachmann (1994, p. 183) argues that the K neurons (or later stage gnostic units) generate responses—for example, perceptual categorization and recognition—to their corresponding stimuli. In fact, in the appendix to his monograph (Bachmann, 1994, p. 221, Equations 20 and 21), where the PR model is given mathematical expression, the probability or efficiency of correct recognition of a stimulus is related to the response of the command neurons activated by their respective stimuli. We feel that this ambiguity in the PR model, although minor, should be resolved in favor of the latter version, which accords better with existing data. Correcting these "tuning" problems with Bachmann's (1994) PR model would be easy and would enhance its explanatory power.[1]

In addition to an enhanced visibility of the second of two stimuli, the PR model predicts that the second stimulus will appear in consciousness faster, when compared with an isolated presentation of the same stimulus. Bachmann (1994) presents data putatively supporting this prediction.[2] As a corollary, the PR model also predicts that the phenomenal order of appearance of a target and a following mask stimulus can be *reversed* with an appropriate choice of SOAs, such as those giving rise to maximum metacontrast suppression. To our knowledge,

no extant data bear on this prediction. The relevant experiment remains to be conducted.

Francis's Analysis of the Boundary Contour System Model

The BCS is a model of a cortical neural network proposed by Grossberg and Mingolla (1985a, 1985b) to account for spatial aspects of the visual perception of stationary, steady-state stimuli. By exploring its dynamic properties, Francis (1996a, 1996b, 1997; Francis & Grossberg, 1996a, 1996b; Francis, Grossberg, & Mingolla, 1994) has recently demonstrated its ability to account also for a variety of empirical findings obtained when visual inputs change rapidly over time. The model is a cooperative–competitive network incorporating, besides the obvious afferent feedforward excitatory drive, three key properties: excitatory feedback, feedforward inhibition, and inhibitory feedback. Since the BCS—at least with respect to form perception of stationary stimuli—relies on the cortical P pathway (Grossberg, 1994), one can consider these mechanisms to operate within a single channel. Figure 4 schematizes the BCS model. At the earliest levels of processing, it consists of unoriented, contrast-specific filters (on-center, off-surround cells) whose outputs feed into oriented, contrast-specific simple cells. The rectified outputs of these units, in turn, feed into complex cells selective for the same orientation but insensitive to contrast polarity. At the first competitive stage, the complex cells project their outputs via on-center, off-surround connections to first-level hypercomplex cells. Because of the off-surround connections, these hypercomplex cells, while remaining orientation selective, additionally are selective for end-stopped stimuli (Hubel, 1988; Hubel & Wiesel, 1965). At the next stage, competition among orientations results when higher order hypercomplex cells are activated via antagonistic inputs from the lower order hypercomplex cells tuned to different orientations. The outputs of these higher order hypercomplex cells, which specify the location of oriented stimulus boundaries in the visual field, feed into cooperative bipole cells. The bipole cells generate feedback that, on the one hand, excites location- and orientation-consistent patterns of activity and, on the other, inhibits inconsistent patterns of activity.

The temporal dynamics of this model account for a number of empirical regularities characterizing visual persistence (Francis, 1996a, 1996b). Persistence arises through activation of the excitatory feedback loops, resulting in long-lasting reverberatory activity in the BCS network. When stimuli change or move, this persistent activity would be problematic in that it could give rise to forward masking (Breitmeyer, 1980, 1984) or to motion smear (Chen et al., 1995). In the BCS model, such persistence can be curtailed in one of two ways: (1) by a gated-dipole mechanism (Francis et al., 1994) that, at stimulus offset, produces a reset signal inhibiting the persisting reverberatory activity or (2) by lateral inhibition operating mainly at the first competitive stage (Francis, 1996a). According to Francis (1996a, 1997), the latter mechanism of lateral inhibition is the more significant contributor to the dynamics of stimulus boundary erosion over time under metacontrast conditions.

Figure 5 shows schematically how the model accounts for key properties of metacontrast. The left panel depicts the effect of the mask on the target at short SOAs. The solid bold curve illustrates the response of a target-activated hypercomplex cell. Because hypercomplex cells receive positive feedback signals, their response persists after the offset of the stimulus. The lateral feedforward inhibitory signal generated by the mask at the first competitive stage is, however, outside the feedback loop, and its duration is shorter. Consequently, the effect of the mask is to reduce transiently the activity of the hypercomplex cells responding to the target, as is shown by the dashed curve. The net response to the target, after taking into account the inhibitory effect of the mask, is shown by the dotted area in Figure 5. As the SOA increases, this transient suppression starts to occur at the weaker portions of the response, as is shown in the middle and right panels of Figure 5. The amount of suppression of the target activity is weak for short SOAs, owing to the presence of the strong feedback signal. As the strength of this feedback signal decays at midrange SOAs, the amount of suppression becomes stronger. At longer SOAs, the net effect of inhibition becomes smaller, because the target-generated activity has already decayed (Figure 5, right panel). The model uses the linking assumption according to which the final visibility, or perceptual quality, of the target is proportional to the *duration* of the boundary signals generated by the target. Therefore, the magnitude of masking will be given by the *change in this duration*. As is shown in the left panel of Figure 5, although the mask suppresses part of the target-generated responses at short SOAs, this suppression does not have any significant effect on the duration of the signal, thereby yielding very weak masking. Similarly, at long SOAs, only part of the inhibitory signal overlaps with the decaying target activity, and the change in duration will be relatively small. As a result, the strongest masking is obtained at intermediate SOAs, yielding a U-shaped masking function. From this simplified analysis, one can see that any change in target stimulus parameters that modifies the neural response to the target (e.g., changes in target energy, by varying either its luminance [Weisstein, 1972] or its duration [Schiller, 1965]) will affect the shape of the metacontrast function. Similarly, since the strength of feedforward inhibition is directly related to the strength of the mask stimulus, changes in mask stimulus parameters (energetic or spatial) are also predicted to affect metacontrast (Breitmeyer, 1978b; Di Lollo, Bischof, & Dixon, 1993; Sherrick & Dember, 1970). Finally, several spatial and temporal properties of target recovery (disinhibition) produced when introducing a second mask to the target–mask metacontrast sequence (Breitmeyer, 1978a; Breitmeyer, Rudd, & Dunn, 1981) are explainable by the component of the inhibitory signal arising from

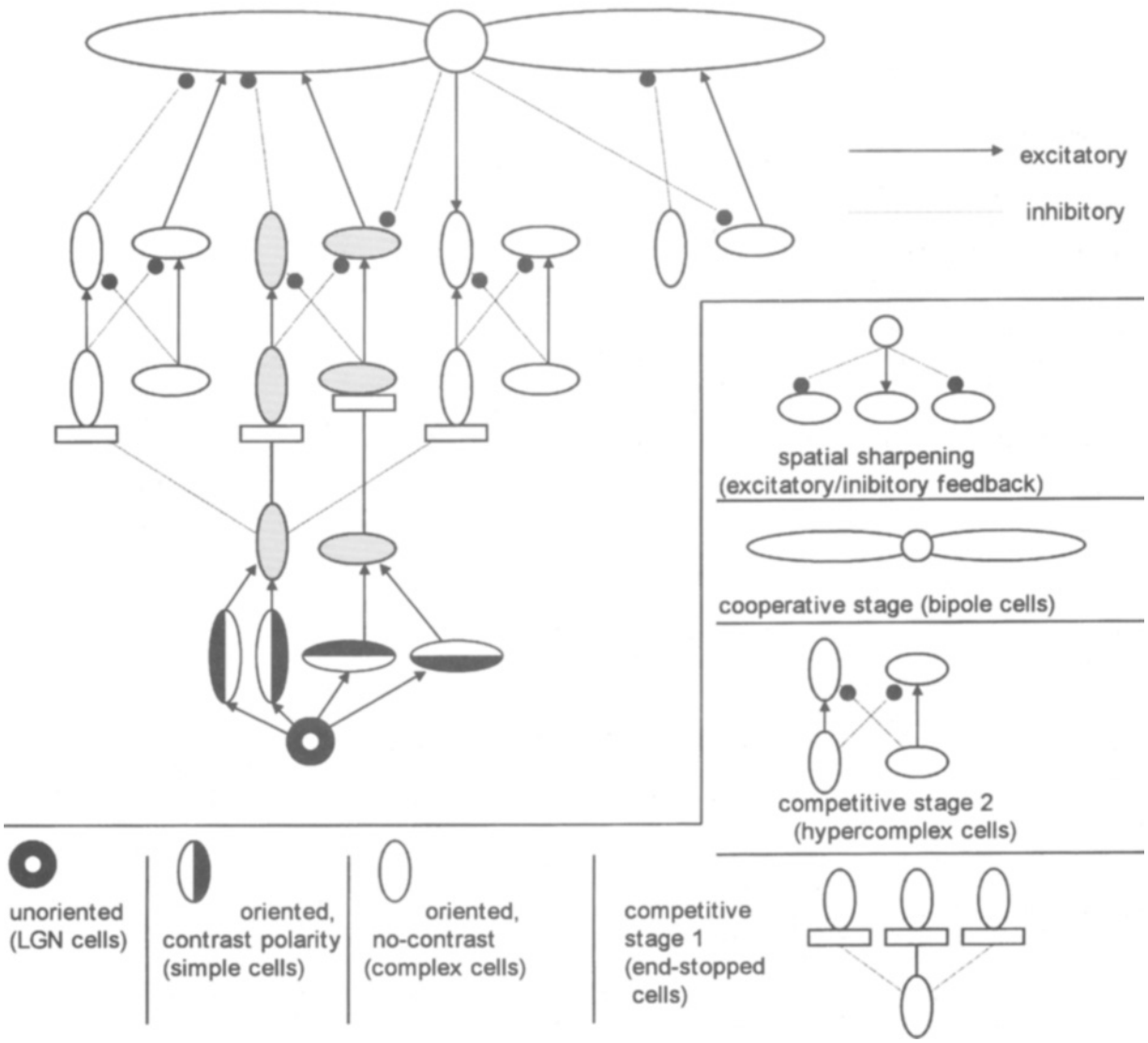

Figure 4. The boundary contour system model. Heavier and lighter lines represent excitatory and inhibitory connections, respectively. The first competitive stage consists of feedforward on-center off-surround connections among spatially neighboring cells that have the same orientational tuning. The second competitive stage is a *push–pull* type interaction at every retinotopic position among cells with opponent orientation preference (e.g., horizontal vs. vertical). The bipole cells pool the outputs of hypercomplex cells in an orientation-selective manner. The on-center off-surround feedback from bipole cells implements spatial sharpening. From "Cortical Dynamics of Lateral Inhibition: Metacontrast Masking," by G. Francis, 1997, *Psychological Review*, *104*, p. 574. Copyright 1997 by the American Psychological Association. Adapted with permission.

the inhibitory feedback pathway implementing spatial sharpening.

A comparison of the BCS and the RECOD models shows that, in both models, feedforward and feedback inhibition within the sustained pathway are used for spatial sharpening. The major temporal dynamics in the BCS model are proposed to occur at the cortical level, which enjoys a highly detailed description, whereas the retinogeniculate system is approximated by steady-state equations. In contrast, the RECOD model, although having a much simpler (lumped) representation of postretinal areas, incorporates detailed retinal dynamics. Hence, in BCS many properties of backward masking are explained by cortical feedforward lateral inhibition, whereas RECOD uses transient-on-sustained inhibition, as in previous dual-channel models (Breitmeyer & Ganz, 1976; Matin, 1975; Weisstein et al., 1975).

As was mentioned above, the extant findings of backward masking are voluminous, revealing not only the main and highly replicated empirical regularities, but also more specific effects. So far, the BCS model has been able to account in a robust manner for a number of these regularities and specific effects. However, much work remains to be done to account for the vast remainder of

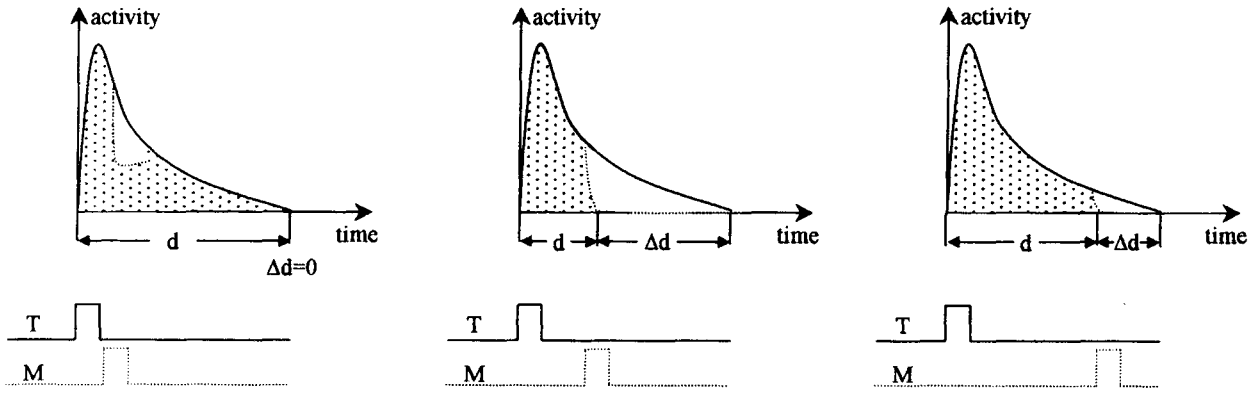

Figure 5. Simplified depiction of temporal interactions leading to metacontrast effects in the boundary contour system model. The left, middle, and right panels illustrate target and mask stimuli at short, midrange, and long stimulus onset asynchronies, respectively. The temporal profile of the target (T) and mask (M) stimuli are shown by the traces at the bottom of the graphs. The solid curve depicts the response that the target would generate in the absence of the mask. The dashed curve shows the inhibitory influence coming from the mask stimulus. The dotted area is the net response to the target stimulus after taking into account the inhibition exerted by the mask stimulus. The duration of this net response is shown by *d*. The change in this duration owing to the presence of the mask, Δd, is used to compute the magnitude of masking.

findings. Unfortunately, a practical limitation of this model, as well as of similar network models, is that the solutions to the network's differential equations are very time consuming (see the appendix of Francis, 1997), and this may prohibit extensive testing of the model. Despite this practical limitation, the model already manifests a wide explanatory scope, providing an integrative scheme accounting for major findings on visual persistence, temporal integration, metacontrast masking, and apparent motion (Francis, 1996a, 1996b, 1997, 1999; Francis & Grossberg, 1996b; Francis et al., 1994). Moreover, the model makes several explicit predictions regarding psychophysical and neurophysiological findings. A particularly strong psychophysical prediction is that optimal metacontrast masking ought to occur at a constant interstimulus interval (ISI) between target offset and mask onset, despite variations of the target duration. This prediction can be visualized from Figure 5 by noticing that the suppressive effect of the mask becomes significant when the target-generated activity starts to decay and becomes weak, which in turn occurs after the offset of the target. Therefore, the optimal delay for the mask can be measured from the offset of the target stimulus irrespective of the duration of the target, provided that changes in the duration of the target do not significantly alter the poststimulus activity. A prior explanation of metacontrast has explicitly (Kahneman, 1967) taken the SOA, rather than the ISI, to be the critical temporal parameter specifying optimal backward masking. Since SOA = D_t + ISI (where D_t = duration of the target), this model predicts that the optimal ISI should decrease, rather than remain constant, as target duration increases. We take up the discussion of this and related issues at a later, more opportune point.

RECENT PSYCHOPHYSICAL FINDINGS

Type B Masking Functions and Contour-Sensitive Interactions

Enns and Di Lollo (1997) recently reported results that they, although not specifying neural mechanisms, attribute to high-level processes of object substitution, rather than to more traditional and, presumably, lower level contour-sensitive mechanisms of metacontrast masking. In their study, the contour discriminability of a diamond-shaped target with either the left or the right corner missing was measured when masked by a surrounding diamond-shaped mask or by a mask made of four dots falling at the corners of a notional square that would enclose the diamond target. Without going into the details of the findings, the key results showed that the four-dot mask, like the contour-adjacent diamond-shaped mask, could produce substantial Type B masking when (1) the target and the mask were presented 3° peripherally, but not when they were presented foveally, and (2) attention was distributed over large areas of visual space—that is, when attention could not be readily focused on a given target–mask location. Enns and Di Lollo argued that their data cannot be explained by contour-based mechanisms of metacontrast because (1) the four-dot masking did not show significant sensitivity to target-contour–mask-contour separation, (2) none of the masking models based on contour-sensitive mechanisms predicts differential masking outcomes as a function of attention, and (3) under conditions of distributed attention, not only backward masking is obtained, but also simultaneous and forward masking.

As was noted by Enns and Di Lollo (1997), these findings pose problems for masking models based strictly on

contour-sensitive mechanisms. We agree, but we hold that the following facts also need to be given consideration. We suggest that the findings of object substitution outside the fovea, but not at the fovea, can be alternately explained by realizing (1) that, as was shown previously (Breitmeyer, Battaglia, & Weber, 1976; Breitmeyer, Love, & Wepman, 1974), target–mask contour proximity is not a necessary condition for obtaining metacontrast masking, particularly when discrimination of target contour is used to measure masking magnitude; (2) that metacontrast masking is stronger and more immune to target–mask contour separations as viewing eccentricity increases (see Breitmeyer, 1984, pp. 108–109, 219); (3) that there is a close but not unalterable relationship (Posner, 1980) between the fovea as an anatomical and as a functional locus of visual attention; and (4) that contour discriminability and other forms of stimulus visibility, whether in masking (Havig et al., 1998; Ramachandran & Cobb, 1995; Shelley-Tremblay & Mack, 1999) or in other psychophysical tasks (Bashinski & Bacharach, 1980; LaBerge, 1995; Posner, 1980; Sagi & Julesz, 1985), benefit from focused attention. For instance, consider the finding that attending to the locus of a stimulus increases its detection sensitivity, d' (Bashinski & Bacharach, 1980). The fact that attention modulates the sensitivity of the visual system in a detection task is no warrant for ruling out the role of lower level detection units in signal detection. Likewise, a modulation of target visibility when attentional allocation to it varies does not warrant ruling out the role of lower level contour inhibitory mechanisms. By offering this alternative way of explaining the results reported by Enns and Di Lollo, we are not ruling out the possible role of object substitution as a masking mechanism. We are simply asserting that their results are not decisive in choosing exclusively between explanations based on object substitution and those based on contour-inhibitory interactions between target and mask neural activities. The latter interactions, in conjunction with variations of attentional focus and the attendant possibility of various levels of "inattentional blindness" (Mack & Rock, 1998), also can account for their results (Shelley-Tremblay & Mack, 1999).[3]

Nonetheless, an important upshot of Enns and Di Lollo's (1997) findings for visual-masking theories is the sharper refocus they place on late object-specific (Williams & Weisstein, 1981) and attentional (Michaels & Turvey, 1979) levels of cortical processing that cortical contour-interactive processes alone cannot explain (Breitmeyer, 1984; pp. 256–261). Such an approach dovetails nicely with evidence for an interruptive mechanism of visual masking, located in or no later than the inferotemporal (Kovács et al., 1995; Rolls & Tovée, 1994; Rolls et al., 1999). Since spatial selective attention powerfully modulates the responses of visual cells, especially in extrastriate and later cortical areas (Moran & Desimone, 1985; Motter, 1993; Sato, 1988; Schiller & Lee, 1991), such interruptive masking would be particularly consistent with the view of an attention-modulated masking at these later levels of cortical processing and, specifically, with a mechanism of object substitution (Enns & Di Lollo, 1997).

Distinctions Between Contour and Area Suppression in Metacontrast Masking

An interesting aspect of metacontrast masking is that it suppresses not only the visibility of stimulus contours (Breitmeyer, 1978b; Breitmeyer et al., 1974; Burchard & Lawson, 1973; Westheimer & Hauske, 1975), but also the brightness or contrast of a spatially uniform stimulus (Alpern, 1953; Flaherty & Matteson, 1971; Weisstein, Jurkens, & Onderisin, 1970). In particular, as was shown by Stoper and Mansfield (1978), metacontrast suppresses the visibility of the contourless area enclosed by, and well away from, the edges or boundaries of a uniform target stimulus. Stoper and Mansfield called this type of masking *area suppression* and argued that masking results depend on two contrast mechanisms. One mechanism processes the contour or boundary contrast of a stimulus, and the other, related to perceptual filling-in (Gerrits & Timmerman, 1969; Gerrits & Vendrik, 1970), processes the brightness or area contrast. The distinction between boundary and area contrast has been around for some time (Hering, 1878; Mach, 1865) and has been attributed to "Mach-type" and "Hering-type" lateral inhibition by von Békésy (1969). More recently, it has been correlated with cortical neural responses to the boundary of a stimulus that precede, by about 100 msec, the neural responses to the interior of the stimulus (T. Lee, Mumford, & Schiller, 1995). Paradiso and Nakayama (1991) investigated the spatial and temporal parameters affecting brightness perception and filling-in, using a forward/backward masking paradigm. On the basis of their extensive findings, Paradiso and Nakayama noted that their results pose problems for several extant models of masking and metacontrast that rely on lateral contour-inhibiting processes. Like Stoper and Mansfield, they propose the necessity of filling-in processes to explain their results.

Although the BCS model accounts for contour-dependent contrast effects, but not for area contrast effects, Grossberg and co-workers (Cohen & Grossberg, 1984; Grossberg, 1983, 1994; Grossberg & Todorović, 1988) have proposed a complementary model that includes a feature contour system (FCS) for the description of perceptual surface attributes, such as the brightness, color, and texture of areas internal to the boundaries of visual stimuli. Although Paradiso and Nakayama (1991) argued, on qualitative grounds, that their results could not be accommodated by the BCS/FCS model, Arrington (1994) subsequently showed that it, in fact, can provide an adequate quantitative account of their results. It seems, therefore, that an elaboration of current models of visual masking is required. Such a model, incorporating not only boundary contrast effects, but also effects of area contrast and filling-in, is needed to account for the results reported by Stoper and Mansfield (1978) and Paradiso and Nakayama. To extend their explanatory scope to include these results, both Francis's (1997) analysis of the BCS

model and Ogmen's (1993) dual-channel model of retinocortical dynamics would have to incorporate either the FCS directly or some other component specifying the processing of internal stimulus areas.

Grossberg (1994) draws an explicit correspondence between the BCS and the FCS, on the one hand, and the cortical P-interblob and P-blob processing streams, on the other (for overviews of cortical processing streams, see DeYoe & Van Essen, 1988, and Van Essen, Anderson, & Felleman, 1992). Thus, both boundary contrast effects and area contrast effects can be viewed as mask-induced modifications of activity in the cortical P pathways. This view would be consistent with either the BCS/FCS or the RECOD model of cortical dynamics—in the former, by relying on intrachannel (sustained-on-sustained) inhibition; in the latter, by relying on interchannel (transient-on-sustained) inhibition. In this connection, however, it should be mentioned that the mechanisms not only for boundary contrast effects, but also for area contrast effects may be operative at earlier than cortical levels of processing. Li, Pei, Zhow, and von Mitzlaff (1991) recently demonstrated how mathematically modeled properties of retinal and geniculate X cells, particularly of the extensive disinhibitory regions surrounding their classical receptive fields, can account for area or Hering contrast effects (but see Rossi & Paradiso, 1999, for counterindicative findings). Even if subcortical processes are involved, the mask-induced modifications of area contrast found by Stoper and Mansfield (1978) and by Paradiso and Nakayama (1991) nevertheless would require cortical mechanisms, not only for theoretical reasons, but also because these effects, as well as the standard boundary contrast masking effects, can be obtained dichoptically.

Wavelength Effects on Metacontrast Masking

We noted that the X and Y cells investigated in cats originally were taken as neural analogues of human sustained and transient channels. Owing to developments over the past 20 years in the study of parallel pathways in monkey vision, the sustained–transient approach to masking, in revised form (Breitmeyer, 1992), takes the parvocellular (P) and magnocellular (M) pathways as neural analogues of these channels. It is sometimes found (Hicks, Lee, & Vidyasagar, 1983; Krueger, 1979; B.B Lee, Martin, & Valberg, 1988, 1989a, 1989b) and, hence, claimed (Livingstone & Hubel, 1988; Ramachandran, 1990; Skottun & Parke, 1999) that stimuli produced by isoluminant hue substitutions produce little, if any, activity in the M pathway. However, neurophysiological evidence indicates that M cells indeed can respond to isoluminant stimuli varying only in wavelength, provided that the cone contrast is sufficiently high—as it is with stimuli at red–green (or blue–yellow) isoluminance (Krueger, 1979; B. B. Lee et al., 1988; Schiller & Colby, 1983). In particular, B. B. Lee et al. (1989b) note that nonlinearities occurring at or before the summing of medium- and long-wavelength cone inputs to the M cells could produce their brisk responses to red–green isoluminant borders. Hence, transient-masking paradigms should yield masking effects with stimuli consisting not only of luminance changes, but also of isoluminant hue changes that also produce large cone-contrast changes. Strong masking effects with red–blue hue substitutions have in fact been reported in the Crawford (masking-by-light) paradigm by Glass and Sternheim (1973), and strong metacontrast masking effects were reported also with red–green hue substitutions by Reeves (1981) and by Breitmeyer, May, and Heller (1991).

An interesting feature of the foveal and parafoveal M pathway is the long-wavelength sensitivity of receptive-field inhibitory surrounds and the consequent suppression of M pathway activity by diffuse red light (De Monasterio, 1978a, 1978b; De Monasterio & Schein, 1980; Dreher, Fukuda, & Rodieck, 1976; Livingstone & Hubel, 1984; Marrocco, McClurkin, & Young, 1982; Van Essen, 1985; Wiesel & Hubel, 1966). Assuming that transient-on-sustained inhibition contributes to backward pattern masking, one should find weaker metacontrast masking when stimuli are presented on red backgrounds than when presented on equiluminant white, green, or blue backgrounds. Such results have been reported in a number of metacontrast studies (Breitmeyer et al., 1991; Breitmeyer & Willliams, 1990; V. T. Edwards, Hogben, Clark, & Pratt, 1996; Williams, Breitmeyer, Lovegrove, & Gutierrez, 1991). Moreover, these effects of background wavelength on human transient M pathway activity and, thus, on metacontrast recently have been corroborated in a number of other psychophysical paradigms, including simple RT (Breitmeyer & Breier, 1994), choice RT discriminations between categorical and coordinate spatial stimuli (Roth & Hellige, 1998), and choice RT discriminations between local and global stimuli (Michimata, Okubo, & Mugishima, 1999).

Parameters Specifying the Peak Masking Effects in Type B Masking Functions

The results of a recent study of metacontrast reported by Macknik and Livingstone (1998) compared human psychophysical masking functions to responses of single cells in cortical area V1 of the monkey. The psychophysical results showed that, for targets of 140 msec and briefer, the SOA at which peak masking occurred did not remain constant but, rather, ranged from 20 to 200 msec, depending on the durations of the target and the mask. When the same results were plotted in terms of ISI, the ISI at which peak masking occurred ranged from about −50 to 40 msec. Finally, when these results were plotted in terms of stimulus termination asynchrony (STA: the time between the offset of the target and the offset of the mask), the STA at which peak masking occurred ranged from 80 to 120 msec. On the basis of these results, Macknik and Livingstone concluded that, for the targets used by them, "STA is the best descriptor of the time of peak backward masking, as the dispersion of the peak masking times was lowest when plotted on the STA scale" (p. 145). Other parameters, such as the SOA, as proposed by Kah-

neman (1967), or the ISI, as the BCS model predicts, were less adequate.

Although we find Macknik and Livingstone's (1998) results and interpretations to be novel and theoretically interesting, we feel that they raise the following points that need to be addressed. It is far from clear which, if any, temporal parameter—SOA, ISI, or STA—is the best or the critical one for explaining the peak masking effect during backward masking. Their results tend to support this tentative view, since there also was noticeable (although less) variability in the peak STAs, as compared with the ISIs and SOAs. It should also be mentioned that the peak SOA, and by extension the peak ISI, is a function of several variables, including light adaptation level (Alpern, 1953; Purcell, Stewart, & Brunner, 1974; Stewart & Purcell, 1974), the ratio between target and mask energies (Fehrer & Smith, 1962; Hellige, Walsh, Lawrence, & Prasse, 1979; Kolers, 1962; Stewart & Purcell, 1974; Weisstein, 1972), the degree to which the mask depicts a three-dimensional (3-D) object (Williams & Weisstein, 1981), the degree to which the mask's figural and semantic features resemble those of the target (Hellige et al., 1979; Michaels & Turvey, 1979), and under dichoptic viewing, whether the target is presented to the dominant or the nondominant eye (Michaels & Turvey, 1979). Therefore, to speak of the SOA or the ISI as though backward masking functions should yield peaks at a fixed value is clearly unrealistic. It should be noted here that the BCS model, like the sustained–transient model (Breitmeyer, 1978b), can account for changes of peak masking produced by changes in target/mask energy ratios. Moreover, the importance of STA, as well as of ISI, emerges from considerations of the BCS model. Although the model predicts that the peak in metacontrast masking occurs at a constant ISI even as target duration varies, the model also predicts that the particular ISI at which peak masking occurs shifts to lower values as mask duration increases (compare panels C and D of Figure 14 in Francis, 1997). Note (1) that STA = ISI + D_m, where D_m is the mask duration, and (2) that a decrease in peak ISI is associated with an increase in D_m. Consequently, the peak STA would tend to remain constant, consistent with the results reported by Macknik and Livingstone.

When discussing any of these temporal parameters, the target/mask energy ratio is of crucial importance (Fehrer & Smith, 1962; Hellige et al., 1979; Kolers, 1962; Stewart & Purcell, 1974; Weisstein, 1972). Note that STA = ISI + D_m = SOA − D_t + D_m, where D_t is the duration of the target. Other things being equal, in Macknik and Livingstone's (1998) study, the target/mask energy ratio was directly proportional to D_t/D_m. As this ratio increases (e.g., as D_m decreases or D_t increases), one would expect to get shifts in the peak masking toward higher SOAs; conversely, as the ratio decreases, the shift ought to be toward lower SOAs. In terms of SOA, Macknik and Livingstone's results support this trend. Moreover, any of the temporal parameters specifying peak masking, in order to attain empirical generality, must be able to accommodate changes in the peak as light adaptation level changes (Alpern, 1953; Purcell et al., 1974; Stewart & Purcell, 1974).

RECENT ELECTROPHYSIOLOGICAL FINDINGS

Temporal Properties

Onset and offset responses. In addition to their psychophysical findings, Macknik and Livingstone (1998) also reported electrophysiological results. In particular, they reported that, in addition to neural responses to the onset of stimuli, responses to the offset of the target and the mask stimuli are important correlates of visibility. Macknik and Livingstone recorded primarily from V1 complex neurons (Macknik, personal communication, February 15, 2000), which not only responded briefly to the onset of a stimulus but also responded with a discharge after the offset of a stimulus. In these neurons, a spatially flanking forward mask (paracontrast) produced strong suppression primarily of the onset response and only occasionally of the offset response; however, a spatially flanking backward mask (metacontrast) suppressed only the after-discharge. Macknik and Livingstone concluded that changes of target visibility during paracontrast are correlated with changes in the neural onset response, whereas changes of target visibility during metacontrast are correlated with changes in the after-discharge.

Since these interpretations proposed by Macknik and Livingstone (1998) posit that U-shaped backward masking is correlated selectively with changes in the after-discharge of V1 (complex) neurons, it is important, for the sake of generality, to establish that such after-discharges exist for stimulus durations equal not only to the 60 msec or longer used by Macknik and Livingstone, but also for stimulus durations as short as 1 msec (Bischof & Di Lollo, 1995). Bridgeman's (1975, 1980) single-cell studies of cat and monkey cortical cells indicate that such after-discharges ought to be found for brief stimulus durations, as do the results of Duysens, Orban, Cremieux, and Maes (1985), Rolls and Tovée (1994), Kovács et al. (1995), and Macknik's extensive dissertation studies (Macknik, personal communication, February 15, 2000).

Latency and transience of neural responses. Of the three models discussed above, the sustained–transient approach to masking places emphasis on two temporal parameters defining neural responses: (1) latency (i.e., the time it takes to reach the peak response for a step stimulus) and (2) transience (i.e., the ratio of the initial peak response to the subsequent steady-state [plateau] response for a step stimulus). In the primate visual system, the cells in the M pathway generally exhibit shorter latencies and higher transience than do cells in the P pathway, although there exists a considerable overlap in terms of population properties (DeMonasterio, 1978b; Maunsell, 1987; Maunsell et al., 1999; Maunsell & Gib-

son, 1992; Maunsell & Schiller, 1984; Nowak, Munk, Girard, & Bullier, 1995; Petersen, Miezin, & Allman, 1988; K. Purpura, Tranchina, Kaplan, & Shapley, 1990; Schiller & Malpeli, 1978; Schmolesky et al., 1998). In terms of transience, neurophysiological data show a clear distinction between P and M cells at the level of both the retina and the LGN (K. Purpura et al., 1990; Schiller & Malpeli, 1978). In terms of latency, although most recent studies show a consistent latency difference between M and P neural responses (Maunsell, 1987; Maunsell et al., 1999; Maunsell & Gibson, 1992; Maunsell & Schiller, 1984; Nowak et al., 1995; Petersen et al., 1988; Schmolesky et al., 1998), K. Purpura et al.'s investigation of retinal M and P cells failed to find consistent differences. It should be noted that the studies reporting consistent latency differences (1) recorded form cortical or geniculate cells, whereas K. Purpura et al. recorded from retinal cells, and (2) used a "direct" method of measuring latencies to the onset of flashed stimuli, whereas Purpura et al. indirectly estimated latencies by applying a linear cascade model to derive neural impulse responses from the steady-state responses recorded to drifting sine-wave gratings. Despite these methodological differences possibly accounting for the differences in latency measures, it is also possible that the fast-transient and slow-sustained distinction corresponds only to some subpopulations of M and/or P pathways. For example, "X-like" and "Y-like" subpopulations of the M system have been proposed (Benardete, Kaplan, & Knight, 1992; K. Purpura et al., 1990; Shapley, 1992). Moreover, at the retinal level, even sustained P ganglion cells exhibit ratios of (initial) peak to (lagging) plateau for step responses (K. Purpura et al., 1990) that can qualify as "transient."

Electrophysiological Findings and the Locus of Metacontrast Mechanisms

Mechanisms proposed to explain the U-shaped function have included peripheral ones, such as retinal interactions among photoreceptors (Alpern, 1953, 1965), up to high-level cortical mechanisms, such as PR (Bachmann, 1984, 1994) or object substitution (Enns & Di Lollo, 1997). The fact that pronounced U-shaped metacontrast effects can be obtained when the target and the mask are presented dichoptically (Breitmeyer & Kersey, 1981; Kolers & Rosner, 1960; May, Grannis, & Porter, 1980; Schiller & Smith, 1968; Weisstein, 1971; Werner, 1940) is a strong indication that the mechanism is located cortically at or beyond the site of binocular integration. Nonetheless, as was noted by Breitmeyer (1984, chap. 4), the exact shape, magnitude, and optimal masking SOA is also influenced by properties of mechanisms located as early as the receptor level (Foster, 1976, 1978, 1979; Foster & Mason, 1977). Although the mechanisms at the peripheral level of visual processing may affect the shape or magnitude of the metacontrast function, the consensus among current theoretical approaches to metacontrast (see above) is that the mechanism responsible for the U-shaped masking effect is cortical. What remains controversial is specifying more precisely its level in the cortical processing stream.

Evidence from animal studies: Early cortical levels. Although Breitmeyer and Ganz (1976), in their initial sustained–transient approach to visual masking, included the LGN as a locus of interchannel, transient-on-sustained inhibition, an updated version of the approach, which takes into account differences between X and Y pathways in the cat and P and M pathways in primates (Lennie, 1980; Singer & Bedworth, 1973), assumes that the interchannel inhibition is of cortical origin. In particular, Breitmeyer (1984) subsequently proposed primary visual cortex (V1) as the site for metacontrast masking. Moreover, the BCS model also predicts that metacontrast suppression occurs at relatively early stages (V1/V2) of cortical processing. Currently available evidence supporting the hypothesis that the metacontrast mechanism is located at early cortical levels is not unanimous. For instance, Bridgeman (1975, 1980) does obtain evidence of metacontrast effects in the responses of single cells in the striate cortex of the cat and monkey to a target stimulus, but only for cells, similar to the one studied by Macknik and Livingstone (1998), that showed late, secondary discharges that could be suppressed by an aftercoming mask stimulus. In contrast, in a recent study, von der Heydt et al. (1997) found no evidence for metacontrast effects in the responses of 20 single cells recorded in V1 and V2 of monkey. However, it should be noted[4] that these cells failed to show any late secondary responses like those investigated by Bridgeman (1975, 1980) and by Macknik and Livingstone. Breitmeyer (1984, pp. 183–184, 226) specifically hypothesized that the most likely site of response suppression effects of metacontrast masking ought to be found in P cells located in the upper layers of V1 (and beyond). Of the 16 cells recorded in V1 by von der Heydt et al., 13 were located in Layers 2–3, and most were color-selective cells (von der Heydt, personal communication, February 15, 2000). Since color selectivity implies P cell input, these results indicate that V1 P cell activity is not inhibited by a metacontrast mask. Macknik (personal communication, February 15, 2000) similarly reported that response suppression was not found predominantly in P cells in the Macknik and Livingstone study. Despite the fact that both of the above electrophysiological studies failed to investigate concomitant behavioral indices of masking in their monkeys (Macknik/von der Heydt, personal communications, February 15, 2000), these findings pose clear problems for Breitmeyer's (1984) cortical transient-on-sustained inhibition hypothesis. Moreover, as was noted above, Francis's (1997) analysis, by incorporating the main features of Grossberg's (1994) BCS architecture, also predicts that neural inhibition produced by metacontrast should be found in cell activities of the cortical P pathway. The electrophysiological results reported by Macknik and Livingstone and by von der Heydt et al. thus also pose difficulties for the BCS or an elaborated BCS/FCS model.

Let us, however, note that indirect evidence supports the existence of inhibitory interactions that are relevant to backward masking. A study by Maunsell and Gibson (1992) in the macaque visual system showed that selective ablations of M layers of LGN result in an increase in the number of cells, with sustained responses in area V1. This finding can be interpreted as evidence for transient-on-sustained inhibition, whose effect becomes manifest in V1. Moreover, a current source density analysis of electrically evoked potentials (Mitzdorf & Singer, 1979), to be discussed further below, found that intracortical inhibition produced by one of two electrical stimuli applied to the optic radiation fibers suppressed activity elicited by the other stimulus in the cells of supragranular layers of area V1 in the visual cortex of the macaque monkey.

The RECOD model in its original form (Ogmen, 1993) uses a lumped representation for postretinal areas and therefore is unable to predict the specific loci of the transient-on-sustained inhibition. However, given the predicted oscillations in the metacontrast masking function, in conjunction with the related and previously cited results reported by Kruse and Eckhorn (1996) and by Fries et al. (1997), we tentatively take the neural inhibition of the sustained γoscillations by transient responses to be a neural correlate of the suppression of target visibility during metacontrast masking. Although this places the correlate within a cortical network, rather than within a single-cell framework, we do not want thereby to skirt the problem raised by the failure to obtain neural correlates of metacontrast either exclusively (Macknick, personal communication, February 15, 2000) or clearly (von der Heydt et al., 1997) at the early cortical P cell level. After all, single cells and their connections make up neural networks. In this regard, since γ oscillations are found in the single-cell responses of the monkey (Livingstone, 1996; Maunsell & Gibson, 1992) and in the cortical evoked visual potentials (CEVPs) of humans (Tallon-Baudry et al., 1996), the finding of Kruse and Eckhorn provides an excellent lead for similar investigations of the possible correlates of visual masking in the oscillating responses of individual cortical neurons in the macaque monkey and of the CEVPs in humans.

Evidence from animal studies: Late cortical levels. The aforementioned neurophysiological studies probed changes in activities at early cortical levels in correlation with either expected or measured changes in the strength of backward masking. In a recent study, Thompson and Schall (1999) took a different perspective to the problem. During electrophysiological experiments, they monitored the performance of monkeys and adjusted the SOA to obtain 50% correct performance. They then compared neural responses recorded in the frontal eye field (FEF), a subdivision of the prefrontal cortex, under *hit*, *miss*, *correct rejection*, and *false alarm* cases. Under all four conditions, there was significant neural activity in the FEF. However, the magnitude of activity, in particular when averaged during a time interval of duration equal to the SOA and immediately preceding the mask response, correlated with the behavioral response. The highest activity corresponded to hits, and relatively lower levels of activity, in decreasing order, corresponded to misses, false alarms, and correct rejections. Thus, although their study did not address directly the effect of stimulus timing, a crucial parameter in backward masking, it shed light on the relation between neural responses and the behavioral report. That there exist neural responses under conditions in which the monkey reports not seeing the stimulus has important implications for the nature of coding strategies used by the nervous system and, as such, for the linking assumptions used by theoretical models. For example, according to the sustained–transient approach, substantial responses are assumed to exist in the transient pathway even under strong backward masking conditions. Because the FEF is reciprocally connected to both dorsal and ventral pathways, according to this theory, it is possible that the responses recorded in the FEF have their origin in the transient pathway. In order to assess this possibility, one needs to determine whether/how task-related information is encoded in neural responses. However, one limitation of Thompson and Schall's study was that both target and mask stimuli generated responses in the neurons under study and the isolation of the *pure target* response was not possible.

This problem has been bypassed by other recent studies that recorded neural responses in the macaque temporal cortex under backward masking conditions (Kovács et al., 1995; Rolls & Tovée, 1994; Rolls et al., 1999; Rolls, Tovée, Purcell, Stewart, & Azzopardi, 1994). Since the temporal cortex contains neurons with a high degree of shape selectivity (see, e.g., Desimone, Albright, Gross, & Bruce, 1984; Gross, Rocha-Miranda, & Bender, 1972; Rolls, 1992), one can use a mask stimulus that generates negligible responses in the neurons under study.

Kovács et al. (1995) recorded from the monkey inferior temporal cortex (IT) under a backward pattern masking paradigm. The task was shape discrimination. To assess the task-related information, they computed the difference between the responses to the shapes that generate the strongest and the weakest activity in the cell. This *response difference* was significant under all conditions, including those that induced strong backward masking in psychophysical experiments. Both masked and unmasked conditions generated approximately the same number of spikes in the early part of the response. However, when the response was integrated over longer time intervals, the response difference between masked and unmasked conditions became significant. Consequently, backward masking effects can be observed in IT neurons if one assumes that the neuronal responses are temporally integrated. The role of temporal integration was also supported by a receiver-operating characteristic analysis of their data. The earliest part of the response (approximately 20 msec) did not contain reliable information for the accurate discrimination of shapes, necessitating integration of information. The effect of the mask was to suppress the information available in the later part of the response,

thereby reducing the performance by making temporal integration ineffective.

Another finding was that a difference (relative increase) of a few spikes in an integration interval of 80 msec was sufficient to reach a high degree of discrimination. This finding is in agreement with information-theoretic analyses of spike trains, suggesting that a single spike can carry several bits of information when the system is subjected to time-varying stimuli (Rieke, Warland, de Ruyter van Steveninck, & Bialek, 1997). Indeed, as a continuation of their earlier studies (Rolls & Tovée, 1994; Rolls et al., 1994), Rolls et al. (1999) applied information-theoretic analysis to neural responses in the monkey temporal cortex and quantified the amount of information available under backward masking conditions. They found significant responses, even under conditions yielding strong masking, as measured by a previous study (Rolls et al., 1994), and concluded that the information is encoded in the *difference* of activities and not by the simple presence of absence of activity. When the firing rate or the cumulated number of spikes was considered, only the responses to the most effective stimulus changed as a function of SOA. The average response of all the cells and the response to the least effective stimulus did not change significantly as a function of SOA. However, when the *amount of information* was calculated, using Shannon's (1948) formulation, SOA had a significant effect not only on responses to most effective stimulus, but also on average responses. The attenuation generated by the mask was strongest in the part of the response carrying the peak information about the stimulus. Consequently, the effect of the mask was more significant in terms of amount of information than in terms of firing rate.

Summary and discussion of animal studies. Backward masking effects are found at late cortical levels, including the temporal cortex and FEFs. Significant neural activities were recorded even under strong masking conditions. Therefore, backward masking is not simply related to the presence or absence of activity but, rather, depends on quantitative, and often small, differences in the activity. The details of neural coding strategies become important for an analysis of these differences. Methods from signal detection theory, as well as from information theory, suggest that task-related information is temporally distributed in neural responses and that temporal integration is necessary to obtain reliable information. The effect of the mask in backward masking is to make temporal integration ineffective. The distribution of information across cell assemblies and its relation to backward masking need further study (Kovács et al., 1995; Rolls et al., 1999), and as was mentioned before, an application of the study by Kruse and Eckhorn (1996) to backward masking would be very informative.

If the information is temporally distributed in neural responses, it remains to be determined whether it is the *same* information that is distributed or whether different types of information are represented at different phases of the response. The three-phase operation proposed in the RECOD model is an example of how information can be multiplexed in neural responses. The responses in the feedforward phase reflect coarse boundary information signaled by afferent connections, whereas the responses in the feedback phase reflect fine boundary information sent by efferent connections (see, e.g., Figure 3 in Purushothaman et al., 1998). A recent study provides evidence that the initial part of the responses in the macaque IT codes coarse information (monkey face vs. human face vs. shape), whereas the finer information about stimulus attributes (identity and expression of the face) is signaled in the part of the response starting approximately 50 msec later (Sugase, Yamane, Ueno, & Kawano, 1999). If information multiplexing is carried out through "traveling waves" of activity among different neural centers, backward masking might involve multiple neural loci, depending on the SOA, the task, and the criterion content. When corrected for latencies, backward masking effects are found in the earlier parts of neural responses in the temporal cortex (within approximately the first 80 msec) but in the later parts of V1 responses (150 msec and higher). This observation consolidates the suggestion that the suppression of late activities observed in V1 might be a manifestation of activities beyond V1 (Bridgeman, 1980).

Evidence from human studies. At first glance, the lack of backward masking effects in early cortical levels also seems to be supported by human electrophysiological results reported by Jeffreys and Musselwhite (1986). In their visually evoked potential (VEP) study of metacontrast, these investigators looked at the effects of a metacontrast mask on the C1 and C2 components (Jeffreys & Axford, 1972a, 1972b) of the cortical VEP to the target. They assumed that the C1 and C2 components of the VEP originate from the striate cortex (area 17/V1) and the extrastriate cortex (area 18/V2 [or beyond?]), respectively. Their results showed that these components of the target's pattern-specific cortical VEP are not affected by an aftercoming mask. Here, we issue several caveats. First, Jeffreys and Musselwhite note that it is difficult to compare their VEP results with those of neurophysiological studies of metacontrast, since the relation of scalp potentials to underlying neural activity is not known. Hence, the ability to localize the components is limited in precision. It is therefore entirely possible that the C1 and C2 components are generated by cortical pattern-specific mechanisms responding prior to the suppressive effects of the mask. Moreover, there is not even a clear consensus as to the general site of neural generation of the C1 and C2 components. Whereas Jeffreys and Musselwhite favor striate area 17/V1 and area 18/V2 (and possibly beyond), respectively, Drasdo's (1980) and Maier, Dagnelie, Spekreijse, and van Dijk's (1987) analyses of cortical VEPs, in contrast, favor area 18 (or 19) and area 17, respectively. In either case, other VEP studies of backward masking and metacontrast (Andreassi, De Simone, & Mellers, 1975; Schiller & Chorover, 1966; Vaughn & Silverstein, 1968) do indicate that later (N2 and P2) components of the target's cortical VEP are suppressed by the aftercoming mask, particularly at SOAs of 30–60 msec,

where masking is optimal. Bridgeman's (1988) reanalysis of Jeffrey and Musselwhite's results tend to confirm this finding. However, as was noted by Jeffreys and Musselwhite, interpretations of such results, in turn, are complicated by the fact that the target-evoked and mask-evoked responses are not clearly separable in the later VEP components.

Bachmann (1994) interprets the VEP findings discussed above as conclusive evidence against the class of contour-inhibitory models of backward masking. We take issue with this claim, for the following reasons. As was noted above, the fact that analyses of early VEP components do not yield evidence for metacontrast masking may simply mean that these early components are generated by responses of cortical cells to the target before the inhibitory effects of the mask occur. In particular, according to Breitmeyer's (1984) proposal, the sustained P cells in layers $4C_\beta$ will generate powerful responses to the target pattern. Should these responses contribute significantly to the early components of the target's cortical VEP, as was argued by Schroeder, Tenke, Givre, Arezzo, and Vaughn (1991), it is not surprising that these components are not affected by the aftercoming mask. According to Breitmeyer (1984), metacontrast is not due to a suppression of the responses of these low-level cortical P cells but, rather, to the transmission of their output signals to cells of the P pathway located at higher levels of cortical processing. This interpretation is consistent with Mitzdorf and Singer's (1979) current source density analysis of electrically evoked potentials in the visual cortex of the macaque monkey. Mitzdorf and Singer found that intracortical inhibition produced by one of two electrical stimuli applied to the optic radiation fibers suppressed activity elicited by the other stimulus in the cells of the supragranular layers of area V1; however, no such response suppression to the latter stimulus was found in layers $4C_\alpha$ and $4C_\beta$ of V1. Consistent with Breitmeyer's (1984) hypothesis, Mitzdorf and Singer concluded that "*intracortical inhibition* has presumably prevented the intracortical relay of afferent activity to the supragranular layers" (p. 78).

On the basis of this analysis, a similar response to Bachmann's (1994) interpretation of the VEP results can be made in relation to the contour-inhibitory BCS model. Here, powerful responses to the target could be generated at the simple-cell and complex-cell levels before the feedforward inhibition at the first competitive stage of the BCS takes effect. Hence, strong VEP signals would be generated at the excitatory responses at the simple- and complex-cell levels of processing occurring prior to the inhibitory effects at the first, but hierarchically later, competitive stage.

CONCLUSIONS

The preceding review illustrates amply that visual backward masking is not a unitary phenomenon. The masking effects one can obtain depend on the choice of stimulus and task parameters and, thus, on the theoretically or empirically motivated choices of these parameters. Hence, one can obtain evidence for masking effects at a number of levels of visual processing, from the earliest preattentive and preconscious levels to levels that engage attentional and top-down processes (Bachmann, 1994; Breitmeyer, 1984; Enns & Di Lollo, 1997; Klotz & Wolff, 1995; Michaels & Turvey, 1979; Ramachandran & Cobb, 1995; Shelley-Tremblay & Mack, 1999; Turvey, 1973; Williams & Weisstein, 1981). As an example, the existence of attentional influences in visual masking was explicitly incorporated by Michaels and Turvey but was already implicit in several prior empirical studies (see Breitmeyer, 1984, pp. 256–261). This is not an unexpected or surprising development, since attention would be expected, and is known, to affect the quality and quantity of processing not only in the visual domain, but also in a broad range of other information-processing domains (Posner & Petersen, 1990; Posner & Rothbart, 1994). Hence, although there must be an attentional *component* to an adequate theory of or approach to visual masking, there hardly can be an exclusively attentional theory of masking. For instance, several recent investigations (Enns & Di Lollo, 1997; Havig et al., 1998; Ramachandran & Cobb, 1995; Shelley-Tremblay & Mack, 1999) have shown that although attention to the configurational or positional aspects of the target can decrease the magnitude of metacontrast, the typical U-shaped masking function is nonetheless still obtained. Thus, the signature nonmonotonicity as a function of SOA remains a fundamental characteristic of metacontrast; and although it *might* be explained solely by properties of attention, it more likely is explained as well by other—for example, contour-inhibitory—processes modulated by attention. The same line of reasoning applies to either the bottom-up modulatory effects of stimulus wavelength (Breitmeyer et al., 1991; Breitmeyer & Williams, 1990; Foster, 1976, 1978, 1979; Reeves, 1981) or the top-down effects of Gestalt and configurational contexts within which the target–mask sequence is presented in a metacontrast paradigm (King, Hicks, & Brown, 1993; Williams & Weisstein, 1981). Here again, although the magnitude of metacontrast and the optimal metacontrast SOA are affected by such bottom-up or top-down influences, the very existence of the nonmonotonicity remains to be explained.

Although the above examples illustrate that variations of visual masking effects legitimately can be regarded as conceptually and empirically motivated variations of masking regimens that can readily be accommodated into any extant theory of masking, there nonetheless are fundamental differences between theories of visual masking. Most relevantly, the recent neural net models proposed by Bachmann (1994), Francis (1997), and the present authors to account for the U-shaped nature of the metacontrast function pose genuinely conflicting conceptual approaches to the study of visual masking. Rather than addressing different regimens of masking, they address the same regimen from different conceptual perspectives. We have reviewed some of the significant positive and negative

aspects of these recent theoretical approaches and summarize them below.[5]

The strengths of the sustained–transient approach to backward pattern masking are its extensive coverage of data, its strong neurophysiological underpinnings for many of its mechanisms, and its links to a large literature of related data (e.g., RTs, motion, color and brightness, visible persistence, and spatial scale). However, when the original model is updated and tied to activity in sustained P and transient M channels, it lacks direct neurophysiological evidence for inhibition of activity in the cortical P pathway. Another notable weakness of the original version of this approach was that it was based on a qualitative or descriptive model. The development of its current version in terms of the RECOD neural net model will provide the quantitative detail to its major descriptive components.

The strengths of the PR model are its coverage of a lot of the masking data, its neurophysiologically plausible mechanisms, and its quantification. Weaknesses of the model are its (easily correctable) ambiguity as to the neural level associated with conscious registration of stimulus properties and its current lack of application to findings outside of those obtained in masking studies.

The BCS model is currently the most sophisticated from a quantitative standpoint. It has a variety of simulated results to back up its properties and also covers the major metacontrast masking results. Another strength of the model is that it is connected to a large literature in other domains (e.g., spatial and 3-D vision, texture segmentation, illusory contours, brightness perception, motion, and visible persistence) investigated by Grossberg and co-workers (Francis, 1996a, 1996b; Francis & Grossberg, 1996b; Francis et al., 1994; Grossberg, 1983, 1994; Grossberg & Mingolla, 1985a, 1985b; Grossberg & Todorović, 1988). Moreover, its mechanisms are neurophysiologically plausible but not as well supported as those for the sustained–transient approach. A weakness, however, is that its data coverage is not as extensive as that of the sustained–transient model or that of the PR model. Like the sustained–transient approach, it also is faced with a lack of direct neurophysiological evidence supporting cortical levels of inhibition in the P pathway. Moreover, the simulations of the BCS model are extremely time consuming, and that itself may be a practical limitation to its data coverage. Finally, the linking hypothesis used in the current approach needs to be refined or revised, since the duration of boundary signals is not related to perceived brightness or percept quality in a simple or direct way, as is demonstrated by the *inverse* relationship between visible persistence and stimulus intensity (Coltheart, 1980; Di Lollo & Bischof, 1995).[6]

Although we cannot preview forthcoming empirical developments or theoretical modifications to these models, we confidently look forward to elaborations of these models in response to new and critical empirical findings. Two topics for future research seem to be of particular importance. One concerns the role of attention and focused arousal in backward masking; the other concerns the site(s) of cortical inhibition in backward masking, particularly in the Type B variety. As to the first topic, in Bachmann's (1994) model, focused arousal (Sheer, 1984; Singer & Gray, 1995) as an attentional activation arising from subcortical (reticular and brainstem) sites seems to be part and parcel of the metacontrast mechanism. Such an intimate connection between attention and U-shaped backward masking also characterizes Enns and Di Lollo's (1997) and Shelley-Tremblay and Mack's (1999) explanations of metacontrast. In contrast, in both the sustained–transient/RECOD model (Breitmeyer, 1984; Ogmen, 1993) and the BCS model (Francis, 1997), attention seems to be relegated to an ancillary role in which it simply modulates neural activity after the contour-inhibitory processes deemed responsible for metacontrast have occurred. Hence, an important topic for future research in backward masking is to specify and investigate more thoroughly the role of attention or focused arousal. As to the second topic, and assuming that the sustained–transient and the BCS models' emphasis on contour-inhibitory processes is correct, future research focusing on where in the cortical visual centers such inhibitory processes occur will reveal whether or not the hypothesized inhibition suppresses activity in the P pathway, as both models imply.

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NOTES

1. Regarding explanatory power, another noteworthy problem arises from Bachmann's meta-analytic survey of empirical findings as they relate to competing theories in visual masking. Bachmann (1994) expands upon a taxonomic system developed by him previously (Bachmann, 1984) to classify and compare a total of 78 findings—including highly replicated empirical regularities, more specific psychophysical effects, and psychophysiological results—obtained in masking with respect to 18 theoretical approaches to masking. One upshot of the meta-analysis is that Bachmann's model fared better than any other. Although we applaud the ambition and goals of such meta-analytic efforts, we feel that Bachmann's analysis is flawed in several ways. First, the selection of empirical regularities, specific psychophysical effects, and physiological findings was determined by Bachmann. Although other researchers, including us, may agree with a majority or even all of these selections, we are equally impressed with the regularities, specific effects, and psychophysiological findings that were not included in Bachmann's meta-analysis. After all, the empirical literature on visual masking and closely related topics is literally voluminous (see Breitmeyer, 1984). Hence, a limited selection of findings is bound to be too exclusive and is likely to be biased. Second, even if we allow Bachmann's limited selection of empirical findings, the interpretation and appraisal of these findings is solely his. Unfortunately, it is fraught with some significant errors. We carefully examined Bachmann's evaluation of the 78 empirical findings

only in relation to the sustained–transient model and found that at least 18 findings that Bachmann deemed either ambiguous (0) or contradictory (−) can be explained by the sustained–transient model. To convey the gist of our criticism of Bachmann's meta-analysis, we will focus on a sample of empirical findings where our disagreement with Bachmann is particularly evident. (1) Relying strictly on Growney's (1978) results, Bachmann claims that metacontrast effects are independent of the spatial frequency contents of the stimuli. What Growney in fact reported is that both the magnitude of masking and the optimal masking SOA do depend in a complex manner on the spatial frequency composition of the target and the mask. Although some results were consistent with the sustained–transient model, others were not. Moreover, Rogowitz's (1983) subsequent and more extensive study of spatial frequency effects in metacontrast showed that variations in the obtained metacontrast masking functions depend on the spatial frequency compositions of the target and the mask and were consistent with predictions based on latency differences between slow-responding high-spatial-frequency channels (sustained) and fast-responding low-spatial-frequency channels (transient). Similar results have been reported by Breitmeyer (1975). (2) Bachmann claims that the sustained–transient model is undecisive regarding disinhibition or target recovery phenomena in metacontrast. On the contrary, from the model one can derive explicit predictions that were confirmed by results reported by Breitmeyer (1978a) and by Breitmeyer (1981). Moreover, these results were discussed extensively by Breitmeyer (1984, pp. 270–286) in the context of other studies reporting target recovery in a variety of visual-masking paradigms. (3) Likewise, Bachmann claims that the sustained–transient model is ambiguous or undecisive regarding the effects of criterion content on the observer's evaluation of the target stimulus and, as a related point, that it is inconsistent with the discrepancies between psychophysical measures on phenomenally reported target contrast and those relying on behavioral measures (e.g., RT measures). Neither claim is valid. The original version of the sustained–transient model proposed by Breitmeyer and Ganz (1976) explained (on p. 26) the lack of U-shaped masking when simple RT or detection measures of target presence were used and the presence of U-shaped functions when target identification or contrast-rating measures were used on the basis of different sources of information (criterion contents) provided by transient and sustained channels. Related findings were discussed by Breitmeyer et al. (1981) and were given further discussion and elaboration by Breitmeyer (1984, 1992). The upshot of this criticism is that a meta-analytic evaluation of theories of visual masking in relation to extant findings requires a consensus on which selections and interpretations of those findings are to be adopted. Such an analysis remains to be done.

2. We say putatively, because the results (Bachmann, 1994, Figure 46) actually do not reveal such an absolute facilitation but, rather, a minimal RT to the second stimulus at SOAs (60–80 msec) optimal for backward masking. This minimal RT, however, never reached the lower RT obtained with the target presented alone. Bachmann (1994) explains this discrepancy as being due to response interference, since a forced-choice, go/no-go task was used, in which subjects responded only if a stimulus designated as the target appeared but withheld their responses otherwise. However, it seems to us that such response interference ought to prevail also when the target is presented in isolation, because trials in which two stimuli were presented sequentially and trials in which a single stimulus was presented were randomized within the same block of trials.

3. Like Enns and Di Lollo's (1997) results, Shelley-Tremblay and Mack's (1999) findings indicate that attention can modulate the shape and magnitude of the metacontrast function. Although we agree with this conclusion, we take issue, for the reasons given in the discussion of Enns and Di Lollo's conclusion, with their additional conclusion that this poses serious problems for explanations invoking lower level contour-inhibitory interactions. Such explanations can invoke supplementary attentional processes while otherwise remaining intact. Moreover, with regard to Shelley-Tremblay and Mack's findings and conclusions, according to Crick and Koch (1995), activity in cortical area V1 is preconscious in that it does not correlate with visual awareness, whereas activity in higher cortical areas does. Yet, recent electrophysiological findings (Di Russo & Spinelli, 1999; Roelfsema, Lamme, & Spekreijse, 1998; Somers, Dale, Seiffert, & Tootell, 1999; Watanabe et al., 1998) strongly indicate that cortical activity as early as that in area V1 can be modulated by attention. Hence, attention can modulate activity at low, preconscious levels of processing and thus yield a variable input to the later levels of processing that, according to Crick and Koch, are correlated with awareness. This implies that, at lower levels of cortical processing, responses of target-activated neurons decreased by inhibition from mask-activated neurons can, at later levels of cortical processing, lead to various states in the observer's awareness of the target, depending on the attentional state of the observer relative to the target.

4. We thank an anonymous reviewer for pointing this detail out to us.

5. We thank an anonymous reviewer for suggesting this summary and contributing to its contents.

6. Indeed, earlier simulations of the BCS model showed that increasing the luminance of the stimulus caused a decrease in the duration of boundary signals, suggesting an *inverse* relationship between perceived brightness and the duration of boundaries (Francis et al., 1994). The linking assumption used in the simulations of backward masking was, however, a *direct* relationship between perceived brightness and the duration of boundaries (Francis, 1997). Simulations that take into account interactions between BCS and FCS (e.g., Francis & Grossberg, 1996a) or alternative linking assumptions might be used to rectify this problem.

(Manuscript received April 26, 1999;
revision accepted for publication February 8, 2000.)