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A Comparison and Elaboration of Two Models of Metacontrast

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Metacontrast has been the subject of two neural network simulations by Weisstein and Bridgeman. We compare and elaborate on the two models, correct flaws not inherent in the models' conceptualizations, and discuss the remaining shortcomings. The idea behind how U-shaped metacontrast functions are generated is similar in both models, but the assumptions about how the visual system is organized are quite different. In one of the models temporal ringing, combined with a complex and implausible linking hypothesis, is necessary in order for masking to be obtained; this model assumes a single spatial and temporal channel. In the other model, masking does not depend on temporal ringing; this model assumes multiple spatial and temporal channels and a simple linking hypothesis. We show that of the two models only the second adequately predicts empirical metacontrast functions, and we relate this model to recent evidence that the visual system contains multiple channels. Sometimes treated as a puzzling and somewhat singular phenomenon, isolated from the "mainstream" of visual data and theory, metacontrast may, on the contrary, turn out to be one of the more interesting manifestations of a multiple-channel visual system.

Metacontrast backward masking has been the subject of two neural network simulations in this journal (Bridgeman, 1971; Weisstein, 1968). Metacontrast would seem innocuous enough as a backward-masking design, and might not generate such interest were it not for the data it produces. If a briefly flashed target (generally a disc or rectangle) and a briefly flashed mask (generally an annulus or two rectangles that flank the target) are above threshold and of roughly equal energy, the greatest reduction in apparent brightness of the target will occur when the mask is presented as much as 125 msec after the target has been turned off (Alpern, 1953). But when the target and mask are presented simultaneously, little if any reduction in apparent brightness occurs. This reduction in apparent brightness with increasing delays between target and mask is illustrated in Figure 1. It is the U in this function that generates such interest in metacontrast. Why should a target dis-

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appear *only* if a mask is delayed by some 60–125 msec and not before?[1]

LATE ACTIVITY AS AN EXPLANATION FOR METACONTRAST

Most theories attempting to explain the U-shaped functions in metacontrast have a common assumption, namely, that the kinds of masks used in metacontrast suppress or alter the late portion of the response to the target, and do not interfere with earlier portions. If so, in order for masking to occur, the mask must be delayed until its presentation will interfere with the appropriate (late) portion of the target response.

The response to the target may be divided into early and late portions if for no other reason than that a brief stimulus flash produces neural activity which may last well over 150–200 msec after the stimulus goes off (Sturr & Battersby, 1966; measurement of the gross potential).[2] In addition, the early portion of this activity looks different from the later portions. If early and late correspond to different perceptual events, then the late-activity explanation for metacontrast may make sense. In metacontrast, something about the *appearance* of a target is affected, while its detectability is generally unchanged: Its apparent brightness or contrast decreases, and its contours break up or fragment. The loss in detectability is held to a minimum by the metacontrast design: Both target and mask are well above threshold, and they are adjacent to each other rather than superimposed on the same retinal location. If we suppose appearance to be connected with the late portion of neural activity, then a mask, to be effective in metacontrast, might have to coincide with the late portions of neural activity to the target. Hence U-shaped masking functions would be predicted. In somewhat loose terms, then, two adjacent, briefly flashed, above-threshold stimuli might not be able to affect each other's appearance unless the activity produced by one of the stimuli "hits" the right portion of the activity produced by the other stimulus; if this portion of activity is late activity, then U-shaped functions will be generated.

NEURAL MODELS AND VISUAL-SYSTEM ORGANIZATION

To tie these ideas down to specific predictions, one may make a model of neural response or visual-system activity. This model should generate metacontrast functions, be consistent with what is known about mammalian visual-system organization, and make reasonable *linking hypotheses* —that is, make reasonable hypotheses about how neural response might correspond to the perceptual data (Brindley, 1970).

Before considering each metacontrast simulation in particular, a few comments are in order about two somewhat competing views of general visual-system organization, the

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[1] One way around this puzzle is to assume that U-shaped functions in metacontrast do not really exist (e.g., Eriksen, Becker, & Hoffman, 1970; Lefton, 1973a). However, proponents of this position will admit that there is a U-shaped reduction in apparent brightness of the target (Eriksen et al., 1970; Lefton, 1973b); their main criticism is that *detection* functions in metacontrast are not U-shaped. This is generally true (but see Cox & Dember, 1972); however, it is an irrelevant argument. There are a number of similar paradigms which demonstrate that detection and apparent brightness do not covary. Cornsweet and Teller (1965) found that increment thresholds remained constant even though the apparent brightness of a steadily presented disc varied depending on the brightness of a surrounding annulus. Few would claim that although the increment threshold to the disc did not change, simultaneous brightness contrast is not "real": discs do look brighter when surrounded by dark annuli than when surrounded by bright annuli. One simply has to use a response indicator appropriate to the phenomenon one wants to measure.

Another approach is to assume a priori that complex phenomena such as metacontrast are not worthy of serious attention (e.g., Sekuler, 1973). However, it would seem that we progress by exploring, rather than evading, that which is puzzling.

[2] As early as 1966, Sturr and Battersby noticed the possible relevance of the later portions of neural activity to backward masking. They hypothesized that the entire 150–200 msec of neural activity had to occur undisturbed in order for a stimulus to escape masking. This is a somewhat more general form of the theories developed to account for metacontrast; additional assumptions, about what happens when, have to be made in order to predict U shapes.

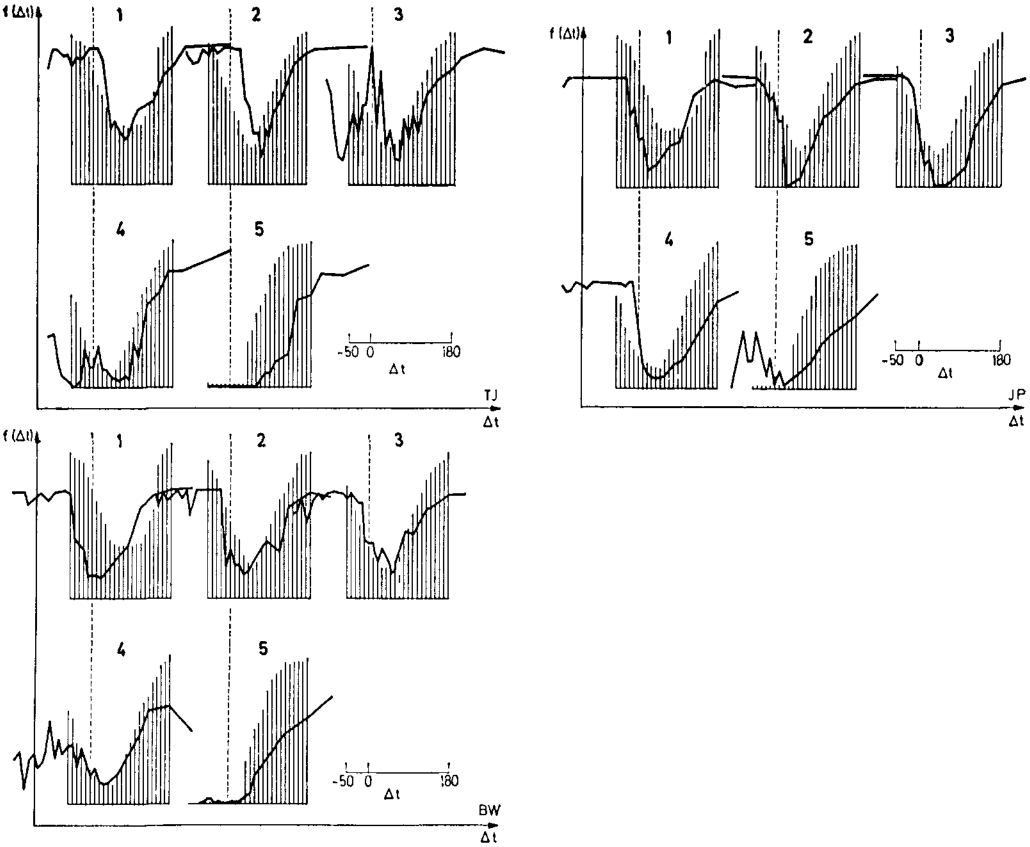

FIGURE 1. Metacontrast functions for each of three subjects, TJ, BW, and JP, as indicated on the figure (solid curves) at each of five target-to-mask energy ratios, T/M (functions 1 through 5; from Weisstein, 1972). The mask was always a lighted 16 msec, 16 footlambert (54.8 cd/m^2) annulus; the target was a lighted, 16 msec disc whose luminance decreased respectively from 1 through 5 as follows: 16, 8, 3.2, 2, and 1 footlambert (54.8, 27.4, 10.9, 6.8, 3.4 cd/m^2). Each point on the solid curves represents a geometric mean of five magnitude estimations of the apparent brightness of the disc at each of 27 Δts, beginning with $\Delta t = -100$ msec and proceeding in 10 msec increments to $\Delta t = 100$ msec; thereafter in 25 msec increments to $\Delta t = 200$ msec, and finally, 250 and 300 msec. U shapes are found for each subject for each T/M except the lowest one or two. Dotted vertical lines indicate simultaneous presentation of target and mask, or $\Delta t = 0$. If the function falls below the amplitude at the dotted vertical line, then it can be said to be U-shaped. The height of the solid vertical lines represents the points predicated by Weisstein's (1972) model. (From "Metacontrast" by Naomi Weisstein, in D. Jameson & L. Hurvich (Eds.), *Handbook of Sensory Physiology* (Vol. 7, part 4: *Visual Psychophysics*). Copyright 1972 by Springer-Verlag. Reprinted by permission.)

single-channel view and the multiple-channel view.

Single Channel

A single-channel view assumes that visual-system response can be characterized by one set of parameters. To put it in terms of neurons, a single spatial and temporal channel (or, equivalently, filter) would correspond to the assumption that the visual system responded as if all single units had the same receptive field size and time course of excitation and inhibition. Then one would simply need one function to describe the visual system's spatial properties and one function to describe the visual system's temporal properties. The single-channel assumption is not necessarily contradicted by the evidence that single units in the visual system have different receptive field sizes and different rates and latencies of firing, since all units could contribute to a composite size and response rate. (See Nachmias, 1968, for a discussion of a composite visual-system spatial response, and see Cornsweet, 1970, for an excellent discussion of how if one assumes a single channel, one can obtain an overall estimate of the visual system's temporal and spatial response by utilizing frequency-response techniques.)

Multiple Channel

A multiple-channel view assumes that to characterize visual-system response, families of parameters are necessary. Many different channels may be active in the visual system, depending on whether a stimulus is small or large, periodic or aperiodic, transient or sustained. These channels do not contribute to one composite, overall function; rather, at some stages in the visual system their activities are independent of each other. Thus, for instance, at threshold it would not be the sum of the activity of a number of different channels that determines threshold, but the activity of a particular channel that has reached threshold, activity not directly influenced by the subthreshold mumblings of other channels that may be active. (The threshold itself may be lowered somewhat by subthreshold mumbling due to probability summation, but this would reflect the independent activities of a number of channels rather than the change in activity of a particular channel as a result of the activity in another. See Graham, in press, for an excellent discussion.)

An above-threshold view of a multiple-channel model might assume that different channels are responsible for different features of a stimulus—some channels for the edges of the stimulus, for example, and others for the inside. We will have more to say about this later. In terms of neurons, a spatial multiple-channel model would assume that units with different receptive field sizes respond to stimuli that are somewhere within the range of their field size, and that when a stimulus is too small or too large, the unit will simply not respond. (Spatial multiple-channel models have usually been phrased in terms of parallel spatial filters, each responding to a band of spatial frequencies, but we prefer, for the moment, to use a somewhat looser notion.) A temporal multiple-channel model would assume that units would respond at different rates, and this implies that some units might prefer transient stimulation and other units might prefer sustained stimulation. Again, we will have more to say about this later. The multiple-channel model doesn't mean that only one channel will respond if only one stimulus is presented; a particular stimulus may produce response in a number of channels. The important point is that these channels are looking at different aspects of the stimulus, and they are responding at different rates.

THE TWO METACONTRAST SIMULATIONS

Of the two attempts to quantify the hypothesis that metacontrast is the suppression of the late activity or slow portion of visual-system response to a stimulus, one (Weisstein, 1968, 1972) is a multiple-channel model, and the other (Bridgeman, 1971) is a single-channel model. We will examine the two models to see which one seems the more plausible. Neither model is adequate as it stands, and in considering which model better fits the data, we need to expand and elaborate on both models. First, we will present Bridgeman's simulation, which assumes a single channel, taking the steps necessary for it to generate metacontrast functions (this was not done in his paper). As we examine this model, some serious shortcomings that have previously escaped notice will become apparent. Then we will consider Weisstein's simulation (1968, 1972), which assumes multiple channels, expanding it so that it can deal with spatial interactions. We will show that two shortcomings noted for this model as it was formulated may be more apparent than real. Finally, we will present an elaboration of this model, which associates the fast and

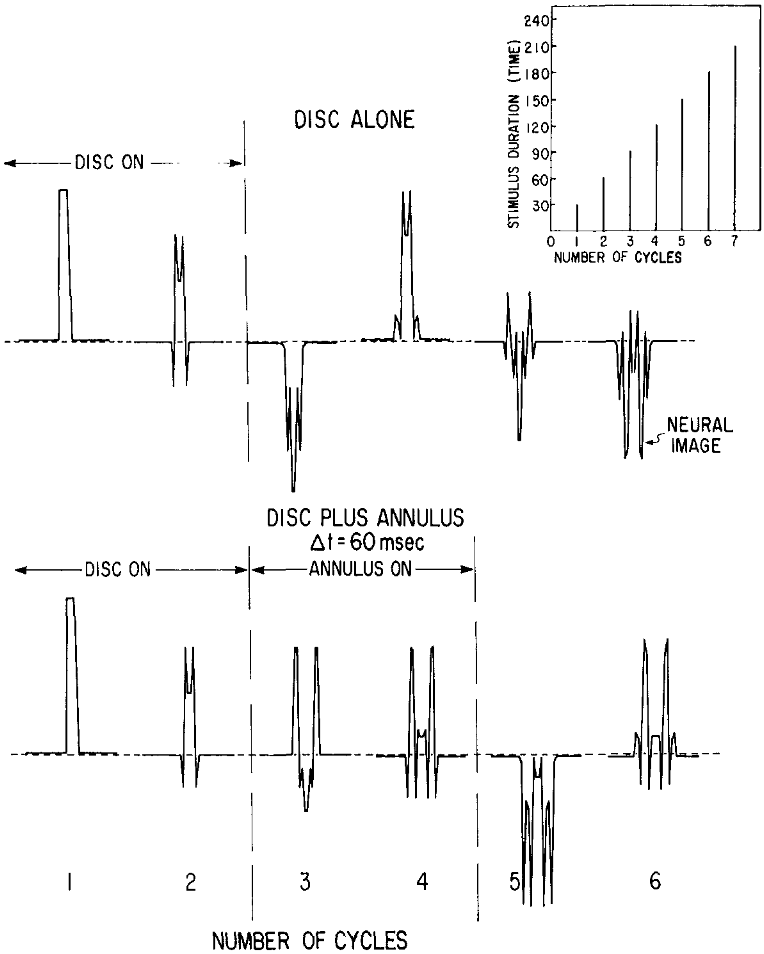

FIGURE 2. The activity of Bridgeman's (1971) neural network (the neural images) for each of six successive time periods. The top row of Figure 2 shows neural images that occur during and after a disc is presented. The bottom row of Figure 2 shows neural images that occur during and after a disc is presented, followed by an annulus. The main thing to notice is that the neural images of cycles 3 through 6 for disc alone (top row) versus disc plus annulus (bottom row) do not resemble each other: This is the basis of the metacontrast interaction. Time is a discrete function of number of cycles: This is plotted in the inset.

slow responses assumed by Weisstein with the recent work on transient and sustained mechanisms (channels) in the visual system.

Metacontrast Simulation 1

Bridgeman (1971) hypothesized that metacontrast occurs because the late neural activity to a target followed by a mask differs from what it would be to a target alone.

To obtain a picture of neural activity, Bridgeman uses the one-dimensional neural network developed by Ratliff and his coworkers (1965), in which each neuron inhibits and is inhibited by its neighbors at recurrent intervals. A neuron in the system inhibits its neighbors in proportion to its own activity and to its distance from its neighbors. In addition, inhibition is subject to time delays: Adjacent neurons are inhibited with a time lag of 30 msec and neighbors one removed are inhibited with a time lag of 60 msec. To simulate this digitally, Bridgeman uses the Ratliff equations (1965, p. 110), with parameters chosen from Barlow's (1969) study of *Limulus*. This network is single channel because it has a single set of spatial and temporal parameters—a spatial weighting function consisting of a point excitation and a spread of inhibition on either side, and a temporal weighting function that is simply a step for excitation and inhibition from adjacent units, and a step with a one-period lag for inhibition from units one removed.

The Network

The Ratliff (1965) equations represent a discrete form of repeated inhibition: Each recurrence of inhibition "spreads" a signal laterally so that one by one, units further and further removed from the site of stimulation become involved. The activity of the neurons in the network, what we may call the *neural image,* is shown in Figure 2, during and after stimulation, for each successive recurrence of inhibition of the network. The top row shows a network responding to a target alone, and the bottom row shows a network responding to a target and then a mask, which is presented after the initial response and one cycle of inhibition of the network. The inhibition delay defines time in this network; that is, one cycle means that up to 30 msec have passed, two cycles means that up to 60 msec have passed, and so forth. This quantization or "stepping" of time is shown in the inset of Figure 2.[3]

As shown in Figure 2, when the stimulus is on, the corresponding neural images remain fairly similar to the physical stimulus through successive cycles, that is, through successive 30 msec intervals at which the inhibition recurs. Each successive recurrence can be seen to spread the signal, and edges and troughs develop at the borders of the stimuli. (Edges and troughs generated by networks like these are the basis of many explanations of Mach bands and related effects.) Bridgeman adopts the term *homophotic* from Stigler (1910) to describe the set of neural images during stimulation. The term *metaphotic* describes the set of neural images that occur when the stimulus is removed. Although the metaphotic images no longer resemble the physical stimulus, they are uniquely related to it.

Masking

The set of neural images that occurs when the target is presented by itself comprises the reference to which all other conditions are compared. The amount of masking is assumed to depend on the extent to which the set of neural images formed when both target and mask are presented differs from the set of neural images formed when the target is presented by itself. This measure

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[3] This figure was prepared using the output from our own simulation program, which repeats the calculations described in Bridgeman (1971). The figure agrees with Bridgeman's Figure 3 (p. 533) with two exceptions (see below), and for the sake of brevity we have left out his third row, which shows the network response to the annulus alone. Our Figure 2 also differs from Bridgeman's because we adjusted the neural images so that they each had the same maximum peak-to-trough amplitude; this way, it is easier to see what the neural images look like for the later cycles where the total signal is very small. (Adjusting the peak-to-trough amplitude, or normalizing these functions, does not change the measure of similarity that Bridgeman used and we have replicated, since the measure itself normalizes all functions.) While this effectively makes all the neural images the same size, it should be stressed that by the seventh or eighth cycle, the actual neural image is almost indistinguishable from noise.

There are some minor differences between our top-row cycles (3 and 4), and Bridgeman's. Below, there are some minor differences between our similarity function (Figure 3), and Bridgeman's. We have gone through extensive checking procedures on our own simulation (following Bridgeman's description of how he did his simulation) and are convinced that our calculations are correct. The differences may be due to the following factors: We used different computers, which could have led to a difference in word size, number of significant digits, rounding errors, plotter and plotting routine; and there may have been some small drafting errors in Bridgeman's figures. In any case, none of these differences are sufficiently large to affect the arguments that we make in this paper.

of similarity includes the entire neural image, from one end of the network to the other: The activity of *every* element in the network is considered, rather than, for instance, just the activity in and around the target area. We shall have more to say about this below. The similarity comparison is made cycle by cycle, and a similarity function is produced, which gives a value of similarity for pairs of neural images (target, target + mask) for each cycle.[4]

Bridgeman (1971) divides masking into two types: The type associated with the homophotic neural images (forward and simultaneous masking) and the type associated with the metaphotic neural images (backward masking and metacontrast). According to Bridgeman, homophotic neural images allow subjects to make *detection* decisions, while metaphotic neural images have to do with the *appearance* of the target. It is not clear how the visual system is able a distinguish homophotic from metaphotic images; the images themselves cannot provide a unique criterion for whether or not a stimulus is physically present. But for the purposes of argument, let us assume that this problem can be solved. If we assume, then, that the system is somehow able to tell the difference between homophotic and metaphotic, in those instances where the mask is presented before or simultaneously with the target, the homophotic images of the target are somewhat different from those of the target and mask together. In this case, detection drops. When the mask is presented later than the target, the homophotic images are identical, but the metaphotic images of the target alone are different from those of the target-plus-mask, as can be seen in Figure 2. Here, then, the appearance of the target—its apparent brightness—is assumed to change, and one has the makings of a metacontrast siutation. (Actually, the model predicts that whenever detection drops, the appearance of the target also changes for those instances where the target is detected, since if the homophotic neural images of target and target-plus-mask are dissimilar, so are the metaphotic images. But the important point is that one can have appearance changes without detection changes, and this forms the basis of the metacontrast predictions.)

Thus the appearance of a stimulus is mediated by the late neural images (i.e., the late neural activity), and metacontrast occurs when these late neural images differ from what they would be if a target had been presented alone. How is a metacontrast function constructed from this formulation?

Constructing the Metacontrast Function

A metacontrast function is a plot of some measure of the change in appearance of the target (generally apparent brightness) for each delay between target and mask (for each Δt). No plots of this kind appear in Bridgeman's (1971) simulation. Rather, neural image similarity is computed cycle by cycle for one Δt in the backward masking range, one in the forward masking range, and one at simultaneous masking. The similarity function obtained is different for these three cases; in particular, for the backward masking case the later, metaphotic neural images of the target do not resemble the corresponding images of the target-plus-mask. This similarity function is shown in Figure 3.

A similarity function is not a metacontrast function, because it provides informa-

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[4] These similarity comparisons are called cross-correlations, but the reference appears to be to a Pearson r. That is, if one computes the actual cross-correlations for the output generated by Bridgeman's (1971) network for the 6 cycles illustrated in Figure 2, they are, in order, 1600, 256, −394, 172, 16, and −5. This bears no resemblance to Bridgeman's Figure 6. Next we tried various variations on our cross-correlations, but none of them got us near Bridgeman's Figure 6. We thought perhaps he had normalized the cross-correlations in some way, so we computed a "constant of correlation" (Lathi, 1965): $F(x) * G(x)/F(x) * F(x)$, where * denotes convolution (for symmetrical stimuli, this is the same as cross-correlation), $F(x) =$ the neural image in response to the target alone, and $G(x) =$ the neural image in response to the target-plus-mask. Again, these numbers in no way resembled Bridgeman's Figure 6. We then computed Pearson rs [the normalized cross-correlation for the points F(0), G(0)], and finally our results agree in all major respects with Bridgeman's Figure 6 (for the minor respects in which they do not agree, see footnote 3).

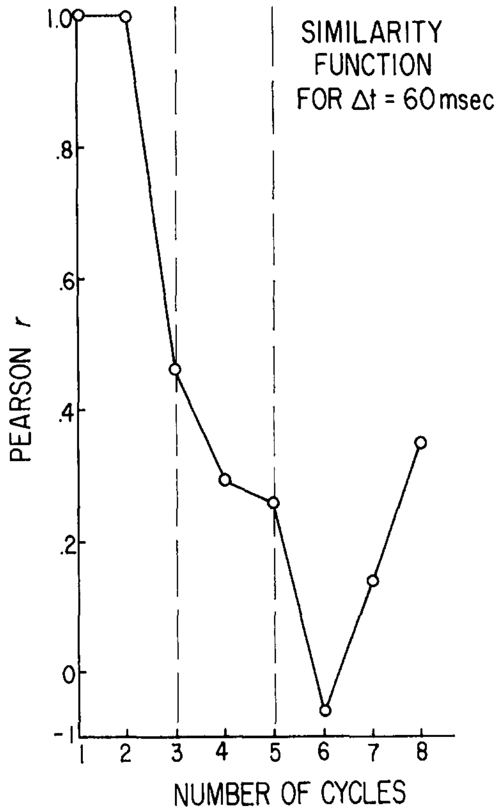

FIGURE 3. The similarity function (what Bridgeman, 1971, calls the cross-correlations) for what is assumed to be the metacontrast or backward masking case (actually, this represents only the point $\Delta t = 60$ msec). The points on this function are Pearson *r*s for the neural image pairs (disc alone, disc-plus-annulus) which occur during the same cycle. Each column in the top and bottom row (each cycle) in Figure 2 allows calculation of another Pearson *r*.

tion about only one delay (or value of Δt). But the information from the similarity function can be suitably processed to obtain a metacontrast function. We can decide on an appropriate measure for masking (a measure that computes from the similarity function for a particular delay a number that corresponds to target apparent brightness for that delay) and then we can repeat this calculation on similarity functions for a sufficient number of delays to give us a metacontrast function.

If we take these calculations step by step, we must first decide on what information to compute from the similarity function. The computation will yield a number, which will be assumed to correspond to target apparent brightness. One should remember that the model assumes that the apparent brightness of a target is mediated by its metaphotic images, and that masking is related to how similar the neural images that occur when a target is flashed in conjunction with a mask are to the neural images that occur when a target is flashed alone. However, the set of metaphotic images cannot be a necessary condition for target apparent brightness, since a steadily presented disc of light has a distinct apparent brightness, and therefore homophotic stimulation is sufficient for the perception of apparent brightness. (This is related to the problem of how the system is able to distinguish homophotic from metaphotic stimulation in any case.) So a suitable measure of apparent brightness for a target for a particular Δt might consider both homophotic and metaphotic neural images, and simply sum *the similarity values, that is, sum the points* computed for the similarity functions for that Δt. The sum would run from the onset of stimulation (the first cycle) through the seventh or eighth cycle (the just-distinguishable signal).[5] The greater the similarity, the greater the value of the sum, the brighter the target, and hence the less the masking.

The results of these calculations are shown in Figure 4. Figure 4a shows calculations for three points in the original network; in Figure 4b, we calculated all the other $+\Delta t$s that can be generated by the network. These are 30, 90, 120, 150, 180, and 210 msec. (The entire set of $+\Delta t$s is limited to about eight, due to the quantization of time and the fact that, at Δts longer than 210, the target signal can not be distinguished from

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[5] It should be noted that we also tried summing various other combinations of cycles: three through seven (i.e., just the metaphotic cycles), four through six, and so forth. None of these sums changes the argument. However, one should not restrict measurement to the later cycles, and indeed, measurement should stop at about the seventh or eighth cycle, since after this even though the Pearson *r* may still take on large negative or positive values, the actual signal is indistinguishable from noise.

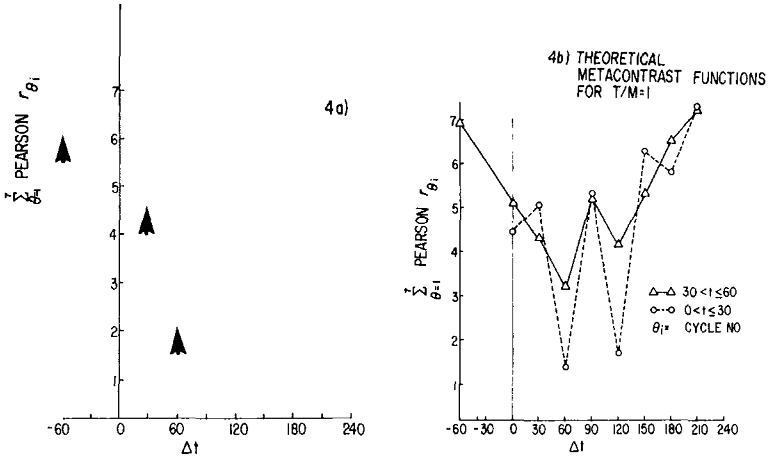

Figure 4. Masking, as computed from Bridgeman's (1971) similarity functions. Each point represents the sum of Pearson *r*s at a particular Δt from the first through the seventh cycle. Figure 4a shows what one would obtain if attention were restricted simply to the three values of Δt that Bridgeman considered; Figure 4b shows the masking functions one obtains when one computes the sum of points on each similarity function for all the other Δts that can be generated by Bridgeman's network. The dotted line shows the masking function obtained when both target and mask are presented for one cycle of the network; the solid line shows the masking function obtained when both target and mask are presented for two cycles of the network (allowing the network one application of inhibition before stimulation is removed). These can be thought of, respectively, as a target and mask presented for durations of $0 < t \leq 30$, and $30 < t \leq 60$, where $t =$ duration.

noise.) In addition, we calculated the same set of Δts for another duration, namely, a duration less than or equal to 30 msec, or the condition where the network does not have a chance to initiate a second cycle before the stimulus is turned off.

The arrows in Figure 4a suggest the beginnings of a U. But when one extends these calculations to include the complete set of Δts possible in this network, it becomes apparent that the function reverses itself two or three times (rather than just once, to produce the U) and thus does not resemble metacontrast functions which are empirically obtained. Let us examine this problem, and related temporal problems, in a little more detail.

Shape of the Metacontrast Function

Networks such as the one in Figure 2, with inhibitory feedback occurring after some short, discrete delay, are subject to damped oscillations or temporal "ringing." That is, a point on the neural image will, with sustained stimulation, first fire at a certain rate; on the next cycle, it will fire less; on the succeeding cycle, it will fire somewhat more again; and so on. This ringing allows strong masking to develop. Pairs of neural images (target, target-plus-mask) at particular cycles can become quite dissimilar, with corresponding points on the neural image even having differing signs (see Figures 2 and 4, cycle 6 for instance: The Pearson *r* for this cycle goes negative). But ringing also results in the metacontrast function itself oscillating a number of times. If, for a given Δt, the neural images produced by a target-plus-mask resemble the neural images produced by a target alone, then for the next Δt, the neural images of the target-plus-mask will be out of phase with those of the target alone, and hence metacontrast masking at successive Δts will first be weak, then strong, then weak again, then strong again, and so on. But metacontrast functions do not oscillate (Alpern, 1953; Growney & Weisstein, 1972; Schiller & Smith, 1966; Weisstein, 1971, 1972; Weisstein, Jurkens, & Onderisin, 1970). It is unlikely that if metacontrast functions had two or three major troughs and peaks they would have been missed by the sampling of Δts utilized by the various studies. For instance, Figure 1 shows empirical metacontrast functions (solid lines) which contain 27 Δts, 21 of them spaced in 10 msec increments, from -100 to $+100$ Δt. In the design from which these data are drawn, all the Δts are presented in random order five times per Δt per target luminance per subject. While there are some ragged edges in Subject TJ's functions, there is no evidence of the kind of abrupt reversals shown in Figure 4b.

Other Temporal Properties of the Network

What matters for a metacontrast function is the *ratio* of target duration to mask duration (with luminance held constant), which we will call T/M. When the durations of target and mask are equal, that is, when T/M = 1, the same U-shaped metacontrast function is obtained, no matter what the durations of target and mask, that is, no matter what T and M (Kahneman, 1967; Weisstein, 1972; Weisstein & Growney, 1969). Thus a target and mask can be presented for 25 msec each or for 125 msec each, and the difference in the metacontrast functions is negligible (Kahneman, 1967). On the other hand, as $T/M \rightarrow 0$, metacontrast functions change shape (they become monotonic, increasing with increase in Δt, as in Figure 1). Neither of these properties of metacontrast is generated by Bridgeman's (1971) network.

Target-to-mask duration ratio approaches zero ($T/M \rightarrow 0$). Time is a discrete function of number of cycles in this network. While there is nothing wrong with approximating time by discrete steps, the assumed duration of each step is much too large to simulate the pertinent features of metacontrast. This is especially true for the first 30 msec, when T/M is the most sensitive to variations in duration. For instance, if one presents a target for 1 msec and a mask for 16 msec, a monotonic function of $+\Delta t$ will be obtained; if one presents a target for 16 msec and a mask for 16 msec, a U-shaped function of $+\Delta t$ will be obtained (Weisstein, 1971). This network cannot distinguish between these two cases.

Target and mask of equal duration (T/M = 1). At the jump points of the network —30 msec, 60 msec, 90 msec—the neural images change. This means that if one holds T/M constant at 1, but multiples of 30 msec are added to both target and mask (that is, one changes the number of cycles the network goes through while each stimulus is present), different metacontrast functions will be obtained. Figure 4b illustrates this: The solid line shows a masking function obtained by allowing the network to receive the target (30 msec) and then recycle once while the target is still present, for a total target duration of $30 < t \leq 60$. The dotted line shows the masking function obtained by allowing the network no recycling while the target is on: It simply receives the target ($0 < t \leq 30$). In each case, the mask is presented for the same duration intervals as the target. Since T/M = 1, the masking functions should be identical. However, as Figure 4b shows, the functions are quite disparate. This contradicts a considerable body of empirical evidence (Weisstein, 1972).

These temporal problems are not caused by the fact that time is quantized in this network, but rather by the dependence of the recurrence of inhibition on the stepping of time. Hence, the problems are not solved by changing the cycle time, that is, by decreasing or increasing the duration between steps. Making the cycle time longer will produce masking at Δts which are too great to correspond to what has been found empirically, and it will only increase the difficulties in predicting monotonic functions as $T/M \rightarrow 0$. Making the cycle time shorter will only translate the metacontrast functions leftward and increase oscillation frequency. This can be illustrated in Figure 4b: Shortening the cycle times, for example, would be equivalent to rescaling the x-axis. If this were done, and each cycle time were assumed 5 msec long (so that the intervals were -10, -5, 0, $+5$, $+10$, and so on) it would solve neither the problem of oscillation nor the problem of differing metacontrast functions for $T/M = 1$ for different target–mask durations. But shortening the cycle time would introduce additional problems: Masking would occur too soon, and, at the delay at which masking *should* occur, the network would have already gone through so many cycles that the signal would be too damped to be distinguished from noise.

Spatial Properties

The network exhibits spatial as well as temporal ringing. This means that it does not predict the spatial properties of metacontrast either. Empirically, as the separation between target and mask increases, metacontrast decreases monotonically. The network, on the other hand, produces abrupt reversals in masking as the separation between target and mask is increased, unit by unit. So, for instance, masking is *greater* at spatial separations of three neural units between target and mask than it is at separation of two units.[6] These abrupt reversals are again a function of the ringing of the network, especially at the edges of the target and mask; when these neural images are in phase, masking is slight; when they are out of phase, masking is heavy.

The Linking Hypothesis: Correlation as an Index of Apparent Brightness

Even if the temporal and spatial properties of metacontrast were better predicted by this network, the way neural image similarity is measured cannot be justified. For each cycle, a Pearson r is computed for the neural images obtained in response to the target alone versus those obtained in response to the target-plus-mask. What this means is that the value of each element in the network for a target neural image at a particular cycle is multiplied by the same element in the network for a target-plus-mask neural image at that cycle, and then the products are summed across the entire network and normalized. Consequently, the measure will produce masking no matter what is added to the target, as long as it is presented somewhere else in the network (and, at least until the cycles spread the neural image to the location in the network where the additional stimulus is located, it doesn't matter *where* else in the network the additional stimulus is presented). That is, for this measure, the magnitude of masking is not affected by the separation between target and mask until the signal is spread far enough. Thus, within some finite number of cycles one could simply add a second stimulus at the very edge of the network, or some place in the middle, and the same amount of masking would be produced. Indeed, one could add two, three, many stimuli at the edges of the network and produce lower and lower correlations.

This does not agree with the evidence. Adding flanks in metacontrast only decreases the apparent brightness of a target when the flanks are close to it (Alpern, 1953; Weisstein & Growney, 1969). As the spatial separation between target and mask increases, masking declines sharply. Furthermore, when disc and annulus are presented simultaneously at the same luminance and duration, enhancement may occur (Matthews, 1974).

It might be more reasonable to inspect the change in neural activity only for those units in and around the location of the target.[7]

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[6] The sum of Pearson rs for $\Delta t = 60$ msec (Bridgeman's condition, 1971) for seven cycles at a separation of two units between target and mask is 3.75; at a separation of three units between target and mask it is 2.94. If one inspects the metacontrast function for this condition (solid lines in Figure 4b), it is clear that relative to the rest of the points, this is a large difference.

[7] It could be argued that the information about the target might not be restricted to a retinally localized area. Such distributed encoding schemes are discussed by Weisstein (in press). Distribution of target information over an extensive retinal area may be a possibility, but then, generally,

When this is done, the correlations remain near 1.00 for both the simultaneous condition and for 60 msec Δt. Cycle by cycle, for 60 msec Δt, they are 1.0, 1.0, 1.0, .97, .932, .997, .999. This hardly constitutes a reduction in similarity.

Summary of Metacontrast Simulation 1

This model fails to simulate a metacontrast function, and it predicts neither the spatial nor the temporal properties of metacontrast. In addition, masking is predicted only by the use of an implausible linking hypothesis.

Many of these flaws have specifically to do with the way in which this particular simulation is constructed and the assumptions behind it. But some of the difficulties might beset any single-channel network, which essentially repeats the physical stimulus (with some modification). Such a network preserves information, but it can hardly be thought of as processing information. That is, different features of a stimulus, which might provide some basis for deciding what kind of stimulus has been presented and when that stimulus has been presented, are not independently represented, at least at the level where the network is still single channel.

We can use Bridgeman's (1971) network as an example of the problems this may create. In Bridgeman's network, apparent brightness is distinguished from detection by a stimulus-dependent event—the stimulus is turned off. But this assumption is untenable for two reasons. First, with a single channel alone, it is not possible to distinguish which is the homophotic and which is the metaphotic signal. Second, the assumption that metaphotic activity results in apparent brightness contradicts the obvious fact that if a stimulus is left on long enough for a subject to make a response to it, the subject will respond that it has a distinct apparent brightness. However, making things stimulus-dependent may be one of the few ways one can distinguish between different stimulus properties while preserving the notion of a single channel.

It is entirely possible that a more convincing single-channel model can be constructed that will overcome some of these problems. Meanwhile, however, it might be worthwhile to turn attention to the other metacontrast simulation (which assumes multiple channels and a rather straightforward linking hypothesis) and see if this simulation does any better.

Metacontrast Simulation 2

The other metacontrast simulation (Weisstein, 1968, 1972) assumes that there is mutual inhibition between fast- and slow-responding neural populations. In particular, for metacontrast it assumes that units whose response is fast inhibit units whose response is slower. Hence, one must delay the stimulus for the fast-responding units in order to have them interfere with a prior stimulus.

Quantification and Criticisms

These assumptions were worked out quantitatively in a neural network incorporating Rashevsky-Landahl two-factor neurons—neurons whose temporal response is a combination of excitation and inhibition (Weisstein, 1968, 1972). This network successfully predicted a number of properties of metacontrast, such as (a) the shape of the metacontrast function and its change of shape as T/M changes (as in Figure 1); (b) the reappearance of the target when target and mask are repeatedly presented at appropriate Δts (as in Schiller & Smith, 1966); and (c) for 34 out of 35 data points that appeared in the metacontrast literature, the Δt at which the minimum of the U occurred. This model is continuous, so any combination of target and mask luminance, duration, and Δt can be simulated.[8] It does

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more than one channel is assumed, and one must use a more plausible linking hypothesis to relate such distributed encoding to perceptual events.

[8] Inspection of the predictions from Weisstein's (1968, 1972) model in Figure 1 might, at first glance, seem to indicate that the same type of quantization of time applies to her model as well. But this is not the case. Weisstein's network was simulated on an analog computer, and within the resolution of the computer, any duration and Δt can be simulated.

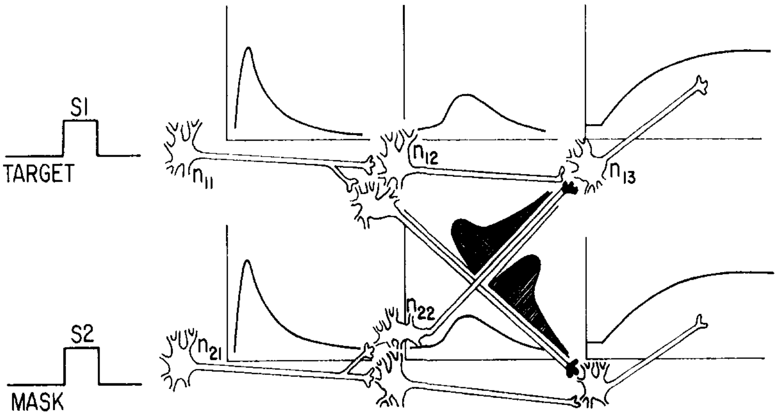

FIGURE 5. Expanded version of Weisstein's (1968, 1972) model, which clarifies the spatial symmetry implicit in that model. The subscripted neurons are those which also appear in Weisstein (1968, 1972). Neuron n_{22} responds faster than n_{12}; therefore, stimulation for n_{22} must arrive later than stimulation for n_{12} in order that the response of n_{13} be suppressed. If one traces responses in the unlabeled neurons, it is clear that an identical effect will occur for the bottom neuron similar to n_{13} if the stimulus in the top row is delayed. Thus one can interchange target and mask and produce the same result.

not depend on temporal ringing to produce masking, and thus it is not subject to the same temporal difficulties as the network considered earlier. In addition, the measure of target apparent brightness is proportional to the frequency of firing of the unit designated as carrying information about the target, so the linking hypothesis is not quite so problematic as it is in Bridgeman's (1971) network.

However, there are two criticisms of this simulation. One has to do with what are assumed to be the spatial constraints imposed by the model, and the other with a temporal assumption behind some of the parameters chosen for individual neurons.

Spatial Interaction

The criticism here has been that "the simulation will not work if target and mask are interchanged" (Bridgeman, 1971, p. 530). This seems to us easily solved. Weisstein's diagram for the metacontrast interaction (1972, p. 248) is *not* symmetric, but symmetry is implicit, and it is easily obtained without making any additional assumptions. Figure 5 depicts the following: Somewhere, some place in the central nervous system, two neurons receive the same excitatory input. The response of one of these neurons is faster than the response of the other, and there is cross-inhibition from these fast responders. That is, the neurons that are responding faster inhibit their successive neighbors. Because their response is fast, inhibition will be effective only if the stimulus for these fast responders is delayed. Working through the various combinations, it is immediately obvious that the network is symmetrical: All that is required for one stimulus to suppress response to another is that it follow the other. (Although the schematic shows only one line of units responding to target and one line of units responding to mask, it is assumed that there will be a collection of units responding to any stimulus.)

The Assumption of Fast Inhibition

Consider now the temporal assumption. Weisstein chose rate constants for the inhibitory factors in most of the neural units that were faster than the rate constants for the excitatory factors. Since the inhibitory and excitatory rate constants are supposed to correspond to inhibitory postsynaptic potentials (IPSPs) and excitatory postsynaptic potentials, (EPSPs), respectively, this amounted to an assumption that IPSPs were faster than EPSPs. Although there is some evidence for fast IPSPs (Creutzfeldt & Ito, 1968), in general the assumption of IPSPs being faster than EPSPs appears tenuous.

However, there are other ways to produce fast and slow responses. With two-factor neurons, the particular rate and shape of the neural temporal response depends most critically on the relationship between the ratios of excitatory gain to excitatory rate constant versus inhibitory gain to inhibitory rate constant, and only secondarily on the rate constants themselves. One can produce a fast response in a neuron—that is (as illustrated by n_{22} in Figure 5), a response in which firing frequency reaches some criterion amplitude rapidly after stimulus onset and decreases to some criterion amplitude rapidly with stimulus offset, so that the latency of response is short, and the temporal resolution is high—by adjusting the gains in that neuron (A, B), the rate constants (a, b), or both. This can be seen by solving Equations 2 and 3 in Weisstein (1972), which would give the functions for ϵ and j, that is, for the excitatory and inhibitory factors that combine to give the neural temporal response. The solutions for a step function input, setting initial conditions to zero, are the familiar exponentials,

$$\text{Response} = \epsilon(t) - j(t) = [(A/a)(1 - e^{-at})] - [(B/b)(1 - e^{-bt})],$$

where A is the gain and a is the rate constant for the excitatory factor, and B is the gain and b is the rate constant for the inhibitory factor, and $e = 2.718$. Now, if one assumes, for instance, that $a \gg b$ (that is, EPSPs that are considerably faster than IPSPs), and $A \gg B$, such that A/a is slightly larger than B/b, a fast, sharp transient excitatory response will be produced, which will then die down to some minimal firing slightly above spontaneous level for as long as a stimulus is maintained.

On the other hand, if one assumes $a > b$, and $A > B$, such that $A/a > B/b$, a slower, more sustained response will develop. Since a number of afferent orders are assumed to be traveled before the metacontrast interaction, one could enhance these differences by further convolution (the fast response would get somewhat slower; the slow response would get a great deal slower). Thus, there are a number of different choices of parameters that will give fast and slow responses. But one does not even have to adjust parameters relative to each other to produce Weisstein's results. One could obtain fast and slow responses simply by assuming slower conduction times in the slower responding neurons. Either (or both) of these alternate ways of producing a fast response would only minimally affect the predictions from the Weisstein network, since the narrowing of the functions and their shift towards the origin, with $T/M \rightarrow 0$, as well as the other temporal properties of metacontrast that were predicted, are due not so much to the temporal *shapes* of the slow and fast neurons (responses that are influenced by the rate constants), but to the neurons' nonlinearity (Weisstein, 1972, Equation 4, p. 246) and to the fact that the response to a stimulus, even if it were transient, would nevertheless smear out somewhat in time when the energy of the mask is high.

The nonlinearity means that the functions will first be wide and then narrow as mask energy increases; the "smearing" of the mask in time means that one will not have to delay presentation of the mask as much, as its energy increases, in order to have it interfere with the target, and thus the metacontrast functions will shift their minima toward the origin, as in Figure 1.

In short, alternate ways of producing a fast response are easily available; the focus on fast IPSPs, or "fast inhibition" (Weisstein, 1972), obscures the main point that the only thing that has to be fast in Weisstein's model is a fast excitatory response, which then inhibits a slow excitatory response. To construct this, one can choose parameters in several ways, only one of which involves choosing faster IPSPs.

One could decide on one or another of the alternative ways of producing a fast response by drawing on the accumulating data about the properties of transient and sustained neural populations in the visual system (see below), and the equations for the two-factor neurons could be revised in light of this evidence. However, for now, since alternate constructions will yield roughly the same results, we prefer simply to note the equivalence of the different ways of looking at what was called fast inhibition.

Further Elaboration of Metacontrast Simulation 2

Edges and blobs. The main assumption in this model is that more than one temporal weighting function is needed to characterize visual system response (i.e., fast versus slow response). So the simulation involves multiple temporal channels, two classes of which are assumed to interact. But there are spatial assumptions as well. Weisstein (1968, 1971, 1972, 1973) proposed that the units that mediated detection of a stimulus had different time constants than the units that carried information about contour or edge. She assumed, as Werner had suggested (1935) that the basis of metacontrast was the suppression of edges or contour information, and that apparent brightness decreased as some function of the suppression of edges. Edges have long been known to play a critical role in the perception of apparent brightness (e.g., Cornsweet, 1970; Land & McCann, 1971; O'Brien, 1958; Shapley & Tolhurst, 1973).

If apparent brightness is largely dependent on edges, then "edgeless" stimuli should be more resistant to metacontrast masking. This seems to be the case: Growney (in press) has shown that one obtains negligible amounts of metacontrast masking when the sharp edges of a target are blurred. Masking is not as negligible when the target edges are sharp and the edges of the mask are blurred. Thus relatively "edgeless" stimuli (they are not completely edgeless, especially if one analyzes edges in terms of the amount of high spatial-frequency components they contain) do not get masked very much.[9]

The slow response in Weisstein's model (1968, 1972) was assumed to have something to do with contour or edge. Evidence has since accumulated that high and low spatial frequencies may be processed by different temporal channels (e.g., Kulikowski & Tolhurst, 1973), and that these different temporal channels may respond with differential speed and resolution (e.g., see discussion by Breitmeyer, in press; Breitmeyer, Love, & Wepman, 1974; and Breitmeyer, Note 1; also see Hood, 1973). A high spatial-frequency response may be thought of (very roughly) as a response to edges, and a low spatial-frequency response may be thought of (just as roughly) as a response to non-edges—blobs, or large, blurred shapes, as in a defocused picture.[10] Thus, given a single stimulus, the response to its edges may be slower than the response to its "blob presence." Hence, both an edge response and a non-edge response may be tied to the accumulating evidence for the presence in the visual system of channels that respond to transient, low spatial-frequency stimuli, and channels which respond to sustained, high spatial-frequency stimuli. (This direct association of fast and slow responses in metacontrast with transient and sustained channels was first suggested by Breitmeyer

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[9] Growney (in press) has presented quite interesting evidence that the specific type of edge in metacontrast in both target and mask can change the amount of masking obtained. While some of this is easily explained by the edge-blob scheme discussed below, some of this may require further elaboration.

[10] Actually, both edges and blobs will have low frequency components, but edges will also contain more high frequency components. Edges and blobs can be specified much more precisely, of course—for any given edge or any given blob, one can calculate its spatial frequency or Fourier spectrum. Similarly, the units sensitive to edges and blobs can be defined by the spatial frequencies to which they respond. But until we know considerably more about these units' spatial frequency response—bandwidth, channel density, variation of temporal response with center frequency (see Graham, in press, for an elegant summary of our ignorance on these matters, and an excellent discussion of how to overcome it)—we lose nothing in precision by talking about edges and blobs, and we may gain in comprehensibility.

et al., 1974, and Breitmeyer, Note 1. A different approach to the relationship between masking and activity in the sustained and transient channels has been suggested by Matin, 1974, and Matin, Notes 2 and 3.)

It should be noted that transient responses do not necessarily imply speed, nor do sustained responses imply delay; however, there is evidence that the higher spatial-frequency channels may require a longer integration time than low spatial-frequency channels (e.g., see Breitmeyer, in press; Hood, 1973; Breitmeyer, Note 1), and there is neurophysiological evidence that the latencies and conduction velocities of neural populations that prefer transient, low spatial-frequency stimuli are faster than the latencies and conduction velocities of those neural populations that prefer sustained, high spatial-frequency stimuli (the Y and X cells, respectively—Enroth-Cugell & Robson, 1966. For evidence concerning conduction velocity and latency of these populations, see Cleland, Dubin, & Levick, 1971, and Fukada, 1971; for evidence that these populations may mutually inhibit each other, see Singer & Bedworth, 1973).

Breitmeyer also has gathered independent evidence from reaction times, which suggests that edges may be processed more slowly than blobs or non-edges (Breitmeyer, in press). He reported that reaction times to high spatial-frequency sinusoidal gratings are greater than reaction times to sinusoidal gratings of lower spatial frequency. These reaction-time data may reflect processing times for edges versus non-edges, since edges contain more high spatial frequencies.

Masking in General

The association of slow and fast responses with the accumulating evidence for sustained and transient, high and low spatial-frequency channels, which we will call the edge–blob scheme, has enormous integrating potential. It may make it possible to explain masking phenomena that have not yet adequately been treated; and it may help explain the relation between apparent movement and metacontrast (see also Breitmeyer et al., 1974; Kahneman, 1967, 1968; Weisstein & Growney, 1969; Matin, Notes 2 and 3, for discussions).[11]

As long as channels are near enough to each other to inhibit each other, the edge–blob scheme works for metacontrast, as stated: U-shaped backward masking occurs because the fast part of the response to the mask inhibits the slow part of the response to the target. This can be easily extended to nonmetacontrast designs, where U-shaped masking functions are also obtained (Purcell & Stewart, 1970; Weisstein, 1971; Turvey, 1973), that is, designs where the target and mask partially or completely superimpose on the same retinal areas. Separate channels are assumed mutually inhibiting; as they approach each other's retinal locations (including superimposition), inhibition will become stronger or remain the same, depending on one's specific spatial-frequency assumptions (e.g., Weisstein, in press). For forward masking, a U shape would be predicted if the slow part of the response to the mask inhibited the fast part of the response to the target.

Here, one might assume that since the target blob response would be inhibited (rather than the target edge response, as in backward masking), the forward analog of metacontrast—paracontrast, where the mask precedes the target—would not be as great as metacontrast, since it is the edges of a

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[11] It should be noted that although the scheme generalizes beyond typical metacontrast backward masking, it does not (nor is it meant to) account for masking that is thought to be more peripheral—masking that decreases detection, depends primarily on overlap of the target and mask, produces montonic functions, and so forth (see Kahneman, 1968, and Weisstein, 1972, for a review of the different types of masking). That is, although blobs might play a role in the detectability of the target, the edge–blob distinction does not commit the blobs to target detection, as might be assumed from the earlier model (Weisstein, 1968, 1972) where the basic distinction was between contour interactions and detection. Rather, masking that involves simple detection may occur as a result of different mechanisms than the ones suggested here; in any case, at the level of processing we are considering, we assume that both blobs and edges play a role in target appearance.

stimulus rather than its blob presence that seem to play such an important role in determining apparent brightness and the other features of the appearance of the target which get disrupted in metacontrast. On the other hand, if the appearance of the target is even minimally dependent on blobs as well, then, although one might not expect much paracontrast until the mask became quite strong, when the mask did become strong, forward U shapes would be expected. Finally, if the mask produced a response that was much greater than the target response, one might predict the same kind of shift of the bottom of the U that one finds in backward masking. This seems to be what happens. In Figure 1, the forward U is either absent or appears as a minor dip compared to the backward U when T/M ≈ 1. It only reaches a respectable size as T/M decreases substantially; then, with further decreases in T/M, the bottom of the U begins to shift towards the origin. So, the edge–blob scheme might be able to account both for forward and backward Us, and for their asymmetry.

Finally, there has always been the nagging suspicion (see footnote 1) that metacontrast does not really exist. In a metacontrast design, while it is clear that target apparent brightness decreases, it is also clear that there is something going on in the location of the target, and indeed, detection measures rarely produce U-shaped metacontrast functions. Also, apparent movement is generally noticed in metacontrast, even when the target apparent brightness is negligible. The edge–blob explanation could account for both of these things: If only the edges of a target are suppressed, something indeed remains. That something would be the target blob. But since blobs produce a stronger response in the transient channels, those which prefer moving stimuli, there is reason to suppose that the continued activity of these channels during metacontrast masking would lead to the impression of movement.

CONCLUSION

A stimulus produces prolonged neural activity. The notion that different perceptual events are associated with different portions of this activity is intriguing, and it lies at the basis of all of the theories of metacontrast discussed in this paper. The major difference in the various theories has to do with assumptions about visual system organization. Here, Bridgeman's (1971) model appears to fail in important respects, whereas Weisstein's (1968, 1972) model does not. While much of this may be due to the particulars of the way Bridgeman set up his model, some of the failure does appear intimately connected to the general notion that a single spatial and temporal channel describes visual system activity. On the other hand, Weisstein's model (with subsequent elaboration) not only offers the prospect of simulating many of the relevant spatial and temporal aspects of metacontrast, but agrees with much current investigation indicating both the presence in the visual system of multiple spatial and temporal channels (Campbell & Robson, 1968; Graham & Nachmias, 1971; Kulikowski & Tolhurst, 1973; Pantle, 1971), and also (for spatial channels at least) their mutual inhibition (Tolhurst, 1972). It is possible that by conceptualizing metacontrast in terms of multiple spatial and temporal channels, we may get a clearer idea of the variety of perceptual functions served by these different channels.

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