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Orientation-specificity and disinhibition in type B pattern masking.

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ORIENTATION-SPECIFICITY AND DISINHIBITION IN TYPE B PATTERN MASKING

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DEDICATION

To Arnie Well,

advisor, teacher, colleague, and friend.

Thank you for your belief in my abilities

and your constant moral support.

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ABSTRACT

Orientation-Specificity and Disinhibition in Type B Pattern Masking

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Breitmeyer and Ganz (1976) proposed that Type B paracontrast is due to intrachannel inhibition of sustained channels while Type B metacontrast is due to interchannel inhibition of sustained by transient channels. This theory of Type B masking yields a number of predictions, two of which are that 1) masking under paracontrast conditions should be more orientation-specific than under metacontrast conditions, and 2) the introduction of a second mask should produce disinhibition under paracontrast but not under metacontrast conditions. These two predictions were investigated in the present study using a 4 c/deg target square-wave grating and two 4 c/deg mask square-wave gratings (mask one and mask two). The degree of masking was represented by the probability of error in detecting the target grating in a forced-choice procedure (on 50% of the trials target gratings appeared, on the other 50% blank fields of the same mean luminance and size as the target grating appeared).

In Experiment 1 orientation-specific masking was studied by presenting mask one at four orientations while holding the target orientation constant. Mask one consisted of two square-wave gratings which

flanked the target. SOAs varied from -90 to 90 msec. Under metacontrast conditions the masking function was strongly U-shaped for three subjects and weakly U-shaped for one subject. Paracontrast conditions led to a strong U-shaped masking function for one subject and a weak U-shaped masking function for a second subject. The paracontrast masking function for the other two subjects was non-monotonic, but not U-shaped. No evidence of orientation-specific masking was found under paracontrast or metacontrast conditions for any subject. This result may reflect the large amount of variability in masking at each mask orientation. An alternative explanation in terms of an interaction between inhibition due to channels tuned to the same orientation and inhibition due to channels tuned to different orientations is also discussed.

The target stimuli, mask one, and mask two were presented in Experiment 4 in order to investigate disinhibition. Mask two was two 4 c/deg square-wave gratings which flanked mask one. Under paracontrast conditions mask two preceded mask one and the target, while under metacontrast conditions mask two followed mask one and the target. At a number of paracontrast and metacontrast SOAs for all subjects it was found that masking was less than would be predicted assuming independence of mask one and mask two effects. This result is contrary to the theory of Type B masking proposed by Breitmeyer and Ganz (1976). Possible modifications of this theory which would account for these results are discussed. These include the occurrence of transient intrachannel inhibition during paracontrast and interneuron mediated transient inhibition of transient channels during metacontrast disinhibition.

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INTRODUCTION

Under the proper conditions the ability to report a visual stimulus (target) can be reduced by the presentation of a spatially adjacent stimulus (mask). Type B pattern masking is defined as occurring if this reduction is at a maximum when a pattern mask either follows or precedes the target. Paracontrast is a reduction in the ability to report the target stimulus when it is preceded by the mask, while the occurrence of this reduction when the mask follows the target is known as metacontrast. Various explanations of Type B paracontrast and metacontrast have been proposed (e.g., Lindsley, 1961; Kahneman, 1968; Bridgeman, 1971; Weisstein, 1972). The present study was an investigation of a theory which attempts to explain Type B masking in terms of interactions between sustained and transient visual channels (Breitmeyer and Ganz, 1976).

A great deal of psychophysical and physiological evidence suggests that visual processing may occur via two independent sets of channels: 1) sustained channels which transfer high spatial-frequency and low temporal-frequency information, and 2) transient channels which transfer low spatial-frequency and high temporal-frequency information (e.g., Cleland, Levick and Sanderson, 1973; Ikeda and Wright, 1975a, 1975b; Kulikowski and Tolhurst, 1973; Tolhurst, 1975). Sustained channels are composed of sustained cells (also called X-cells or tonic

cells) which have a prolonged increase in neural activity to a stimulus presented to a cell's receptive field (e.g., Sherman, Wilson, Kaas, and Webb, 1976). Transient channels are composed of transient cells (also called Y-cells or phasic cells) which yield a brief increase in neural activity to the onset or offset of a stimulus in a cell's receptive field (e.g., Scobey and Horowitz, 1976).

Breitmeyer and Ganz (1976) proposed that Type B paracontrast is due to intrachannel inhibition of target sustained channels by mask sustained channels, while Type B metacontrast is due to interchannel inhibition of target sustained by mask transient channels. If this is true then the addition of a second mask to the normal masking display of a target and one mask should yield disinhibition under paracontrast but not under metacontrast conditions. In other words, when the second mask precedes the first mask and target the masking of the target should not be independent of the effect of the second mask on the first. However, when the second mask follows the first mask and the target the masking of the target should be independent of any effect of the second mask on the first mask. These predictions are tested in the present study, as is a prediction (also derived from Breitmeyer and Ganz, 1976) concerning orientation-specific masking under paracontrast and metacontrast.

As the Breitmeyer and Ganz Theory utilizes sustained and transient channels a review of the evidence concerning these channels is presented in the present paper. This is followed by a review of the Type B masking findings and theories, and then a review of the disinhibition literature.

Sustained and Transient Channels

The physiological cornerstone of these channels lies in the two types of cells first observed by Enroth-Cugell and Robson (1966). In their study the response of ganglion cells to sinusoidal gratings was investigated using microelectrode recordings from optic-tract fibers of the cat. Response properties of the cells indicated two cell types. For the first type, spatial summation over the cell's receptive field (RF) was approximately linear, gratings moved across the RF produced little response, and certain positions of a grating within the RF produced no response. Spatial summation for the second cell type was very non-linear, response frequency was greatly increased by moving gratings across the RF, and any grating position within the RF always produced a response. Enroth-Cugell and Robson (1966) referred to the former as X-cells and the later as Y-cells. Since this time X-cells have also become known as sustained cells (due to the sustained response of such cells to visual stimuli) while the Y-cells have also been termed transient cells (due to the transient response of such cells) (Hoffman, Stone, and Sherman, 1972; Cleland, Levick, and Sanderson, 1973; Ikeda and Wright, 1975a).

This sustained-transient dichotomy has also been found to exist at the lateral geniculate nucleus (Hoffman, Stone, and Sherman, 1972) and at the visual cortex (Dow, 1974). Furthermore, it has been found that transient retinal neurons project to transient neurons of the lateral geniculate nucleus (LGN), which in turn project to transient visual cortex neurons (Hoffman and Stone, 1971; Hoffman, Stone, and Sherman,

1972). Similar excitatory projections have been found for sustained cells at these levels of the visual pathway (Hoffman and Stone, 1971; Hoffman, Stone, and Sherman, 1972). Thus a physiological structure for parallel and independent sustained and transient channels extends from the retina to the visual cortex.

Electrophysiological and psychophysical studies have consistently found a large number of characteristics distinguishing sustained from transient channels. The characteristics are in terms of responses to various stimulation and physical properties of the X- and Y-cells. Sustained neurons have been found to be more sensitive to high spatial frequency visual stimulation while transient neurons respond more readily to low spatial frequencies (Cleland, Levick, and Sanderson, 1973; Ikeda and Wright, 1974; Fukuda and Saito, 1971). In the temporal domain, sustained neurons respond more readily to stationary or low frequency stimuli, while transient neurons respond to high frequency or rapidly moving stimuli (Singer and Bedworth, 1973; Movshon, 1975; Ikeda and Wright, 1975a).

Ikeda and Wright (1972) have shown that transient cells are not sensitive to refractive errors or image blur while sustained cells do show such a sensitivity and respond best to sharply focused images. This result is consistent with the spatial frequency characteristics of each cell type. Psychophysical studies also indicate two independent channels; one transferring high spatial and low temporal frequency information, the other sensitive to low spatial and high temporal frequencies (Kulikowski and Tolhurst, 1973; Breitmeyer and Julesz, 1975; Keesey, 1972; Pantle, 1970).

Kulikowski and Tolhurst (1973) studied the sensitivity of the human visual system to various spatial-frequency sine wave gratings which were temporally modulated at various frequencies. Subjects performed a flicker threshold detection task (simply increasing the contrast of a grating until it was seen as flickering) and a pattern recognition detection task (increasing the contrast until the spatial frequency of the grating could be reported). Two distinct thresholds were found for a temporally modulated grating: flicker could be detected at a low contrast, while a higher contrast was needed to detect the spatial structure of the stimulus. The flicker detection threshold and pattern recognition threshold varied independently as functions of the spatial and temporal frequencies. Sensitivity of flicker detection was greatest for low and medium spatial frequencies and poor at low temporal frequencies, while the sensitivity of pattern recognition was greatest at high and medium spatial frequencies with no decline in sensitivity at low temporal frequencies. These results suggested to Kulikowski and Tolhurst (1973) that there are two independent systems of channels; one system transferring low spatial and high temporal frequencies, the other responsible for high spatial and low temporal frequencies.

Single cell recordings have demonstrated that sustained neurons have a longer response latency to visual stimuli than transient neurons. Cleland, Levick, and Sanderson (1973) recorded post-stimulus histograms of neural impulses per second from sustained and transient ganglion cells of the cat retina. For transient neurons the distribution (of

summed neural responses to fifty stimuli presentations) reached a maximum at approximately 40 msec. after stimulus onset, while the distribution for sustained neurons reached a maximum at about 80 msec. Ikeda and Wright (1975b), recording from Area 17 of a cat's cortex, found that the latency of the beginning of a response histogram was 40 msec. for transient neurons and 60 msec. for sustained neurons. Dow (1974), recording from the striate cortex of rhesus monkeys, reported a latency difference of about 50 msec. between two classes of cells which may have been sustained and transient neurons.[1]

Breitmeyer (1975) reported a corresponding psychophysical finding. Simple reaction time to sinusoidal gratings increased by 46 to 80 msec. as the spatial frequency was increased from 0.5 to 11.0 cycles/degree (c/deg), suggesting that low spatial frequency (transient) channels respond faster by several tens of milliseconds than sustained channels.

A study by Tolhurst (1975) demonstrated that detection reaction time to a low frequency grating (0.2 c/deg) is faster than reaction time (RT) to a higher frequency grating (3.5 c/deg), but only when the response to the low frequency grating is to the onset of the stimulus. Threshold gratings of 3.5 and 0.2 c/deg were presented with three onset-offset combinations: 1) sudden onset and sudden offset, 2) sudden onset and gradual offset, and 3) gradual onset and sudden offset. The type of onset-offset had little affect on the distribution of reaction time responses to the 3.5 c/deg grating (this distribution was unimodal with a maximum at a reaction time of 800 msec.).

However, onset-offset type did affect the reaction time (RT) distribution to the 0.2 c/deg grating. The RT distribution for either gradual onset or gradual offset of the 0.2 c/deg grating was unimodal with a maximum occurring approximately 500 msec. after the sudden transition. However, with sudden onset-sudden offset the RT distribution was bimodal with about 2/3 of the responses occurring approximately 500 msec. after onset and 1/3 occurring approximately 500 msec. after offset. The reaction times to the 0.2 c/deg offset in both the unimodal and bimodal distributions were no faster than the reaction times to the 3.5 c/deg grating. This evidence suggests that the faster latency of the transient channel may occur only when this channel responds to the onset of a stimulus.

Sustained neurons also have a longer response persistence or integration time. Electrophysiological studies of the cat have shown prolonged increases in the rate of neural impulses for sustained cells to visual stimuli of short duration--2 msec. (Cleland, Levick, and Sanderson, 1973)--or long duration--500 msec. (Ikeda and Wright, 1975b). Transient neurons of the cat were found to have a relatively brief response persistence (Enroth-Cugell and Robson, 1966; Cleland, Levick, and Sanderson, 1973; Ikeda and Wright, 1975b). Sherman, Wilson, Kaas, and Webb (1976), recording from the dorsal LGN of owl monkeys, found that the response of sustained (termed X-cells by these researchers) cells persisted for the length of time the stimulus was shown (30 to 50 seconds). On the other hand, transient cells (termed Y-cells) responded for no more than 1 or 2 seconds to such stimuli. Similar

differences in response persistence between sustained and transient cells in primates have been reported by other researchers (Gouras, 1968; Marrocco, 1976; Schiller, Finlay, and Volman, 1976; Scobey and Horowitz, 1976).

Again psychophysical studies yield corresponding findings. Breitmeyer and Ganz (1976) have interpreted Kulikowski and Tolhurst's (1973) finding that high spatial frequency gratings have a lower critical fusion frequency as reflecting a longer response persistence or integration time for sustained channels. The results of Tolhurst's (1975) reaction time experiment show a much wider distribution of reaction times for high spatial frequencies than for low frequency gratings. If as Tolhurst states "reaction time is related to the time at which threshold is first exceeded in a trial" then this difference in the range of the two distributions indicates a prolonged persistence of response to high spatial frequency information.

Estimations of short-term visual storage (STVS) also provide evidence that activity is prolonged in high spatial frequency channels. Maguire and Meyer (1977) presented subjects with various gratings each of 50 msec. duration followed by a blank field. Subjects gave a judgement of the length of the blank interval for which the stimulus seemed continuously present; this provided an estimate of STVS length. Maguire and Meyer found that judged STVS duration increased several hundred milliseconds as the spatial frequency of sine waves increased from 0.4 to 6.4 c/deg.

Single-cell recordings have shown that transient neurons have a faster impulse propogation than sustained neurons. Dreher, Fukada,

and Rodieck (1976) found that with electrical stimulation of the optic chiasm of two species of primate no X-like (sustained) cell in the LGN had a latency shorter than 1.7 msec., while no Y-like (transient) cell had a latency longer than 1.6 msec. Similar findings in primates have been reported by Gouras (1969), Marrocco (1976), and Sherman, et al., (1976). Cleland, Levick, Morstyn, and Wagner (1976) demonstrated similar results in the cat LGN. As Stone and Hoffman (1971) have shown that fast conducting neurons at the cat's LGN project to fast conducting neurons of the visual cortex (and similarly for slow conducting neurons) the difference in impulse propagation between sustained and transient neurons appears to extend to the visual cortex.

Hoffman, Stone, and Sherman (1972), recording from the dorsal LGN of the cat, found that within any localized region of the retina the RF centers of sustained cells were smaller than the RF centers of transient cells. For example, the mean RF center size of 15 sustained cells projecting from an area 0 to 3 degrees from the fovea was approximately 0.5^{o}, while for 29 transient cells projecting from the same area the mean RF center diameter was 1.1^{o}. This result has been supported by Fukada (1971), Cleland, Dubin, and Levick (1971), Cleland, Levick, and Sanderson (1973), and Sherman et al., (1976). The mean size of the entire receptive field of both sustained and transient cells has been found to increase with increasing eccentricity from the fovea (Wiesel, 1960; Stone and Fabian, 1966; Sanderson, 1971).

The relative frequency of transient ganglion cells (expressed as a percentage of the total transient and sustained cell population

in a given retinal area) has been found to increase with increasing eccentricity from the fovea (Hoffman, Stone, and Sherman, 1972; Fukada and Stone, 1974; Ikeda and Wright, 1975b). Hoffman, Stone, and Sherman (1972), recording from the LGN of a cat found that this percentage of transient cells (projecting to the LGN) increased from 34% in the area 0 to 3^{o} from the fovea to 73% in the area 45 to 70^{o} from the fovea. Gouras (1968) reported that transient cells (termed "phasic" by Gouras) were relatively more common toward the periphery while sustained cells (termed "tonic") were more common toward the fovea of the rhesus monkey's retina.

The antagonistic center-surround organization of visual receptive fields yields intrachannel inhibition in both sustained and transient neurons. There are two types of center-surround organization--on-center and off-center. In on-center cells the neural response is increased when a light is presented to the center of the RF and diminished when a light is presented to the periphery of the RF (Cornsweet, 1970). Off-center cells experience excitation when a light is presented to the periphery and are inhibited by a light presented to the center of the RF (Cornsweet, 1970). There are both sustained on- and off-center as well as transient on- and off-center cells (Cleland, Levick, and Sanderson, 1973).

The latency of response of a cell to a stimulus delivered to the center of the cell's RF is somewhat shorter than the latency of response to a surround-delivered stimulus (Poggio, Baker, Lamarre, and Sanseverino, 1969; Maffei, Cervetto, and Fiorentini, 1970; Singer

and Creutzfeldt, 1970). Maffei et al., (1970) recording from the cat retinal ganglion cell estimated that this latency difference is on the order of 20 msec. Singer and Creutzfeldt (1970) recording on- and off-center cells in the LGN of cats reported similar findings. They found that both the on-center excitatory and off-center inhibitory responses of neurons was 20 to 30 msec. shorter than the responses elicited by surround stimulation. Poggio et al., (1969) reported that surround inhibition of neurons in the LGN was most effective when the surround stimulus is presented 10 to 30 msec. before the center stimulus.

A psychophysical study by Fiorentini and Maffei (1970) reported a wider range for this latency difference. Subjects determined the modulation threshold for a light spot surrounded by an annulus of light. The spot and annulus were presented at the same temporal frequency, but this frequency varied from 1 to 16 cycles per second (cps). The phase difference between the two stimuli was also varied. Maximum modulation threshold occurred at a phase difference of 45^{o} for stimuli modulated at 0.8, 1.5, and 6.0 cps. This indicates a surround latency that is from 20 msec. (for 6.0 cps) to 160 msec. (for 0.8 cps) slower than the central latency.[2]

In the studies of center-surround latency difference discussed above no attempt was made to discover whether transient or sustained channels were being investigated. However, Winters and Hamasaki (1976) did distinguish between transient and sustained neurons in a study of surround inhibition in ganglion cells of the cat retina. Winters and Hamasaki found that maximum inhibition for an on-center sustained neuron

occurred when an annulus was presented to an RF surround on the average of 7 msec. (S.D.=3.8) before presenting a spot to the RF center. For an on-center transient cell, maximum inhibition occurred with an average surround-center latency difference of 38 msec. (S.D.=5.9). Winters and Hamasaki also found that this "best delay" (i.e., the surround-center delay which maximizes inhibition) decreased with increasing luminance. As the spot and annulus intensity was varied from 0.4 to 1.6 log units above threshold the average best delay for sustained neurons fell from about 22 msec. to 3 msec., while the average best delay for transient neurons fell from about 53 to 29 msec. Enroth-Cugell and Lennine (1975) found similar results for sustained cells. They reported that the latency difference decreased from 30 msec. during dark adaptation to 20 msec. during light adaptation. It thus appears that the surround-center latency difference is a function of stimulus luminance and the type of channel used.

The results of some studies would seem to indicate that surround inhibition is stronger for sustained cells. Fukada (1971) found that transient cells gave moderate responses to diffuse light over the entire RF while the sustained cells gave a weak or no response. Hickey, Winters, and Pollack (1971) reported that for sustained cells the response to a central RF spot of light was reduced to a greater extent when a peripheral annulus was presented than was true for transient cells. Winters and Hamasaki (1976) point out that in both of these studies peripheral stimulation was presented simultaneously with central stimulation. Since the optimal latency difference for sustained

cells is very near simultaneity (7 msec. according to Winters and Hamasaki), stronger inhibition would be expected for sustained cells in these studies. Winters and Hamasaki could find to difference in the strength of sustained and transient surround inhibition when the appropriate "best delay" was used in presenting central and peripheral stimulation to transient and sustained cells. Equivalent strength of sustained and transient intrachannel inhibition is supported by others (Cleland, Levick, and Sanderson, 1973; Enroth-Cugell and Lennine, 1975). Thus the evidence currently appears to favor a relative equivalence of surround inhibition in both the transient and sustained cells.

Besides intrachannel inhibition, sustained and transient cells appear to exhibit interchannel inhibition. Hoffman, Stone, and Sherman (1972) found that the response of sustained neurons in the LGN of a cat were inhibited by stimulation of transient neurons and transient neurons were inhibited by stimulation of sustained neurons. Hoffman et al., did not report any difference in the strength of these two types of interchannel inhibition, but there study does not appear to have been designed with such a difference in mind.

Singer and Bedworth (1973) found strong support for transient inhibition of sustained cells. Cells of a cat's LGN were classified as X and Y (sustained and transient) and their RF's mapped. The cells were then stimulated via spots of light to their RF or by electrical stimulation at the optic tract, optic chiasm, or superior colliculus. Singer and Bedworth found that when the RF center of a sustained and a transient cell coincided, the sustained cell was inhibited by a

fast moving stimulus (300^{o}/sec.) while the transient cell increased its firing rate. The sustained inhibition was maximal when the transient excitation was maximal; furthermore, the sustained inhibition had the same time course as the transient excitation. Singer and Bedworth also found an early IPSP (inhibitory post-synaptic potential) in sustained cells occurring before the first EPSP (excitatory post-synaptic potential). Considerations of conduction velocities of sustained and transient neurons (Hoffman and Stone, 1971; Stone and Hoffman, 1971) plus the assumption that post-synaptic inhibition in the LGN is mediated via interneurons (Burke and Sefton, 1966a, 1966b) led Singer and Bedworth to the conclusion that the early sustained IPSP must be a result of transient excitation. IPSP's were also produced in sustained cells at stimulus intensities well below threshold for sustained cell EPSP, but at or above transient cell EPSP threshold.

In regard to sustained mediated inhibition of transient cells Singer and Bedworth concluded that "indirect evidence implies that the occurrence of X mediated inhibition of Y cells is quite likely." As support they offer the finding that the overall amplitude of the IPSP in transient cells was considerably increased when the electrical stimulation of the optic chiasm was increased from EPSP threshold of transient cells to the EPSP threshold of sustained cells. As further support they point to the findings of Singer, Pöppel, and Creutzfeldt (1972) who found prolonged inhibition of transient cells to a light stimulus of long duration. Since the quickly adapting responses of transient cells are not consistent with prolonged inhibition, this

finding may be taken as evidence for sustained mediated inhibition of transient cells.

Unfortunately psychophysical evidence does not appear to support interchannel inhibition in humans. Stromeyer and Julesz (1972) found that masking of a sine wave grating by a low-pass noise band of gratings decreased linearly as the upper cut-off of the noise band was decreased. This was found for target gratings of 2.5, 5.0, and 10.0 c/deg; the low-pass noise band never having a frequency component higher than the target. For the 5.0 and 10.0 c/deg target gratings masking was virtually nonexistent when the noise band contained no frequencies higher than 1.5 and 2.5 c/deg respectively. If sustained channels are inhibited by transient channels then masking by lower frequency noise should have been fairly strong.

Square-wave gratings were found to be best masked by adjacent square-wave gratings of approximately the same frequency in a study by White and Lorber (1976). A 6 c/deg grating appeared least "clear" when followed by a 4 c/deg grating, while a 12 c/deg grating was least clear when followed by a flanking 12 c/deg grating. In both cases masking decreased rather monotonically as lower masking frequencies were used. As the degree of masking was low at low spatial frequencies this study indicates that at best interchannel inhibition of sustained by transient channels is very weak. Legge, Cohen, and Stromeyer (in press), using a signal detection method, could find no evidence of low frequency masking of high frequency gratings in a backward masking task. Legge (1978) also found no evidence of high frequency masking

by low frequency gratings. Legge (1978) presented a high frequency grating of 100 msec. duration which was immediately preceded and followed by a 20 msec. exposure of a low frequency mask. A forced choice method showed no masking.

A final property of the sustained-transient dichotomy which we shall consider is orientation-specificity. It has been known for some time that many cells in the mammalian visual system increase their firing rates maximally when a line of a certain orientation appears in their RF (e.g., Hubel and Wiesel, 1962, 1968). This preferred orientation varies between cells and can be any one of the possible range of orientations in two dimensions (i.e., 0 to 360^{o}). As the orientation of a line is varied from a cell's preferred orientation the rate of neural firing decreases. For some cells the rate of the decrease as the orientation changes is more rapid than for other cells; that is, some cells are more orientation-specific than others.

Breitmeyer and Ganz (1976) contend that sustained cells are more orientation-specific than transient cells. They cite a study by Dow (1974) in support of this argument. Using single cell techniques Dow recorded the activity of 234 cells in the foveal projection area of striate cortex in fifty rhesus monkeys. Dow derived five classes of cells based on responses to moving stimuli of various speeds, response latency, orientation-specificity and other criteria. Dow's Class V cells seem to correspond to transient cells as they gave phasic responses to turning a light on or off, had short latencies (50 to 60 msec.), high spontaneous activity, and responded vigorously to fast movement

across the RF. Class V cells also responded to a wide range of orientations of stationary stimuli (lines of light).

Class II cells, on the other hand, respond only to stationary stimuli of a precise orientation. Breitmeyer and Ganz probably interpreted these as sustained cells for Class I lacks orientation-specificity and Class III and Class IV do not respond to stationary stimuli. If Class II cells do correspond to sustained cells and Class V to transient cells, then the contention of greater orientation-specificity for sustained cells is supported by Dow's study. Dow, however, did not specifically classify cells as sustained or transient and so provided no direct evidence as to the orientation-specificity of sustained and transient channels.

Dow stated that Class II cells " . . . probably correspond to the classic simple cells of Hubel and Wiesel." while Class V cells "constitute a third subset within the class of complex cells" and may " . . . conceivably belong to a transient (phasic) system." Such statements along with the studies of Stone and Hoffman (1971) and Hoffman and Stone (1971) could lead one to conclude that the sustained cells are more orientation-specific. Stone and Hoffman (1971) demonstrated that ganglion cells with fast conduction velocities (transient cells) innervate LGN cells which have fast conducting axons projecting to the visual cortex. Similar connections were found for cells with slow conduction velocities (sustained cells). No interconnections were found between fast and slow conducting axons. Hoffman and Stone (1971) presented evidence that fast afferents synapse onto complex cells

while slow afferent synapse onto simple and hypercomplex cells. No innervations of complex cells by slow afferents were found, nor innervations of simple or hypercomplex cells by fast afferents. These findings indicate that transient LGN cells project to complex cells while sustained LGN cells project to simple or hypercomplex cells. Studies by Maffei and Fiorentini (1973) and Movshon (1974) also support this view.

Pettigrew, Nikara, and Bishop (1968), Hubel and Wiesel (1962), and Rose and Blakemore (1974) have conducted single cell recordings which strongly indicate that complex cells are less orientation-specific than either simple or hypercomplex cells. If complex cells are exclusively innervated by transient cells while hypercomplex and simple cells are exclusively innervated by sustained cells then the sustained channel would be more orientation-specific. However, studies by Ikeda and Wright (1975a, 1975b) suggest that the innervations are not exclusive. Ikeda and Wright (1975a) recording from the cat's visual cortex, classified cortical cells as transient if their firing rate returned to a spontaneous level within 5 sec. of stimulation time, while those responding at 3-4 spikes/sec. above mean spontaneous level after 5 sec. were classified as sustained. Simple cells were defined as those which gave a modulated response to a drifting sinusoidal grating and had either an on-center, off-flank, or off-center, on-flank RF. Complex cells were defined as those having an unmodulated response to the drifting grating and an on-off receptive field (i.e., the cell responded to a light turned on or off over the entire RF). These

criteria were based on studies by Hubel and Wiesel (1962) and Maffei and Fiorentini (1973).[3] Based on this classification system 55% of sustained cells were also "simple" while 45% were "complex." Transient cells were classified as 60% "simple-transient" and 40% "complex-transient."

Ikeda and Wright (1975b) then investigated the orientation tuning of sustained, transient, simple, and complex cortical neurons. A comparison of the sustained vs. transient orientation tuning curves yielded no significant difference, but the same comparison was significantly different ($p<.05$) for complex vs. simple neurons. Ikeda and Wright (1975b) concluded that "There is thus a functional distinction based on orientation between simple and complex cells which is independent of the functional distinction based on spatial and temporal features between sustained and transient classes of cells." If Ikeda and Wright are correct then there is no difference between the orientation-specificity of sustained and transient channels. However, Ikeda and Wright (1975b) admit that in regard to sustained and transient cells "The criteria for classifying the cells are somewhat arbitrary. . . ." If these criteria led to incorrect classifications of sustained and transient cells then the evidence they present is inconclusive. Thus at this time it appears that more research is needed on the orientation-specificity of sustained and transient channels in order to clarify this issue.

Visual Masking--Metacontrast and Paracontrast

Visual masking occurs when the visibility of a stimulus (referred to as the target or T) is reduced by the presentation of another stimulus (the mask or M).[4] The procedure can consist of displaying T and M either: 1) concurrently in the same spatial position, 2) successively in the same spatial position, 3) concurrently in adjacent spatial positions, or 4) successively in adjacent spatial positions. When the mask follows the target in time backward masking is said to occur. Forward masking arises when the mask preceeds the target in time.

The temporal interval between onsets of the target and mask is known as the stimulus onset asynchrony (SOA). Negative SOA values indicate that the mask precedes the target, while positive SOAs indicate that the target precedes the mask in time.

The degree or amount of masking of the target stimulus is generally indicated by a reduction in the brightness, contrast, or identification of the target. The manner in which masking varies as a function of SOA depends on the mode of stimulus presentation, the subject's task (Kahneman, 1968), and the target to mask energy ratio where energy is related to the luminance, duration and size of the stimulus (Weisstein, 1972). Basically two types of masking are produced; Type A in which degree of masking decrease monotonically as absolute SOA increases and Type B in which degree of masking varies in a non-monotonic, U-shaped fashion. In Type B, masking reaches a maximum at an SOA greater than 0 msec. for backward masking and at an SOA less than 0 msec. for forward masking. In Type A, maximum masking occurs at an SOA of 0 msec. (see Figure 1).

Lefton (1973) defines metacontrast as "the phenomenal suppression of a visual stimulus by a second stimulus which falls in an adjacent retinal area within a critical time period. It is the case in which the two stimuli fall on nonoverlapping retinal areas. . . ." This definition of metacontrast should be clarified by stating that metacontrast occurs when the masking stimulus follows the target stimulus in time (a case of backward masking). Paracontrast occurs when the target is visually suppressed by a spatially adjacent stimulus which precedes the target in time (a case of forward masking). As the present investigation is concerned with Type B metacontrast and paracontrast we shall now restrict the discussion to these two topics.

Paracontrast. Kahneman (1968) states that, "In Type B . . . forward masking is weak or absent. . . ." Alpern (1953), using a brightness matching procedure, found relatively weak paracontrast in comparison to metacontrast. In Alpern's study a rectangular target of variable luminance was presented, preceded or followed by two flanking rectangles (the mask). The subject's task was to adjust the luminance of the target until it appeared to match the luminance of a comparison standard rectangle set at 10.6 foot-Lamberts (ft-L). The maximum value of adjusted target luminance was about 18 ft-L for negative SOAs (paracontrast), but was over 100 ft-L for positive SOAs (metacontrast).

Weisstein (1972) reported strong paracontrast for one subject when subjective estimations of the magnitude of masking where used as the indicator of masking. In this study Weisstein varied the ratio of target to mask luminance (T/M) from 1.0 to 0.0625. For subject TJ

at T/M ratios of 0.2 and 0.125 paracontrast was found to be as strong as metacontrast (see below for a discussion of the target to mask ratio). Masking was Type B for subject TJ under these conditions. However, for the other two subjects in this study paracontrast was monotonic (Type A) at these ratios, indicating little forward masking. These findings could lead one to speculate that paracontrast is very susceptible to individual differences in visual processing. Some subjects show no Type B paracontrast, others weak paracontrast, and still others strong paracontrast.

Research by Kolers and Rosner (1960) demonstrated that paracontrast could be obtained dichoptically, eliminating the possibility that paracontrast is a purely retinal effect. In Kolers and Rosner's paracontrast condition a disk was presented to one eye followed by the presentation of a ring (or no stimulus) to the corresponding surrounding area of the opposite eye. The subject's task was to report when the ring had appeared. Masking was indicated by the probability of detecting the ring. It was found that probability of detection was a U-shaped function, with maximum masking at -40 to -55 msec. SOA, when the diameter of the ring was much greater than the diameter of the disk. Masking was quite strong in such cases as the probability of detection reached a minimum of about .30.

Type B metacontrast. As has been discussed above metacontrast is usually found to be a much stronger effect than paracontrast (Alpern, 1953; Weisstein, 1972). Even when paracontrast is totally absent (as in two of Weisstein's subjects) metacontrast is strong (Weisstein, 1972).

Breitmeyer and Ganz (1976) point out that, "In metacontrast one can obtain suppression of brightness or spatial contrast (Alpern, 1953; Growney and Weisstein, 1972; Weisstein, 1972), of contour and contour detail (Breitmeyer, Love, and Wepman, 1974; Burchard and Lawson, 1973; Sukale-Wolf, 1971) and of form identity (Averbach and Coriell, 1961; Mayzner et al., 1965; Weisstein and Haber, 1965)."

The later case--suppression of form identity--is perhaps the greatest indicator of the strength of metacontrast. In studies performed by Mayzner and his colleagues (e.g., Andreassi, Mayzner, Beyda, and Waxman, 1970; Mayzner and Tresselt, 1970; Mayzner, Tresselt, and Helfer, 1967) letters which occur first in a display are so effectively masked by the occurrence of subsequent letters that the former are very rarely reported. Thus the display CHAIR, with H and I occurring first in time, is most often reported as C A R (Mayzner, Tresselt, and Helfer, 1967).

Metacontrast is affected by a variety of stimulus variables. Weisstein (1972) proposed that, all other variables being held constant, the target to mask energy ratio (T/M) determines the shape of the metacontrast function. The energy of a stimulus is a function of the duration, luminance, and size of the stimulus. Research has shown that changes in any of these three variables has noticeable effects on metacontrast. Alpern (1953) and Kolers and Rosner (1960) found decreases in metacontrast with increases in target duration and increases in metacontrast with increases in mask duration. Alpern (1953) reported no metacontrast when the luminance of his masking stimulus was equal to

the target luminance; but as the luminance of the mask was increased, metacontrast likewise increased. Weisstein (1972) also found that increasing the mask luminance increased metacontrast and furthermore resulted in a shift in the masking function from Type B to Type A. Battersby, Oesterreich, and Sturr (1964) and Matteson (1969) reported increases in the amount of masking with increasing mask size. Mayzner, Blatt, Buchsbaum, Friedel, Goodwin, Kanon, Keleman and Nilsson (1965), however, found that metacontrast decreased with increased surround width.

Since 1935 (Werner, 1935) intercontour distance (the visual angle subtended by the distance between the outside of the target and the inside of the mask) has been believed to influence metacontrast. It is generally agreed that the extent of masking is inversely related to increases in intercontour distance (Alpern, 1953; Toch, 1956; Kolers and Rosner, 1960; Cox, Dember, and Sherrick, 1969; Weisstein and Growney, 1969). However, while some argue that with increasing intercontour distance the SOA at which maximum masking occurs becomes less (Alpern, 1953; Streicher and Pollack, 1967), others feel that this SOA value increases (Weisstein and Growney, 1969; Kolers and Rosner, 1960).

The retinal location of target and mask also affects the magnitude of metacontrast. Alpern (1953) found no evidence of metacontrast when the target was presented foveally. Stewart and Purcell (1970) recorded similar results when subjects were asked to identify letters in a metacontrast paradigm. Kolers and Rosner (1960) found only a small

degree of foveal metacontrast when compared to metacontrast obtained with peripheral stimuli. Such studies have led some investigators to conclude that metacontrast is weak or absent foveally and the effect progressively increases as the stimuli are displayed at increasingly peripheral positions (e.g., Breitmeyer and Ganz, 1976).

Such a conclusion, however, ignores a large number of studies which find fairly strong foveal masking. In a number of studies of orientation-specific masking, Gilinsky (Gilinsky, 1967, 1968, 1971; Gilinsky and Doherty, 1969) obtained very strong foveal masking. Sekuler (Houlihan and Sekuler, 1968; Sekuler, 1969) also found strong foveal masking in studies similar to Gilinsky's. Schiller and Smith (1965) presented letters masked by a surrounding ring and found strong metacontrast when the letters were presented foveally. White and Lorber (1976) found spatial-frequency-specific metacontrast with square wave gratings presented foveally. Mayzner et al., (1965) reported U-shaped masking when target letters were only 0.5^{o} from a fixation point. Studies by Eriksen and Marshall (1969) and Lefton (1970) also support foveal metacontrast. Thus, while it is agreed that peripheral metacontrast is generally stronger than foveal metacontrast, the strength of foveal metacontrast does not appear to be as weak as some researchers conclude.

Internal contours of the target and mask affect the degree of metacontrast. Increasing the number or complexity of contours in the masking stimulus has been shown to increase metacontrast (Schiller and Smith, 1965; Johnson and McClelland, 1973). Concerning target

contours, it is generally found that the more complex a target (large number of line segments, many angles, etc.) the more difficult it is to mask. Dember and his colleagues have repeatedly reported such findings (Dember, 1971; Dember and Stefl, 1972; Ellis and Dember, 1971). Lefton (1975), using magnitude estimation, found that high frequency square wave gratings were difficult to mask. As such gratings contain many contours this supports the work of Dember. However, in a separate study (Lefton, 1974), using a forced choice procedure, Lefton found that masking increased as more contours were introduced in the target. In this later study metacontrast was monotonic (Type A), which may indicate that the effect of internal contours is confounded with the type of masking being studied. Perhaps complex targets are more difficult to mask under Type B masking and easier to mask under Type A.

Certain measures are not much affected under metacontrast. Fehrer and Raab (1962) demonstrated that simple reaction time to a target is not affected by the presentation of a mask. Schiller and Smith (1966) found that positional information was not lost. In this study subjects had to chose in which of two positions a target disk had been shown; both positions were followed by masking annuli. No change in the choice reaction time was found as a function of SOA. Pollock (1972) found that the accuracy of slant detection of lines was not affected under a metacontrast-like procedure known as sequential blanking (see Mayzner and Treseelt, 1970). Fotta (1976) found evidence that under sequential blanking certain features of target letters and sometimes the general shape (e.g., round vs. angular) of target letters could

be discriminated. Thus it appears that the occurrence of the target, its position, and certain target features are not suppressed under metacontrast.

Theories of Type B Masking

The theories of Type B masking can be classified under two general headings: interruption theories and integration theories. Both interruption and integration theories assume that the processing of a stimulus into a conscious visual representation takes a certain amount of time. Interruption theories assume that the masking stimulus stops the processing of the target stimulus during this critical time period. Integration theories assume that mask and target information become summed during this critical time period in such a way that the target information is degraded or totally lost. As the present study involves an investigation of an integration theory of masking we shall review these types of theories in more detail.

Interruption theories. There are two basic versions of interruption theories. In one version (Averbach and Coriell, 1961; Sperling, 1963) it is assumed that a visual image is formed at the level of some temporary visual storage. This information must be read into a more permanent store and it is assumed that this "read-in" is serial. The mask interrupts this "read-in" process replacing the target representation with its own representation in the temporary visual store.

In the second version of interruption theories the target information never reaches the level of a temporary visual storage (Lindsley,

1961; Lindsley and Emmons, 1968). According to this version the neural activity caused by the masking stimulus arrives at the cortex as the neural activity of the target is being consolidated into a visual representation. The masking activity interfers with this consolidation process so that the target image is never formed. Lindsley (1961) referred to the destruction of the target image as being " . . . like taking a photographic negative out of the developer too soon, which leaves the image unformed."

Various researchers have pointed out inconsistencies between interruption theories and masking data. First, interruption theories can not account for paracontrast (Kahneman, 1968). Second, these theories can not account for the shifts in the maximum masking SOA which occur as changes are made in various stimulus conditions (Weisstein, 1972). Third, theories assuming short-term visual storage can not account for masking of one item by another at SOAs as long as 100 msec. (Weisstein, 1972). Finally, the serial read-in process creates difficulties for interruption theories. Weisstein (1966) increased the number of stimuli in which a target was embedded and found that masking did not increase linearly as predicted by a serial read-in interruption theory. Furthermore, research by Sperling (1967) indicates that this read-in must be at least partially parallel. Interruption theories as they now stand can not explain masking of parallel processed data.

Integration theories. We will consider four models of integration which predict Type B masking. Three of these models are based on

inhibitory interactions, the fourth relates metacontrast to apparent motion. Apparent motion is the perception of motion between two spatially separated stimuli when the presentation of the second stimulus follows the offset of the first by a certain critical time (on the order of 100 msec.). Kahneman (1967, 1968) proposed that metacontrast was a case of perceived "impossible motion." According to Kahneman (1967), a target flanked by two objects provides cues for an impossible motion of the target in two directions at once. In the disk-annulus procedure the disk is made to grow and disappear at the same time. Kahneman believes that the perceptual system suppresses the input of the target since such target motion is clearly "impossible."

Kahneman (1968) supports his position with studies by himself (Kahneman, 1967) and by Mayzner and his colleagues (e.g., Mayzner, Tresselt, Adrignolo, and Cohen, 1967; Mayzner, Tresselt, and Cohen, 1966). Kahneman (1967) had subjects estimate metacontrast and apparent motion in different conditions. The functions obtained were very similar. Metacontrast and apparent motion seemed to both be functions of the SOA and not systematically related to exposure durations.

Kahneman (1968) interprets the sequential blanking of Mayzner's studies in the following manner:

> Even the two extreme letters of a word may be suppressed when they cannot be incorporated in a coherent percept of motion. The suppression is invariably a U-shaped function of presentation speed. On the other hand, all letters are seen in the many different sequences that permit the perception of a regular flow of motion. (Kahneman, 1968, p. 413)

Kahneman (1968) also reports another similarity between apparent motion and metacontrast. In both cases at SOAs too short for optimal motion or suppression the first object is seen as dimmer than the second. Kahneman reported that this dimming effect was first noted by Wertheimer (1912) in motion displays.

Although there are similarities between metacontrast and apparent motion there are some important differences. Weisstein and Growney (1969) found that the metacontrast function decreased in amplitude and changed shape with increases in the visual angle between stimuli, but the apparent motion function did not similarly change. Metacontrast was affected by energy manipulations while apparent motion was not. Eriksen and Colegate (1970) found that apparent movement did not reduce the discriminability of the first stimulus (i.e., no metacontrast occurred). Stoper and Banffy (1977) reported that introducing a second masking stimulus reduced metacontrast to a much greater extent than apparent motion (the latter was sometimes even enhanced). Furthermore, Stoper and Banffy found that peripheral presentations and close spacings of target and mask gave strong metacontrast while completely eliminating apparent motion. If metacontrast is due to "impossible" apparent motion then it is reasonable to assume that metacontrast and apparent motion should exist under the same conditions.

Thus although apparent motion and metacontrast are affected similarly by the manipulations of some variables, important differences between the two make an explanation of metacontrast in terms of apparent motion rather untenable. Many investigators, however, feel

that the two phenomena share some common mechanisms (Lefton, 1973; Weisstein and Growney, 1969; Breitmeyer and Ganz, 1976).

Bridgeman's theory. Bridgeman (1971) proposed a theory of metacontrast based on lateral recurrent inhibition using the equations for such inhibition developed by Hartline and Ratliff (see Ratliff, 1965). In this model the inhibition a neuron exerts on neighboring neurons is proportional to its firing rate and the proximity to its neighbors. Furthermore it is assumed that inhibition is subject to time delays. Assuming that neurons are separated from each other by some discrete units of distance and assuming a time delay of t msec., then directly adjacent neurons are inhibited with a time lag of t msec., those neurons two units away with a time delay of $2t$ msec., etc.

Bridgeman (1971) used a computer simulation of a network of such neurons to examine metacontrast and paracontrast. Disks and annuli were presented as stimuli to the simulated network and the frequency of firing of the simulated network was plotted as a function of the time since presentation of the first stimulus. Reduced frequency of firing to the disk plus annulus--as compared to the disk alone--indicated that both metacontrast and paracontrast were predicted by this system.

Lefton (1973), in a review of the metacontrast literature, wrote rather highly of this model stating that it, " . . . may prove to be the most quantitative and precise one that is available." Lefton pointed out that the model could account for: a) the effect of intercontour distance on the strength of metacontrast, b) the effects of changes in the energy of the target and mask on metacontrast and, c) the occurrence of metacontrast under dichoptic presentations.

The prediction of paracontrast under appropriate conditions is also an advantage of this model.

Weisstein, Ozog, and Szoc (1975) criticize Bridgeman's model on a number of grounds. First, Weisstein et al., argue that the similarity function which Bridgeman used to infer metacontrast is not a true metacontrast function. The similarity function was a cross correlation of the activity generated by the disk and the activity generated by the disk plus annulus simulation. This function was not a function of the SOA, but rather a function of the time interval since the first stimulation of the network. Such a function provided information about only one SOA, and hence is not a metacontrast function.

Secondly, when Weisstein et al., simulated Bridgeman's model and varied SOA the model predicted temporal oscillations, i.e., there were a number of SOAs maximizing metacontrast. Thirdly, the assumption of discrete inhibition yields incorrect predictions of the shape of the masking function as T/M is varied. Finally, Weisstein et al.'s simulation of the model exhibited spatial oscillations; for example, masking was greater at spatial separations of three neural units between target and mask than at two units. These criticisms diminish the plausability of Bridgeman's model to an extent that the model appears unacceptable in its present form.

Weisstein's theory. Weisstein's model of metacontrast is also based on inhibitory interactions (Weisstein, 1968, 1972; Weisstein, Ozog, and Szoc, 1975). Neurons in which excitation and inhibition develop at different rates and combine to yield the firing frequency

of each neuron form the basis of Weisstein's model (Weisstein, 1968). Such neurons are known as two-factor neurons (Rashevsky, 1948). Weisstein (1972) originally formulated her model with five two-factor neurons using assumptions and equations for the rates of excitation and inhibition developed by Rashevsky (1948) and Landhal (1962, 1967). However, as a recent revision of the model (Weisstein et al., 1975) incorporates a sixth neuron, we will discuss the six-neuron model here.

In the Weisstein et al., (1975) model the first two neurons represent transmission of information in the periphery of the visual system (retina, optic nerve). One conveys excitatory information about the mask, the other excitatory information about the target. Each of these neurons synapse onto two more central neurons (second-order neurons); one of these neurons responds faster than the other and is inhibitory, the other is slower responding and excitatory. Finally there are two "decision" neurons whose strength of response is translated into some psychophysical response measure. The target "decision" neuron receives input from the second order target excitatory neuron and from the second order mask inhibitory neuron. Similarly, the mask "decision" neuron receives input from the second-order mask excitatory neuron and from the second-order target inhibitory neuron. The inhibitory activity is algebraically added to the excitatory activity and this sum is responded to by the decision neuron. Thus when the mask is presented after the target the fast-responding inhibitory activity will interact with the slower-responding excitatory activity producing little or no response of the target decision neuron.

Weisstein et al., emphasized that this network successfully predicted; a) the shape of the metacontrast function and its change of shape as T/M changes, b) the reappearance of the target when the target and mask are repeatedly presented at appropriate SOAs (Schiller and Smith, 1965), and c) the SOA at which maximum masking occurred for U-shaped functions in 34 out of 35 such functions occurring in the literature reviewed by Weisstein (1972). Furthermore, the assumption of faster-responding inhibitory neurons has received a great deal of support from the sustained-transient literature (cf. Cleland, Levick, and Sanderson, 1973; Hoffman, Stone, and Sherman, 1972; Ikeda and Wright, 1975b; Singer and Bedworth, 1973; Stone and Hoffman, 1971).

Weisstein (1972) offers criticism of her own model. First, the network does not predict paracontrast. Any model of metacontrast should yield predictions of paracontrast as these two types of masking are, most likely, subsets of the same phenomenon. Secondly, the model does not take into consideration the spatial properties of metacontrast such as intercontour distance and the effects of internal contours.

Weisstein et al., (1975) proposed that paracontrast could occur if the slow-responding mask neuron inhibited the fast-responding neuron of the target. Even though such an hypothesis receives some support from physiological studies (Hoffman, Stone, and Sherman, 1972; Singer and Bedworth, 1973), Weisstein has not yet quantified this hypothesis in terms of her model. Until this is completed and the spatial properties of metacontrast can be accounted for, this model remains somewhat incomplete. It does, however, seem to this writer that

Weisstein's model is a much more promising formulation of metacontrast than Kahneman's (Kahneman, 1968), Bridgeman's (Bridgeman, 1971), or any interruption theory (Averbach and Coriell, 1961; Lindsley, 1961).

The Breitmeyer and Ganz theory. Breitmeyer and Ganz (1976) proposed that inhibitory interactions within and between sustained and transient neurons could account for masking phenomena. Although this model offers an explanation of Type A and Type B masking (as well as addressing attention and saccadic suppression) we will focus our discussion on the model's description of Type B masking.

Breitmeyer and Ganz (1976) proposed that Type B paracontrast is a result of the antagonistic center-surround organization of sustained visual receptive fields. Intrachannel inhibition occurs in this mechanism as the surround affects the center via lateral inhibition (see above; also, Cleland, Levick, and Sanderson, 1973; Cornsweet, 1970; and Hubel and Wiesel, 1962). Paracontrast is predicted from such inhibition since the inhibitory response of the surround lags behind the excitatory response of the center. If stimuli are shown concurrently to the center and surround of a sustained cell's RF the surround inhibition will reach a maximum after the excitation of the center has reached a maximum. In order to most effectively inhibit the center response the surround stimuli must be presented before the central stimuli (see above). When this is done paracontrast occurs, according to Breitmeyer and Ganz.

Most studies have found a center-surround latency difference between 10 and 30 msec. (Singer & Creutzfeldt, 1970; Maffei, Cervetto,

and Fiorentini, 1970; Poggio, et al., 1969) although one study extends this range to over 100 msec. (Fiorentini and Maffei, 1970) (for a more complete discussion of these studies see above). These results are compatible with the results of paracontrast studies which find that maximum masking occurs in the SOA range of 20 to 70 msec. (Weisstein, 1972; Alpern, 1953; Kolers and Rosner, 1960).

Although the antagonistic mechanism can account for monoptic paracontrast an additional assumption is needed to account for dichoptic paracontrast (Kolers and Rosner, 1960). Breitmeyer and Ganz (1976) state that: "If it is assumed that this asynchrony in response latencies to center and surround stimulation also characterizes the largely binocularly activated striate cortex cells (Hubel and Wiesel, 1962, 1968), dichoptic paracontrast effects are readily explainable." However, Breitmeyer and Ganz offer no supporting evidence for this assumption.

Breitmeyer and Ganz exclude transient neurons from involvement in paracontrast on the basis of a study by Fiorentini and Maffei (1970). As discussed above Fiorentini and Maffei found forward masking for a disk and annulus modulated at temporal frequencies up to 6 cps. However, for temporal frequencies of 8 cps and above backward masking effects were obtained. Breitmeyer and Ganz argued that at low temporal frequencies the target and mask activate predominately sustained channels while at intermediate to high temporal frequencies--approximately 7 cps and above--the target and mask activate both transient and sustained channels with transient channels probably predominating (Ikeda

and Wright, 1975b; Keesey, 1972; Kulikowski and Tolhurst, 1973). Since Fiorentini and Maffei (1970) found paracontrast only for low temporal frequencies, Breitmeyer and Ganz reasoned that paracontrast must involve only sustained neurons.

Breitmeyer and Ganz (1976) proposed that the mechanism of metacontrast is realized in the interchannel inhibition of sustained cells by transient cell inhibitory activity. The essential aspects of this mechanism involve the differences in response latency and persistence between transient and sustained channels as shown in Figure 2. In this figure the stimuli (both target and mask) are represented by the rectangular bars (target = T, and mask = M), the transient activity of each stimulus is represented by the spike which immediately follows the stimulus in time, and the sustained activity of each stimulus is represented by the inverted U-shaped curves following the transient activity. Breitmeyer and Ganz also assume that progressively higher spatial frequency channels have greater response latencies, lower response amplitudes, and a longer response persistence (Cornsweet, 1970; Davidson, 1968). This assumption is illustrated in Figure 2 by the three different sustained response curves. The solid line represents the activity of intermediate spatial-frequency channels; the dashed line, high spatial frequency channels; and the dotted line, very high spatial frequency channels.

Furthermore, Breitmeyer and Ganz assume (although it is not represented in this figure) that transient units are not as orientation-specific as sustained units, but this limited orientation-specificity is an important feature of metacontrast.

Breitmeyer and Ganz make two basic assumptions about the process of interchannel inhibition:

1) They assume that interchannel inhibition is strongest when the preferred orientations of transient and sustained channels activated by the target and mask are the same. This inhibition decreases with increasingly divergent orientations of target and mask. Such orientation-specific inhibition has been shown by Blakemore and Tobin (1972) to exist at the cat's visual cortex.

2) Interchannel inhibition is most pronounced when the inhibitory activity of mask transient channels is temporally superimposed upon the excitatory activity of target sustained channels (see Figure 2c and 2d). According to Breitmeyer and Ganz, cortical transient activity precedes sustained activity by 50 to 100 msec. (Dow, 1974). Thus optimal interchannel inhibition should occur when the mask onset is delayed by 50 to 100 msec. relative to the onset of the target.

Assumption 2 finds support in the sustained-transient literature (see discussion above). Breitmeyer and Ganz believe that assumption 1 finds support from findings that:

1) There is a columnar organization of striate-cortex cells that are functionally related in terms of orientation selectivity and the region of retinal space represented (Brooks and Jung, 1973; Hubel and Wiesel, 1962, 1968, 1974).

2) Transient and sustained neurons are found in the same cortical column (Dow, 1974).

3) Neural inhibition among different columns has been shown to exist (Benevento, Creutzfeldt, and Kuhnt, 1972; Hess, Negishi, and Creutzfeldt, 1975).

Based on these findings Breitmeyer and Ganz (1976, p. 16) state:

> Under such conditions, the inhibition of sustained cells by transient cells in the same column or in neighboring columns that show similar orientation specificity would result in the high degree of spatial and structural specificity found in Type B metacontrast effects. . . . (Uttal, 1970, 1971; Weisstein, 1972; Werner, 1935)

Perhaps the most crucial feature of the Breitmeyer and Ganz theory is the faster response latency for transient channels. When mask onset occurs before target onset (Figure 2a) the transient activity generated by the mask precedes the sustained activity generated by the target there is no interchannel inhibition and the target is perceived. Similarly for a simultaneous presentation of target and mask (Figure 2b). Only when the mask onset follows the target onset by a time interval approximately equal to the difference between the sustained and transient latencies of response will interchannel inhibition occur causing Type B masking (Figure 2c and 2d).

As can be seen from Figure 2 progressively longer SOAs will result in interchannel inhibition of progressively higher spatial frequency channels. Breitmeyer and Ganz argue that this implies the existence of a family of Type B masking curves. The specific curve obtained is a function of the spatial frequency composition of the target and the nature of the perceptual task which determines what spatial frequency information is necessary for the psychophysical response.

Finally, Breitmeyer and Ganz assume that transient neurons do not directly inhibit sustained neurons, but do so via an internuncial neuron--a neuron excited by a transient neuron and inhibiting a

sustained neurom. Transient channels alone generate brief activity so the inhibition of sustained channels would be correspondingly brief. Since sustained channels respond in a prolonged manner, a brief inhibition would not seem sufficient for the strong masking effects of metacontrast (Alperm, 1953; Mayzner and Tresselt, 1970; Weisstein, 1968). The internuncial neuron would generate the prolonged inhibition necessary to account for strong metacontrast effects.

The Breitmeyer and Ganz (1976) model appears to be able to account for many of the findings in the metacontrast literature. Some of these are:

1) The decrease in Type B effects as spatial separation between target and mask increase (Alpern, 1953; Weisstein and Growney, 1969).

2) Progressively greater metacontrast as the target is located at increasingly parafoveal eccentricities (Kolers and Rosner, 1960).

3) The finding of Type B contour masking effects during stroboscopic motion (Breitmeyer, Love, and Wepman, 1974).

4) The immunity of some target information to masking (Fehrer and Raab, 1962; Pollock, 1972).

5) The shift of peak metacontrast effects to lower SOAs as T/M decreases (Weisstein, 1972).

Disinhibition

Disinhibition has been defined as occurring under either of two conditions: 1) when the masking effect produced by two masks is less than the sum of their separate effects (Hartline and Ratliff, 1957;

Alpern and David, 1959; Dember and Purcell, 1968), or 2) when the masking effect of two masks is less than the masking effect of one of the masks--usually the one yielding the greatest masking of the two (Robinson, 1966, 1968; Long and Gribben, 1971; Barry and Dick, 1972).

Disinhibition was first "discovered" by Hartline and Ratliff during their work on the inhibiting influences of receptor cells in the eye of the horseshoe crab, Limulus (Hartline, Wagner, and Ratliff, 1956; Hartline and Ratliff, 1957, 1978; Ratliff and Hartline, 1959). It was found that the inhibition which a cell exerted on a neighboring cell (target) could be reduced if the inhibiting cell was itself inhibited by a third cell far enough removed from the target cell so as not to inhibit the target (Hartline and Ratliff, 1957). Hartline and Ratliff (1958) also found that when the two inhibiting cells were near the target so that both had inhibitory effects on the target, then the combined inhibitory effect of the two was less than the sum of their separate effects--indicating mutual inhibition. Hartline, Wagner, and Ratliff (1956) reported that as the area of an inhibiting stimulus increased the increase in inhibition of the target cell first increased markedly, but then subsequent area increases led to smaller inhibitory increases. This may have indicated that the cells responding to the outer portion of the stimulus while inhibiting the target cell were also inhibiting cells responding to the inner portion of the stimulus.

A study in brightness contrast effects by Alpern and David (1959) gave the first indication that disinhibition was present in the human visual system. Alpern and David presented subjects with a target rectangle whose brightness was to be matched to a comparison standard.

Two flanking rectangles were shown either very close to the target (15') or separated by a greater distance (45'). The inhibitory effect (defined in terms of the brightness at which the subject set the target) of the flanking rectangles was noted at each position and compared to the effect when all four flanking rectangles were presented. This later effect was less than the sum of the individual components, most notably at lower intensities of the flanking rectangles. This is consistent with Hartline and Ratliff (1958). Alpern and David also found that increasing the size of two contiguous flanking rectangles did not systematically increase the inhibiting effect; the increase in inhibition became more gradual with increasing flank size similar to the finding of Hartline, Wagner, and Ratliff (1956). Finally, Alpern and David noted that with one subject there was no combined inhibitory effect when two flanking rectangles which inhibited the target were presented with two flanking rectangles too far removed to inhibit the target. This later result is predicted from the findings of Hartline and Ratliff (1957).

A similar study by MacKavey, Bartley, and Casella (1962) extended the effects of disinhibition to much greater spatial separations in the human eye. Once again a target patch had to be matched to a comparison stimulus. Inducing patches of light were presented either alone at target-center to inducing-center distances of 1, 2, 3, or 4 degrees or in pairs at various different distances (e.g., 1 degree and 2 degrees, 1 degree and 3 degrees, but never 1 degree and 1 degree). The inhibitory effect was measured similarly to Alpern and David (1959).

The inhibitory effect of the single inducing patch was found to decrease rather monotonically with increasing distance from the target, asymptoting at about 4 degrees. But more significantly it was found that the inhibitory effect of two inducing patches was generally less than that of the single inducing patch. This reduction in inhibition increased as the distance of the further patch was increased. For example, the inhibitory effect of patches at 1 and 5 degrees was much less than the effect of patches at 1 and 3 degrees. Thus MacKavey et al., (1962) presented evidence that disinhibition could be obtained when the inhibiting stimuli were separated by as much as 3.25 degrees.

During the late 1960's and early 70's a number of researchers investigated disinhibition in humans via masking studies. These studies differed from the two just reviewed in that: 1) target and mask stimuli were presented for a very short time (i.e., from 1 to 50 msec.), 2) the onset of the target and mask were usually not simultaneous, and 3) the inhibitory or masking effect on the target are usually measured via a detection and not a brightness criteria.

In the first of these studies Robinson (1966) presented subjects with three concentric overlapping discs (23', 46', and 92') at SOA1s[5] of 45 to 120 msec. and an SOA2 of 40 msec. Correct detections of the target disc increased monotonically with increasing SOA1 under both 2 disc and 3 disc conditions (the latter having two masks). However, the target detection rate was always greater under the 3 disc condition.

Dember and Purcell (1967) investigated disinhibition effects on target letters (D and O) with a first mask (hereafter M1) a black disc

and the second mask (hereafter M2), a black annulus. They added the condition of T+M2 (i.e., target shown with only M2) which Robinson (1966) did not include. Only one SOA between T and M1 of 31 msec. and one SOA between M1 and M2 of 55 msec. were used. Dember and Purcell found that the mean recognition of T (target) under the T+M1+M2 condition (i.e., T shown with both M1 and M2) was significantly lower than the mean percent recognition score predicted from the scores under T+M1 and T+M2 conditions (assuming masking by M1 and masking by M2 was independent). These researchers concluded that M1 and M2 could not be independently masking T but that M2 must be inhibiting M1, releasing T from masking on some trials.

Robinson (1968) extended disinhibition to interocular presentations. Using the same stimuli as his 1966 study, Robinson presented the stimuli either binocularly (all stimuli to both eyes) or interocularly (T and M1 to the left eye, M2 to the right eye). The SOA1s and SOA2s used were from 15 to 200 msec. Recognition of T was again a monotonic function of SOA1. Disinhibition (increased recognition of T under T+M1+M2 vs. T+M1) was greatest under binocular presentation at short SOA2s and weakened with increases in SOA2. Interocular disinhibition, though somewhat weaker than binocular, was strongest at long SOA2s (75 to 200 msec.) and weakened with decreases in SOA2. These findings may indicate that it takes much longer for M2 inhibitory effects to inhibit M1 interocularly.

Schurman and Eriksen (1969), in a study which attempted to replicate Robinson (1966), did not find disinhibition and brought up some methodological problems with disinhibition studies. As with Robinson's

studies (1966, 1968) three concentric discs were used, but a forced-choice procedure was used with no target disc presented on 50% of the trials. Masking conditions used were T+M1, T+M2, and T+M1+M2. Inter-stimulus intervals (ISI) used were; ISI1 (T offset to M1 onset) of 0, 25, and 50 msec., ISI2s (M1 offset to M2 onset) of 0 and 20 msec., and T offset to M2 onset of 20, 65 and 90 msec. Masking of T was monotonic under T+M1 and T+M1+M2, while no masking of T was found under T+M2. No significant difference was found between masking under T+M1 and masking under T+M1+M2. Schurman and Eriksen concluded that disinhibition had not occurred for these stimuli.

Schurman and Eriksen felt that the design of this study (and also Robinson's) provided cues to the occurrence of T. The first cue was a strong apparent-motion effect that was obtained under presentations of the three stimuli, an effect that was reportedly diminished when only two stimuli (T+M1) were presented. The second cue was the occurrence of simultaneous contrast leading to Mach bands under the condition T+M2. Schurman and Eriksen reported that if the target occurred prior to the presentation of M2 then the boundary of the target was enhanced while dark bands appeared outside the boundary. Such cues may exist in disinhibition studies and confound the already complicated inhibitory effects. In order to control for these effects Schurman and Eriksen designed and ran a second experiment using letters (A, T, and U) as the target, with M1 and M2 the same size as the first study. Again no disinhibition was found.

Uttal (1970) also reported no disinhibition effect in experiment two of his study. The target stimuli were alphabetic characters

composed of dots of light displayed on a cathode ray tube. Mask one and mask two were overlapping noise fields of random dots centered on the target letters. In one condition, T+M1, the ISI1 varied from 0 to 100 msec. In the other condition, T+M1+M2, ISI1 was held constant at 20 msec. while ISI2 varied from 20 to 100 msec. T+M1 produced a monotonic Type A curve with percent correct target detection increasing with increasing SOA. Uttal found that under the T+M1+M2 condition percent correct target detection was no different than detection under T+M1 at the 20 msec. ISI1. From this Uttal concluded that no disinhibition existed in this experiment. However, Uttal did not consider the effect of M2 separately on T. It is very possible that M2 alone had a significant masking effect on target letters. If this was so then the finding of no increase under T+M1+M2 could indicate the presence, not absence, of disinhibition. As Uttal did not take into consideration the effect of M2 alone this study can neither support nor contradict the existence of disinhibition.

Long and Gribben (1971) investigated disinhibition, varying not only the interstimulus interval but also the duration of M1 and M2. The targets were various pairs of letters, and M1 and M2 were white fields of equal size. As usual the targets appeared either alone with M1 or with M1+M2. The ISI1s between T and M1 were from 1 to 50 msec. as were the ISI2s between M1 and M2. M1 and M2 durations varied from 1 to 50 msec. Long and Gribben found an overall main effect of masking condition, i.e., T recognition was better under T+M1+M2. Masking was again a Type A function of ISI1. When M1 duration was 1 msec. M2 always summated with M1 producing more masking than M1 alone.

This effect increased with increasing durations of M2. As M1 duration was increased the effect of M2 reversed; a 1 msec. M2 lead to more masking than longer M2s. However, with M1 greater than 1 msec. all M2 durations disinhibited M1 effects. This finding may indicate that the duration of masks must be considered in dealing with disinhibition effects. However, the duration of M1 is confounded with the SOA between M1 and M2 and it is generally thought that masking is a function of SOA not ISI. The duration interaction here may only indicate the effect of SOA2. This is supported by the finding in this study that increasing ISI2 tended to increase the disinhibition effect of M2 on M1, i.e., increasing correct target detections resulted with increases in ISI2.

Barry and Dick (1972) in a set of two experiments first replicated Robinson (1966) and then varied the fixation point and report criteria. The stimuli were three concentric disks with SOAls of 45, 70, and 95 msec. and one SOA2--45 msec. Masking conditions used were T+M1, T+M2, M1+M2, and T+M1+M2. Barry and Dick found that percent correct target detection under T+M1+M2 was greater than target detection under T+M1 only at an SOA1 of 70 msec. However, these researchers did not take into consideration the effect of M2 alone, even though they had included T+M2 trials and reported percent correct T detection rates under this condition. A cursory examination of their Table 1 shows that this M2 effect was so substantial that had it been considered disinhibition would have most likely been found at the other SOAls. Of even greater interest is the reported percent correct T detections when M1 was not

reported (i.e., M1 was masked) under T+M1+M2. Over the three SOAls this measure increased monotonically from 88 to 100%. This indicates that when M1 was masked, T was seen nearly every time--a very strong indication of disinhibition. Barry and Dick, however, believed that this finding was only a weak indication of disinhibition since masking of M1 occurred only 30% of the time.

In their second experiment Barry and Dick (1972) used the same stimuli at the same SOAs, but the stimuli appeared either concentrically about a fixation point or 5 degrees to the left of a fixation point (experiment 1 had used no fixation point). Furthermore, subjects, beside reporting the occurrence of the target, were asked to give some indication of the brightness of the stimuli--light, dark, or normal. With foveal presentations, and allowing only "light" and "normal" responses, little masking occurred under T+M1 except at an SOA1 of 70 msec. (correct T detection rate 69%). At this SOA1, under T+M1+M2, T was reported with 100% accuracy whenever M1 was not reported--indicating disinhibition. Inclusion of "dark" responses for targets increased percent correct detection at the 70 msec. SOA1 to 96%; no comparison for disinhibition could then be made.

With peripheral presentations, including only light or normal responses, correct detection of the target was much greater at all SOAls under T+M1+M2 as compared to T+M1. However, inclusion of dark responses abolished this advantage, i.e., raised the percent correct T detection under T+M1 to the same level as T+M1+M2.

As a consequence of their study Barry and Dick concluded that the "recovery" (increased correct T reports under T+M1+M2) does not

occur as a disinhibition phenomenon, but rather is due to brightness reversals of stimuli which interact with subject criteria. Contrary to their conclusion this writer feels this study yields evidence for the occurrence of disinhibition for the following reasons: 1) percent correct reports of T when M1 is masked under T+M1+M2 was nearly 100%, 2) consideration of the effect of M2 alone on T would have led to predictions of much greater masking of T under T+M1+M2 than was found (see Dember and Purcell, 1967), and 3) "dark" responses are an indication of masking whether one choses to call these "brightness reversals," inhibition, criterion shifts, etc. and as such they should not be included in a measure of masking (unless one is doing estimations of the degree of masking--see Weisstein, 1968, 1972).

Lovegrove (1976) reported an extension of disinhibition to a forward masking condition (Experiment 3). The target was a vertical line with the mask being either a single vertical line or two such lines, one vertical the other intersecting at its midpoint and rotated away from the vertical from 15 to 90 degrees. The ISI used was -20 msec. (mask before target). Masking was found to be greatest when only the single vertical line appeared as a mask. Masking was significantly less when 2 line masks appeared at all rotations except 45 degrees and was least at a rotation of 15 degrees, which Lovegrove reported as maximum disinhibition. Given the design of the study it appears difficult to clearly establish whether disinhibition actually occurred (i.e., an inhibition of the vertical mask line by the rotated mask line which led to decreased target masking) or whether the results reflect the effect of differences in the construction of the mask stimuli.

An Investigation of Orientation-Specificity and Disinhibition in the Breitmeyer and Ganz Theory of Type B Masking

The purpose of this study was to investigate two aspects of the Breitmeyer and Ganz theory of Type B masking; 1) orientation-specificity in paracontrast and metacontrast, and 2) disinhibition effects in paracontrast and metacontrast. Orientation-specificity was investigated in Experiment 1, while disinhibition was studied in Experiment 4. Experiments 2, 3, and 5 were necessary to establish parameters and comparisons for Experiment 4.

Experiment one. Breitmeyer and Ganz (1976) have proposed that paracontrast is due to intrachannel inhibition of sustained channels, while metacontrast is due to interchannel inhibition of sustained channels by transient channels. Breitmeyer and Ganz (1976) also contend that transient channels are not as orientation-specific as sustained channels. If these assumptions are true then a transient channel should respond (i.e., neural excitation increases) over a wider range of orientations than a sustained channel. If the inhibition caused by a channel is proportional to the excitation of that channel (Cornsweet, 1970) then the transient channel should inhibit a sustained channel over a wider range of orientations than a sustained channel inhibiting a sustained channel. From this argument it follows that paracontrast should be more orientation-specific than metacontrast. In other words, as target and mask orientations increasingly diverge, the Breitmeyer and Ganz theory predicts that paracontrast will decrease more rapidly than metacontrast.

This prediction was tested in the first experiment by presenting to subjects a rectangular-shaped square-wave grating (target) and two flanking rectangular-shaped square-wave gratings (mask). The orientation of the target grating was held constant while the orientation of the mask was varied. The time between onset of target and mask (the stimulus onset asynchrony--SOA) was varied from -90 to +90 msec. in 30 msec. increments. The targets were either square-wave gratings of the same periodicity as the mask or blank fields whose luminance equaled the mean luminance of the mask and target gratings. Fifty percent of the targets were square-wave gratings and fifty percent were mean luminance targets.

The square-wave target gratings were always at an orientation of 45 degrees relative to a subject's horizontal and vertical visual meridians. This orientation has proven to yield strong masking effects in previous studies (Gilinsky and Doherty, 1969; Gilinsky and Mayo, 1971). Masking square-wave gratings were at orientations of 45, 55, 65, and 75 degrees. It was decided to investigate masking in only a 30 degree range for two reasons. First, previous studies indicate that differences in target-mask orientations beyond 30 degrees produce little masking (Gilinsky and Doherty, 1969; Sekuler, 1965). Secondly, a larger number of orientations would add a large number of observations to each subject's task--a task which involves a great many observations (1120) when only four masking orientations are used.

The duration, luminance, and contrast of target and mask gratings were equivalent in order to maximize Type B effects (Weisstein, 1972).

The contrast of the gratings was low. At low contrast the visual system should be responding linearly to the Fourier components of the grating (Cornsweet, 1970). The duration and luminance were also low (but above threshold) in order to insure a linear response to the Fourier components (Cornsweet, 1970). However, the duration must be long enough and the luminance high enough to insure that masking effects will occur. If we had used a threshold measure (50% detection) in order to determine duration and luminance, then we would have produced little masking as a subject could guess with a 50% detection rate in our study, even though precautions were taken to minimize guessing. Pilot studies indicated that an 80% to 90% detection rate (for target grating presented alone) would yield a duration that produces effective masking. Consequently an 85% detection rate was chosen in order to establish the target and mask duration.

The subject's task on each trial was to say whether or not a target grating had appeared. Furthermore, in order to respond "yes" (i.e., that a target grating had appeared) a subject had to feel that he was at least somewhat certain that a target grating had appeared. If a subject felt that a response would be a guess, he was to respond "no." The reason for using this modified forced-choice procedure was to stabilize the false alarm rate (i.e., the rate of "yes" response when a mean luminance target was shown), especially in the paracontrast condition.

Pilot research has shown that the false-alarm rate fluctuates greatly as SOA varies when guessing is permitted in the paracontrast

condition. Changes in the detection rate as a function of SOA will then most likely reflect criterion shifts (see Coombs, Dawes, Tversky, 1970, pp. 165-201) and not masking effects. But when a subject could respond "yes" only when he was at least "somewhat certain" that a grating was shown, then the false-alarm rate was both lowered and stabilized in pilot research. Changes in the detection rate as a function of SOA where then U-shaped and appeared to reflect masking effects. Kolers and Rosner (1960), in one of the few studies to find Type B paracontrast, had subjects respond "yes" only if they were certain that a target had appeared. Strong Type B paracontrast was obtained.

Subjects were asked to rate their "yes" responses from one to three depending on the clarity of the target. If the target appeared dim or very unclear they were to respond "yes-one," if fairly clear "yes-two," and if very clear "yes-three." This was thought to provide a finer measure of masking than a simple yes-no design. Responses of yes-one and yes-two were thought to represent some degree of masking but not as great as a no response, while a "yes-three" was thought to represent no masking. This method is somewhat similar to magnitude estimations of masking which have yielded U-shaped functions (Weisstein, 1972).

The target was displayed foveally. Our major concern here was that we could not control for eye movements. We have found in pilot research that with a parafoveal presentation, the subject attempted an eye-movement away from the fixation point and toward the target. Such eye-movements appear to yield a great deal of noise. If accomplished before target presentation, eye-movements may decrease masking;

but if an eye-movement took place during or immediately after target presentation, masking would be enhanced (Breitmeyer and Ganz, 1976). While foveal target presentations may yield less masking than parafoveal presentations (see discussion above), foveal presentations may eliminate eye-movement noise while still yielding considerable masking effects (Gilinsky, 1971; Schiller and Smith, 1965; Lefton, 1970).

In order to insure that there was no bias toward activating either sustained or transient channels to a greater degree than the other the target and mask gratings were of an intermediate frequency (i.e., 4 cycles per visual degree). At this spatial frequency both transient and sustained channels are activated to approximately the same degree (Kulikowski and Tolhurst, 1973). Choosing a high spatial frequency would bias the visual system toward the use of sustained channels while using a low spatial frequency would create a bias toward transient channels (Kulikowski and Tolhurst, 1973; Keesey, 1972; Pantle, 1970). Furthermore, there are indications from the Visual Science Laboratory at the University of Manchester, England that target and mask gratings of intermediate spatial frequency produce Type B masking (Kranda, 1977; Breitmeyer, 1977--personal communications).

Subjects performed all experiments under conditions of low-level light adaptation. This was done in order to enable us to generalize from our findings to the use of masking in natural conditions. Also, dark-adaptation was found in pilot research to yield after-images and the level of dark-adaptation may have varied as the subject was repeatedly exposed to bursts of light--the target and mask. Finally,

we wanted to be consistent with previous masking studies and most have used light-adaptation (Weisstein, 1968; Greenspon and Eriksen, 1968; Gilinsky and Doherty, 1969; Kolers and Rosner, 1960; etc.).

Experiments two through five. The Breitmeyer and Ganz model also leads to differential predictions of paracontrast and metacontrast in a disinhibition paradigm. If, as Breitmeyer and Ganz (1976) propose, metacontrast is due to the inhibition of sustained channels by transient channels then there should be no disinhibition in metacontrast. This is because only the transient activity of the second mask (M2) should inhibit only the sustained activity of the first mask (M1) having little or no effect on the transient activity of M1. Accordingly, the M1 transient activity will still cause inhibition of the sustained channels activated by the target (T) causing little change in the masking of T. However, according to the Breitmeyer and Ganz model disinhibition is predicted for paracontrast. This follows since the sustained activity of M2 will inhibit the sustained activity of M1, thus masking M1. If paracontrast is due to sustained intrachannel inhibition and the sustained activity of the M1 channels is now greatly reduced there should be little inhibition of the target sustained channels. Hence, the target should now not be effectively masked.

In metacontrast there may even be an increase in masking due to transient activity of M2 affecting the sustained activity of T. Also, there may be some masking in paracontrast even if disinhibition occurs, due to the effect of M2 sustained channels on target sustained channels.

Such effects of M2 directly on the target were considered and dealt with (see below).

In order to test these predictions (no disinhibition for metacontrast, disinhibition for paracontrast) a second square-wave grating was introduced (Experiment 4). However, before introducing this second mask it was necessary to determine: 1) the proper M1 to M2 SOAs to use in order to insure that M2 masks M1, and 2) the masking effect which M2 alone had on the target grating (T). Determining the former was accomplished in Experiment 2, while the latter was determined in Experiment 3.

As masking is rather variable between subjects we thought it best to establish for each subject the SOAs which yielded maximum masking of M1 by M2. These maximizing SOA2s would then be the only SOA2s used in Experiment 4. In order to establish these SOA2s only M1 and M2 were presented in Experiment 2. The contrast, and luminance of M2 and M1 was the same as for T and M1 in Experiment 1 (in fact these parameters were constant throughout all experiments). The durations of M1 and M2 were the same as the last duration used for a subject in Experiment 1 (this duration was then kept constant throughout Experiments 2 through 5). Only one orientation of M1 and one orientation M2 were used. The choice of these orientations is discussed below under Experiment 4. The SOA2s used in Experiment 2 were the same as the SOA1s used in Experiment 1. M2 was, like M1, composed of two square-wave gratings; one of these gratings flanked the left side of M1 and the other flanked the right side of M1. Either M1 or a blank field of mean luminance

and size equal to M1 appeared. The subject's task was to give one of the four responses (discussed above) on each trial for M1.

The maximum probability of error in detecting M1 for negative SOAs determined the SOA2 to be used for the paracontrast trials of Experiment 4, while this same probability for positive SOAs here was used to determine the SOA2 used for metacontrast trials in Experiment 4.

The masking effect of M2 alone on T was assessed in Experiment 3. Only one M2 orientation was used. The SOAs used in this experiment were those that would occur between T and M2 in Experiment 4 for each subject. The subject's task was the same as Experiment 1--a forced-choice response to the target. Again 50% of the trials had T presented and 50% had the blank field presented. The probability of error in detecting T at each SOA here was used to establish a comparison for Experiment 4.

In Experiment 4 three stimuli were presented on each trial: the target (either T or blank field), M1, and M2. The subject's task was again to give one of the four responses to the target stimulus. We had hoped to minimize the effect of M2 on T and yet yield masking of M1 by M2 by chosing the orientations of M1 and M2 such that: 1) M1 would mask T to a significant extent, 2) M2 would mask M1 to a significant extent, and 3) M2 would have little direct masking effect on T.

Such a method of chosing orientations was to be based on the orientation-specific curves found for each subject in Experiment 1. For example, if it was found for a subject that a mask which differed from

T by 10^{o} significantly masked T while a mask which differed by 20^{o} did not, then M1 for that subject would be 55^{o} and M2 would be 65^{o} for Experiments 2 through 5. However, Experiment 1 did not yield orientation-specific masking functions so another method of chosing M1 and M2 orientations was used. M1 and M2 were chosen to be the same orientation as the M1 orientation yielding the greatest masking for a subject in Experiment 1.

In Experiment 5, the target (T or blank field) was presented with M1. The M1 orientation was the same as that which maximized T masking in Experiment 1. This study was done in order to attempt to account for any practice effects which may have occurred between Experiments 1 and 4. The results of this study when averaged with the masking by the same mask orientation in Experiment 1 would seem to provide a better estimate of masking of T by M1 with which to compare the results of Experiment 4.

METHOD

Subjects

Two of the subjects were the author (MF) and his research assistant (JD). Both were males in their twenties with corrected-to-normal vision. Two naive subjects (males 18 and 21 years old) with normal vision were also used and paid for their participation. All subjects participated in all experiments.

Apparatus

The stimuli were shown in a four-channel tachistoscope. Three channels of this tachistoscope were provided by a Scientific Prototype Model Gb 3-channel tachistoscope slaved to a unit which controls the luminance and duration of each channel and the time interval (here SOAs) between onset of each channel. The fourth channel consisted of a half-silvered mirror, mounted between the aperture of the 3-channel tachistoscope and the headrest, a Kodak Ektagraphic slide projector with Kodak Zoom Ektaner lens, a circular piece of white plastic that served as a diffuser, and some black cardboard tubing.

The purpose of this fourth channel was to provide light adaptation. Light was projected from the slide projector to the diffuser. The space between the projector lens and the diffuser was enclosed by a black cardboard tube. The circular image of the diffuser was then positioned

by moving the half-silvered mirror and the diffuser so that this image completely surrounded the display from the three-channel tachistoscope (i.e., the target, mask one and mask two--see below). In the center of the diffuser was a black spot. This served as a fixation point in the image which the subject saw. The size of the spot was 0.6 mm which subtended 7 minutes of arc at the subjects eye. This spot appeared in the center of the target image.

The 3-channel tachistoscope was positioned so that a subject could sit upright and look straight ahead through the half-silvered mirror (of channel four) and into the aperture of the 3-channel tachistoscope. Slide trays mated to channels one and two provided automatic advancement of slides for these two channels.

A headrest whose height and lateral position could be adjusted was used by each subject. A black hood with holes for the left eye, the nose, and mouth, was sewn to the headrest. Clamps were used to hold the frames of the half-silvered mirror and the diffuser in place once the diffuser image was adjusted for each subject.

Stimuli

A black and white square-wave grating was produced by placing 1/4 inch Chartpak black matte tape in strips 1/4 inch apart on a white mattboard. This pattern was then photographed with Kodak High Contrast Copy Film (ASA 64) using a Nikon Ftn camera with a Nikon 50 mm Macro-Lens. The pattern was rotated to various angles so that the resulting negatives were square-wave gratings of the following orientations:

45, 55, 65, and 75 degrees. The negatives were mounted as 35 mm transparencies for viewing in the 3-channel GB tachistoscope. All negatives resulted in a 4 cycle/degree grating when viewed through the 4-channel apparatus.

To produce the appropriate target and masking stimuli, cardboard masks were placed in each channel of the 3-channel tachistoscope. A rectangular mask with a 5 mm wide central gap was placed in channel one. This resulted in a visible portion 5 mm wide in the center of any slide shown in this channel. This yielded a stimuli 1 degree of visual arc at a subject's eye. As the height of any channel in the 3-channel tachistoscope was 3 degrees at the eye, this mask yielded a 1 degree wide by 3 degree high central target stimuli.

In channel two a rectangular cardboard mask was placed which had two 5 mm wide gaps separated by a 5 mm wide piece of cardboard. This resulted in Mask one (M1), two 1 degree wide by 3 degrees high rectangles which flank the target rectangle (one masking rectangle on each side of the target). Only the cardboard masks for channels one and two were used in Experiments 1 and 5.

In Experiments 2, 3, and 4 a third cardboard mask was used, this one in channel three of the 3-channel tachistoscope. This mask had two 5 mm wide gaps separated by a 15 mm piece of cardboard. This resulted in Mask two (M2), two 1 degree by 3 degree rectangles flanking Mask one (see Figure 3). In Experiment 2 only the second and third cardboard masks were used. In Experiment 3 only the first and third cardboard masks were used. In Experiment 4 all three cardboard masks were used.

All square-wave grating slides were produced with a contrast of 0.16 where contrast equals the difference of maximum and minimum luminances of a grating divided by the sum of these two luminances (i.e., contrast = (Lmax-Lmin/Lmax+Lmin)). The maximum and minimum luminances as measured by a Tektronix J16 Digital Photometer were 11.5 nits (cd/m^2) and 9.5 nits respectively. Thus mean luminance was 10 nits which was the luminance of all "blank field" slides.

The light adaptation field had a diameter of 55 mm or 6 degrees of visual arc at the eye. Its luminance was 3 nits.

Procedure

On each day of participation the subject first adjusted the headrest so that he could comfortably view the display field. All channels of the tachistoscope used in the experiment the subject was currently participating in were turned on, with all cardboard masks in place. The subject positioned the headrest so that the fixation point appeared in the center of the target rectangle, all rectangular target and masking fields flanked without gaps, and the total display field was centered in the light adaptation field.

Experiment 1. On the first day of participation a target grating duration which yielded a detection rate of 85% was found. This was done using a modified method of limits. The subject was first told to gaze at the fixation point for three minutes, providing light adaptation. In order to make a preliminary determination of the range of durations to be investigated, the subject was first shown a 45^{o} target grating

at durations increasing from 2 msec. in 1 msec. increments. The midrange of the durations to be used was determined when the subject responded that he had seen the target grating. The range to be investigated was then from 2 msec. below this midrange duration to 2 msec. above the midrange. Five durations were investigated as 1 msec. increments were used.

The subject received 40 trials at each duration using ascending and descending series. For example, if a subject's range was determined to be from 4 to 8 msec. he received trials in the order 4, 5, 6, 7, 8 msec. then 8, 7, 6, 5, 4 msec. then 4, 5, 6, 7, 8 msec., etc. Of the 40 trials at each duration, 20 were presentations of the target grating (the 45^{o} square-wave grating) and 20 were presentations of the slides of mean luminance (target blanks). The order of presentations was randomized. Each subject received the instructions presented in Appendix I.

Percent correct was based on the performance for target gratings only, with performance on target blanks used to assess the false alarm rate. Percent correct detection (i.e., any yes response when the grating appeared, corrected for the false alarm rate) was plotted against duration and the 85% detection rate determined from this graph (rounded to the nearest msec.) was used for mask and target duration. After completing the duration procedure the subject then was ready to proceed with the actual running of Experiment 1. In Experiment 1 there were a total of 56 conditions: 7 SOAs ($\pm$90, $\pm$60, $\pm$30, and 0 msec.), 4 Mask 1 orientations (45, 55, 65, and 75 degrees) and 2 types of target (a

45^{o} 4c/deg square-wave grating and a mean luminance blank field). We presented 20 of each target type at each SOA at each mask orientation, yielding a total of 1120 observations per subject for Experiment 1.

Trials were grouped into blocks of 56 trials. Eight trials were run at each SOA within a block, with two trials at each Mask 1 orientation. Within each block the eight trials at each SOA were also blocked. For example, the eight trials at an SOA of 30 msec. appeared successively; similarly for all other SOAs used. The order of presentation of these SOA blocks was randomized with the stipulation that each SOA could occur no more than three times at any temporal position within the 20 experimental blocks, i.e., each SOA could occur first no more than three times, second no more than three times, etc. This was in order to control for possible practice effects within each experimental block. Presentations of Mask 1 orientations and target type were randomized within each SOA block with each Mask 1 orientation appearing with each target type. Each subject received the instructions presented in Appendix II.

As the duration procedure took about one hour for each subject not many masking observations could take place in the first session. However, it was possible to run one practice block during each subject's first session.

On following days the experimenter again reminded the subject of the response choices. Each subject then received a block of 50 trials with only the central rectangle (half the trials were gratings and half blanks) in order to assess detection rate and provide practice. Four

experimental blocks were run on each of the following five days. As the experimenter changed the order of slides in the trays after each block, the subject received a five minute rest after each block. It took six days to run Experiment 1.

The subject's performance was monitored day by day. If a subject was not responding with at least an average of 70% correct target grating detections on any day, then the following day the duration of the target and mask was increased by 1 msec. If, on the other hand, the subject showed little evidence of masking i.e., a correct target detection rate average of 85% or over then the duration was decreased by 1 msec.

Experiment 2. In this experiment Mask 2 and either Mask 1 or a mean luminance field of size and position equal to Mask 1's were presented on each trial. As explained above, Mask 2 (M2) was two 1 degree wide by 3 degree high 4 c/deg square-wave gratings, one adjacent to the left of M1 and one adjacent to the right of M1. As subjects were still instructed to gaze at the central dot this placed the center of the M2 gratings 2 degrees from the fixation point. The duration for both M1 and M2 was made equal to the last duration for M1 and target used in Experiment 1 for each subject (this duration for target, M1, and M2 was used throughout Experiments 2 to 5). No target gratings or blanks were shown. The orientations of M1 and M2 were chosen to be equal to the orientation of M1 yielding maximum masking in Experiment 1 for each subject. The SOAs used here were the same as Experiment 1. Each subject

received 14 practice trials, 2 at each SOA, then 40 experimental trials at each SOA. Instead of target gratings or blanks, Mask 1 gratings or Mask 1 mean luminance fields (M1 blanks) were shown (50% of presentations were of each type).

Trials were blocked in groups of 70 with 10 trials at each SOA (SOAs were also blocked as in Experiment 1). There were 4 experimental blocks in Experiment 2. The presentation of Mask 1 type (grating or blank) and the order of SOAs were randomized. After each block of 70 trials, the experimenter changed the slides so the subject received a five minute rest between each block. This experiment was run in one session which took approximately one and a half hours per subject.

Again a forced-choice procedure was used. Each subject was to respond "no" if he did not see M1 (grating) occur in a trial or was not sure. Each subject was to respond "yes" and rate the clarity of M1 if he saw M1 in a trial. Clarity was rated on the same three-point scale used for Experiment 1. Each subject received the instructions presented in Appendix III.

The positive SOA which produced the lowest detection rate (correct yes responses) for M1 gratings was used as the SOA2 in the metacontrast condition of Experiment 4. The negative SOA which produced the lowest detection rate for M1 was used as the SOA2 in the paracontrast condition of Experiment 4.

Experiment 3. In this experiment M2 and the target (grating or blank) were presented to the subject. The orientation of M2 for each subject was the same as the orientation of M1 and M2 used for that

subject in Experiment 2. The duration of M2 and targets was the same for each subject as in Experiment 2 for that subject. The SOAs used here also depended on the results of each subject in Experiment 2. The positive SOA2 which was established from Experiment 2 was paired with each of the positive SOAs from Experiment 1 and 0 msec. This would yield the total T to M2 SOA used for metacontrast trials in Experiment 4. The negative SOA2 which was established from Experiment 2 was paired with each of the negative SOAs from Experiment 1 and 0 msec. This yielded the total T to M2 SOA used for paracontrast trials in Experiment 4. Thus for three subjects the SOAs used in Experiment 3 were $\pm$120, $\pm$90, $\pm$60, and $\pm$30 msec. For the fourth subject the SOAs used here were $\pm$120, $\pm$90, $\pm$60, -30, and 150 msec.

Each subject received 14 practice trials, 2 at each SOA, then 20 experimental trials of target grating and 20 experimental trials of target blank at each SOA. Trials were blocked in groups of 70--10 trials at each SOA. There were four experimental blocks in Experimental 3. SOAs were again blocked, but this time 10 trials appeared in each SOA block. As in Experiment 1 the presentation of target type and order of SOAs was randomized. After each block of 70 trials the experimenter changed the slides so the subject received a 5 minute rest at this point. This experiment was run in one session which took approximately an hour and a half per subject.

A forced-choice procedure exactly the same as Experiment 1 was used in Experiment 3. Each subject received the instructions presented in Appendix IV.

Experiment 4. In this experiment disinhibition was investigated by presenting Mask 1, Mask 2, and targets (gratings or blanks) to each subject. The orientations of M1 and M2 for each subject were the same as the orientations of M1 and M2 used for that subject in Experiment 2 and 3. The SOA2 for the metacontrast and paracontrast conditions were determined from Experiment 2. For all subjects the paracontrast SOA2 here was -30 msec. For three subjects the metacontrast SOA2 here was 30 msec. while for the fourth subject this SOA2 was 60 msec. For paracontrast trials the SOAls used were 0, -30, -60, and -90 msec. while the SOAls for metacontrast trials were 0, 30, 60, and 90 msec. The durations of M1, M2, and targets were the same for each subject as the durations for these stimuli in Experiments 2 and 3.

There were 16 conditions in Experiment 4: two target types (grating or blank) X two masking conditions (paracontrast or metacontrast) X four SOAls (as above). There were 80 trials of each target type at each SOA1 of each masking condition. This yields 1280 observations per subject. These observations were broken into 16 blocks of 80 trials each. In half the blocks all metacontrast trials appeared first, in the other half all the paracontrast trials appeared first. SOAls were blocked with 10 trials at each SOA1 in each experimental block. Presentation of target grating or blank was randomized with 50% target gratings and 50% target blanks. Each subject did four blocks of 80 trials per session. As the experimenter changed the order of slides after each block, each subject received a five minute rest at this time. This experiment took four sessions to complete. Each session lasted about an hour and a half.

A forced-choice procedure exactly the same as in Experiments 1 and 3 was used in Experiment 4. Each subject received the instructions presented in Appendix V.

Experiment 5. This experiment was basically a repetition of Experiment 1 using only the M1 orientation for each subject used in Experiment 4. M1 and either a target grating or target blank were presented on each trial. The SOAs used were the same as in Experiment 1. For each subject the duration of M1 and targets were the same as in Experiment 4.

There were 14 conditions in Experiment 5; two target types (grating or blank) X seven SOAs (as Experiment 1). There were 20 trials of each target type at each SOA. Trials were presented in 4 blocks of 70 trials each. As in all previous experiments SOAs were blocked. There were 10 trials at each SOA within each experimental block. Presentation of target gratings or blanks was randomized with 50% of presentations being target blanks and 50% target gratings. The order of SOAs was also randomized for each subject. Subjects first received 35 practice trials--5 at each SOA.

As before each subject received a five minute rest between each block as the experimenter had to change the slides. Experiment 5 took one session of approximately one hour to complete. The subject's task was the forced-choice procedure used in Experiments 1, 3, and 4. Each subject received the instructions presented in Appendix VI.

RESULTS

Experiment 1

For each subject two measures were used to represent the degree of masking: 1) the probability of an error in detecting target gratings--P(E), and 2) the mean clarity ratings. P(E) was calculated using only yes vs. no responses and included a correction for false alarms:

$$P(E) = \frac{1 - P(H)}{1 - P(FA)}$$

where P(H) is the probability of a "hit" or correct target detection (i.e., a response of yes-one, yes-two, or yes-three to the presentation of a target grating) and P(FA) is the probability of a false alarm, i.e., responding to a target blank with any of the above yes responses. (See Appendix VII for a derivation of this formula.)

The mean clarity rating is the mean of the adjusted clarity ratings. Adjusted clarity ratings were calculated separately for each mask orientation during each session. This was done as the adjusted clarity ratings were used primarily to investigate orientation-specificity. The adjusted clarity ratings for each subject were calculated using the equation:

$$\text{Adjusted Clarity Rating} = \frac{H1 + (2 \times H2) + (3 \times H3) - FA1 - (2 \times FA2) - (3 \times FA3)}{4}$$

where H1, H2, and H3 represent the number of correct target detections rated 1, 2, and 3 respectively. Similarly, FA1, FA2, and FA3 represent the number of false alarms rated 1, 2, and 3 respectively. The denominator is 4 as there were 4 target grating presentations and four target blank presentations at each mask orientation during each session. The adjusted (and mean) clarity ratings could thus range from -3.0 (if only four false alarms rated 3 occurred) to 3.0 (only four hits rated three occurred).

For each subject, P(E) as a function of SOA (collapsed over all mask orientations) is shown in Figure 4. The bracket at each SOA represents $\pm$ 1 standard error. The range of standard errors was between 0.02 and 0.06 over all subjects.

Figure 4 shows a Type B masking function for SOAs greater than 0 msec. (metacontrast) for each subject (although the function is rather weak for subject JD). The SOA yielding the maximum P(E) (hereafter the maximizing SOA) varied over subjects; the maximizing SOA was 30 msec. for subjects MF and JD, 60 msec. for subject AH, and 90 msec. for subject BB. It is apparent from Figure 4 that the extent of metacontrast also varied greatly between subjects. Increases in P(E) between a 0 msec. SOA and the maximizing SOA were 0.17 for MF, 0.12 for JD, 0.49 for BB, and 0.62 for AH. Due to the variation in both the maximizing SOA and the extent of masking, the data have not been collapsed over subjects--each subject's data are treated separately.

As can be seen from Figure 4, for SOAs less than 0 msec. a strong Type B paracontrast function was found only for subject MF. JD showed an increase above the 0 msec. P(E) at two negative SOAs, but the function is not U-shaped. AH had a slight increase at a -30 msec. SOA and thus demonstrated a weak Type B paracontrast function. Subject BB had no increase at any negative SOA as compared to the 0 msec. SOA and his paracontrast function appears nearly monotonic. However, since BB's P(E) at -30 sec. equals P(E) at 0 msec. this could indicate that peak paracontrast for BB is between 0 and -30 msec. and the function is actually U-shaped.

For all subjects, the negative SOA yielding the greatest masking was always -30 msec. (although for subject JD, P(E) at -90 msec. is only slightly less than P(E) at -30 msec.). Increases in P(E) between the 0 msec. SOA and the -30 msec. SOA were: 0.13 for subject MF, 0.08 for JD, 0.00 for BB, and 0.04 for AH.

These results are consistent with previous research on Type B masking in which it has been found that greater masking occurs for metacontrast as opposed to paracontrast (e.g., Alpern, 1953; Weisstein, 1972), and that a great deal of variability occurs between subjects (e.g., Weisstein, 1972).

Figure 4 also shows that while metacontrast masking was a great deal less for experienced subjects P(E) means over positive SOAs of 0.24 for MF and 0.31 for JD as opposed to 0.61 for BB and 0.62 for AH), paracontrast masking was greater for the experienced subjects (P(E) means over negative SOAs of 0.20 and 0.25 for MF and JD respectively, versus 0.08 and 0.12 for BB and AH respectively).

In order to measure orientation-specificity we plotted P(E) against each mask orientation at the maximizing metacontrast and paracontrast SOAs of each subject (see Figure 5). These graphs show no indication of orientation-specific masking effects for either paracontrast or metacontrast (there was no orientation-specific masking evident at any other SOAs either). In fact for each subject masking appears to be greater at some mask orientation different from 45 degrees, but there was no consistent trend either between or within subjects.

The possible effect of mask orientation was considered in two other manners, mean P(E) and mean clarity ratings. Mean P(E) is the mean of the P(E)'s of each mask orientation during each of the five sessions. Mean clarity rating has been explained above. Each of these measures was plotted against the maximizing metacontrast SOA and maximizing paracontrast SOA for each subject (see Figure 6 for an example). Unfortunately neither of the two measures yielded any evidence of orientation-specific masking for any subject. Sign tests performed separately on the mean P(E)'s and on mean clarity ratings showed no significant difference in P(E) between a 45 degree mask and any other mask orientation (for each subject $p>.20$).[6]

Experiment 2. The results of Experiment 2 are summarized in the P(M1)[7] row of Table 1, the probability of an error in detecting M1 when M2 was presented. The orientations of both M1 and M2 for Experiments 2 through 5 were chosen to be the same as the orientation yielding maximum masking for a subject in Experiment 1. This orientation was 55

degrees for MF, 45 degrees for JD, 75 degrees for BB, and 65 degrees for AH.

The purpose of this experiment was to find the positive and negative SOAs which maximized masking of M1 by M2 for each subject. These SOAs were then to be used as the SOA2s between M1 and M2 in Experiment 4. For subject MF the positive SOA yielding maximum M1 masking was clearly 30 msec. (see Table 1). However, for negative SOAs there appears to be a tie between -30 and -60 msec. In order to break this tie the clarity ratings for these two SOAs were considered. It was found that the mean clarity rating for -30 msec. was lower, and thus this SOA was chosen as the negative SOA2 for subject MF. Given these findings the positive SOA2 (MSOA2) to be used only with metacontrast trials (SOA1s of 0, 30, 60, and 90 msec.) for MF in Experiment 4 was 30 msec., while the negative SOA2 (PSOA2) to be used only with paracontrast trials (SOA1s of 0, -30, -60, and -90 msec.) was -30 msec.

As Table 1 shows the -30 msec. SOA clearly yielded maximum M1 masking among negative SOAs for subjects JD, BB, and AH. Thus, this SOA was chosen as the PSOA2 for all subjects in Experiment 4. Among positive SOAs 30 msec. yielded maximum M1 masking for subjects JD and AH, while 60 msec. lead to maximum M1 masking for BB. Thus, 30 msec. was chosen as the MSOA2 in Experiment 4 for subjects MF, JD, and AH, while for subject BB the MSOA2 was 60 msec. See Figure 7 for a diagram of these SOA presentations.

Experiment 3. The results of Experiment 3 are summarized in Table 1 in the P(T/M2) row--the probability of an error in detecting T when M2 was presented. The SOAs used here were the SOAs, for each subject, which would occur between T and M2 in Experiment 4. Thus for all subjects the negative SOAs here were -30, -60, -90, and -120 msec. (these T-M2 SOAs would occur when the PSOA2 of -30 msec. was paired with the paracontrast SOAls of 0, -30, -60, and -90 msec.). For every subject except BB, the positive SOAs here were 30, 60, 90, and 120 msec. (pairing the MSOA2 of 30 msec. with the metacontrast SOAls of 0, 30, 60, and 90 msec. results in these T-M2 SOAs). For subject BB, whose MSOA2 was established to be 60 msec., the positive T-M2 SOAs here were 60, 90, 120, and 150 msec.

The results indicated by P(T/M2) show the degree of masking of the target grating when only M2 was presented. An estimation of the degree of T masking by M2 was necessary in order to generate a hypothesis of the amount of T masking which would occur when both M1 and M2 were presented assuming no disinhibition (the generation of this hypothesis is discussed below). While the purpose of Experiment 3 was to enable the generation of this hypothesis, it is interesting to note that the masking effect of M2 was generally less than the masking effect of M1 presented at the maximizing orientation in Experiment 1 (the latter is represented in Table 1 by P(T/M1, E1)). As M2 was separated from T by 1^{o} of visual arc while M1 was directly adjacent to T, the finding of less masking by M2 supports previous studies which have found that increasing the intercontour distance between target and mask decreases

the degree of target masking (cf. Alpern, 1953; Toch, 1956; Weisstein and Growney, 1969).

Experiments 4 and 5. As the results of Experiment 5 are used to establish an estimate of M1 masking and this estimate is necessary to generate the hypothesis of M1+M2 masking assuming no disinhibition, we will first consider the results of Experiment 5 and then the results of Experiment 4.

The results of Experiment 5 are summarized in Table 1 as P(T/M1, E5)--the probability of an error in detecting T at each SOA during Experiment 5. Recall that in this experiment only M1 was presented and only at the same orientation used for each subject in Experiments 2 and 4 (i.e., the mask one orientation which maximized masking in Experiment 1). The results of Experiment 1 for this same M1 orientation are presented in Table 1 as P(T/M1, E1). As can be seen from Table 1 some subjects had less masking by M1 (i.e., a lower probability of error in detecting T) in Experiment 5 as opposed to Experiment 1, while others had greater masking by M1 in Experiment 5 (subject BB at SOAs of 30, 60, and 90 msec. provides an example of the former, while subject MF at SOAs of -30 and 30 msec. provides an example of the latter). The average probability of error in detecting T over Experiment 1 and 5 is therefore a more realistic measure of the masking of T by M1 with which to compare the results of Experiment 4 (i.e., masking of T by M1 and M2 combined). This average is presented in Table 1 as P(T/M1)--our estimate of the probability of error in detecting T which is caused by M1 in Experiment 4.

We have now established estimates, for each subject at each SOA, of the degree of masking of T when M1 is presented and when M2 is presented with T. As Dember and Purcell (1968) pointed out, if there is no disinhibition then the effects of M1 and M2 on T can be considered to be independent. Thus when M1 and M2 are presented together (Experiment 4) the joint masking effect of the two masks should be predictable from the formula for combining independent probabilities:

$$P(T/M1+M2)nd = P(T/M1)+P(T/M2) - P(T/M1) \times P(T/M2)$$

where P(T/M1+M2)nd refers to the predicted probability of error in detecting T when M1 and M2 are presented under the assumption of no disinhibition. This predicted value is presented in Table 2 for each subject at each SOA1 used in Experiment 4. The values for P(T/M1), P(T/M2), and the actual value for masking by M1 and M2 combined in Experiment 4--P(T/M1+M2)--are also presented in Table 2.

The probabilities presented in Table 2 are shown under the appropriate SOA1s (the SOA between T and M1 presentation) used in Experiment 4. Some explanation of the design of this table is necessary, especially concerning the SOA1s of P(0) and M(0). For Experiment 4 (P(T/M1+M2)) both of these SOA1s are actually 0 msec.; however, P(0) represents trials in which an SOA1 of 0 msec. was paired with an SOA2 equal to each subject's PSOA2, while M(0) represents trials in which an SOA1 of 0 msec. was paired with an SOA2 equal to the subject's MSOA2.

P(T/M1) in Table 2 represents the estimate of the degree of masking of T by M1 in Experiment 4 (i.e., the probability that an error

in detecting T was caused by masking by M1 only). Thus, P(T/M1) at P(0) is our estimate of the degree of M1 masking of T at an SOA1 of 0 msec. and SOA2s equal to the subject's PSOA2. Similarly P(T/M1) at M(0) is our estimate of the degree of M1 masking of T at an SOA1 of 0 msec. and an SOA2 equal to the subject's MSOA2. Both of these estimates are based on the average performance at an SOA of 0 msec. in Experiments 1 (for the maximizing mask orientation) and 5 and hence are equivalent.

P(T/M2) in Table 2 represents the estimate of the degree of masking of T by M2 in Experiment 4 (i.e., the probability that an error in detecting T was caused only by masking by M2). At this point the reader can infer the meaning of P(T/M2) at P(0) and M(0) based on the above explanations. The P(T/M2)s found in Experiment 3 (Table 1) are simply placed under the corresponding SOA1s in Table 2. For example, for subject MF a T to M2 SOA of -120 msec. corresponds to an SOA1 of -90 msec. as M2 always preceded M1 by 30 msec. (PSOA2 of -30 msec.). Similarly for MF an SOA between T and M2 of -90 msec. corresponds to an SOA1 of -60 msec., and so forth.

P(T/M1+M2) in Table 2 represents the probability of an error in detecting T during Experiment 4. Here we have the actual probabilities which resulted when M1 and M2 were presented together. If disinhibition occurred (i.e., if the effects of M1 and M2 when presented together are not independent) then P(T/M1+M2) should be less than P(T/M1+M2)nd. Using the direct difference method for calculation of Student's t

(Runyon and Haber, 1976) it was found that P(T/M1+M2) was indeed less than P(T/M1+M2)nd for each subject.[8]

The specific results for a one-tailed t-test for each subject were: 1) subject MF- t=2.23, p<.05, 2) subject JD- t=4.19, p<.005, 3) subject BB- t=6.46, p<.001, and 4) subject AH- t=2.55, p<.025.

A one-tained z-test for proportions (Hardyck and Petrinovich, 1969) was then done at each SOAl for each subject. The results indicated that P(T/M1+M2) was less than P(T/M1+M2)nd at each of the following SOAls:

1) Subject MF at SOAls of -60 msec. (z=1.79, p<.05), -30 msec. (z=3.68, p<.001), 30 msec. (z=2.59, p<.01), and 60 msec. (z=4.65, p<.001).

2) Subject JD at SOAls of -90 msec. (z=1.99, p<.025), -60 msec. (z=3.94, p<.001), P(0) (z=3.85, p<.001), M(0) (z=3.78, p<.001), 60 msec. (z=4.13, p<.001), and 90 msec. (z=1.83, p<.03).

3) Subject BB at SOAls of -60 msec. (z=1.67, p<.05), -30 msec. (z=2.89, p<.01), P(0) (z=3.34, p<.001), M(0) (z=4.91, p<.001), 30 msec. (z=5.01, p<.001), 60 msec. (z=6.27, p<.001), and 90 msec. (z=4.12, p<.001).

4) Subject AH at SOAls of -60 msec. (z=3.27, p<.001), 30 msec. (z=4.26, p<.001), 60 msec. (z=4.69, p<.001), and 90 msec. (z=6.01, p<.001).

It thus appears that not only did disinhibition occur, but it occurred at both paracontrast and metacontrast SOAls. This is contrary to the expectations of the Breitmeyer and Ganz (1976) theory of Type B masking, wherein only paracontrast disinhibition is predicted. The

results of Experiment 4 are graphed in Figures 8 through 11. P(T/M1+M2) and P(T/M1+M2)nd are presented for each subject at each SOA1. Arrows below an SOA1 indicate that P(T/M1+M2) was significantly less than P(T/M1+M2)nd at that SOA1.

We also decided to use the assumption of disinhibition to predict the results of Experiment 4 and to compare these predictions to the predictions under the assumption of no disinhibition. As explained above the predicted probability of error in detecting T under the assumption of no disinhibition can be found using the formula:

$$P(T/M1+M2)nd=P(T/M1)+ P(T/M2) - P(T/M1)xP(T/M2)$$

Under the assumption of disinhibition the predicted probability of error is entirely different. As with no disinhibition the effect of mask two is assumed to be simply P(T/M2). However, the effect of mask one must include a term which subtracts from P(T/M1) the amount of T masking by mask one which will be negated by inhibition of M1 by M2. This term can be represented by P(M1)xP(T/M1), where P(M1) is the degree of masking of M1 by M2. This term shows the probability that an error in detecting T will <u>not</u> occur due to masking of M1 by M2. The predicted effect of M1 under disinhibition is thus equal to P(T/M1)- P(M1)xP(T/M1).

To find the predicted values for Experiment 4 we must then add the effects of M1 and M2 and subtract the intersection of these effects, i.e., P(T/M2)x (P(T/M1)- P(M1)xP(T/M1)). Therefore the predicted masking effect of M1 and M2 under the assumption of disinhibition is:

$$P(T/M1+M2)d = P(T/M2)+P(T/M1)-P(M1)xP(T/M1)-P(T/M1)xP(T/M2) +P(T/M1)xP(T/M2)xP(M1),$$

where P(T/M1+M2)d is the predicted probability of error in detecting T in Experiment 4 assuming disinhibition.

The results of the no disinhibition and disinhibition predictions for Experiment 4 are shown in Figures 12 through 15, where they are compared with the actual results of Experiment 4, i.e., P(T/M1+M2). It is apparent from these figures that neither model accurately predicts the results of masking by two masks, under the present assumptions. However, at least under metacontrast the assumption of disinhibition appears to more closely predict the effect of M1+M2. For subject MF (Figure 12) this is true at SOAls of M(0), 30 and 60 msec., while at 90 msec. P(T/M1+M2)nd more closely predicts P(T/M1+M2). For subject JD (Figure 13) disinhibition provides a better predictor at SOAls of M(0), 60, and 90 msec., while at 30 msec. an assumption of no disinhibition leads to a better prediction of M1+M2 masking. For subject BB (Figure 14), P(T/M1+M2)d is closer to P(T/M1+M2) at all metacontrast SOAls. For subject AH (Figure 15), P(T/M1+M2)d provides a better estimate of P(T/M1+M2) only at a metacontrast SOA1 of 90 msec. At M(0) both the assumption of disinhibition and the assumption of no disinhibition are fairly close to P(T/M1+M2), while at 30 and 60 msec. both assumptions led to predictions which are rather distant from P(T/M1+M2).

Predictions concerning paracontrast are less impressive. For subjects MF and BB (Figures 12 and 14) the P(T/M1+M2)d and P(T/M1+M2)nd

paracontrast predictions are virtually equal. Both predictions are very close to the actual P(T/M1+M2) at an SOA1 of -90 msec. for both subjects and at an SOA1 of P(0) for MF. For subject JD, P(T/M1+M2)d is very near the actual value at an SOA1 of P(0) while P(T/M1+M2)nd is distant. At SOAls of -90 and -60 msec. disinhibition leads to a more accurate prediction while at an SOA1 of -30 msec. P(T/M1+M2)nd provides a much better predictor of P(T/M1+M2). For subject AH (Figure 15), P(T/M1+M2)d is a more accurate predictor at SOAls of -90 and -60 msec., while at SOAls of -30 and P(0) msec. P(T/M1+M2)nd is closer to P(T/M1+M2) being very accurate at P(0) msec. Thus over all subjects P(T/M1+M2)d seems to be only a slightly better predictor of P(T/M1+M2) under paracontrast.

Under paracontrast two factors may be affecting P(T/M1+M2) and the predictions of this value: 1) a floor effect--masking under paracontrast may be so low that it is difficult to detect differences under paracontrast, and 2) little masking effect of M2 on M1 for some subjects (MF and BB)--if P(M1) is very low then the difference between P(T/M1+M2)d and P(T/M1+M2)nd will be very small and no differentiation can be made between these two sets of predictions at paracontrast SOAls.

Concerning the inaccuracy of P(T/M1+M2)d there may be two effects which, if they could be estimated, would provide a more accurate predictor. First it is possible that on some trials when neither M1 or M2 alone would have masked T the occurrence of M1+M2 acted to mask T. This masking effect (let us call it P(M1+M2)) would have to be added to both P(T/M1+M2)d and P(T/M1+M2)nd, or to only P(T/M1+M2)nd if it

is assumed that such a masking effect does not occur under disinhibition. There is, however, no way to calculate P(M1+M2) in the present study.

The second possible "ignored" effect is that of M1 on M2. We have assumed that on some trials M1 did not mask T but that M2 did. If on some of these trials M1 masked M2 so that M2 could no longer mask T then our use of P(T/M2) as a measure of the masking effect of M2 would be inaccurate. The values for P(T/M2) would then be somewhat less than the values used to predict P(T/M1+M2). Unfortunately, there was also no manner in which to calculate the effect of M1 on M2 in the present study. Considerations of P(M1+M2) and of the effect of M1 on M2 may have made the disinhibition predictions more accurate. While it is possible that such considerations would have made the no disinhibition predictions more accurate than the disinhibition predictions, the greater inaccuracy of P(T/M1+M2)nd at most metacontrast SOAls makes this seem unlikely. Hopefully, future studies of disinhibition will consider these previously ignored effects.

DISCUSSION

Experiment 1

The major results of Experiment 1 were: 1) the masking functions were Type B, with greater metacontrast than paracontrast masking, and 2) there was no orientation-specific masking apparent for any subject. We will discuss each of these findings separately.

Type B masking. In Experiment 1 the duration, contrast, and luminance of T and M1 were equalized within each subject and the size of each flanking rectangle (1/2 of M1) was equal to the size of T. Thus the resultant T/M1 ratio was near 1.0. The masking functions of the present study, therefore, are in support of Weisstein's (1972) contention that T/M ratios near 1.0 will yield Type B masking.

Considering masking over all subjects, metacontrast was stronger than paracontrast--a result consistent with the Type B literature (e.g., Alpern, 1953; Kahneman, 1968; Weisstein, 1972). However, for subjects MF and JD paracontrast masking was only marginally less than metacontrast masking. Weisstein (1972) found Type B paracontrast to be nearly as strong as metacontrast for one subject, while paracontrast was much weaker than metacontrast for two other subjects. This finding led us to speculate (above) that paracontrast is very susceptible to individual differences in visual processing. While our results may merely reflect

such individual differences, another explanation is possible. Subjects MF and JD were experienced with masking procedures (MF to a greater extent than JD) while subjects BB and AH were not. Practice with masking may reduce the effects of masking. The reduction of MF's T and M1 duration from 7 to 4 msec. through Experiment 1, in order to keep the proper average detection rate (see Method), and the decrease in masking for subjects BB and AH from Experiment 1 to Experiment 5 (see Table 1, P(T/M1, E1) and P(T/M1, E5)), support this contention. However, this practice effect may not be as strong for paracontrast as for metacontrast. This appears to be the case for subjects BB and AH (see Table 1), if we compare the decrease in P(E) under paracontrast SOAs to the decrease in P(E) under metacontrast SOAs. Thus when we attempted to keep an average detection rate over each session for subjects MF and JD we may have used durations at which metacontrast effects were lowered due to practice but paracontrast masking was little affected.

The duration used for each subject only had to produce a detection rate over all SOAs of between 70 and 85%. It did not matter how this average was obtained--whether metacontrast and paracontrast were approximately the same (MF and JD) or metacontrast was much greater than paracontrast (BB and AH). As long as the overall detection rate was in the proper range the duration was not adjusted. A more appropriate course may have been to set the average detection range for metacontrast and paracontrast trials separately.

While such a separate but equivalent criterion would sometimes lead to different durations for metacontrast and paracontrast trials

(and thus confound any interpretation of the relative strengths of the masking effects), it would more properly control for the effects of practice on both metacontrast and paracontrast. This could also lead to more substantive investigations of paracontrast as the duration of stimuli would be such that this weak effect could be enhanced. Previous studies of masking have used the same detection criteria and duration under paracontrast and metacontrast conditions (e.g., Alpern, 1953; Kolers and Rosner, 1960).

The differences in practice in masking between subjects and the average detection range used over all SOAs may have contributed to the variability between subjects in this study. However, the great degree of variability found in this study--variation in amount of masking, the maximizing SOAs, and relative paracontrast to metacontrast strength--has similarly been noted by previous researchers (e.g., Robinson, 1968; Weisstein, 1972; Kranda, 1977; Breitmeyer, 1977). The above suggestion for paracontrast and metacontrast detection ranges and using equally practiced or equally naive subjects may reduce this variability.

The strength of masking in the present study is somewhat surprising in light of previous studies on foveal masking. As discussed above many researchers have found little or no evidence of foveal masking (e.g., Alpern 1953; Kolers and Rosner, 1960; Stewart and Purcell, 1970). In fact there seems to be a prevailing notion (e.g., Breitmeyer and Ganz, 1976) that metacontrast is very weak or absent with foveal stimuli. The present study, however, supports the results of studies

which have found strong foveal masking (e.g., Schiller and Smith, 1965; Lefton, 1970; White and Lorber, 1976; Saunders, 1977). We have extended foveal masking effects to paracontrast as strong forward masking effects were found for MF and to a lesser extent for subjects JD and AH. As the stimuli, the measure of masking, and various experimental conditions vary so greatly between masking studies, it seems likely that the differences reported on the extent of foveal masking are attributable to differences in experimental designs. Certain conditions which existed in this study must have contributed to strong foveal masking. As the present study did not investigate foveal vs. peripheral masking we have no evidence as to which of the experimental conditions (low luminance, low contrast, T/M near 1.0, duration of stimuli set by a specific detection criterion, etc.) contributed to foveal masking. Variations of experimental conditions with varying retinal positioning of the stimuli could yield evidence as to those specific conditions necessary for foveal masking.

Orientation-specificity. As has been pointed out, there was no orientation-specific masking in Experiment 1. In fact for some subjects masking was greater (higher P(E)) when the mask orientation differed by as much as 30° from the target orientation (see Figure 5, P(E) at mask orientation of 75° vs. P(E) at 45°). Such increases in P(E), however, were not found to be significant. As masking under paracontrast and metacontrast was equally nonspecific, there was no evidence to support the hypothesis that paracontrast is more orientation-specific than metacontrast.

As many previous studies have found orientation-specific masking (e.g., Campbell and Kulikowski, 1967; Gilinsky, 1967, 1968; Gilinsky and Dolherty, 1969; Houlihan and Sekuler, 1968; Sekuler, 1969) our lack of orientation-specific masking was totally unexpected. In all of the previous studies cited as mask orientation was increasingly varied away from the target orientation the degree of masking decreased rather monotonically. When the mask orientation differed from the target's by 30 degrees or more there was little, if any, masking. The results of the present study are inconsistent with these findings.

However, all of these previous studies used masks which overlapped the target. The present study used a flanking mask. This difference initially led us to speculate that orientation-specific masking may only occur with overlapping target and mask. However, Weisstein, Harris, Berbaum, Tangney, and Williams (1977) found that a thin black bar, retinally separated by as much as 4 degrees from a target grating, had masking effects on the grating only when the bar and grating were of the same orientation. Lovegrove (1977) found that masking decreased when the relative orientations of a disk grating and a surrounding annulus grating were varied by 15^{o}. In neither of these studies was masking accomplished by overlapping masks, yet orientation-specific masking effects were obtained. These later studies differ from the present study along a number of experimental dimensions--type of stimuli, durations, measure of masking and others (for example, Weisstein et al., used 15 c/deg circular target gratings, a 2' wide bar as mask, a 100 msec. mask duration, a 10 msec. target duration, and magnitude

estimations of masking). Thus the inconsistency between our results and those of Weisstein, et al., (1977) and Lovegrove (1977) may be attributable to differences in experimental design.

It is evident from Figure 6 that there was a large amount of variability at each mask orientation. The lack of orientation-specific masking may be a result of this large variability. The variability here may be due to the design of Experiment 1. In this experiment a number of variables were concurrently manipulated (mask orientation, SOA, target type), blocks within a session were separated by fairly long rest periods, and the experiment was run over five days.

In previous research by this writer (Fotta, 1976), when a number of variables were manipulated concurrently in a visual study the subjects reported that they set up expectations as to what type of presentation would appear next. When these expectations were not met (which usually occurred) their ability to report the target suffered. In the present study subjects may have set up expectations as to the target type and SOAs (even though SOAs were blocked subjects were not informed when SOAs were changed). These expectations could have produced an additional source of variability in the present study.

In order to reduce this variability the present study could be modified so that only two SOAs were used. Pilot research would establish the maximizing paracontrast and metacontrast SOAs for each subject. Mask orientation would then be varied at only these SOAs. A threshold detection task could also be used to remove target-type expectations.

With this design a subject could then receive all the trials in a single session in order to exclude variability between sessions.

Another possible explanation of our lack of orientation-specificity results if we consider the research of Thomas and Shimura (1975). Thomas and Shimura (1975) presented various stimuli to subjects in a detection paradigm. The stimuli were two bars of light, overlapping at the center, with one bar vertical and one bar rotated from the vertical at various orientations from 5 to 90 degrees (imagine X's with various angles between the bars). Thomas and Shimura found that detection sensitivity was least when the orientations of the two components of the stimulus differed by 15 or 25 degrees. The conclusion drawn by the researchers was that the reduced visibility was due to inhibition between channels tuned to different orientations (Andrews, 1965; Atkinson, 1972; Benevento, Creutzfeldt, and Kuhn, 1972) and that this inhibition was greatest when the channels are tuned to orientations which differ by 15 to 25 degrees. Thomas and Shimura pointed out that this conclusion is in agreement with the work of Blakemore, Carpenter, and Georgeson (1970) who found inhibition to be greatest between channels which differed by 15 degrees.

In light of this finding it seems possible that our result of no orientation-specific masking may reflect the interaction of inhibition due to channels tuned to the same orientation with inhibition due to channels tuned to different orientations. If such an interaction does occur then P(E) as a function of mask orientation (Figure 5) actually represents the resultant of two masking functions: 1) a monotonically

decreasing function which reflects only the inhibition due to the same-orientation channels, and 2) a U-shaped function with a maximum occurring when target and mask differ by 15 to 25 degrees; this reflects only the different-orientation channel inhibition. The varying shapes of the functions in Figure 5 and 6 could be accounted for if the slope of the monotonic function and the maximum of the U-shaped function vary between subjects and SOAs. As an example let us consider the functions of subject JD at 60 msec. (Figure 5b) and subject AH at 60 msec. (Figure 5d). JD's function here could be the result of a steep monotonic same-orientation channel function and a U-shaped different orientation channel function that has a maximum when the target and mask differ by about 25^{o}. AH's function could be the result of a gradual monotonic same-orientation channel function and a U-shaped different-orientation channel function which has a maximum when the target and mask differ by about 15^{o}.

Admittedly such an explanation lacks parsimony. However, given the consistent finding of great variability between subjects in Type B masking such a variation in the types of inhibition responsible for the masking seems possible; that is, if there are indeed two types of inhibition (same and different-orientation channel) interacting in masking. Unfortunately, the present study yields no direct evidence as to the occurrence of such an interaction. Proof or refutiation of our speculation must await future studies.

In conclusion, Experiment 1 has failed to yield either paracontrast or metacontrast orientation-specific masking. Considerations of the

Breitmeyer and Ganz (1976) theory of Type B masking led us to a prediction of more orientation-specific masking under paracontrast. The finding of no orientation-specific masking thus leaves us unable to comment on the appropriateness or inappropriateness of the Breitmeyer and Ganz approach to Type B masking (i.e., whether or not sustained intrachannel inhibition yields paracontrast while transient inhibition of sustained channels leads to metacontrast).

Experiments 2 through 5

The discussion of these experiments is grouped together as: 1) Experiment 2 was run merely to provide PSOA2 and MSOA2 for Experiment 4, 2) Experiment 3 was run to provide a measure of masking of T by M2; this measure was used in developing the no disinhibition hypothesis for comparison with Experiment 4, and 3) Experiment 5 was run to provide a more accurate estimate of T masking by M1; this estimate was also used in developing the no disinhibition hypothesis for comparison with Experiment 4. In other words Experiments 2, 3, and 5 were necessary to accomplish the purpose of Experiment 4 (i.e., the testing of our disinhibition hypothesis). The main thrust of this section is thus a discussion of Experiment 4 where disinhibition was found under both paracontrast and metacontrast. However, before beginning a discussion of disinhibition we will consider the finding of Experiment 3 that increasing the intercontour distance between target and mask decreased masking.

Intercontour distance--Experiment 3. Previous studies have repeatedly found that increasing the intercontour distance between target and mask decreases the masking effect (e.g., Alpern, 1953; Cox, Dember, and Sherrick, 1969; Growney, Weisstein, and Cox, 1977). Alpern (1953) found that an intercontour distance of one degree greatly reduced masking effects. Alpern also found that as spatial separation increased the maximizing SOA decreased.

Kolers and Rosner (1960) reported that the probability of detecting a target disk, when the disk to annulus mask separation was 0.63^{o}, was about five times as great as when the disk and annulus were contiguous. However, Kolers and Rosner found that there was still a weak masking effect at this separation while no masking was found at a separation of 1.2^{o}. The maximizing SOA in this study increased as spatial separation increased.

Growney, Weisstein, and Cox (1977) found that while metacontrast was weaker at spatial separations of 1 degree, the masking effect was still strong at this distance and some masking was found up to distances of 3 degrees. Growney, et al., reported that the maximizing SOA did not change as the intercontour distance was increased. Growney and Weisstein (1972) also found no change in the maximizing SOA as the spatial separation of the target and mask were increased. Weisstein, et al., (1977) found fairly strong masking effects at separations up to 4 degrees.

The present study is generally consistent with the finding of less masking at greater intercontour distances. The effect of M2 on T (P(T/M2) in Table 3), at an intercontour distance of one degree, is

generally much less than the effect of M1 on T (P(T/M1) in Table 3) where the stimuli are contiguous. This is true under paracontrast and metacontrast SOAs for subjects MF and BB and under metacontrast SOAs for subject AH. However, for subject JD (P(T/M2) is greater than P(T/M1) at half of the SOAls, and for subject AH P(T/M2) is greater than P(T/M1) at three SOAls less than 30 msec. These latter cases are contrary to previous findings and, outside of the general variability in masking, we can offer no explanation for them at this time.

Considering P(T/M2) (Table 3) it is apparent that except for subject MF, masking, while reduced when the mask is 1^{o} from the target, is evidently still fairly strong at a number of SOAls. This finding is more supportive of the findings of Growney et al., (1977) and Weisstein et al., (1977) than of the studies finding very weak masking at a separation of one degree (Alpern, 1953; Kolers and Rosner, 1960). Furthermore the maximizing paracontrast and metacontrast SOAs did not change for subjects MF and BB with the more distant mask. This is agreement with Growney and Weisstein (1972) and Growney et al., (1977) and contrary to the findings of Alpern (1953) and Kolers and Rosner (1960).

Theories of masking in terms of lateral inhibitory interactions usually assume that the inhibition propogates at a specific speed in terms of the retinal visual angle (Weisstein, 1968; Bridgeman, 1971). The SOA causing maximum masking should, according to these theories, decrease as the mask is moved increasingly further from the target. Neither our results nor those of Growney and Weisstein (1972) and Growney et al., (1977) support such a hypothesis. However, this necessarily only brings into question the assumption of a specific speed of

inhibitory propogation and not necessarily all theories of lateral inhibitory masking. If we assume that inhibition can be caused by neurons with different conduction speeds and response latencies, then our masking results and those of Growney and co-workers can still be accounted for in terms of lateral inhibitory interactions. As reviewed above, the physiological literature supports such an assumption (Stone and Hoffman, 1971; Cleland, Levick, and Sanderson, 1973; Ikeda and Wright, 1975b; Dreher, Fukada, and Rodieck, 1976).

Disinhibition--Experiment 4. The results of the present study indicate that the degree of masking which results from two masks is often less than that which would be expected if each mask independently affected a target stimuli. This disinhibition was found to occur under both paracontrast and metacontrast conditions. Our results are consistent with many previous psychophysical studies (e.g., Alpern and David, 1959; MacKavay, Bartley, and Casella, 1962; Robinson, 1966, 1968; Dember and Purcell, 1967) and are in agreement with the results of physiological studies (Hartline and Ratliff, 1957, 1958; Ratliff and Hartline, 1959; Rentschler and Hilz, 1976).

Our results extend the occurrence of disinhibition in masking to non-overlapping stimuli, to Type B masking, and to forward masking. The previous masking studies of disinhibition did not use completely non-overlapping stimuli, and with the exception of Lovegrove (1976) disinhibition under paracontrast has not been previously examined. Also, masking was monotonic in all previous studies with the exception of Barry and Dick (1971, Experiment 2). With the use of non-overlapping

stimuli the cue of Mach bands (Schurman and Eriksen, 1969) can not be used in detecting the target. Furthermore the present design by using a forced choice procedure with target blanks of mean luminance equivalent to target grating mean luminance renders ineffective any use of apparent motion (Schurman and Eriksen, 1969) or brightness reversals (Barry and Dick, 1971) in order to detect the target. The present study also found strong foveal disinhibition, whereas previous studies with foveal target presentations reported little or no disinhibition (Schurman and Eriksen, 1969; Uttal, 1970; Barry and Dick, 1971).

At each metacontrast SOA1 in the present study disinhibition occurred for at least two subjects. Disinhibition at metacontrast SOAls was found at SOA2s of 30 msec. (subjects MF, JD, and AH) and 60 msec. (subject BB). If we assume that the number of subjects for which disinhibition occurred at an SOA1 is an indication of the strength of disinhibition, then disinhibition was weakest at an SOA1 of M(0)--0 msec.--(where disinhibition existed for only two subjects--JD and BB), somewhat stronger at SOAls of 30 msec. (subjects MF, BB, and AH) and 90 msec. (subjects JD, BB, and AH), and strongest at an SOA1 of 60 msec. (all subjects). These findings are in agreement with previous disinhibition studies which have found disinhibition from an SOA1 of 4 msec. with an SOA2 of 35 msec. (Long and Gribben, 1972) to an SOA1 of 80 msec. with an SOA2 of 55 msec. (Dember and Purcell, 1967). Long and Gribben (1972) also reported that disinhibition was weaker at SOAls of 4 msec. then at longer SOAls. Although we have some indication that disinhibition is weaker at an SOA1 of 0 msec., this weakness may only reflect

the U-shape function of masking in the present study, i.e., less masking occurred at a 0 msec. SOA1 then at longer SOAls.

At each paracontrast SOA1 disinhibition occurred for at least one subject. This paracontrast disinhibition was found at an SOA2 of -30 msec. Following our assumption concerning the strength of disinhibition, it appears that among paracontrast SOAls disinhibition was weakest at an SOA1 of -90 msec. (where it existed for only one subject--JD), somewhat stronger at SOAls of -30 msec. (subjects MF and BB) and P(0)--0 msec.--(subjects JD and BB), and strongest at an SOA1 of -60 msec. (all subjects). No comparison can be made with previous studies as disinhibition under paracontrast conditions has been investigated at only one SOA1, -20 msec., in only one study, Lovegrove (1976).

As has been previously discussed disinhibition under paracontrast can be accounted for by the Breitmeyer and Ganz theory of Type B masking. A model for paracontrast disinhibition which is consistent with this theory is presented below. This model (and the model presented later for metacontrast disinhibition) assumes that the basic cause of disinhibition is that the target is released from masking by M1 on some trials in which M1 is itself masked by M2. In terms of inhibitory reactions it is assumed that M2 neural activity inhibits M1 neural activity on some trials in which M1 activity would normally inhibit T neural activity. When this occurs M1 can not inhibit T; thus masking of T by M1 decreases.

Consideration of Table 2 lends some support to this assumption. Comparing P(T/M1+M2) and P(T/M1) at paracontrast SOAls it can be seen

that masking by M1+M2 (P(T/M1+M2)) is less than masking by M1 (P(T/M1)) at some SOAls for each subject; specifically, at SOAls of -90 msec. (subject BD), -60 msec. (subject AH), -30 msec. (subjects MF and BB) and P(0) (subjects JD and BB). If, in order to maximize the possible contribution of M1 to masking of T under M1+M2, we assume that M2 does not mask T at all under M1+M2, then masking by M1 must be less at these SOAls (for the appropriate subjects) when M2 is presented than when M2 is not presented. The assumption of any contribution of M2 masking of T leads to the same conclusion.

A model for paracontrast disinhibition. According to Breitmeyer and Ganz (1976) paracontrast is hypothesized to occur due to intrachannel sustained inhibition arising from the antagonistic center-surround organization of the RF's of sustained cells. As the surround inhibitory response is slower than the central excitatory response, a stimulus delivered to the surround before a stimulus delivered to the center of the RF will cause maximal inhibition and hence masking of the central stimulus. In the present study due to the size of the target stimulus (3^{o} high by 1^{o} wide) we would have to assume that the target falls on the RF center of a number of cells which mediate a response to the target, while the masking stimuli (M1) falls in the RF surround area of these same cells. If we assume that the inhibitory response of the surround is proportional to the excitatory response of a second set of sustained cells whose RF centers mediate the same retinal area as the surround of the first set, then paracontrast disinhibition can

be accounted for. Disinhibition would occur when a stimulus (M2) falls on the surround area of this second set of cells before a stimulus (M1) falls on the central area of this second set. The central excitatory response of the second set would be reduced and, following our assumption, this would reduce the inhibitory effect of the surround of the first set leading to increased central excitatory response of the first set. Thus the target stimulus falling on this central area (first set) would be reported more often than when only M1 was shown.

This model must be modified to take into account those cells whose center RF mediate a target response and whose surround extends far enough to respond to the M2 stimulus. Such cells would, by themselves, decrease responses to the target. Thus the combination of M2 inhibition of M1 central response and M2 inhibition of T central response may yield less inhibition, more inhibition, or the same inhibition as M1 alone. One can not be sure unless the M2 effect on T is considered. The present study gathered estimates of the inhibiting effect of M2 alone. Comparisons of the effect of M1 and M2 with the predicted independent effects of M1 and M2 indicated that M2 interfered with M1 masking of T. The remaining inhibition of T can be accounted for in terms of the model just described.

There appears to be at least one problem with this model; that is the assumption that all paracontrast inhibition is mediated via sustained cells. If this is true then the surround-center latency for sustained cells must vary from 60 to 0 msec. within subjects JD and BB for disinhibition is found for these subjects at SOAls from

-60 msec. to 0 msec. While this may in fact be the case, the study by Winters and Hamasaki (1976) casts doubt on this assumption. Recall from our previous discussion of this study that Winters and Hamasaki found that for cat retinal ganglion cells the inhibition of the central response was greatest in sustained cells when the surround preceded the central stimuli by 7 msec. (mean). Inhibition of the central response of transient cells was greatest when the surround stimuli preceded the central stimulus by 38 msec. (mean).

If this latency difference between transient and sustained center-surround RFs extends to humans then paracontrast may be caused by sustained inhibition at short SOAs and by transient inhibition at longer SOAs. The finding of maximum paracontrast at an SOA of -30 msec. for three subjects in this study and at SOAs from -20 to -70 msec. in previous paracontrast investigations (e.g., Alpern, 1953; Kolers and Rosner, 1960; Weisstein, 1972) is consistent with the results of transient cells in the Winters and Hamasaki (1976) study. However, the findings of maximum paracontrast at an SOA of 0 msec. for subject BB in this study, and at this same SOA for many subjects in a variety of studies finding only Type A paracontrast are consistent with the results of sustained intrachannel inhibition in the Winters and Hamasaki study. The finding of weak Type B paracontrast (Kahneman, 1968) may represent the results of transient intrachannel inhibition. If in Type B paracontrast studies only the transient channels (low spatial frequency information) were masked then much of the higher spatial-frequency information, transmitted via sustained channels is left unscathed.

Thus the reduction in target information would be much less than in metacontrast where sustained channels are inhibited. This differential reduction in target information between metacontrast and paracontrast conditions may cause the large difference in their masking effects.

Finally, the assumption of the involvement of both transient and sustained intrachannel inhibition in paracontrast could account for the peculiar paracontrast results of subject JD. As is shown in Figure 4 this subject had a W-shaped paracontrast function with maxima at both -30 and -90 msec. This W-shaped function was found for this subject during each session of Experiments 1, indicating the functioning of some rather consistent mechanism. Perhaps this mechanism is the combination of sustained and transient intrachannel inhibition, with his sustained intrachannel latency at about -30 msec. and his transient intrachannel inhibition at about -90 msec.

The development of a model for metacontrast disinhibition. The hypothesis presented by Breitmeyer and Ganz (1976) that Type B metacontrast is the result of transient inhibition of sustained channel activity does not appear to be entirely correct in light of our finding of disinhibition under metacontrast. According to Breitmeyer and Ganz's theory when a second mask is added the transient activity of M2 should inhibit only the sustained activity of M1; the transient activity of M1 is unaffected, still causes inhibition of the target's sustained activity and hence masking of T. One should then be able to predict masking of T under the two mask case from consideration of independent

masking by M1 and M2. This was clearly not possible in the present study.

Again we are assuming that masking of M1 by M2 leads to disinhibition. There is, however, the possibility that masking of M2 by M1 yields disinhibition. The resultant reduction in M2 masking of T would then be the cause of P(T/M1+M2) so often being less than P(T/M1+M2)nd. Since under metacontrast conditions M1 preceded M2, masking of M2 by M1 would be a case of paracontrast. As this paracontrast could be caused by sustained intrachannel inhibition, an explanation of disinhibition entirely in terms of M2 masking by M1 would be consistent with Breitmeyer and Ganz (1976).

Consideration of Table 2, however, leads us to conclude that such an explanation can not totally account for disinhibition in the present study. First, consider the result of subject BB at an SOA1 of 60 msec. No masking by M2 occurred for this subject at this SOA in Experiment 3 (see P(T/M2)). We thus estimated that no masking occurred due to M2 in Experiment 4; thus, P(T/M1+M2)nd was based only on masking by M1. If disinhibition was caused by masking of M2 by M1 then P(T/M1+M2) should equal P(T/M1+M2)nd and P(T/M1) here. However, the actual degree of masking is much less than that predicted, and less than P(T/M1), indicating that masking of T by M1 has been severely reduced.

Next, comparing P(T/M1) and P(T/M1+M2) at metacontrast SOAls it can be seen that masking by M1+M2 is less than masking by M1 at M(0) for subjects MF and BB, at 30 msec. for subjects MF, BB, and AH, at 60 msec. for all subjects, and at 90 msec. for BB and AH. If we assume

that M2 is so totally masked by M1 that M2 does not contribute to masking of T, i.e., to P(T/M1+M2), then masking of T by M1 must be less when M2 is presented than when M2 is not presented at these SOAls. The assumption of any contribution of M2 masking to P(T/M1+M2) leads us to the same conclusion, i.e., M1 masking is reduced under M1+M2.[9]

Thus an explanation of disinhibition in terms of only masking of M2 seems implausible given the present data. We will therefore proceed with an explanation of disinhibition in terms of masking of M1 by M2. Previous disinhibition studies have also considered masking of M1 by M2 as the cause of disinhibition.

It seems likely that the metacontrast which occurred in this study resulted from the inhibition of sustained channels activated by the target stimuli. Given the U-shaped functions obtained it does not seem plausible that metacontrast resulted from the inhibition of transient channels activated by the target. As transient channels have a fast latency and a short persistence, inhibition of transient channels would yield a Type A function. The Type A function would result since beyond very short SOAs the target transient activity would be ended by the time any mask activity could cause inhibition. The assumption that metacontrast results from the inhibition of target sustained channels is consistent with Breitmeyer and Ganz (1976).

Theoretically target sustained inhibition could arise from either M1 sustained activity or M1 transient activity. However, our Type B metacontrast functions (Experiment 1) do not support M1 sustained activity inhibiting T sustained activity. As the mask and target

sustained channels have equal latencies Ml sustained inhibition of T sustained channels would be greatest at an SOA of 0 msec. In other words a Type A function would have occurred. However, since transient channels have a faster latency of response than sustained channels inhibition of T sustained channels would be greatest at an SOA greater than 0 msec., i.e., a Type B function would result (see above for a more detailed explanation). As the finding of Type B masking in the present study is consistent with inhibition of target sustained channels by mask transient channels, we will assume this type of inhibition occurred. We thus agree with Breitmeyer and Ganz (1976) as to the cause of T sustained inhibition under Type B metacontrast.

We will also assume, as did Breitmeyer and Ganz (1976), that the inhibition generated from transient channels far outlasts the initial transient activity caused by a stimulus. Metacontrast could only be due to transient inhibition of sustained channels if the transient generated inhibition was itself rather sustained. However, transient neurons have only a brief response to stimuli (Enroth-Cugell and Robson, 1966; Scobey and Horowitz, 1976). Breitmeyer and Ganz (1976) postulated that transient neurons generated tonic inhibition of sustained neurons via interneurons (Burke and Sefton, 1966a,b) whose response to transient excitation far outlasts the period of stimulation by transient neurons. There is recent evidence that such is, in fact, the case for certain interneurons of the cat LGN (Dubin and Cleland, 1977). Our model of disinhibition incorporates tonic interneuron inhibition as mediating transient inhibition of sustained channels. Hereafter, when using

the term "transient inhibition" we will be referring to the tonic interneuron inhibition generated by transient channels.

If the M1 transient activity inhibits target sustained activity then the occurrence of disinhibition indicates that M2 must somehow reduce the transient inhibition arising from M1. If the inhibition exerted by a neural channel is proportional to the activity of that channel (Hartline, Wagner, and Ratliff, 1956; Cornsweet, 1970; Bridgeman, 1971) then the inhibition of M1 transient channels will reduce the inhibition of T sustained channels. In terms of the sustained-transient dichotomy this reduction could result either from M2 sustained channels or M2 transient channels inhibiting M1 transient channels.

If we consider the latency difference between onsets of the transient and sustained channels activated by M2, it then appears more likely that the reduction in M1 transient inhibition is caused by M2 transient than M2 sustained activity. This conclusion is based on evidence that the onset of sustained channels follows onset of transient channels activated by a stimulus by 40 to 100 msec. (Dow, 1974; Ikeda and Wright, 1975b; Cleland, Levick, and Sanderson, 1973). If M2 sustained onset follows M2 transient onset by a similar time period, then M2 transient inhibition would have a greater period of time (than M2 sustained inhibition) in which to reduce M1 transient activity. The longer M1 transient activity is itself inhibited, the less chance M1 transient activity will have to inhibit T sustained activity. As M2 transient inhibition could provide up to 100 msec. more inhibition of

M1 than M2 sustained inhibition, it seems more likely that M2 transient inhibition is the cause of the reduction in M1 transient inhibition which yields disinhibition. It is possible that M2 sustained inhibition contributes to the reduction of M1 transient inhibition, but this contribution would be much less than that of M2 transient inhibition.

Admittedly, our entire argument is rather speculative as we have not yet discussed possible mechanisms for the inhibition of transient channels under metacontrast conditions. A recent study by Dubin and Cleland (1977) presents a possible mechanism for transient inhibition of transient channels. This study will be discussed later, as will evidence for sustained and transient inhibition under metacontrast conditions. Figure 16 diagrams the possible inhibitory interactions between stimuli in our model of disinhibition.

Consideration of the data of subject BB in terms of the relative latency of channel onsets for each stimulus also leads one to conclude that M2 transient inhibition of M1 is more plausible. In order to consider these relative latencies let us represent the latencies of transient onsets as t(TrN) and the latencies of sustained activity onsets as t(SuN), where N represents the appropriate stimulus--0 for the target, 1 for M1, and 2 for M2. The latencies of the onsets of all transient and sustained activity generated by the stimuli in Experiment 4 can then be represented by the equations:

$$t(Tr0) = t0$$

$$t(Su0) = t0 + t1$$

$$t(Tr1) = t0 + SOA1$$

$$t(Su1) = t0 + SOAL + t1$$

$$t(Tr2) = t0 + SOA1 + S)A2$$

$$t(Su2) = t0 + SOA1 + SOA2 + t1$$

where t0 represents the latency of onset of transient activity (assuming this is the same for all stimuli) and t1 represents the difference in latency between the activation of transient and sustained channels for the same stimulus (again assuming this difference is the same for all stimuli). As we are dealing only with metacontrast, SOA1s and SOA2s are always positive. Physiological studies also indicate that t1 is always positive, i.e., the sustained latency is longer (e.g., Cleland, Levick, and Sanderson, 1973; Ikeda and Wright, 1975a, b).

For subject BB strong disinhibition occurred at an SOA1 as long as 90 msec. with an SOA2 of 60 msec. We will substitute these time periods for SOA1 and SOA2 in the above equations. As t0 is a constant over all equations we need not be concerned with an estimation of this value. However, we do need an estimate of t1. The maximizing SOA for a subject can be used as this estimate. This is so as an SOA greater than 0 msec. provides for a delay of the onset of mask transient inhibition. When this delay is equivalent to the difference in latency between sustained and transient channels then mask transient onset coincides with target sustained onset. The simultaneous onset of

transient mask channels and sustained target channels should result in maximum masking as mask transient inhibition will then be concurrent with target sustained activity. Thus the SOA which yields maximum masking provides an estimate of the latency difference--t1. For subject BB the SOA yielding maximum masking of T by M1 was 60 msec. (see Table 2--P(T/M1)). Therefore, we will assume t1 equals 60 msec. for this subject. The equations for the relative latencies of transient and sustained channels under disinhibition at an SOA1 of 90 msec. are then:

$$t(Tr0) = t0$$

$$t(Su0) = t0+60 \text{ msec.}$$

$$t(Tr1) = t0+90 \text{ msec.}$$

$$t(Su1) = t0+90 \text{ msec.} +60 \text{ msec.}$$

$$t(Tr2) = t0+90 \text{ msec.} +60 \text{ msec.}$$

$$t(Su2) = t0+90 \text{ msec.} +60 \text{ msec.} +60 \text{ msec.}$$

If M2 sustained (Su2) inhibition of M1 transient activity (Tr1) is the cause of disinhibition then Tr1 has had 120 msec. in which to inhibit target sustained activity (Su0) before Tr1 is itself inhibited. Cleland, Levick, and Sanderson (1973) estimated that the response persistence of sustained channels in a cat to a short stimulus (2 msec.) is about 100 msec. If this is also true of human physiology then Tr1 inhibition should have completely suppressed Su0 activity over 120 msec. If the target sustained activity is completely suppressed by the time M2 is presented then no disinhibition could occur.

On the other hand consider M2 transient (Tr2) inhibition of M1 transient channels. Here Tr1 has had only 60 msec. in which to inhibit SuO before Tr1 was itself inhibited. If Tr1 inhibition is reduced at this point this would leave about 40 msec. of inhibition-reduced or inhibition-free processing time for target sustained channels. During this time the probability of seeing the target would be increased, i.e., disinhibition would occur.

Our general argument against sustained inhibition of M1 transient activity as the cause of disinhibition is based on the prolonged length of time allowed for target inhibition under the assumption of sustained inhibition of M1. Such an argument is based on logical and not empirical grounds. Turning to the metacontrast literature there is evidence for the involvement of both transient and sustained inhibition in Type B masking. However, the evidence for transient inhibition seems to be stronger.

First consider the consistent finding of weaker masking with foveal stimuli (Alpern, 1953; Kolers and Rosner, 1960; Stewart and Purcell 1970; Barry and Dick, 1971). As the relative frequency of sustained neurons has been found to decrease with increasing eccentricity from the fovea (Fukada, 1971; Hoffman, Stone, and Sherman, 1972), stronger peripheral masking does not support the involvement of sustained inhibition. Stronger peripheral masking, on the other hand, supports the involvement of transient inhibition as the relative frequency of transient cells increases with increasing foveal eccentricity (Fukada, 1971; Hoffman, Stone, and Sherman, 1972).

The finding that a mask offset can produce masking (Holzworth and Doherty, 1971; Turvey, Michaels, and Kewley-Port, 1974) is consistent with transient mediated inhibition as transient neurons respond to light offset as well as onset (Enroth-Cugell and Robson, 1966). Bowen, Pokorny, and Cacciato (1977) found that Type B masking occurred only if the mask was a luminance transient (i.e., an increase in luminance over the pre-mask field). With a mask of no transient luminance (the mask hue was changed from that of the pre-adapting field) no Type B masking occurred. As transient cells have been associated with achromatic channels and sustained cells with chromatic opponent-color channels (Ingling and Drum, 1973), Bowen et al., concluded that sustained channel inhibition was not involved in Type B masking.

Growney's (1976) report that blurring a mask (i.e., removing the high frequency components) does not decrease masking is inconsistent with sustained mediated inhibition. The finding of contour suppression under stroboscopic motion (Breitmeyer, Wepman, and Love, 1976) supports the involvement of transient neurons in masking as these respond more readily to motion.

Sherrick, Keating, and Dember (1974) found that a black target is masked equally well by a white ring as by a black ring. Obviously the contrast of the mask relative to the target was not critical. This can be accounted for if one assumes transient inhibition, as transient responses do not appear to be contrast specific whereas sustained activity does appear to be contrast specific (Enroth-Cugell and Robson, 1966).

There are, however, findings in the metacontrast literature which seem to support the involvement of sustained inhibition in metacontrast. First, it has been found that increasing the mask complexity increases the degree of metacontrast (Schiller and Smith, 1965; Johnson and McClellend, 1973). As increased complexity increases the high frequency components of the mask, the sustained activity generated by the mask is, most likely, increased (Kulikowski and Tolhurst, 1973). Secondly, Breitmeyer (1977b) in a recent paper presented at the Psychonomic Society Meeting reports disinhibition when the second mask was two flanking bars which were on continuously as the target and mask were flashed briefly (8 msec.). Breitmeyer assumes that the second mask activates only sustained channels while the target and first mask activate both sustained and transient channels. As masking is U-shaped Breitmeyer concludes that this disinhibition indicates the involvement of sustained inhibition of transient channels. However, as mask two was on before mask one (and stayed on continuously during and after the display of mask one and target) we may actually have here a case of paracontrast disinhibition or more correctly paracontrast disinhibition of metacontrast. It seems possible that the disinhibitory effect on mask one may be caused by sustained intrachannel inhibition (one of the two types of intrachannel inhibition occurring in our paracontrast disinhibition model, see above) and not necessarily by sustained inhibition of transient channels as Breitmeyer infers.

The findings of White and Lorber (1976) also appear to support the involvement of sustained inhibition in Type B masking. White and

Lorber (1976) found that as increasingly lower mask frequencies were presented following a 6 or 12 c/deg target, masking decreased rather monotonically. If transient inhibition was responsible for Type B masking then masking should have been greater at lower spatial frequencies as transient channels respond readily in this range (see discussion above). Masking for the 12 c/deg target was greatest when the mask was also 12 c/deg. At this spatial frequency sustained channels respond readily and transient channels respond little if at all (Kulikowski and Tolhurst, 1973; Legge, 1978). These findings would seem to indicate that sustained inhibition is involved in Type B masking. Kitterle[10], however, has criticized this study as only one SOA--100 msec.--was used. Thus, we can not be sure that White and Lorber (1976) were producing Type B masking. Type A masking may have been produced, even though this seems improbable given the long SOA.

Thus, the evidence appears to support the involvement of both transient and sustained inhibition in Type B masking. There does, however, appear to be more evidence supporting transient in inhibition. This could possibly reflect a weaker inhibitory effect for sustained channels in Type B masking, i.e., both transient and sustained inhibition may be involved but the predominate cause of masking is transient inhibition.

Unfortunately none of the studies just reviewed directly address the question of transient vs. sustained inhibition of <u>transient</u> channels. It seems doubtful whether any metacontrast studies do directly address this question as such studies either 1) demand a response that

is based on sustained activity, for example in studies dealing with contour suppression (Breitmeyer, Wepman, and Love, 1976) or pattern recognition (White and Lorber, 1976), or 2) demand a response in which it is unclear whether transient or sustained activity is used, for example brightness discrimination (Alpern, 1953). Metacontrast studies seem only to be able to show the relative involvement of transient and sustained inhibition without specifying how each is related to inhibition of transient channels.

Consideration of recent research by Dubin and Cleland (1977) also supports the occurrence of transient inhibition and may provide a mechanism for our hypothesized transient inhibition of transient channels. These researchers, recording from the LGN of the cat, found evidence for the existence of two classes of interneurons--intrageniculate and perigeniculate. The following properties were found for intrageniculate interneurons: 1) they received direct excitation from a small number of ganglion cells, 2) a ganglion cell could simultaneoulsy excite an intrageniculate interneuron (IG interneuron) and an LGN relay cell (a cell extending from the LGN to the visual cortex, 3) they were innervated by either sustained or transient ganglion cells (but never was one IG interneuron innervated by both types), 4) their receptive field was of the on-center or off-center type, and 5) transient cell excitation of an IG interneuron produced only a transient response.

The perigniculate interneurons were found to have the following properties: 1) they received inputs from recurrent collaterals of relay cell axons, 2) they received binocular input, 3) only transient

cell innervation of perigeniculate (PG) interneurons could be found, 4) they responded well to rapidly moving stimuli across their RF, 5) they responded equally well to black or white stimuli, 6) their RF size was about 5 degrees--much larger than IG interneurons RFs, and 7) a transient excitatory response generated a tonic PG interneuron response. The evidence indicated that both PG and IG interneurons inhibited LGN relay cells.

Considering the evidence supplied by this study, the perigeniculate interneurons seem to be the likely candidate to supply the tonic inhibition necessary for transient inhibition of sustained channels as hypothesized by Breitmeyer and Ganz (1976). The RFs of PG interneurons have the same properties of transient neurons--response to fast movement, equal response to on or off, large RF size--are innervated by only transient neurons, and generate extended inhibition to a brief stimulation. Furthermore they are binocularly driven, which is consistent with dichoptic metacontrast (Kolers and Rosner, 1960) and dichoptic disinhibition (Robinson, 1968).

The PG interneurons could also mediate disinhibition effects. If the PG interneuron activity generated by the first mask (PG1), while it is inhibiting sustained target activity, is itself inhibited by the PG interneuron activity generated by the second mask, then this could release the target sustained activity from inhibition. It is also possible that transient activity generated by M2 directly inhibits the PG interneuron activity of M1 thus causing disinhibition. However, due to the phasic persistence of transient neurons and the tonic persistence of the PG interneurons this latter possibility seems remote.

Our model of disinhibition under Type B metacontrast incorporates the basic concepts of the PG interneuron (see Figure 16). The interneurons in this model (I1 for the interneuron activity generated by M1, and I2 for the interneuron activity generated by M2) are innervated by the transient channels and deliver tonic inhibition to the sustained channels. Again sustained channels are represented by SuN and transient channels by TrN, where N is replaced by 0 for the target, 1 for mask one, and 2 for mask two.

In Figure 16 the large arrow to the right of the Su0 channel represents the output of the activity of this channel. The greater this output the greater is the probability of detecting the target. When only T is presented at a supra-threshold level this output will be very great and T will usually be detected. However, when M1 is presented following T this output is reduced. This follows as the transient activity of M1 (Tr1) excites a set of interneurons (I1) which produce tonic inhibition of Su0. As the output of Su0 is reduced, T is detected less often, i.e., masking occurs.

The presentation of M2 generates transient activity (Tr2) which excites a second set of interneurons (I2). This second set of interneurons inhibits the activity of the first set of interneurons. This reduction of I1 activity yields a corresponding reduction in the inhibition of Su0. As the inhibition of Su0 is reduced the output of Su0 is increased and T will be more easily detected than when only M1 is shown.[11]

Although it is not shown in this model we feel that the Su2 channel may also play a role in disinhibition. Given our previous line

of reasoning concerning this channel we believe this role is rather minor. At any rate, the involvement of Su2 may also occur via interneurons, i.e., Su2 activity innervates a set of interneurons which inhibit I1.

Summary

The occurrence of disinhibition under metacontrast conditions in the present study can not be accounted for strictly in terms of the Breitmeyer and Ganz (1976) theory that transient channels inhibit sustained channels in Type B masking. However, a modification of this theory which incorporates the use of interneurons mediating both transient-sustained inhibition and transient-transient inhibition can account for the present findings. We concluded that the major cause of disinhibition is due to inhibition generated by M2 transient channels which reduces the inhibition generated by M1 transient channels. A minor role in disinhibition may also be played by inhibition arising from M2 sustained channels. Our model of metacontrast disinhibition assumes that: a) disinhibition occurs due to inhibition of some M1 activity, b) masking in the present study was the result of inhibition of sustained target channels, c) the inhibition of sustained target channels is accomplished via inhibition generated by M1 transient channels, and d) inhibition generated by transient channels far outlasts the initial transient excitation.

Our model of paracontrast disinhibition is generally consistent with the inhibitory interactions under paracontrast proposed by

Breitmeyer and Ganz (1976). We have, however, concluded that transient intrachannel inhibition may occur in paracontrast disinhibition and, most likely, under paracontrast.

Finally, we did not find any evidence of orientation-specificity. Thus we could not test the assumption made by Breitmeyer and Ganz (1976) that sustained channels are more orientation-specific. Our lack of orientation-specific masking may represent the interaction of inhibition due to channels tuned to the same orientation with inhibition due to channels tuned to different orientations.

FOOTNOTES

[1]The differences in latencies reported can most likely be attributed to differences in experimental design. As has been explained, different measures--e.g., beginning of response vs. maximum response--are used to assess latency. Also the stimuli vary between experiments both in type--dot of light, bar of light, flashed repeatedly, flashed once, etc.--and in intensity.

[2]The figures reported here for the latency differences were calculated by this writer based on Figure 3 of Fiorentini and Maffei (1970). It should also be noted that at 3 cps in this figure this writer can discern no appreciable difference in modulation threshold between the various phase angles.

[3]Maffei and Fiorentini (1973) found that simple cells responded to a moving grating by a modulation of their discharge frequency. In other words, the increase in frequency is not constant, it rises a number of times from pre-stimulus discharge to a peak discharge rate in a sinusoidal fashion. Complex cells were found to respond to a moving grating by an overall constant increase of their discharge rate, i.e., no modulation occurred.

[4]Although the visibility of the mask may also be reduced (Kolers and Rosner, 1960) this reduction is not great and perhaps as a consequence has not received much attention in metacontrast studies.

[5]SOA1 is the SOA between onset of the target stimulus and the first mask. SOA2 is the SOA between onsets of the first and second masks.

[6]Clarity ratings are not reported beyond this point as they yielded results which essentially replicated those reported via P(E).

[7]All probabilities of error reported in this study where calculated using the equation for P(E). For P(M1), however, we were concerned with the probabilities of a hit and a false alarm to the M1 stimulus.

[8]In calculating these t-tests, the differences between P(T/M1+M2)nd and P(T/M1+M2) at each SOA1 was computed for each subject. The mean of these differences and the standard error of these differences was then calculated for each subject. These two terms were then used to calculate a t-ratio for each subject.

[9]Via a z-test for proportions, P(T/M1) was significantly greater than P(T/M1+M2) at the following SOAls for the following subjects:

a) -30 msec. for subject BB ($z = 1.97$, $p<.03$),

b) P(0) for subject BB ($z = 1.78$, $p<.04$),

c) 30 msec. for subject BB ($z = 1.84$, $p<.04$),

d) 60 msec. for subjects MF ($z = 2.23$, $p<.02$), BB ($z = 3.86$, $p<.001$), and AH ($z = 2.09$, $p<.02$),

e) 90 msec. for subjects BB ($z = 2.20$, $p<.02$), and AH ($z = 1.68$, $p<.05$).

[10]Fred Kitterle, personal communication, March 1977.

[11]This model does not consider the masking of T by M2 which occurred in the present study. As we felt that the major component of disinhibition concerned the reduction in M1 inhibition of T, we did not wish to further complicate this model by the addition of M2 inhibition. Such inhibition of T could be represented by an arrow from Tr2 to a set of interneurons which then inhibit SuO.

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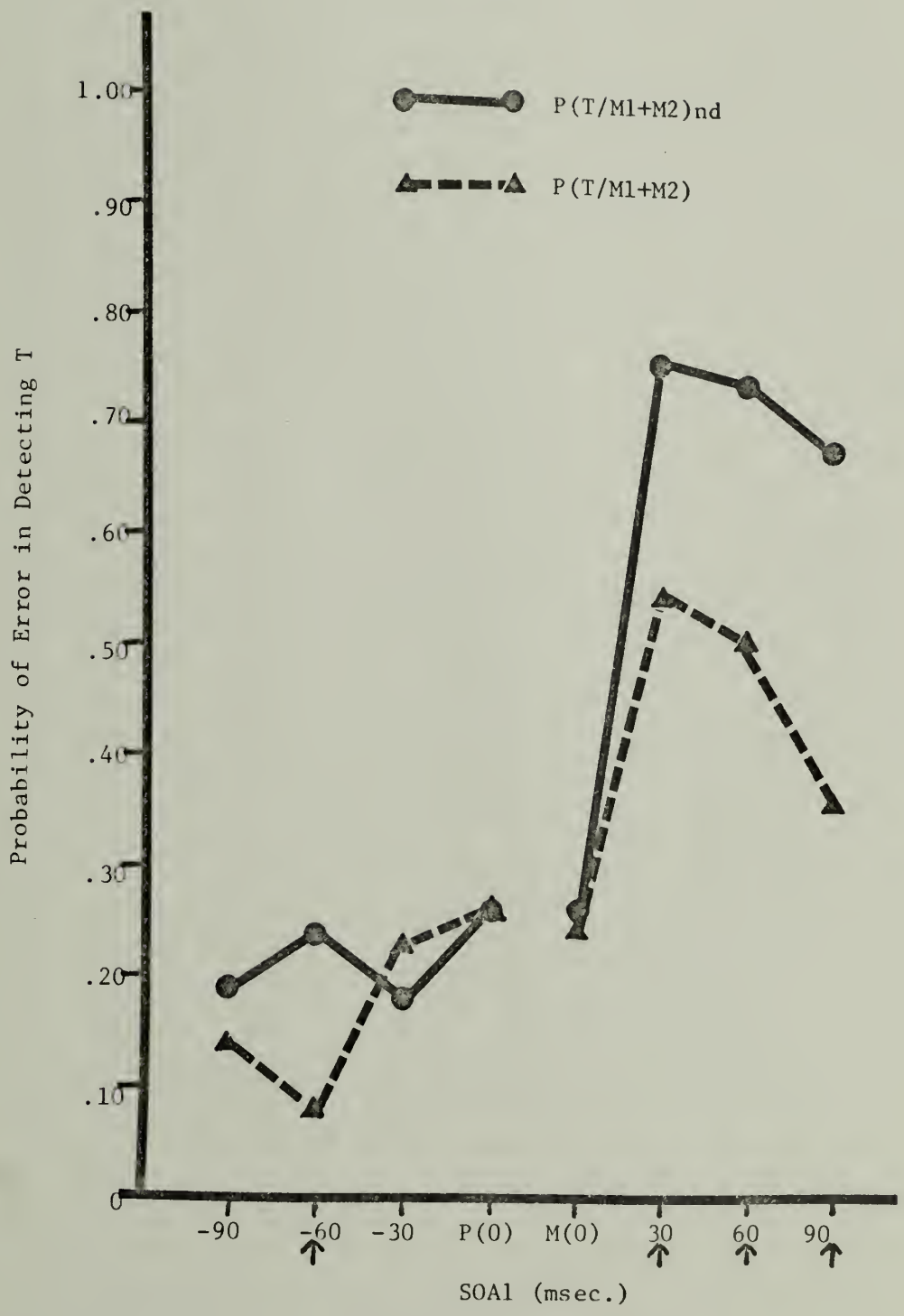


FIGURE 11. Subject AH

TABLE 1

PROBABILITY OF ERROR IN DETECTING T + M1
(EXPERIMENTS 1, 2, 3, AND 5)

Subject	Probability	SOA (msec.) -120	-90	-60	-30	0	30	60	90	120	150
MF	P(T/M1,E1)		.10	.15	.25	.18	.45	.40	.25		
	P(T/M1,E5)		.10	.15	.50	.26	.74	.47	.25		
	P(T/M1)		.10	.15	.38	.22	.59	.44	.25		
	P(T/M2)	.05	.10	.15	.05	---	.05	.10	.10	.00	
	P(M1)		.00	.05	.05*	.05	.25*	.15	.05		
JD	P(T/M1,E1)		.29	.28	.16	.06	.33	.50	.38		
	P(T/M1,E5)		.20	.00	.26	.35	.26	.30	.15		
	P(T/M1)		.24	.13	.21	.21	.30	.39	.19		
	P(T/M2)	.05	.30	.10	.25	---	.30	.05	.25	.25	
	P(M1)		.67	.59	.71*	.28	.67*	.22	.37		
BB	P(T/M1,E1)		.00	.05	.25	.25	.55	.75	.78		
	P(T/M1,E5)		.20	.10	.20	.20	.25	.30	.15		
	P(T/M1)		.10	.08	.22	.22	.40	.52	.45		
	P(T/M2)	.00	.10	.00	.05	---	---	.25	.20	.00	.05
	P(M1)		.00	.05	.10*	.00	.20	.25*	.18		
AH	P(T/M1,E1)		.05	.05	.17	.11	.71	.86	.71		
	p(T/M1,E5)		.05	.25	.10	.06	.56	.56	.32		
	P(T/M1)		.05	.15	.14	.08	.64	.71	.52		
	P(T/M2)	.15	.10	.05	.20	---	.20	.32	.11	.32	
	P(M1)		.10	.25	.53*	.26	.59*	.29	.22		

*P(M1) for SOA2s used in Experiment 4

TABLE 2

ESTIMATES OF THE PROBABILITY OF ERROR IN DETECTING T AND ACTUAL PROBABILITY OF ERROR IN DETECTING T (EXPERIMENT 4)

Subject	Probability	SOA1 msec. -90	-60	-30	P(0)	M(0)	30	60	90
MF	P(T/M1)	.10	.15	.38	.22	.22	.59	.44	.25
	P(T/M2)	.05	.10	.15	.05	.05	.10	.10	.00
	P(T/M1+M2)nd	.14	.24	.46	.26	.26	.63	.50	.25
	P(T/M1+M2)	.12	.15	.26	.25	.18	.49	.24	.34
JD	P(T/M1)	.24	.13	.21	.21	.21	.30	.39	.19
	P(T/M2)	.05	.30	.10	.25	.30	.05	.25	.25
	P(T/M1+M2)nd	.28	.39	.29	.39	.45	.34	.54	.39
	P(T/M1+M2)	.18	.18	.27	.18	.24	.33	.31	.29
BB	P(T/M1)	.10	.08	.22	.22	.22	.40	.52	.45
	P(T/M2)	.00	.10	.00	.05	.25	.20	.00	.05
	P(T/M1+M2)nd	.12	.17	.22	.26	.41	.52	.52	.48
	P(T/M1+M2)	.10	.10	.09	.10	.14	.24	.18	.25
AH	P(T/M1)	.05	.15	.14	.08	.08	.64	.71	.52
	P(T/M2)	.15	.10	.05	.20	.20	.32	.11	.32
	P(T/M1+M2)nd	.19	.24	.18	.26	.26	.76	.74	.68
	P(T/M1+M2)	.14	.08	.23	.26	.24	.55	.51	.36

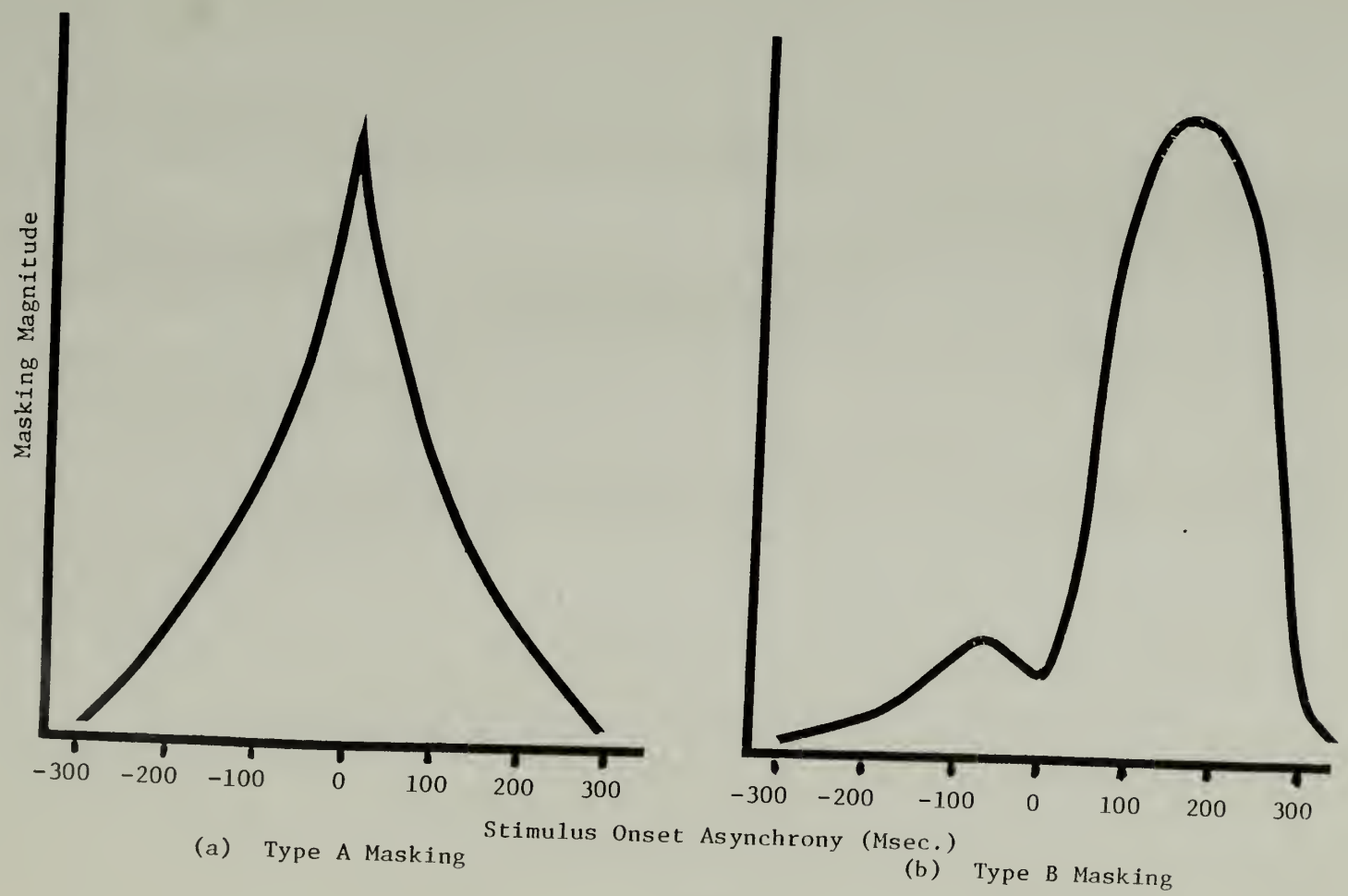

Masking Magnitude
-300
-200
-100
0
100
200
300
(a) Type A Masking
-300
-200
-100
0
100
200
300
Stimulus Onset Asynchrony (Msec.)
(b) Type B Masking

FIGURE 1

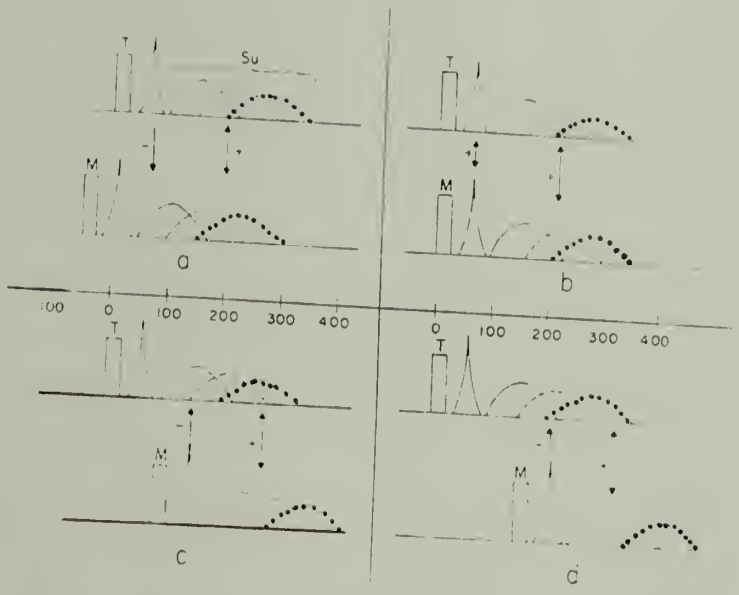


FIGURE 2. This figure illustrates the response latency, persistence and inhibitory interactions of the sustained and transient channels activated by a target and mask stimulus. The letter representations are: M -mask stimulus, T- target stimulus, and Su- sustained channels. Transient channel activity is represented by the spike. Arrows indicate the direction of inhibition. (a) mask precedes target, no inhibition of target, (b) mask and target presented simultaneously, again no inhibition of target, (c) mask follows target at relatively short SOA, inhibition of intermediate sustained spatial-frequency channels of the target, (d) mask follows target at longer SOA, inhibition of high sustained spatial-frequency channels of the target. In the Su curves the solid line curve represents intermediate spatial-frequency channels, the dashed line represents high spatial-frequency channels, and the dotted line represents very high spatial-frequency channels (from Breitmeyer and Ganz, 1976).

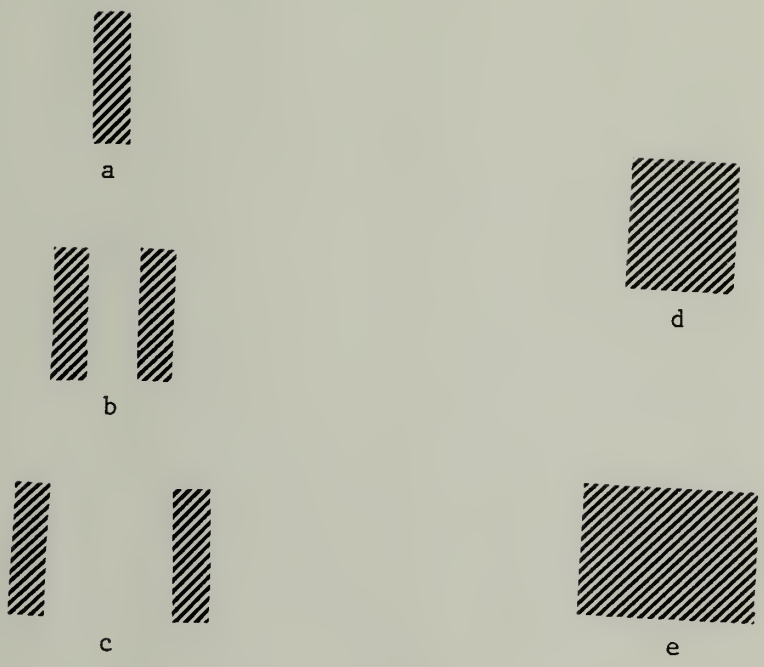


FIGURE 3. An example of target (a), Mask one (b) and Mask two (c) showing the relative horizontal distance between each. All gratings shown here are 45°. In Experiment one only the target and Mask one were presented--(d) gives an example of target grating and a 45° Mask one at an SOA of 0 msec. In Experiment two target, Mask one, and Mask two were displayed--(e) shows an example of the target grating, a 45° Mask one grating, and a 45° Mask two grating displayed concurrently.

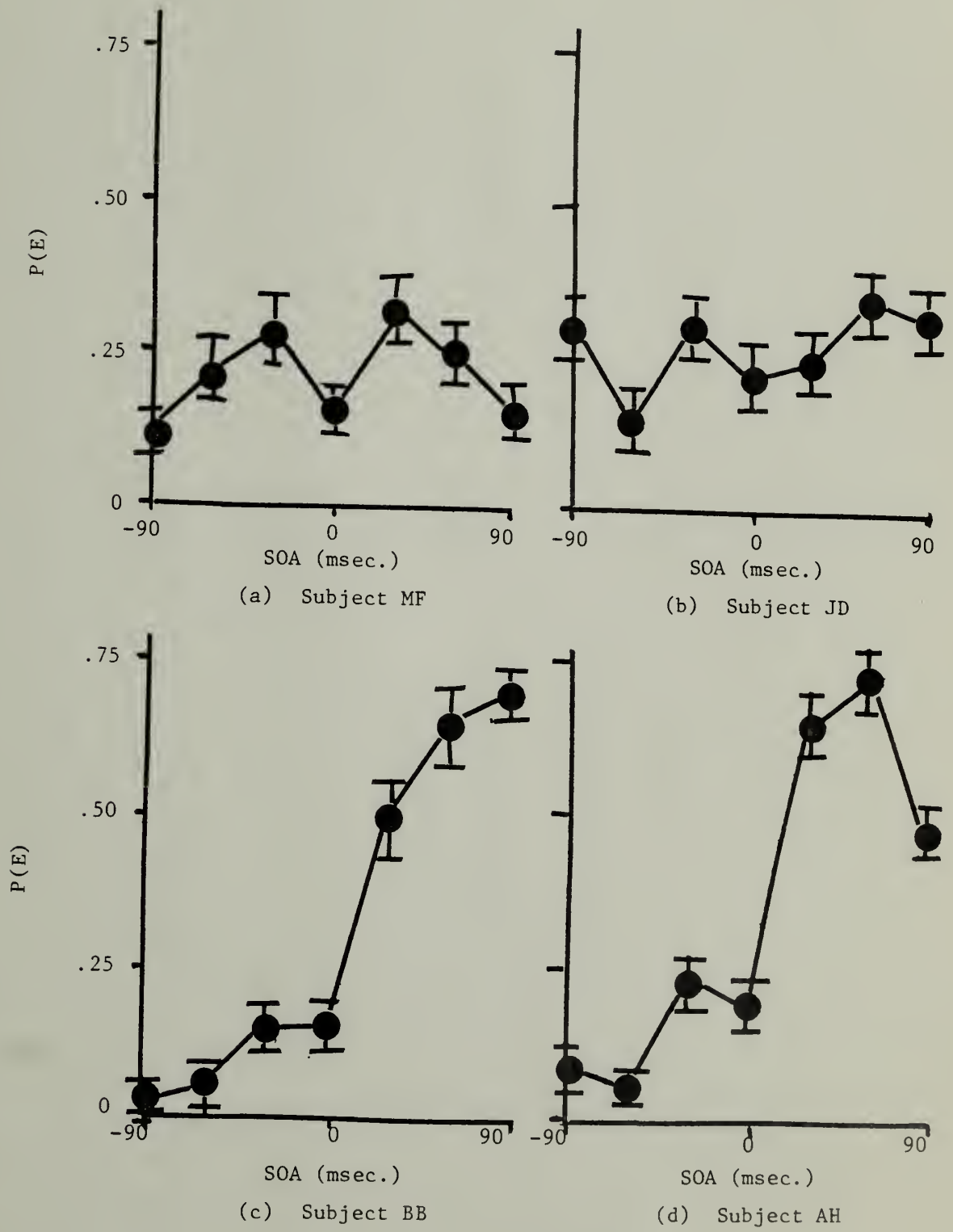

.75
.50
.25
0
P(E)
-90
0
90
SOA (msec.)
(a) Subject MF
(b) Subject JD
(c) Subject BB
(d) Subject AH

FIGURE 4

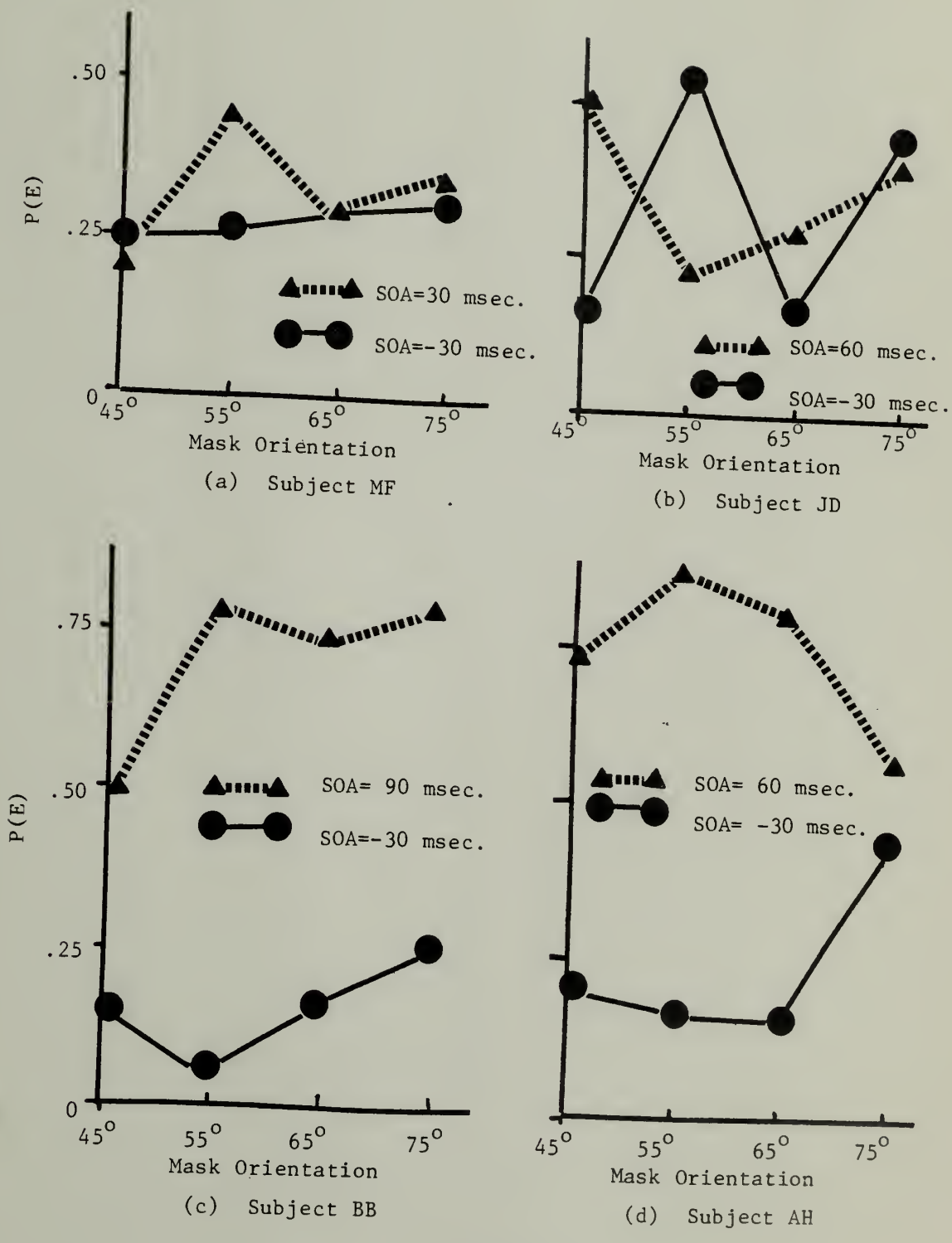

.50
.25
0
P(E)
SOA=30 msec.
SOA=-30 msec.
45°
55°
65°
75°
Mask Orientation
(a) Subject MF
SOA=60 msec.
SOA=-30 msec.
45°
55°
65°
75°
Mask Orientation
(b) Subject JD
.75
.50
.25
0
P(E)
SOA= 90 msec.
SOA=-30 msec.
45°
55°
65°
75°
Mask Orientation
(c) Subject BB
SOA= 60 msec.
SOA= -30 msec.
45°
55°
65°
75°
Mask Orientation
(d) Subject AH

FIGURE 5

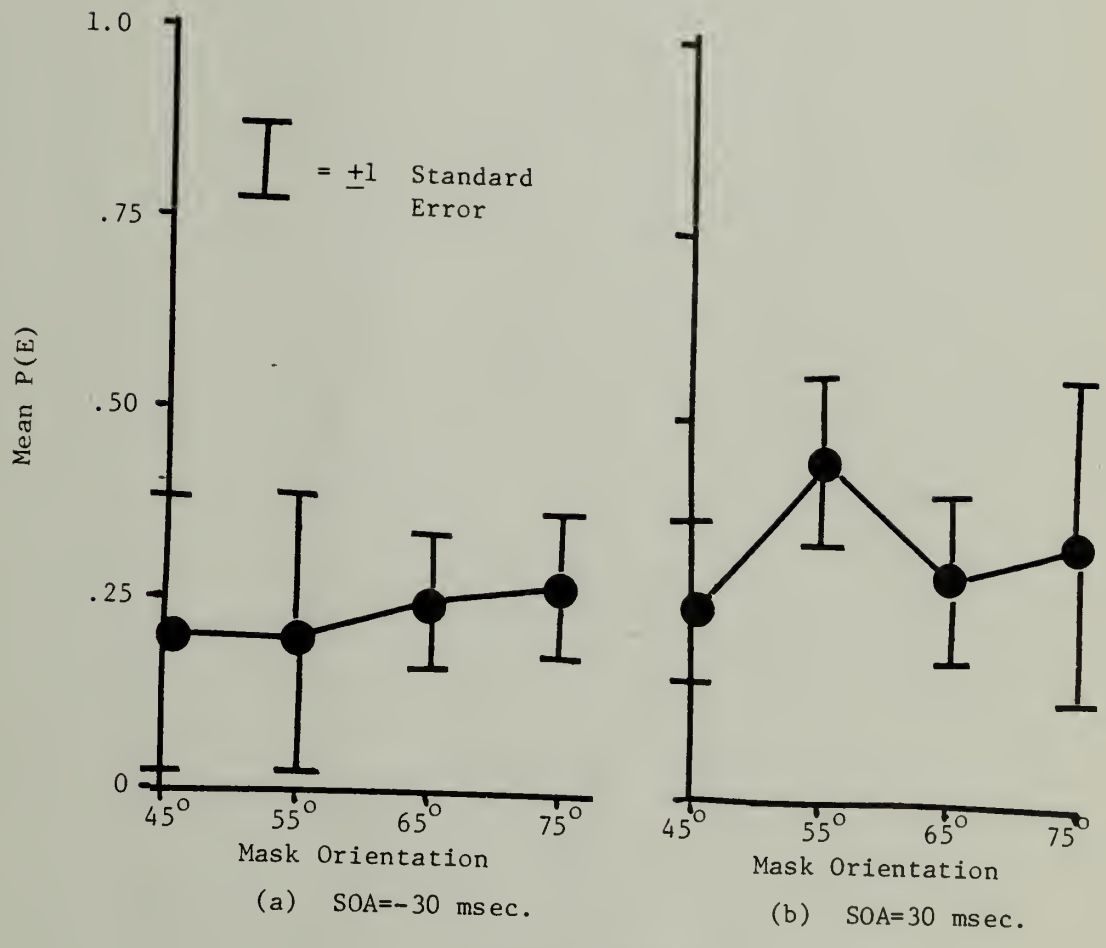
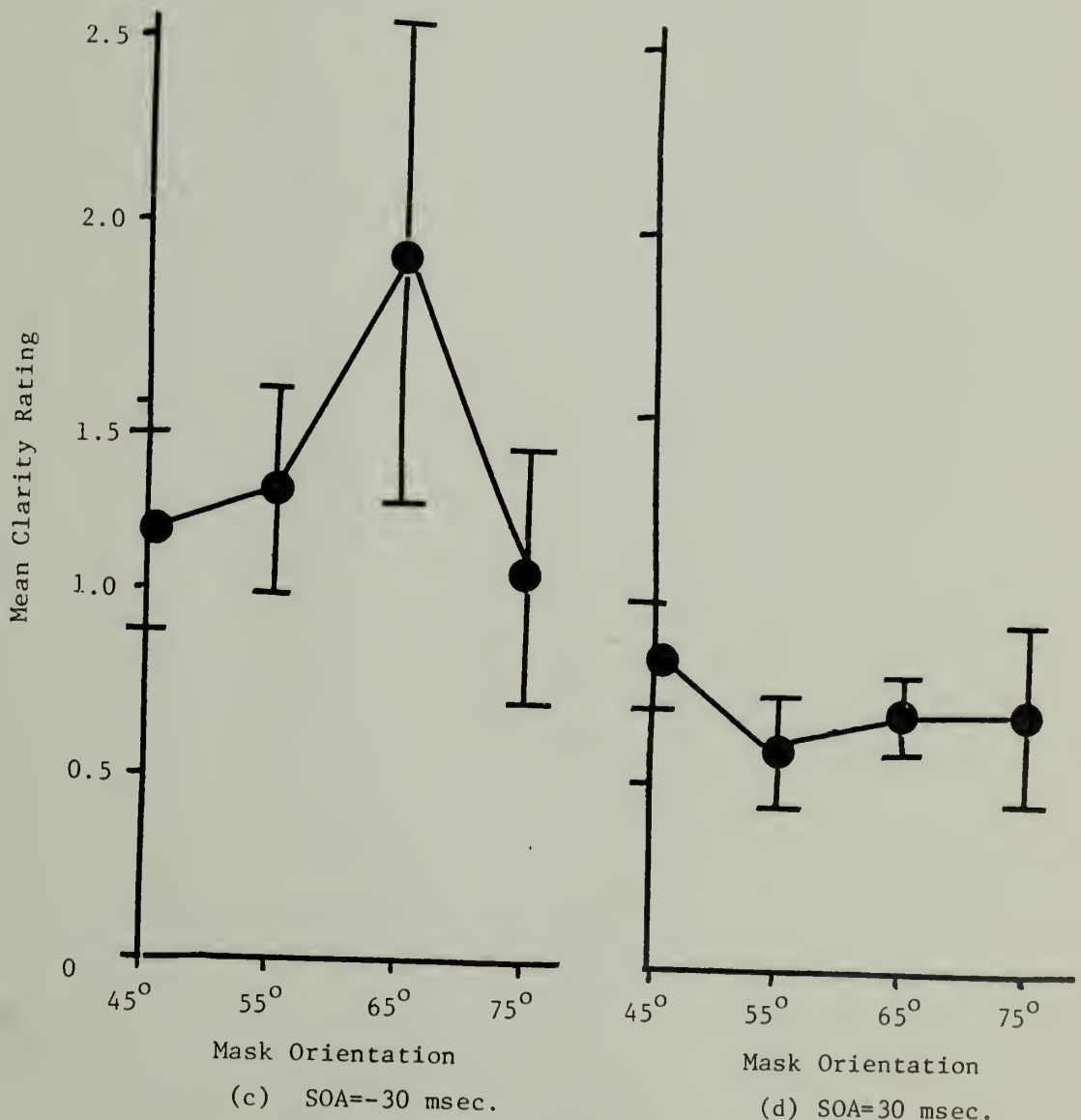


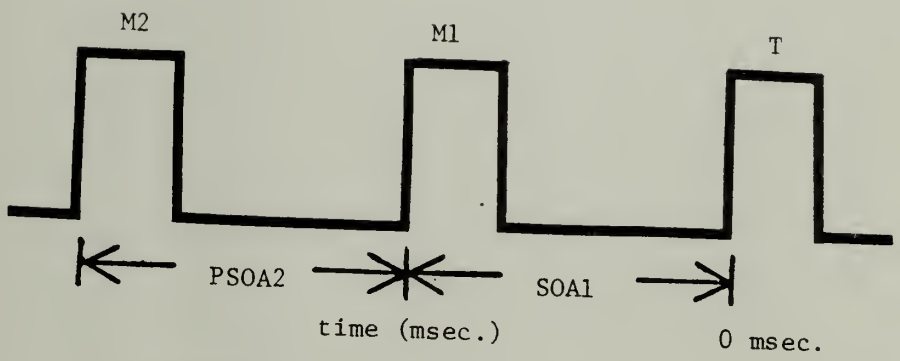
FIGURE 6. Subject MF



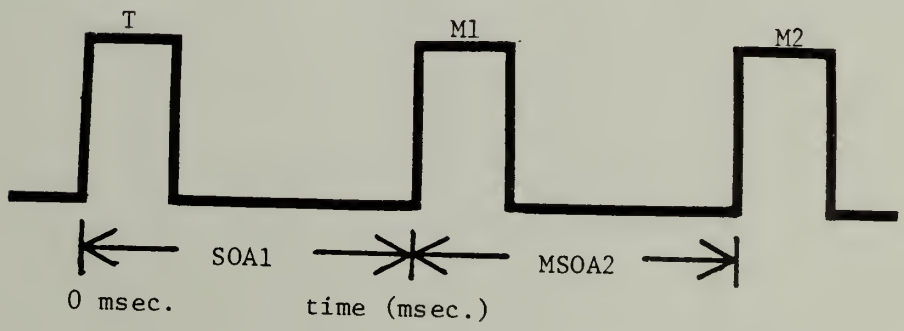
(c) SOA=-30 msec.

(d) SOA=30 msec.

FIGURE 6 (Cont'd) Subject MF



(a) Presentation of M2 with paracontrast SOAls.



(b) Presentation of M2 with metacontrast SOAls.

FIGURE 7

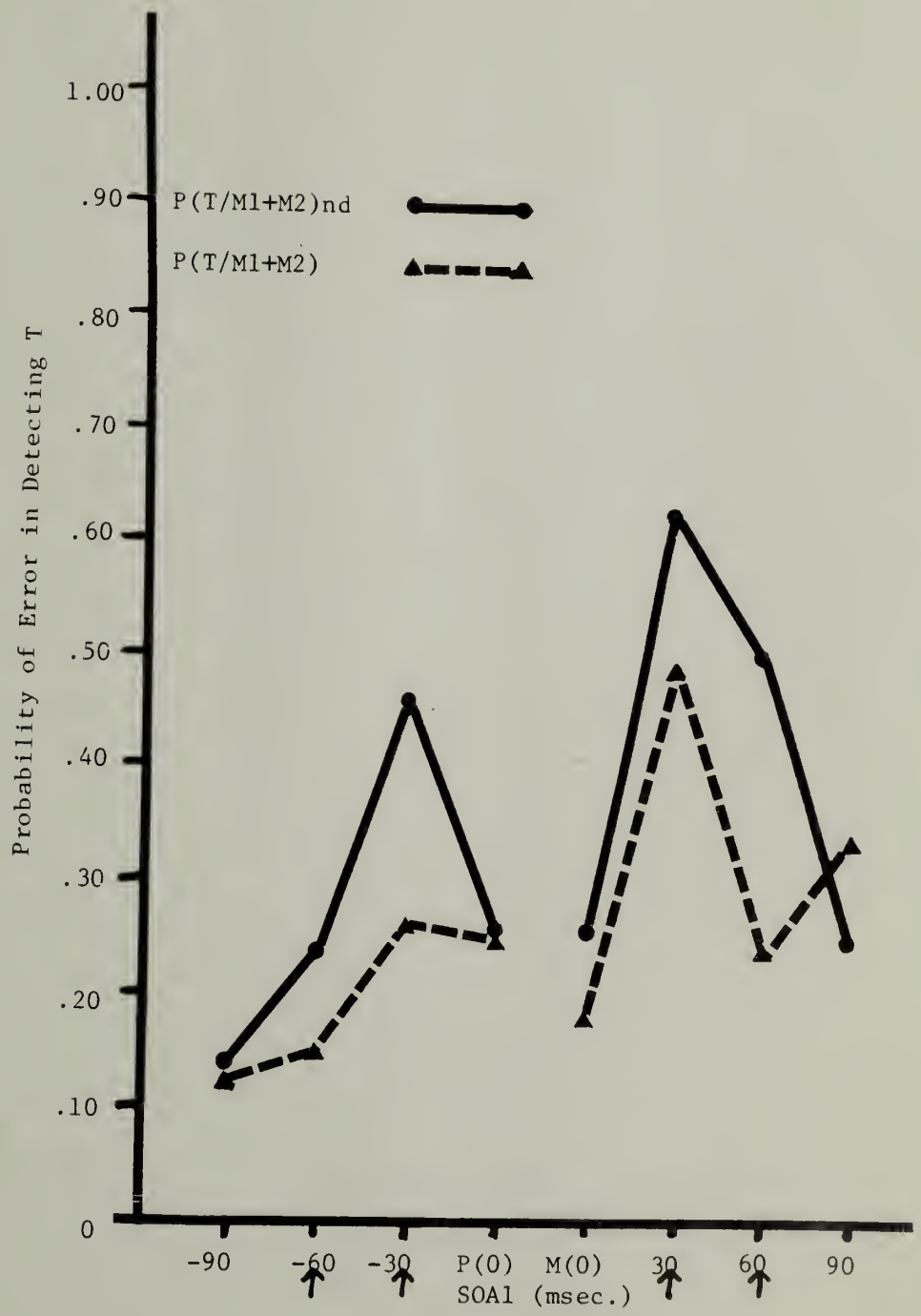


FIGURE 8. Subject MF

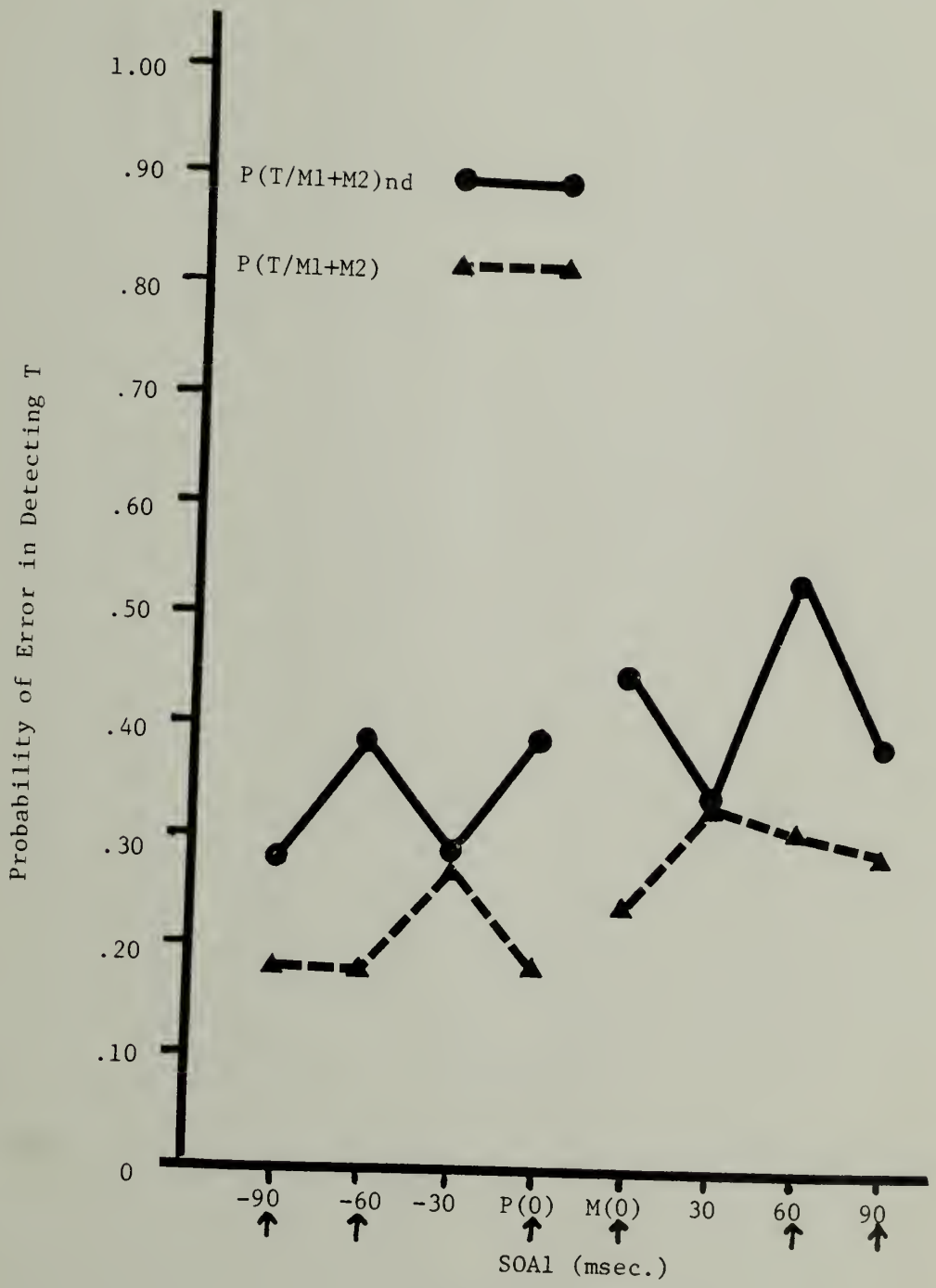


FIGURE 9. Subject JD

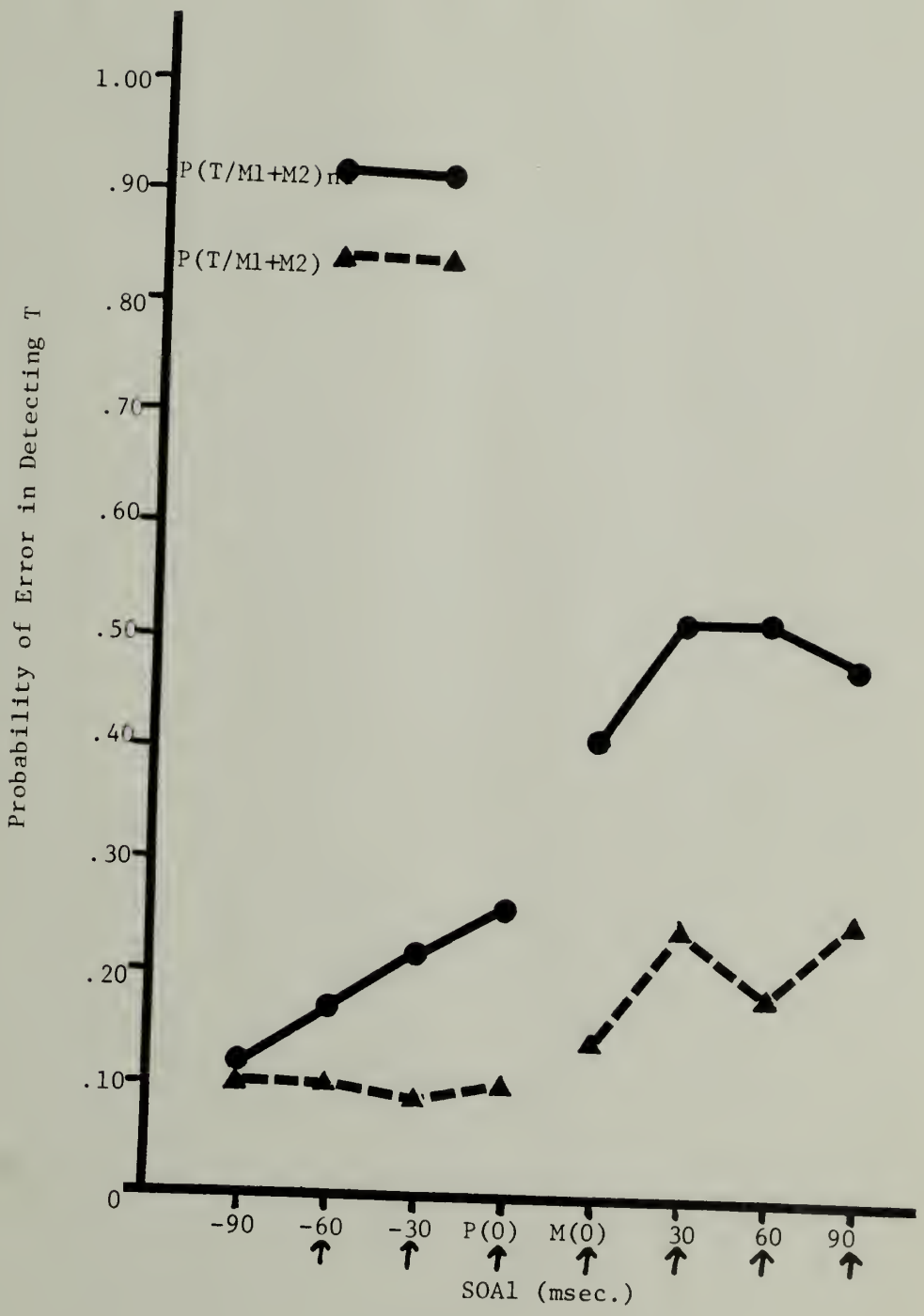


FIGURE 10. Subject BB

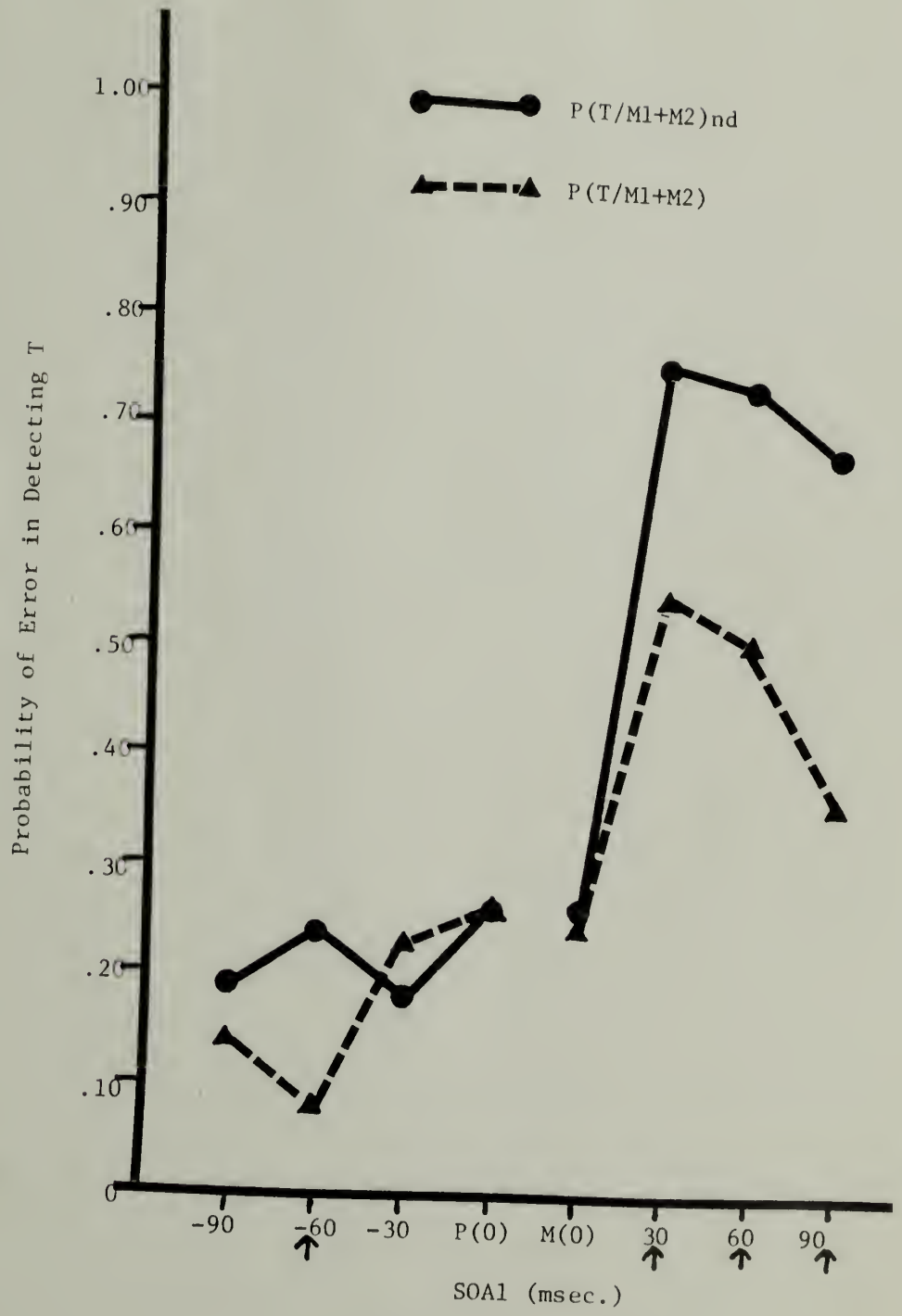


FIGURE 11. Subject AH

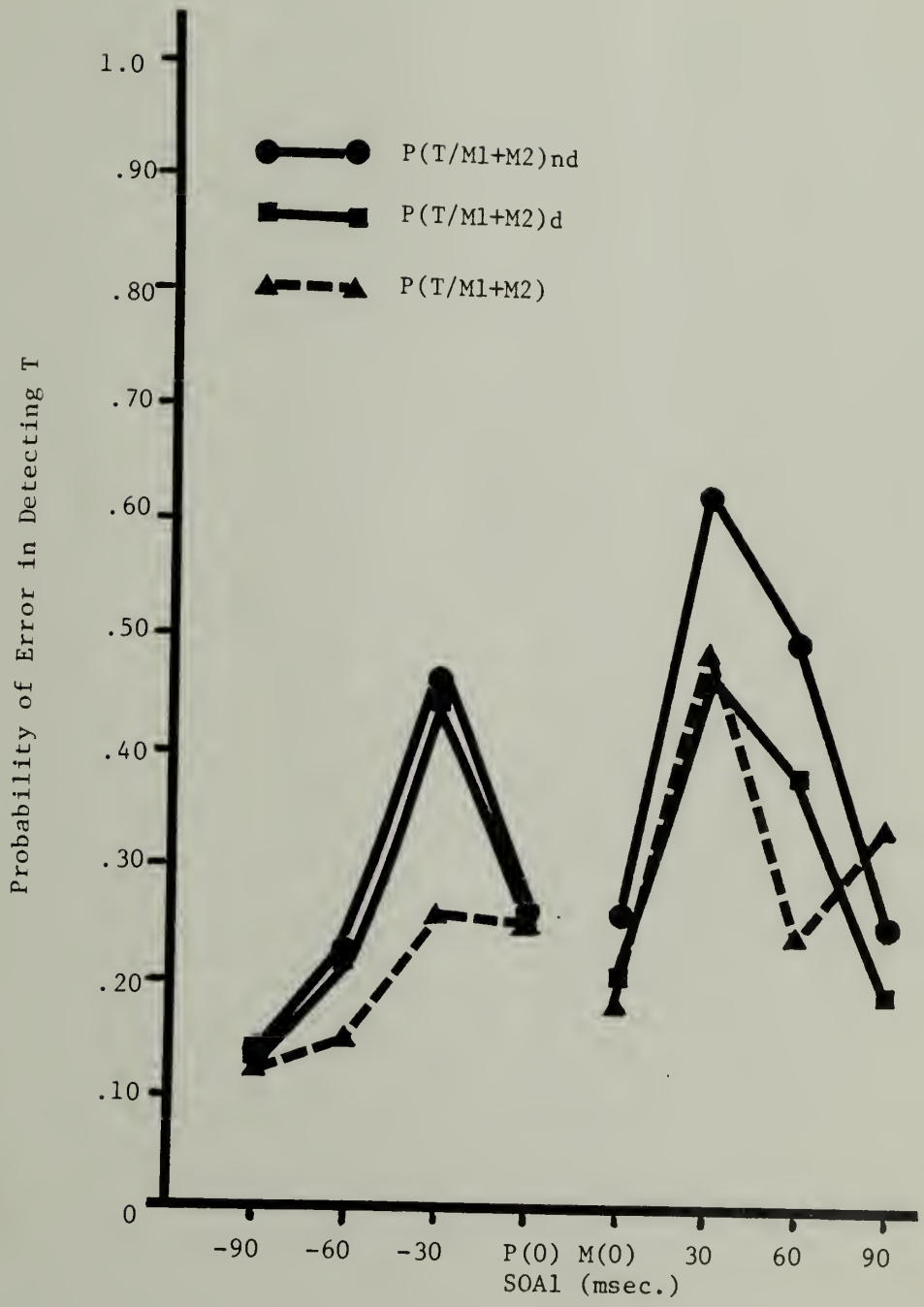


FIGURE 12. Subject MF

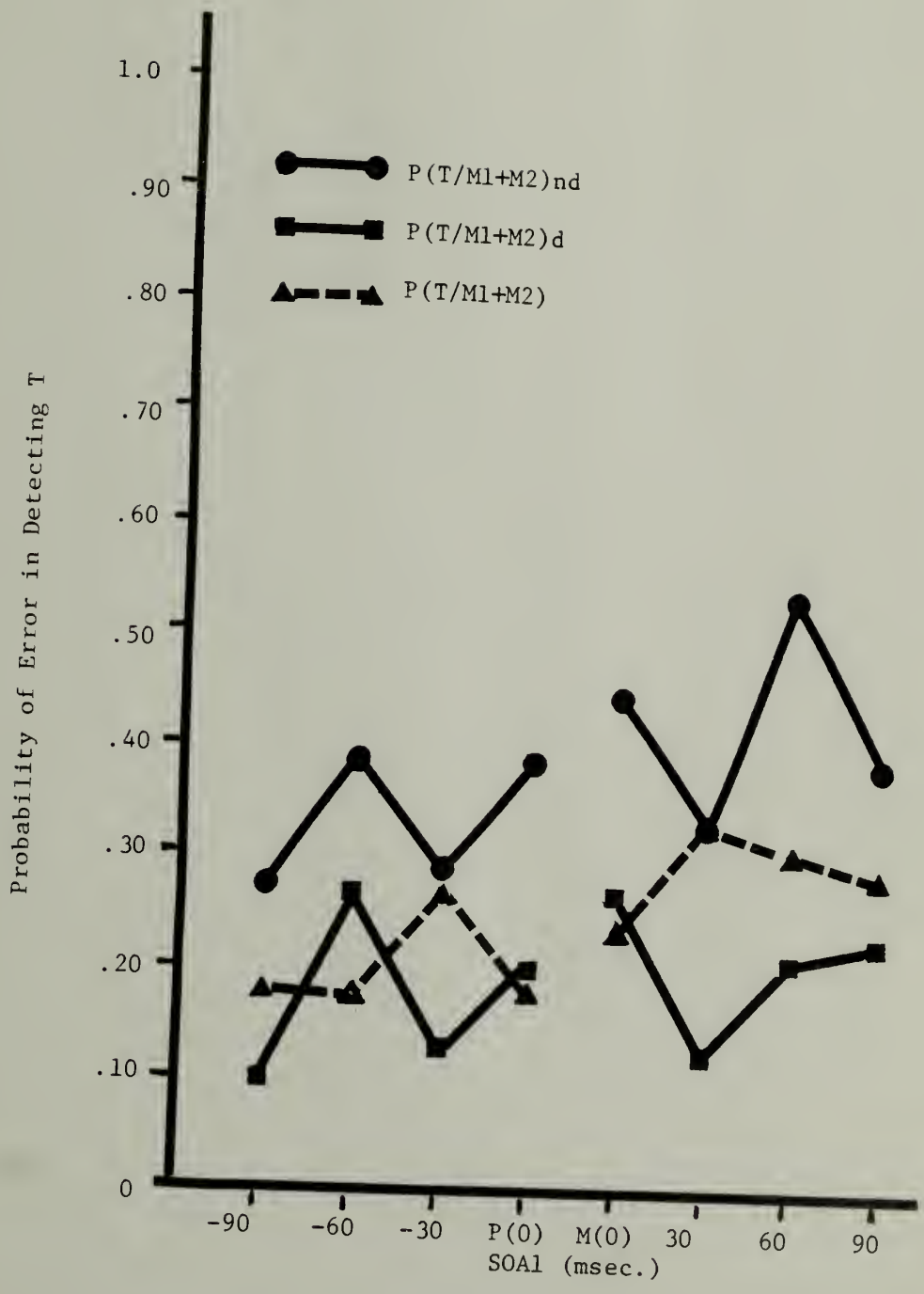


FIGURE 13. Subject JD

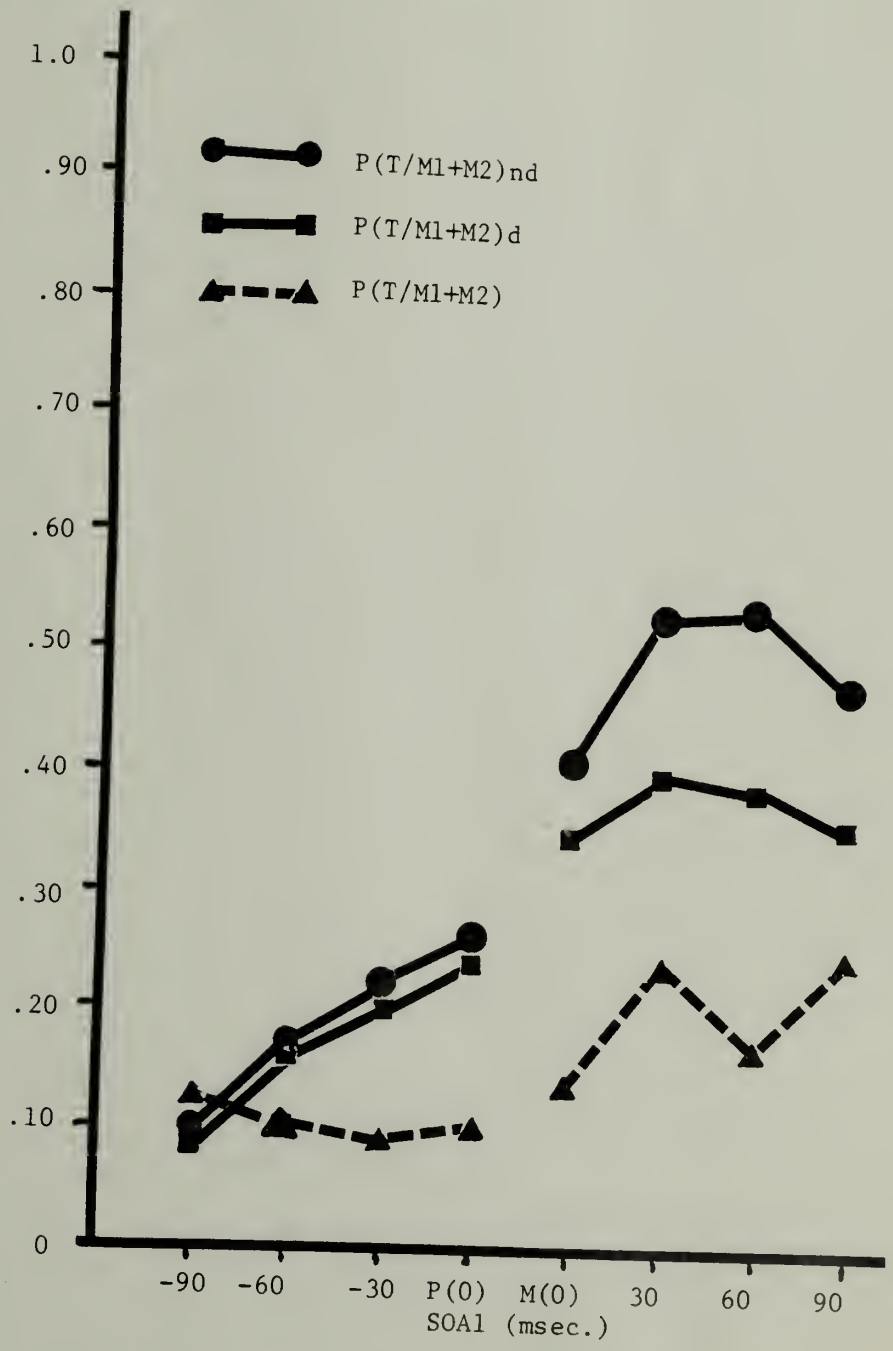


FIGURE 14. Subject BB

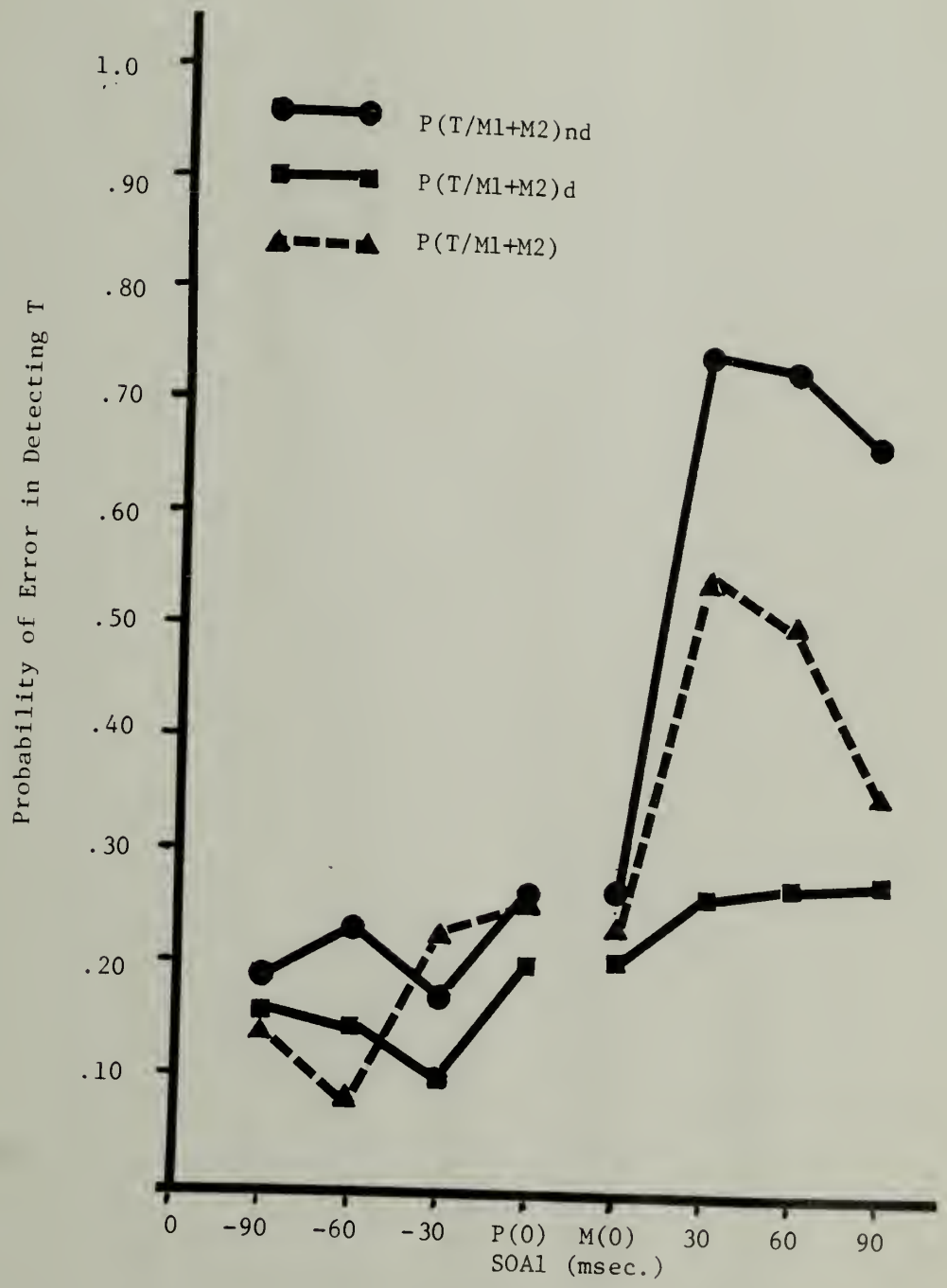


FIGURE 15. Subject AH

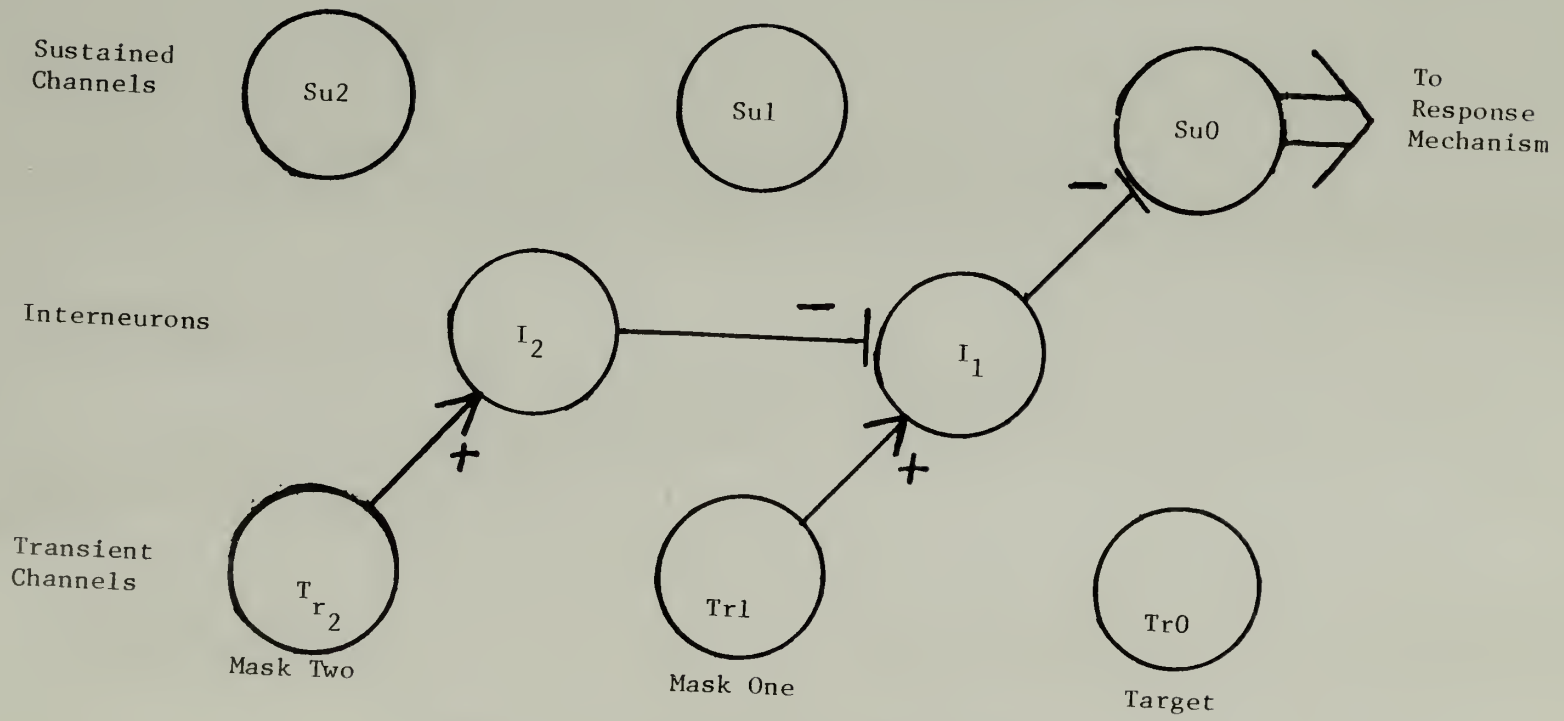


FIGURE 16. Metacontrast Disinhibition Model

APPENDIX I

Instructions for the Duration Procedure

You are going to be shown a series of trials to determine your rate of detection of a square-wave grating as the duration of this grating is varied. This is what the square-wave grating will look like (E turns on channel one)--a series of slanted lines. We will refer to this as the target grating from now on. On each trial either this target grating or this blank field will appear (E advances slide tray to present a mean luminance slide). This we will refer to as the target blank. Your task is to respond "yes" whenever you see the target grating and "no" whenever you do not see the target grating on a trial. Sometimes you will respond "no" because a target blank was shown and sometimes you will respond "no" when a target grating was shown but you did not see it. It does not matter which was the case, a "no" response simply means you did not see a target grating on that trial. In order to respond "yes" you should feel that you have seen the target grating with some degree of certainty. In other words if you feel that on a particular trial a "yes" response would be the guess, then you should respond no. Furthermore, we would like you to give a rating of how clear a target grating appeared on each trial that you respond yes. To do this after each yes response we would like you to respond with either the number one, two, or three. Respond "one" if the target grating appeared to be dim or not very clear; respond "two" if the target grating appeared to be fairly clear; respond "three" if the target grating appeared to be very clear. So you will be giving four possible responses; "yes-one" if the target grating appeared to be dim, "yes-two" if the target grating appeared to be fairly clear, "yes-three" if the target grating appeared to be very clear, or "no" if you did not see the grating or feel that you would be guessing. I will say "ready" before the presentation of each trial. This should serve as a warning to you to gaze at the dot and to pay attention. In fact you should try to gaze at the dot at all times. You will receive a five minute rest after you have completed 70 trials and another rest after the next 65 trials. However, if you feel that your eyes are straining or that you are very fatigued at any time please tell me and we will rest at that time. Do you have any questions? If not, please gaze at the circle for two minutes to become used to this light level.

APPENDIX II

Instructions For Practice Block and Experiment 1

As before center your gaze on the dot. On the next series of trials beside seeing a target grating or blank you will see a grating appearing on either side of the central rectangle (E turns on channels one and two). However, your task is as before to respond to the central rectangle--the target. On each trial you will make one of the four responses which you used previously. Respond "yes-one" if the target grating appeared to be dim or not very clear, respond "yes-two" if the target grating appeared to be fairly clear, respond "yes-three" if the target grating appeared to be very clear, and respond "no" if you did not see the target grating or feel that you would be guessing. Remember that you are to make your responses based on the central rectangle, not the two side rectangles. All of the rectangles will be flashed very briefly and at different rates so your task may seem difficult at times. Just do the best you can. As before I will say "ready" before each presentation and you should make sure that you are gazing at the dot. Today the 56 trials which you will receive will be practice trials and will not count in your results. This will conclude your participation in this session. For the next five sessions you will receive 224 trials of this sort per session. Do you have any questions? If not please gaze at the circle for two minutes to become used to this light level.

APPENDIX III

Instructions for Experiment 2

Once again gaze at the dot in the center of the field. This study is a little different than the previous ones. There will be no central rectangle displayed in the following trials. You will see two rectangles (E turns on channel two) each flanked on the outside by another rectangle (E turns on channel three). The two innermost rectangles will either be gratings as you see now or blanks (E adjusts the slide tray of channel two) as you now see. Your task is to respond to these innermost rectangles--a "no" response if you did not see a grating in the innermost rectangles, and either a "yes-one", a "yes-two" or a "yes-three" response if you did see a grating. Respond "yes-one" if the gratings in the innermost rectangles appeared dim or unclear, respond "yes-two" if the gratings appeared fairly clear, respond "yes-three" if the gratings in the innermost rectangles appeared to be very clear. Or respond "no" if you did not see gratings in the innermost rectangles or if you feel that you would be guessing. I will again say "ready" before the presentation of each trial; this should serve as a warning to you to gaze at the dot and to pay attention. You will first receive 14 practice trials. Then you will receive 70 experimental trials at a time with a five minute rest after each 70 trials. You will do four such blocks of 70 trials. Again please gaze at the circle for two minutes to become used to this light level.

APPENDIX IV

Instructions for Experiment 3

Again please gaze at the dot. In this study, as before, either a target grating or target blank will appear in the center (E turns on field one). Also two gratings will be presented one on either side of the target and some distance away from the target (E turns on channel three to show M2). Your task is again to give one of the four responses to the target rectangle. You are to respond yes only if you feel that you have seen the target with some degree of certainty. Furthermore, please rate each yes response as before: "yes-one" if the target grating appeared dim, "yes-two" if the target grating appeared to be fairly clear, and "yes-three" if the target grating appeared to be very clear. Respond "no" if you did not see the target grating or feel that you would be guessing. As before I will say "ready" before the presentation of each trial. This will serve as a warning to you to gaze at the dot and pay attention. You will first receive 14 practice trials. Then you will be presented with 70 experimental trials at a time with a 5 minute rest after each 70 trials. You will do four blocks of 70 trials. Again please gaze at the circle for two minutes to become used to this light level.

APPENDIX V

Instructions for Experiment 4

Please gaze at the dot once again. In this study you will see a central rectangle of either a grating or a blank (E turns on field one), two rectangular gratings which flank this central rectangle (E turns on field two), and two rectangular gratings which flank these last two rectangles (E turns on field three). Your task is again to respond to only the central rectangle--the target. On each trial you will make one of the four responses you have used previously. Respond "yes-one" if you saw the grating in the central rectangle but it appeared dim, respond "yes-two" if this grating appeared fairly clear, respond "yes-three" if this grating appeared to be very clear, and respond "no" if you did not see a grating or feel that you would be guessing. Remember that you are to make your response based on the central rectangle and not any of the other rectangles. All of the rectangles will be flashed very briefly and at various rates so your task may seem difficult at times. Just do the best you can. Again I will say "ready" before each presentation. This will serve as a warning to make sure that you are looking at the dot and paying attention. You will first receive eighty practice trials. After these practice trials you will receive four blocks of eighty trials each, with a five minute rest after each block. Then for the next four days you will do four blocks of trials per day. Please gaze at the circle to become used to this light level.

APPENDIX VI

Instructions for Experiment 5

Please gaze at the dot again. In this study you will be shown the central rectangle either grating or blank (E turns on field one) and the two gratings directly flanking the central rectangle (E turns on field two). Your task is again to make one of the four responses to the central rectangle. Respond "yes-one" if the target grating appeared to be dim or not very clear, respond "yes-two" if the target grating appeared to be fairly clear, respond "yes-three" if the target grating appeared to be very clear, and respond "no" if you did not see a grating or feel that you would be guessing. As before all of the rectangles will be flashed briefly and at various rates so your task may sometimes seem difficult. Just do the best you can. First you will receive 35 practice trials. Then after a five minute rest we will start the experimental trials. These will be in blocks of seventy trials and you will have a five minute rest at the end of each block. You will do four of these blocks of seventy and this will conclude your participation in this experiment. Please gaze at the circle for two minutes to become used to this light level.

APPENDIX VII

Derivation of formula for P(E)

We first assumed that the probability of detecting T could be represented by:

$$P(D) = \frac{P(H) - P(FA)}{1 - P(FA)}$$

where P(D) is the probability of detecting T, P(H) is the probability of a yes response to the presentation of T, i.e., the probability of a hit, and P(FA) is the probability of a false alarm. This equation is presented in Coombs, Dawes, and Tversky (1970), p. 187.

Now assuming that the probability of an error is simply 1.0 minus the probability of detection, P(E) = 1-P(D), and substituting for P(D) we obtain;

$$P(E) = 1 - \frac{P(H) - P(FA)}{1 - P(FA)}$$

We then simplify the equation as follows:

$$P(E) = \frac{1-P(FA) - (P(H) - P(FA))}{1-P(FA)}$$

$$P(E) = \frac{1-P(H)}{1 - P(FA)}$$