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Notes and Comment

Visual evoked potentials: Concomitants of metacontrast in late components

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Metacontrast is the masking of a briefly flashed target by a subsequent mask that shares common or nearby contours with the target. It is of interest because the effect seems to work backward in time, with a later mask obscuring an earlier target. The delay in masking suggests that the internal representation of a stimulus (the target) remains vulnerable for a time to interference in the nervous system. Thus, metacontrast provides a psychophysical tool for the dissection of temporal aspects of visual coding.

In addition to psychophysical studies, the visual evoked potential (VEP) is a source of information used to differentiate the metacontrast theories currently proposed. The VEP has shown promise in revealing where and when in the brain metacontrast interactions occur. A recent paper on electrophysiological correlates of metacontrast showed no VEP correlates of metacontrast in a "C1" component that reflected activity of striate cortex (Jeffreys & Musselwhite, 1986). However, while C1 is an early potential that reflects afferent activity, extensive evidence, much of it published in *Perception & Psychophysics*, has shown metacontrast to be a complex phenomenon that is influenced by repetition, practice, binocularity, and other effects to be expected at a higher level (Schiller & Greenfield, 1969; Schiller & Smith, 1968; Ventura, 1980; Weisstein, 1971). Thus, we would expect correlates of metacontrast in later VEP components, where higher level influences can be seen. This paper will show that although correlates of metacontrast are indeed present in the Jeffreys and Musselwhite data, they occur in later components, where other studies would lead us to expect them.

Several studies have found a suppression of later components in the VEP that matches psychophysical U-shaped metacontrast functions, even while early components are unaffected (Andreassi, DeSimone, & Mellers, 1975; Vaughan & Silverstein, 1968). Jeffreys and Musselwhite (1986) added to this literature by using electrode placements and stimulus locations that isolated responses of either striate or extrastriate visual cortex (Jeffreys, 1971; Jeffreys & Axford, 1972); the components are termed C1 and C2, respectively. The method is based on the different anatomical orientations of dipoles in circumscribed topographic regions of striate and extrastriate cortex.

Using this technique, Jeffreys and Musselwhite (1986) reported "no discernible modification of either of the two initial VEP components C1 and C2" (p. 631) under stimulus conditions in which they found U-shaped metacontrast masking functions psychophysically. Indeed, their early, prominent C1 and C2 components showed no changes with metacontrast. However, they did not analyze the later components of their VEPs, where others have found correlations with psychophysical metacontrast functions. Because the C1 and C2 potentials are more precisely localized than those in previous studies of metacontrast-related potentials, it is useful to extend the search for VEP correlates of metacontrast to the later components in this study. This paper will show that metacontrast-related components do exist in Jeffreys and Musselwhite's VEP data following the C1 component.

Because Jeffreys and Musselwhite (1986) published VEPs out to a poststimulus latency of 300 msec (their Figure 2) and 350 msec (their Figure 4), some of the late components can be measured directly from their published data. Late components of the visual evoked response were measured, in the present analysis, for the target-alone condition, for target-mask stimulus onset asynchronies (SOAs) of 20-100 msec with a multielement target (from their Figure 2), and for SOAs of 10-110 msec with a single-element target and a four-element mask (from their Figure 4).

To restrict the data to those obtainable from the published figures, the dependent variable used was the instantaneous amplitude of the average VEP in microvolts at a latency of 250 msec after target onset. This latency falls within the range of the component found by Vaughan and Silverstein (1968) to covary with psychophysical metacontrast (about 160-260 msec), and also falls within the range of poststimulus latencies where Bridgeman (1975, 1980) found metacontrast-like functions in the activities of single neurons in cat and monkey striate cortex, respectively. If metacontrast correlates were to be found anywhere, they should be found at about this latency.

The choice of dependent measure was arbitrary, but not critical. An inspection of the published VEPs shows that similar results can be obtained by sampling VEP amplitude at other latencies near 250 msec, or by integrating the area under the VEP curve between 250 and 300 msec. VEP amplitudes were measured directly from enlargements of the published figures.

Because Jeffreys and Musselwhite (1986) gave evoked potentials for their C2 (extrastriate) component at only two SOAs—70 and 100 msec—it is impossible to generate similar metacontrast curves in extrastriate cortex. Both

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of the published latencies fall near the broad peak of the psychophysical masking functions, so that the shapes of electrophysiological concomitants of the psychophysical masking curves cannot be determined. The published data, though, made it seem unlikely that any such correspondence would be found.

Experiment 1

Figure 1 shows the change in amplitude of the VEP for targets and masks varying from 10 to 100 msec SOA. The VEP was sampled for Subject H.D., with a multielement target and mask. The target consisted of three rows of four identical squares, each 24′ arc square and separated by 36′ arc. The mask had four rows of identical squares, so that each target square was flanked on both sides by a mask square (Jeffreys & Musselwhite, 1986, Figure 1A).

The late components, although small in amplitude, show a regular decrease with target-mask SOA, reflecting the initial flank of a U-shaped metacontrast curve. Because the longest SOA reported is 100 msec, and psychophysical curves for other subjects under these conditions continue to show strong metacontrast at this latency (Jeffreys & Musselwhite, 1986, Figure 1C), the expected upward-sloping leg of the function at longer SOAs is beyond the range of the available data. The decrease in evoked potential at earlier SOAs, however, corresponds fairly well with the psychophysically determined functions, to the degree that psychophysical magnitude estimations can be compared with VEP amplitudes. Unfortunately, there is no subject for whom both psychophysical and VEP data are available.

Experiment 2

Figure 2 shows the electrophysiological concomitants of metacontrast for Subjects H.D. and M.J.M. for a small one-element metacontrast target and a four-element mask. Here a wider range of SOAs is available, and the corresponding psychophysical functions peak before 100 msec SOA (Jeffreys & Musselwhite, 1986, Figure 3), so that both flanks of the U-shaped masking curve are visible in the electrophysiological results.

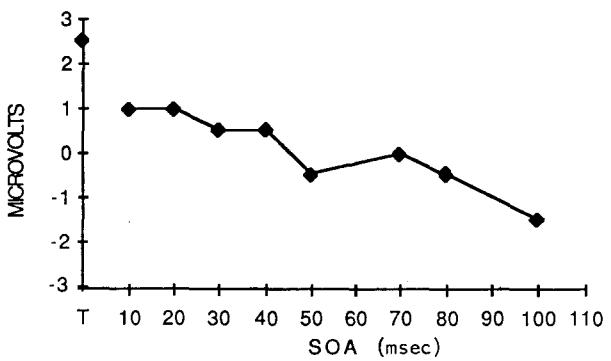

Figure 1. Amplitude of VEP for Subject H.D. at 250 msec after target onset for multielement target and mask. T = target alone.

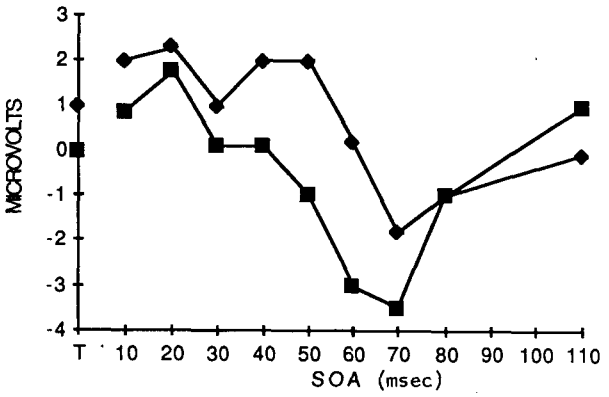

Figure 2. Amplitude of VEP for 2 subjects at 250 msec after target onset, for one-element target with four-element mask. Diamonds = Subject H.D. Squares = Subject M.J.M. T = target alone.

The metacontrast-related components are again small in amplitude, but they show reliable changes that correspond to the psychophysical masking functions for the same stimuli. The U-shaped metacontrast curves are probably narrower than the curve in Experiment 1 because of the smaller size of the stimulus. Subject M.J.M., the only one for whom both electrophysiological and psychophysical data are available, shows a peak of masking at about 70 msec SOA with both psychophysical and VEP measures.

Discussion

Far from contradicting the earlier studies of evoked potentials in metacontrast, the Jeffreys and Musselwhite (1986) VEPs showed no effects of metacontrast masking at short latencies, and clear effects at longer latencies. Thus they confirmed the gross evoked potential results of others (Andreassi et al., 1975; Vaughan & Silverstein, 1968). The present results are also consistent with the finding that single-cell correlates of metacontrast are found only in the late activity of neurons in striate cortex (Bridgeman, 1975, 1980). Single-cell data (Bridgeman, 1980) in the monkey showed changes in the firing of most striate cortex neurons; these changes correlated with psychophysical metacontrast measured in the same monkeys. The changes in single-cell firing were evident as long as 400 msec after target onset. An early burst of firing, at a poststimulus latency corresponding to the traditional receptive field, was unchanged by metacontrast stimuli. The strength of the early burst was uncorrelated with the strength of the late burst, however, and the two bursts had different spatial distributions within striate cortex.

To the degree that the geometry of electrode placement and visual field stimulation isolate striate cortex, the VEP results also show that metacontrast-related late potentials in humans can be ascribed to striate cortex. It is possible that the later potentials have a spatial distribution that is different from that of C1, possibly nonstriate in origin (D. Jeffreys, personal communication, August 12, 1986).

However, because the observed VEP correlates of metacontrast occur in the time range in which the single-cell data from monkey striate cortex would lead us to expect them, the interpretation that is most consistent with both monkey and human data is that the late components are of striate origin. This interpretation leaves open the possibility that the human early and late potentials represent differently distributed striate activity.

The results further confirm the prediction of Bridgeman's (1971, 1977, 1978) lateral inhibitory model. This prediction postulates that correlates of metacontrast should first be found in late components of the evoked potential. The model invokes parallel distributed processing rather than discrete detectors. In this model, the earliest evoked activity is adequate only for detection, a condition in which U-shaped functions are not obtained psychophysically. This activity is unchanged by metacontrast masks in computer simulations of the model. U-shaped masking is obtained by integrating a longer epoch of poststimulus activity.

Both the VEP results given here and the monkey data contradict the prediction of Breitmeyer and Ganz (1976) and Breitmeyer (1984), which postulates that a component at the latency of sustained-cell activity (about 100 msec) should be suppressed in metacontrast. Psychophysical data, however, show metacontrast to be more complex than either the models and theories cited above or the electrophysiological correlates would imply. An effect that none of the present models can explain with their proposed neural interactions is the near disappearance of metacontrast with enough practice (Ventura, 1980). Uttal (1971) gives other, more general reasons why current neurophysiological models are unsatisfactory.

The long latency of metacontrast-related potentials in both human and monkey cannot be explained by interactions within striate cortex. Rather, earlier striate activity must leave that region, undergo processing elsewhere, and return to the striate cortex again. Where these signals go, and what happens to them there, remains unknown. It is likely that the practice effect and other influences on metacontrast have their effect between the early potentials in striate cortex and the late potentials that show concomitants of metacontrast.

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