It is proposed that inputs to binocular cells are gated by reciprocal inhibition between neurons located either in the lateral geniculate nucleus or in layer 4 of striate cortex. The strength of inhibitory coupling in the gating circuitry is modulated by layer 6 neurons, which are the outputs of binocular matching circuitry. If binocular inputs are matched, the inhibition is modulated to be weak, leading to fused vision, whereas if the binocular inputs are unmatched, inhibition is modulated to be strong, leading to rivalrous oscillations. These proposals are buttressed by psychophysical experiments measuring the strength of adaptational aftereffects following exposure to an adapting stimulus visible only intermittently during binocular rivalry.

1 Introduction

Binocular rivalry refers to the alternating periods of dominance and suppression that occur when unmatched stimuli are presented to the two eyes. For example, if a vertical grating is presented to one eye and a horizontal grating to the other, then vertical and horizontal stripes are seen successively in an oscillating manner, and not both simultaneously in the form of a plaid. This distinctive response to uncorrelated images may help us understand how images are matched during stereopsis, and in general shed light on the organization of binocular vision. In this article we attempt to connect psychophysically based models of rivalry with the anatomy and physiology of early visual pathways, in particular the microcircuitry of the striate cortex and lateral geniculate nucleus.

Neural models of binocular rivalry (Matsuoka 1985; Lehky 1988; Blake 1989; Mueller 1990), while differing in emphasis and detail, have agreed that the suppressive circuitry by which the signal from one eye blocks that of the other involves monocular neurons organized to form reciprocal feedback inhibition between left and right sides, prior to binocular convergence. Figure 1 shows one form of such a circuit. The inhibitory neurons are assumed to be monocularly driven because that is the most parsimonious arrangement by which inputs from one eye could selectively suppress inputs from the other. The circuit is believed to involve feedback rather than feedforward inhibition in order to produce oscillations.

However, the psychophysical data outlined below cause problems for this class of model. These data compare the strength of various adaptational aftereffects when the adapting stimulus is either continuously visible or only intermittently visible during rivalry. One might think that since a stimulus is visible for less time during rivalry, it would cause less...
adaptation than a continuously visible one. However, all studies to date indicate that this is not so. These include measurements of:

1. Contrast threshold elevation aftereffect (Blake and Fox 1974a; Blake and Overton 1979)
2. Spatial frequency shift aftereffect (Blake and Fox 1974b)
3. Tilt aftereffect (Wade and Wenderoth 1978)

In every case the strength of the aftereffect is the same whether or not the adapting stimulus is undergoing rivalry. The implication is that all adaptation to all classes of stimuli occurs early in visual pathways, prior to the site of the monocular suppressive circuitry. This seems implausible given the complexity of the aftereffects (orientation specific, direction specific). Both Lehky (1988) and Blake (1989) have highlighted this set of adaptation data as troublesome for binocular models.

It happens, however, that all the above studies measured aftereffects to rivalrous stimuli visible 50% of the time. Although no decrease in adaptation was apparent under that condition, perhaps a decrease could be found under a more extreme condition, comparing a continuously visible stimulus with a rivalrous stimulus visible, say, only 10% of the time. In that situation we find that there is indeed a significant difference in adaptation strength, as measured by contrast threshold elevations to gratings. Before discussing this experiment and its implication for binocular vision, we shall outline a simple model that allows quantitative interpretation of the data.

2 Data Analysis Model

The basic assumption is that inhibition during rivalry has one particular locus, whereas the potential for adaptation may be distributed over a number of sites. If that is so, three possible situations are:

1. All adapting neurons are located before the site of rivalry suppression.
2. All adapting neurons are located after the site of rivalry suppression.
3. Adapting neurons are distributed both before and after the site of rivalry suppression.

Under the first mode, adaptation strength is constant regardless of the fraction of time the rivalrous stimulus is visible, and equal to the adaptation caused by a continuously visible stimulus. Under the second mode, adaptation strength is proportional to the fraction of time a stimulus is visible. It is not linearly proportional, because adaptation strength as a function of adaptation time shows a compressive nonlinearity (Magnussen and Greenlee 1986; Rose and Lowe 1982). Finally, the third mode is intermediate to the two extremes described above. Adaptation is still proportional to predominance, but follows a flatter curve than the second mode.

All this can be expressed by equation 2.1:

\[ c = [1 - c_0][x + (1 - x)f]^p + c_0 \]  

(2.1)

where:

- \( c \) = threshold contrast after adaptation
- \( c_0 \) = threshold contrast before adaptation
- \( x \) = fraction of time stimulus is visible during rivalry
- \( f \) = fraction of adaptation located prior to site of suppression
- \( p \) = exponent defining power law time course of adaptation

The term inside the first square bracket is a normalization constant keeping \( c \) from exceeding 1.0. The first and second terms in the second square bracket indicate relative adaptation rates during dominant and suppressed phases of rivalry, respectively. The equation requires that the values of \( c \) and \( c_0 \) be normalized so that threshold contrast = 1.0 following adaptation to a continuously visible stimulus.

In applying the model, the time course of adaptation is assumed to follow a square root law, so \( p \) is set to 0.5. This appears to be a reasonable estimate based on data in the literature (Magnussen and Greenlee 1986; Rose and Lowe 1982). The parameter \( c_0 \) is set from the data. The goal is to estimate the value of the parameter \( f \), based on the shape of the experimental \( c \) versus \( x \) curve. A flatter curve implies a larger value of \( f \).

3 Methods

The general procedure was to induce rivalry by presenting orthogonal gratings to the two eyes, and afterward measure threshold contrast for the eye viewing the adapting grating. The predominance of the adapting grating (fraction of time it was visible) was varied for different runs by changing the grating contrast to the opposite eye, while holding the contrast of the adapting grating constant.

Stimuli were 3.0 cdeg \(^{-1}\) sinusoidal gratings, horizontal to the left eye and vertical to the right, within circular apertures 1.0° in diameter. No-nius lines were present at the perimeter of the apertures. These stimuli, presented on a pair of Tektronix 608 oscilloscopes, were viewed through a Wheatstone stereoscope. The left eye grating was the adapting stimulus, and its contrast was held constant at 0.15. The right eye grating
contrast was either 0.0, 0.15, or 0.53 during different runs. This resulted in rivalry in which the fraction of time that the left eye predominated was either 1.0, 0.5, or 0.1, respectively. There were two subjects, SL, the author, and MB, who was unaware of the purposes of the experiment.

The method of adjustment was used to determine contrast thresholds. Following a 1 min adaptation period during which rivalry ran freely, the screens went blank to mean luminance. The subject immediately adjusted a 10-turn logarithmic potentiometer controlling left eye contrast to what was judged as threshold, and pushed a button to indicate this decision to the computer. The grating used to measure postadaptation threshold was identical in spatial frequency and orientation to the adapting grating. Unadapted thresholds were measured in the same way, following binocular viewing of blank screens for a duration equal to the adaptation period of the other conditions. All conditions were replicated 10 times for each subject, with at least 15 min between trials to allow recovery from adaptation.

4 Results

Figure 2 shows contrast sensitivities (the inverse of contrast thresholds) following adaptation to stimuli with different predominances. There is a difference in sensitivity following adaptation to gratings with 0.5 and 1.0 predominances, but it is not statistically significant. This is compatible with previous failures to find an effect of rivalry on adaptation strength. It is only when comparing the most extreme conditions, with predominances of 0.1 and 1.0, that the effect becomes significant ($p < 0.05$). Even though only the most extreme conditions are significantly different, an overall trend is apparent in the data: increased predominance of the adapting stimulus during rivalry leads to decreased contrast sensitivity.

The data in Figure 2 are replotted in Figure 3 as contrast threshold versus predominance, where threshold has been normalized to equal 1.0 when predominance = 1.0.

5 Discussion

The basic experimental observation is that an adapting stimulus that is intermittently visible during rivalry produces a weaker adaptational aftereffect than a continuously visible stimulus. Since adaptation strength is a decelerating function of adaptation time, the claim is that this effect of rivalry is apparent only when the predominance of the adapting stimulus is small, much less than the 0.5 predominance used by studies in the past. These conclusions, drawn from the single experiment presented here, should be regarded as preliminary until subject to a number of confirmatory studies. However, if one accepts that rivalry does affect the strength of adaptation, as these data indicate, then this has implications for the organization of binocular pathways as will be detailed below.

Predictions of the data analysis model (equation 2.1) are superimposed on the data in Figure 3. It shows $c$ plotted as a function of $\tau$ for three values of $f$, where these variables were defined above. Inspection shows that the curve for which rivalry suppression precedes all adaptation ($f = 0$) corresponds best to the data.

Accepting this, one can suggest specific anatomical sites for rivalry suppression in accord with the following argument. First, it should be noted that adaptation to gratings is orientation-specific (Blakemore and Nachmias 1971). Given that information, the two premises of the argument are:

1. Suppression occurs prior to adaptation, which is orientation specific (based on data presented here).

2. The site of orientation-specific psychophysical adaptation coincides with the site of orientation-specific neurons.

From these it follows that suppression precedes the appearance of orientation specificity in the visual system. Thus to the earlier requirement for a valid model that the suppressive neurons be monocular, we are now adding the requirement that they be nonoriented as well.
Retinal ganglion cells fit these requirements but can be excluded because there are no opportunities for binocular interactions there. Neurons in the striate layer receiving direct magnocellular input, 4Co, although monocular, are oriented (Hawken and Parker 1984; Livingstone and Hubel 1984), and are therefore also excluded. These considerations leave either the lateral geniculate nucleus, or those layers of striate cortex receiving direct parvocellular input (4A and 4C\(b\)) as the location of suppression during binocular rivalry to unmatched spatial patterns. The location of suppression may be different for unmatched motion or color. The inability to further localize suppression to either the LGN or parvocellular layer 4 reflects the lack of known physiological differences among neurons in those structures. (Note that if there were a significant subpopulation of nonadaptable orientation-specific neurons, that could render these localization arguments less secure.)

Figure 3: Data of Figure 2 is replotted to show normalized contrast thresholds (threshold = 1/sensitivity). Squares on the vertical axes indicate unadapted thresholds. The lines are plots of equation 2.1 for three values of \(f\), where \(f\) is the fraction of adaptation occurring before rivalry suppression. In all cases the parameter \(p\) was fixed at 0.5. The data correspond best to \(f = 0\), showing that rivalry suppression precedes adaptation. Since adaptation to gratings is orientation specific, it is argued that suppression precedes orientation specificity in the visual system.

If rivalry suppression occurs in the LGN, a plausible substrate would be inhibitory interneurons between adjacent layers of that structure. Such inhibitory interactions were reviewed by Singer (1977), who also suggested they may be involved in rivalry. An alternative possibility, reciprocal inhibition mediated by the feedback loop to the LGN from layer 6 of V1 cortex, must be rejected because layer 6 outputs are binocular and oriented, while, again, units mediating suppression are postulated here to be monocular and non-oriented. On the other hand, if the locus of suppression is in layer 4, then it would likely involve inhibitory interneurons between adjacent ocular dominance columns.

In addition to this "gating circuitry," which can selectively block signals originating from one eye during rivalry, there must be "matching circuitry," which controls the state of the gate depending on whether inputs to the eyes match or not. Lehky (1988) proposed a model specifying the interactions between the gating and matching circuits. Without repeating the underlying reasoning, the essence of the model is that the same reciprocal inhibitory gating circuitry (Fig. 1) underlies both binocular fusion and rivalry. The difference between the two states depends on the strength of inhibitory coupling. Weak coupling leads to stable fusion while strong coupling produces rivalrous oscillations. Under this model, the output neurons of the matching circuitry (not pictured in Fig. 1) act to modulate the strength of reciprocal inhibition in the gating circuitry as a function of the correlation between left and right eye signals.

It seems reasonable to believe that the output neurons (though not necessarily the intrinsic neurons) of the binocular matching circuitry are binocularly driven. If that is the case, then to find these outputs one must look for binocular units that feed back on monocular units (of the gating circuitry). According to the available data, neurons of layer 6 of area V1 have this property uniquely. Layer 6 sends major projections only to structures with preponderantly monocular cells, namely the LGN as well as layers 4C\(a\) and 4C\(b\) (but not 4B, which is binocular), and none of those monocular regions receives major binocular inputs other than from layer 6. This relationship becomes apparent on matching the physiology (reviewed by Livingstone and Hubel 1987) with the anatomy (Blasdel 1985; Fitzpatrick et al. 1985; Lund and Boothe 1975). Therefore, whether binocular gating occurs in the LGN or layer 4 (or both), it is the layer 6 output that is the most likely candidate for controlling the gate.

Figure 4 summarizes the proposed organization, illustrating the case in which gating may be occurring in the LGN. The other possibility, that of gating occurring in layer 4, could be illustrated analogously with ocular dominance columns replacing LGN layers. It is possible that the circuitry discussed here is important not only for suppression during rivalry, but also during other binocular processes, such as pathological suppression during amblyopia or the elimination of false matches during stereopsis.
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Figures 4: Summary diagram of the model, in essence a more elaborate version of Figure 1. Signals from the left and right eyes are binocularly gated by reciprocal inhibition, either between adjacent layers of the LGN (as illustrated here) or between adjacent ocular dominance columns in those parts of striate layer 4 receiving direct parvocellular inputs (not shown). The restriction to parvo-driven parts of layer 4 is based on data presented here involving rivalry induced by unmatched spatial patterns, and the situation may be different for unmatched motion. Whatever the case, the strength of inhibitory coupling is modulated by layer 6 units, which are the outputs of binocular matching circuitry. If the stimuli to the two eyes match, layer 6 neurons modulate reciprocal inhibition in the gating circuitry to be weak, leading to fused binocular vision. If the stimuli do not match, inhibition is modulated to be strong, producing rivalry. (The modulatory feedback loop is not included in Figure 1.)

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