The Time Course of Interocular Suppression in Normal and Amblyopic Subjects

Sigrid de Belsunce and Ruxandra Sireteanu*

The authors measured the time course of interocular suppression of five normal subjects and eleven patients with amblyopia (strabismic and/or anisometropic). Orthogonal gratings were presented dichoptically for durations that ranged from 10–6000 msec. All normal observers reported fusion or superimposition of the orthogonal gratings for short stimuli and reported binocular rivalry for stimuli longer than 150 msec. At long presentation times, all amblyopes constantly suppressed the pattern that was presented to their amblyopic eye. Six amblyopes showed superimposition of the two patterns at short presentation times. Of these, three had time courses similar to those of normal observers; the other three had a much shorter onset of suppression (about 80 msec). The remaining five amblyopes perceived only the pattern of the dominant eye at short stimulus durations; at intermediate durations, they reported partial superimposition of the stimuli, whereas at the longest stimulus durations, again only the stimulus of the dominant eye was perceived.

The results suggest that binocular rivalry in normal observers and strabismic suppression in amblyopes are mediated by different mechanisms. The heterogeneity of the time courses of suppression in amblyopes might result from differences in the disturbances of early visual development (age at onset of strabismus and/or anisometropia, origin, and therapy). Invest Ophthalmol Vis Sci 32:2645–2652, 1991

Binocular rivalry occurs when different stimuli are presented dichoptically and simultaneously to corresponding loci of the two eyes. At each position in the visual field, only one of the two monocular inputs is perceived at a time, whereas the other is suppressed. The pattern of dominance and suppression changes periodically. However, when dichoptic stimuli are briefly flashed, they appear to fuse into a single percept. Wolfe measured the rise-time of suppression in normal subjects and found that dichoptic stimuli appear to be fused when flashed for less than 150 msec. In a later study, Wolfe compared normal subjects and constant suppressors and found no difference in their temporal properties. He therefore suggested, in line with other researchers, that the interocular suppression that occurs in unilateral amblyopes is based on similar neural mechanisms as binocular rivalry.

Subjects with anomalous binocular vision (strabismus and/or anisometropia) continuously suppress their affected eye. For short presentation times, interocular suppression is reduced in amblyopic observers (Sireteanu, unpublished observations). Nevertheless, it seems surprising for the phenomena of suppression to be the same in normal and amblyopic eyes. Several psychophysical studies show that the stimuli for strabismic and rivalry suppression are different and that the pattern of sensitivity loss also differs in strabismic suppression and binocular rivalry. Thus, the two suppression phenomena might involve different neural pathways.

This study reexamined these controversial findings by repeating Wolfe's experiment in normal observers and in subjects with known disorders of binocular vision.

Materials and Methods

Subjects

We tested five normal observers, aged 21–40 yr. All had or were corrected to full visual acuity (corrected visual acuity ≥ 1.0) and had stereopsis of at least 40", as assessed by Titmus and TNO-Test. Three of these normal observers were unaware of the purpose of the experiment; the other two were the authors of this report.

The experiments were further performed on 11 amblyopic subjects, aged 22–45 yr. The refractive status was assessed for all of the subjects subjectively with trial lenses at a distance of 6 m. For RMM, Rst, JW, BW, and MF data were also obtained objectively.
with the aid of a refractometer. Visual acuity was tested at 6 m with Snellen optotypes (letters and figures). Fixation was tested with a visuscope. The angle of strabismus was measured at 6 m and at 30 cm. The cover test was used when fixation was central; in subjects with eccentric fixation, the corneal reflexes were monitored to measure the angle of strabismus. The binocular status was assessed with the TNO, Titmus, Randot, and Lang stereo tests. Detailed results are only given for randomized patterns of TNO and Randot test. The angle of anomaly was assessed with several methods: the striated glasses of Bagolini, dark and light red filter in combination with the Maddox cross, the dichoptic stimuli of the experiment, but could not be performed.

The results of the orthoptic examination are shown in Table 1. The subjects wore their best correction during testing. All patients with amblyopia fully perceived the stimulus presented to their weaker eye when tested monocularly. In binocular conditions, all suppressed constantly the image of the amblyopic eye. All amblyopes were naive to the purpose of the experiments. Informed consent was obtained before participation in the study.

**Apparatus**

Horizontal and vertical square-wave gratings were projected on a tangent screen. The spatial frequency of the gratings was 0.8 c/deg, the mean luminance: 6.8 cd/m². The stimuli were circular with a diameter of 5.8°. The large field and low spatial frequency were chosen to ensure that the patterns were resolved monocularly, even by the observers with deep amblyopia (Rst, JW, MF). The projection was simultaneous and

<p>| Table 1. Orthoptic status of the subjects |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|</p>
<table>
<thead>
<tr>
<th><strong>Subject</strong></th>
<th><strong>Eye</strong></th>
<th><strong>Refraction</strong></th>
<th><strong>Visus c.c</strong></th>
<th><strong>Fixation</strong></th>
<th><strong>Strabismus</strong></th>
<th><strong>Correspondence</strong></th>
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<tbody>
<tr>
<td><strong>Group I</strong></td>
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<tr>
<td>PZ</td>
<td>RE</td>
<td>+1.75-0.5/180°</td>
<td>1.0</td>
<td>foveolar,</td>
<td>far +6°</td>
<td>angle of anomaly 1° (non-harmonious)</td>
</tr>
<tr>
<td></td>
<td>LE*</td>
<td>+4.5-1.25/20°</td>
<td>0.4</td>
<td>unsteady</td>
<td>near +6°</td>
<td>first Rx at 5 yr, occlusion therapy at 5-6 yr, family history</td>
</tr>
<tr>
<td>JH</td>
<td>LE*</td>
<td>+4.5-1.5/40°</td>
<td>0.8-1.0</td>
<td>foveolar,</td>
<td>near +1°</td>
<td>family history</td>
</tr>
<tr>
<td>SL</td>
<td>LE*</td>
<td>-0.75-0.75/180°</td>
<td>1.25</td>
<td>unsteady</td>
<td>near +1.5°</td>
<td>angle of anomaly 1° (harmonious)</td>
</tr>
<tr>
<td></td>
<td>-1.0/160°</td>
<td>0.6-0.8</td>
<td></td>
<td></td>
<td>near +1.0°</td>
<td>first Rx at 17 yr</td>
</tr>
<tr>
<td><strong>Group II</strong></td>
<td></td>
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<tr>
<td>RMM</td>
<td>RE</td>
<td>-2.25</td>
<td>1.25</td>
<td>foveolar,</td>
<td>far 0°</td>
<td>initially RE emmetropic</td>
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<tr>
<td></td>
<td>LE*</td>
<td>plano</td>
<td>0.5-0.6</td>
<td>foveolar</td>
<td>near 0°</td>
<td>normal</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>initially LE hyperopic (2D difference), family history</td>
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<td><strong>Group III</strong></td>
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<tr>
<td>JW</td>
<td>RE</td>
<td>+0.25-0.75/150°</td>
<td>1.25</td>
<td>foveolar,</td>
<td>far -2°-VD 2°</td>
<td>angle of anomaly 5° (non-harmonious)</td>
</tr>
<tr>
<td></td>
<td>LE*</td>
<td>+6.25-2.5/8°</td>
<td>0.084</td>
<td>5° nasal,</td>
<td>near +5°-VD 3°</td>
<td>large angle esotropia, surgery at 10-11 yr</td>
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<td></td>
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<td></td>
<td>unsteady</td>
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<td>family history</td>
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<tr>
<td></td>
<td>RE</td>
<td>-0.75-0.5/150°</td>
<td>1.0</td>
<td>foveolar,</td>
<td>far +17°</td>
<td>angle of anomaly 12° (non-harmonious)</td>
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<tr>
<td></td>
<td>LE*</td>
<td>+5.5-0.70/180°</td>
<td>0.6</td>
<td>unsteady</td>
<td>near +20°</td>
<td>occlusion therapy at 6-9 yr, can alternate</td>
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<tr>
<td></td>
<td>RE</td>
<td>+0.75-0.75/15°</td>
<td>1.25</td>
<td>foveolar,</td>
<td>far +4°</td>
<td>angle of anomaly 1-4° (non-harmonious)</td>
</tr>
<tr>
<td></td>
<td>LE*</td>
<td>+1.75</td>
<td>0.12</td>
<td>3°, 5° nasal,</td>
<td>near +10°</td>
<td>family history</td>
</tr>
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<td></td>
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<td>1.5° up,</td>
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<td></td>
<td></td>
<td>unsteady</td>
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<tr>
<td></td>
<td>RE</td>
<td>+3.75-1.0/5°</td>
<td>0.8-1.0</td>
<td>foveolar,</td>
<td>far +10°</td>
<td>angle of anomaly 1° (non-harmonious)</td>
</tr>
<tr>
<td></td>
<td>LE*</td>
<td>plano</td>
<td>1.25</td>
<td>unsteady</td>
<td>near +10°</td>
<td>first Rx at 2 yr, surgery at 6-7 yr, occlusion therapy at 6-7 yr, initial occlusion therapy at 6-7 yr</td>
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<tr>
<td></td>
<td>RE</td>
<td>+0.5-0.25/0°</td>
<td>1.25</td>
<td>foveolar,</td>
<td>far +2°</td>
<td>angle of anomaly 1° (non-harmonious)</td>
</tr>
<tr>
<td></td>
<td>LE*</td>
<td>+0.75-0.25/90°</td>
<td>0.8</td>
<td>unsteady</td>
<td>near +2°</td>
<td>first Rx at 5 yr, occlusion therapy at 10-11 yr</td>
</tr>
</tbody>
</table>

Amblyopic eyes are indicated by asterisks.
dichoptic. The stimuli were dissociated by polarizing filters. Stimulus duration varied from 10–6000 msec by computer-programmed shutters placed in front of the projectors. The subject was 1.14 from the test screen.

Procedure

We used a five-point rating scale to define the perception of the stimuli (adapted from Wolfe\(^6\)). First, each monocular stimulus—one vertical and one horizontal orthogonal grating—was shown to the subject. Both impressions were to be rated 0. We then projected the two stimuli simultaneously; the checkerboard was rated 5.

The subjects were then asked to wear polarizing glasses. Stimuli with durations ranging from 10–6000 msec were presented in random order. The subjects learned to use a five-point rating scale: a fused perception or superimposition of the orthogonal gratings equalled 5; the perception of only one monocular input equalled 0. The intermediate steps 1, 2, 3, and 4 described rivalrous perceptions with different pattern mosaics; 1 and 2 corresponded to a dominance of a monocular perception of one grating; 3 and 4 corresponded to partial superimposition.

This procedure differed slightly from that of Wolfe\(^6\), who gave the rivalrous binocularly experienced pattern the rating value 0 (see the Discussion section).

All subjects had little or no difficulty learning to use this scale. Each session comprised one or two training runs and at least six further runs of 20 different stimulus durations that were presented in random order.

Before stimulus presentation, the subjects were adapted to the background luminance of the screen. Time was allotted between presentations to permit previously induced suppression to dissipate fully. There was no prestimulus input, and, because the stimulus did not produce noticeable after-images, there was no poststimulus flash. The stimuli were projected at the center of a large screen (70° × 50°). The edges of the screen were barely visible and thus the subject's eye. This lateral displacement did not exceed 5° (subject JW).

Results

Normal Observers

All normal observers reported seeing a checkerboard or plaid (superimposition of the orthogonal gratings) for stimuli shorter than 150 msec. Longer stimuli led to binocular rivalry (Figs. 1 and 5). Stimuli longer than 1000 msec appeared to be rivalrous with a strong dominance of one, then the other monocular input.

Amblyopic Observers

Amblyopic observers showed clearly different, but heterogeneous time courses of suppression. According to their responses, they tended to fall in three classes.

Group I: Three amblyopes (PZ, JH, SL) had time courses of suppression similar to those of normal observers; two of them without reaching complete superimposition of the gratings for short stimulus presentations. For stimuli longer than 1000 msec, only the stimulus of the nonamblyopic eye was perceived (Fig. 2). All of these subjects were strabismic, subject PZ was also anisometropic, and subject JH ametropic (Table 1).

Group II: Three other amblyopic observers (RMM, UL, Rst) perceived complete superimposition (checkerboard) for short stimulus durations and had an early (about 80 msec) onset of strabismic suppression (Fig. 3). All three subjects saw for durations of more than 150 msec always only the grating presented to the right eye (in every case their dominant eye). Subjects UL and Rst were strabismic and ametropic; subject RMM was only anisometropic (Table 1).

Group III: The remaining five amblyopes perceived only the image of their dominant eye for short stimuli (less than 30 msec), unstable pattern combinations of the two monocular inputs for longer stimuli, and only the image of the dominant eye for the longest stimuli. The exact time courses differed from one subject to the next. Subject SV rated all flashes of durations shorter than 150 msec 0 and reported mosaic patterns even for our longest stimuli (Fig. 4). Further testing at durations longer than 6000 msec showed perception of the image of the dominant eye only. All five observers included in this group were strabismic; subjects JW and CS were also anisometropic (Table 1). Figure 5 shows an example of each group.
Comparison of the Groups

The graphs of subjects in groups I and II differed in slope and in midpoint: subjects in group II had lower midpoints (65–120 msec) than subjects in group I (150–280 msec). The slopes of the graphs of subjects in group II were much steeper (1.6–2.7 msec⁻¹) than for subjects in group I (0.5–1.2 msec⁻¹). The graphs of normal subjects had midpoints ranging from 250–1000 msec; their slopes were similar to those of subjects in group I (0.5–0.8 msec⁻¹). Thus, the difference between normal subjects and subjects in group I was minor; a large difference occurred between the slopes of normal subjects and subjects in group II. A t-test comparison between the groups is not possible because the variances are different and the number of subjects is insufficient. Nevertheless, an inspection of the distribution of midpoints and slopes suggests that subjects in groups I and II show distinct suppression patterns (Fig. 6).

Control Experiments

The low rating values for briefly flashed stimuli by subjects in group III might be caused either by the weakness of the stimulus presented to their amblyopic eye, or to a delay of information processing in the visual pathway of this eye. To distinguish between these two possibilities, we performed two control experiments.
INTEROCULAR SUPPRESSION IN NORMAL AND AMBLYOPIC SUBJECTS

Fig. 5. Time course of suppression. One case from each group is shown. Plotted points represent the mean of six ratings; bars show the standard deviation.

To test for the possibility that the nonvisibility of the pattern presented to the amblyopic eye at short presentation times was due to the relative weakness of this input, in three normal subjects, one of the two images was progressively weakened with the aid of neutral density filters. The results of this experiment in one subject (SA) are shown in Figure 7. The other tested subjects (RS and SB) produced similar results.

Lowering the luminance of one monocular stimulus produced a loss of perception of this stimulus at brief presentation times. The pattern of suppression was similar to that shown by subjects in group III.

This finding shows that adding neutral density filters in front of one eye of normal observers can produce patterns of suppression similar to those of subjects in group III.

Neutral density filters produce not only a reduction of luminance, but also a temporal delay of the presented stimulus. In another control experiment, we delayed the stimulus of the dominant eye of amblyopic subjects in group III for varying periods. Stimulus duration was 10 and 30 msec. All other conditions were unchanged.

If the low rating values for short flashes are only due to

Fig. 6. Mid-points and slopes of the averaged suppression curves for normal observers and for subjects in groups I and II. Data were obtained by interpolation.
tic stimuli simultaneously. Rivalry begins only for the amblyopic eye, we would expect a perception of stimuli longer than about 150 msec and is complete at which the patterns appeared to be superimposed. The results are difficult to interpret. Further investigations are needed to clarify this point.

This control experiment suggests that the perception of the stimulus in the amblyopic eye in subjects of group III is not simply delayed, but is also smeared in time. The temporal offset between the dichoptic stimuli produces a wave of complex interactions (backward and forward masking, for example), and the results are difficult to interpret. Further investigations are needed to clarify this point.

We rule out the possibility of a simple delay of information-processing of the image in the amblyopic eye as an explanation of the suppression patterns of subjects in group III.

Discussion

Evaluation of the Results

The results of our experiments show that the temporal properties of suppression in normal and amblyopic subjects are different.

Normal observers perceive briefly flashed, dichoptic stimuli simultaneously. Rivalry begins only for stimuli longer than about 150 msec and is complete for stimuli longer than 1000 msec.

In three of eleven subjects with unilateral strabismic and/or anisometropic amblyopia, the time course of interocular suppression was similar to that of normal subjects. The remaining subjects fell into two categories: one group showed a more rapid onset of suppression (about 80 msec); the other showed a prevalence of the preferred eye for short exposure times, followed by partial superimposition of the two stimuli and suppression of the nonpreferred eye.

The exact pattern of results differed from one subject to the next. In group III, subject SV seemed to exhibit a pattern that was the reverse of normal subjects. However, additional tests with longer stimuli showed that this subject also suppressed the stimulus of the deviated eye at the longest stimulus durations. The reason of the extreme processing delay in this subject is unclear.

For the subjects with larger squint angles (BW, CS, MF, and PZ), retinally noncorresponding areas were stimulated in the two eyes (the fovea of the fixating eye and a peripheral area in the deviated eye). All these subjects had nonharmonious anomalous correspondence and perceived the two stimuli as superimposed. With the exception of PZ, these subjects belonged to group III (Table 1).

The pattern of suppression of subject PZ did not differ significantly from that of the two microstrabismic subjects in group I (JT and SL). Subjects BW, CS, and MF were similar to subject JW, whose large-angle esotropia was reduced to microstrabismus by surgery. Thus, the nonfoveal projection of the stimulus in the deviated eye of some subjects does not seem to influence the time course of suppression of this eye.

Our groups II and III differ clearly from normal observers. The results of group II could be due to a weaker input of the amblyopic eye to the rivalry mechanism. However, such an imbalance causes a lowering of the rating values for short stimulus durations rather than an acceleration of suppression. The rapid onset of suppression for subjects in this group is more likely to be an expression of the frequent use of the suppression mechanism to prevent diplopia under everyday viewing conditions.

The nonperception of the stimulus presented to the amblyopic eye at short durations for subjects in group III might depend on a delay of information processing in the visual pathway of the amblyopic eye.

Clinical research\textsuperscript{13,14} showed that sensation and reaction time are significantly prolonged in amblyopic eyes. Visually evoked cortical potentials also show a prolonged latency in some types of amblyopia.\textsuperscript{15}

Our control experiment on normal observers showed that weakening of one monocular image by neutral density filters produces time courses similar to those of amblyopic subjects in group III. Thus, it seems that, at least in some amblyopes, the suppres-
sion of brief stimuli can be explained by the relative weakness of the amblyopic visual pathway. Note, however, that at longer presentation times in amblyopic subjects, the stimulus of the amblyopic eye is suppressed, whereas in normal observers the pattern appears to be rivalrous.

Thus, the low rating values for brief stimuli for subjects in group III are due to their monocular perception of the stimulus presented to the dominant eye (presumably because of the low energy of the stimulus in the amblyopic eye), whereas at long presentation times, the stimulus presented to the amblyopic eye is suppressed (although the data points themselves are indistinguishable).

Further experiments are being performed in our laboratory to determine the temporal properties of rivalry suppression under conditions of artificial weakening of one eye.16

**Relationship to Previous Studies**

The heterogeneity of the suppression courses in amblyopes reinforces the idea that the constant suppression in amblyopes and rivalry suppression in normal observers are mediated by different neural mechanisms.

This conclusion is supported by several other findings. The adequate stimuli for binocular rivalry are orthogonal patterns, whereas strabismic suppression occurs more readily with stimuli of similar orientation;10 the spectral sensitivity functions determined during suppression in binocular rivalry are different from those shown in esotropic subjects during strabismic suppression.12

Our results differ from those of Wolfe,5-6 who showed that normal observers and constant suppressors follow the same time course of suppression. Our amblyopes of group I are similar to some of Wolfe’s constant suppressors, yet this finding is only one of various possible answers of the amblyopic visual system to this kind of stimulus presentation. The question arises whether the difference between the two studies rests on the selection of the subjects: Wolfe’s subjects were stereoblind and constant suppressors, but often only mildly amblyopic. Two of our subjects had some stereopsis (RMM and SV); several others were deeply amblyopic (Rst, JW, MF). However, even when these subjects are eliminated from the analysis, some of the remaining subjects show patterns of suppression that are definitely abnormal (for example, subjects UL, BW, and CS). A careful analysis of Wolfe’s results shows one case of strabismic suppression, which is comparable to the results of our group III (Wolfe’s subject LH).

As described in the Materials and Methods section, we modified Wolfe’s rating scale. This change was caused by the observation of some amblyopic subjects in group III, who experienced briefly presented binocular patterns as much more similar to monocular gratings than to rivalrous long-lasting patterns. To avoid negative ratings, 0 was used in our study for purely monocular patterns. This change produces slightly higher ratings at long-stimulus durations than in Wolfe’s study, but it cannot explain the major differences between the two studies.

**Underlying Neural Mechanisms**

Unfortunately, it is not possible to correlate the three different patterns of suppression with the kind of sensory disturbance of each amblyope (except for the tendency of subjects in group III to have larger preoperative squint angles; see Table 1). However, the actual orthoptic status does not necessarily reflect the status of each subject during the early developmental period. The angle of strabismus can change spontaneously or as a result of surgery; refraction often shows spontaneous changes, and the patterns of fixation and correspondence can change accordingly. Therefore, it is likely that the heterogeneity of the time courses of suppression is due to differences in the early disturbances of visual development (age at onset of strabismus and/or anisometropia, origin, and therapy) of each amblyope. These data are in most cases uncertain.

It thus seems that numerous pathogenetic factors are involved in the strabismic suppression mechanism; they might be localized at several neural levels. Because the goal of strabismic suppression is always to avoid diplopia, it is interesting to speculate about the possible neural substrate of this mechanism.

In normal adult cats, binocular suppression was seen in single neurons in the striate visual cortex.17 Rivalry suppression occurs occasionally in single units of the cat dorsal lateral geniculate nucleus. This phenomenon depends on the function of the visual cortex, thus suggesting a role of the corticofugal projection in binocular rivalry suppression.18,19 No data exist, however, on the locus and mechanisms of strabismic suppression.

The differences we found between normal binocular rivalry and strabismic suppression suggest that the underlying neural mechanisms are different in adult observers. One reason for the difference must be that in normal observers, most striate cortex cells are binocular, including the corticofugals, whereas most squinters can be assumed to be monocular. We cannot exclude, however, that during the early develop-
ment of strabismic suppression and amblyopia, binocu-
lar rivalry might have played an important role. Pre-
sumably, an early squint causes rivalry that, when 
carried over in the continuous suppression of one eye, 
produces changes in the central connections of this 
eye. In the modified visual system, strabismic sup-
pression occurs as a new phenomenon, based on a 
different set of neural connections.

Key words: humans, binocular rivalry, suppression, ambly-
opic patients, temporal properties

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