Fusional vergence eye movements in microstrabismus
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3. Studies on vergence in patients with strabismus

3.1 Introduction

Several studies on the subject of motor fusion in strabismus have been published. Those studies that have much in common with our study will be described in this chapter and will also be discussed in chapter 8. In section 3.2 studies will be mentioned using various methods (with the exception of infrared recording) while in section 3.3 studies using infrared recording are described. A diagram summarizing the studies mentioned is also given (See table 3.1).

3.2 Studies on motor fusion

Burian (1941) described fusion on peripheral stimuli in a large group of patients. The group consisted of 75 selected patients with a permanent horizontal deviation of as much as 12 arc degrees and with nearly equal vision in both eyes. Patients with convergent and divergent squint, as well as patients who had been operated on to reduce the angle of squint were included in this study. None of these patients had normal binocular vision, but a large number of them followed peripheral fusional stimuli, even in case of foveal suppression.

Hallden (1952) investigated fusional vergences in 9 subjects with anomalous correspondence; 6 subjects with a convergent angle, 2 subjects with a divergent angle and one subject without an angle of squint. The purpose of the fusional movements observed was supposed to maintain subnormal binocular vision on the basis of the existing anomalous correspondence. It was demonstrated that these fusional movements were sometimes insufficient to compensate the stimulus completely, and that they were accompanied by a change in the angle of anomaly (sensory fusion partly supplementing motor fusion).

Maraini and Pasino (1964) used an after-image technique. They noted the presence of convergence movements when 11 patients with microstrabismus were tested with 6 prism diopeters (base-out). They found three patterns of response: A change in the angle of anomaly (sensory fusion), a motor fusion response and a combination of the two.

In 1976 Bagolini described experiments in which subjects with postoperative residual esotropia were stimulated with prisms of up to 40Δ. The subjects showed a slow compensation for the effect of the prism (in minutes to hours). This differs from the fast compensation found in normal subjects. Bagolini called the observed movement anomalous fusional movement and compared it with normal fusion. The anomalous movements of Bagolini are slower than the fusional movements such as found by Rashbass and Westheimer (1961), and they are comparable to the change in relative eye position in
prism adaptation experiments.

Bagolini also mentions a clinical problem due to these anomalous movements: The movements are a handicap to the restoration of normal (bifoveal) binocular vision, because they tend to restore the original angle of squint post-operatively. In fact the anomalous movements resemble tonic vergence found in subjects with normal binocular vision, which is an adaptive process as the result of a long lasting disparity (See section 2.2.2).

Stimulation with increasing disparities, by means of an amblyoscope, of subjects with primary microstrabismus was described by Prick (1981). As in Bagolini’s experiments the stimuli were large (up to $25^\circ = 44\Delta$). It may be assumed that as a consequence of the methods used, exposure lasted longer than a few seconds. Therefore tonic vergence may have been measured as well. The stimulus used was a large contrast-rich peripheral stimulus. Three different groups were found:
1. The first group (8 subjects) performed motor fusion that fully compensated the disparity.
2. The second group (11 subjects) predominantly changed the angle of anomaly (perceptual or sensory fusion).
3. Ten subjects showed a combination of sensory and motor fusion. Sensory fusion was present for smaller disparities. At increasing disparity, motor fusion was performed. These subjects started with a motor response when they were stimulated with between 2.5 and 14 degrees.

3.3 Studies on the dynamics of motor fusion

In the studies mentioned in this section infrared recordings were used. Some of these studies provided information on velocity and latency of motor fusion, see table 3.0.

<table>
<thead>
<tr>
<th>Author</th>
<th>Vergence latency</th>
<th>Vergence velocity</th>
<th>Stimulus range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boonstra et al, 1988</td>
<td>about 150 msec</td>
<td>1.37° / sec</td>
<td>2.3° C</td>
</tr>
<tr>
<td>Kenyon et al, 1980</td>
<td>160 - 200 msec</td>
<td>3° / sec</td>
<td>1.5° C/D</td>
</tr>
<tr>
<td>Schoessler, 1980</td>
<td>375 - 1400 msec</td>
<td>not available</td>
<td>-8° - +8° D/C</td>
</tr>
</tbody>
</table>

**table 3.0**

Vergence latency and vergence velocity in (micro)strabismus. (Data obtained from the literature), C=convergence D=divergence.

Schoessler (1980) found a larger percentage of non-responders in subjects with strabismus than in normal subjects. In strabismus the latency was often larger. Responses opposite to the vergence movements that the disparity should have evoked were also found. Vertical
lines (foveal stimuli) were presented as targets by a mirror haploscope (For an explanation of the mirror haploscope see appendix B).

Kenyon, Ciuffreda and Stark (1980a) studied midline or symmetrical vergence in normal subjects and in patients with intermittent strabismus, constant strabismus with amblyopia, and amblyopia without strabismus. The dichoptic targets were fine crosses (foveal stimuli). Normal subjects performed symmetrical vergence, without a saccade. The patient group showed a pattern of accommodative vergence, in which the average vergence amplitude of the dominant eye was approximately 18% of that of the other eye. A binocular saccade accompanied the vergence movement. These responses in strabismus and amblyopia were very similar to those found in the normal subjects when one eye was occluded. The authors concluded that disparity vergence in strabismus and amblyopia is absent or blocked.

In subjects with strabismus and slight amblyopia accommodative vergence was normal, when the dominant eye or the non dominant eye was occluded. However, in case of deep amblyopia (visual acuity about 1/20) the response amplitude decreased rapidly (Kenyon et al., 1980b).

The same authors published on the subject of asymmetrical vergence in 1981. The foveal target was now presented at different depth planes in the visual axis of one eye (instead of along the subject’s midline). Normal subjects made a symmetrical vergence and a binocular saccade (See section 2.3.1.2) while subjects with strabismus made an unequal vergence and non-Hering’s law saccades. All patients with strabismus and some of the patients with amblyopia showed accommodative vergence rather than asymmetrical disparity vergence when attempting to track real targets moving in depth (both eyes open). In general the patient group showed accommodative vergence, rather than disparity vergence.

In 1985 Boman and Kertesz did a similar experiment. They believed that the absence of fusional vergence, found by Kenyon et al. was the consequence of the fact that they only used a foveal stimulus. Therefore Boman and Kertesz described fusional responses to foveal and extrafoveal stimulation in a group of patients with microtropia, esotropia and exotropia. They used a dichoptic projection type video display at a distance of 115 cm. The target consisted of a random dot stereogram (subtending 50 deg by 40 deg) with a vertical line seen by both eyes and two horizontal nonius lines. Disparity steps of 0.5° were given and the eye movements were recorded with an infrared reflection device. Two differences were observed between the responses of strabismics and stereonormal subjects: The first difference was that the esotropes and the microtropes produced less overall motor compensation to full field stimuli than subjects with normal stereoscopic vision. Secondly, subjects with strabismus tested with foveal stimuli had small fusional amplitudes and usually no measurable vergence amplitude to a cross subtending an angle of 1.5 degrees. Boman and Kertesz concluded that small central stimuli were not effective in producing fusional vergence while stimuli that covered the periphery produced responses that were similar to those found in normal subjects. With regard to accommodative vergence in
strabismus the authors were able to confirm the findings of Kenyon et al. These authors also suggested a mechanism that is able to prevent disparity information from being used even when the stimulus image is not suppressed. Boman and Kertesz, however, concluded that the disparity blocking mechanism is restricted to the central visual fields. A summary of the studies mentioned in this section is given in table 3.1.

<table>
<thead>
<tr>
<th>authors</th>
<th>dissociation method, or targets</th>
<th>stimulus</th>
<th>total number of patients</th>
<th>patients showing complete compensation by disparity vergence</th>
<th>patients showing absence of disparity vergence</th>
<th>patients showing incomplete compensation by disparity vergence</th>
<th>remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burian, 1941</td>
<td>Polaroid</td>
<td>+ +</td>
<td>75</td>
<td></td>
<td></td>
<td></td>
<td>large number</td>
</tr>
<tr>
<td>Hallden, 1952</td>
<td>afterimage prism polaroid</td>
<td>+ +</td>
<td>9</td>
<td></td>
<td></td>
<td></td>
<td>9</td>
</tr>
<tr>
<td>Maraini and Pasino, 1964</td>
<td>afterimage prism polaroid</td>
<td>+ +</td>
<td>11</td>
<td>3</td>
<td>4</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Bagolini, 1976</td>
<td>prism (20-30Δ)</td>
<td>- +</td>
<td>85</td>
<td>15</td>
<td>21</td>
<td>49</td>
<td></td>
</tr>
<tr>
<td>Schoessler, 1980</td>
<td>polaroid</td>
<td>+ -</td>
<td>4 normals</td>
<td>5 patients</td>
<td>4 normals</td>
<td>5 patients</td>
<td>motor response not related to stim., higher latencies</td>
</tr>
<tr>
<td>Kenyon et al., 1980a</td>
<td>illumination of lucite plates</td>
<td>+ -</td>
<td>2 normals</td>
<td>11 patients</td>
<td>2 normals</td>
<td>1 patient</td>
<td>The other patients responding by accommodative vergence instead of disparity vergence</td>
</tr>
<tr>
<td>Kenyon et al., 1981</td>
<td>illumination of lucite plates</td>
<td>+ -</td>
<td>2 normals</td>
<td>7 patients</td>
<td>1 amblyopic</td>
<td>1 amblyopic +</td>
<td></td>
</tr>
<tr>
<td>Prick, 1981</td>
<td>amblyoscope</td>
<td>- +</td>
<td>32</td>
<td>8</td>
<td>11</td>
<td>10</td>
<td>3 deviant responders</td>
</tr>
<tr>
<td>Boman and Kertesz, 1985</td>
<td>large red/green dot matrix</td>
<td>- +</td>
<td>11</td>
<td></td>
<td></td>
<td>11</td>
<td>no measurable vergence to a disparity pure foveal stimulus</td>
</tr>
<tr>
<td></td>
<td>small lucite crosses</td>
<td>+ -</td>
<td>2 normals</td>
<td>3 patients</td>
<td>2 normals</td>
<td>2 patients</td>
<td>1 patient</td>
</tr>
</tbody>
</table>

| table 3.1             | Summary of different studies on disparity vergence in (micro)strabismus. (+ means: patients responded now and then) |

3.4 Causes of (micro)strabismus in literature

The cause of strabismus is unknown. The literature on strabismus can roughly be divided into two theories: In the first theory (Crone, Worth) strabismus is caused by an inborn irreversible defect of disparity sensitivity (in the striate cortex). In the second theory (Chavasse, 1939) the postnatal development of binocular vision is impaired.
Anomalous correspondence (ARC) has been suggested to be a primary cause of microstrabismus by Lang. In his book on microstrabismus, Lang mentions four possible causes of microstrabismus, namely: Expansion of Panum’s area, anisometropia, the statistical theory of Goldmann (1967) and genetic factors. Goldmann (1967), proposes a central adaptation mechanism ("Raumwertanderung") as a cause for microstrabismus and a peripheral adaptation as a consequence of feedback mechanisms. Theories on the adaptation of the central nervous system to microstrabismus were also published by Velzeboer and Crone. According to Velzeboer (1957) unilateral squint, with a certain degree of amblyopia and a small and fixed angle of squint promotes the development of an abnormal cortical junction (a coupling of the cortical endings of disparate retinal elements).

Hallden (1952) studied fusional vergence in microstrabismus and found a covariation of the angle of anomaly and the objective angle (a combination of fusional vergence and sensory fusion). Hallden describes "a third component" which is not fusional in character and he suggests that this third component may be found in convergence of near vision. Kerr (1980) published a possible explanation for the accommodative response on disparate stimuli in subjects with strabismus. According to Kerr, subjects with strabismus and ARC have a neurological defect in fusional vergence control. A change in disparity causes fusional vergence. However, because of the anomalous correspondence, the proper feedback signal is missing. This makes disparity vergence an open-loop situation in subjects with ARC. The only mechanism available for subjects with strabismus and ARC to compensate for disparity is accommodative vergence, according to Kerr.

Summary
In studies on fusional vergence, published before 1970, we find sensory as well as motor fusion in patients with strabismus. After 1970 studies on fusional vergence, made with infrared detection, can be divided into two groups: First the studies with small central (foveal) stimuli and secondly the studies with peripheral stimulation. In the studies with foveal stimulation on subjects with strabismus an alternative response is described which resembles accommodative vergence. In the studies with peripheral stimulation subjects with strabismus may show fusional vergence which is comparable to normal fusional vergence. In all the studies now and then an absence of fusional vergence in strabismus is found. Different explanations are given, such as "changing the angle of anomaly" or "absence of disparity detection" or "prevent disparity information from being utilized". Obviously, in strabismus, together with the fusional vergence found on peripheral stimulation, a non-motor mechanism is functioning as well. If there is a difference between normal subjects and subjects with strabismus, it must be the presence of a non-motor component that can compensate for a disparate stimulus and only functions now and then.