Suppression in strabismus—an update

J. A. PRATT-JOHNSON AND G. TILLSON

From the Department of Ophthalmology, University of British Columbia, Vancouver, and the Department of Ophthalmology, Children's Hospital, Vancouver, BC, Canada

SUMMARY Previous reports have described suppression scotomas, suppression varying with the type of strabismus and suppression confined to one half of the retina (hemiretinal suppression). Our findings show that suppression in all varieties of strabismus, with the exception of the monofixation syndrome, involves the whole of the visual field of the deviating eye except for its monocular temporal crescent. In the monofixation syndrome our findings show a small central suppression scotoma involving the fovea but leaving the rest of the visual field of the deviating eye unsuppressed. We could find no evidence to support the concept of hemiretinal suppression but found evidence to support the presence of a trigger mechanism for suppression which operates on a hemiretinal basis.

The sensory adaptations of suppression, anomalous retinal correspondence (ARC), and amblyopia which occur in strabismus with onset during visual immaturity have been extensively studied by a variety of methods. The investigation of all these adaptations has been limited by the difficulty of providing test situations which are close to the patient's normal seeing conditions and yet allow the function of each eye, individually, to be assessed. This problem is particularly significant in the investigation of suppression, which is a binocular condition existing only when both eyes are open and existing only under the conditions for which it was developed during visual immaturity. For these reasons it is extremely difficult to devise experiments which permit the investigation of suppression without disrupting it.

Over the past few years we have been engaged in experiments to explore and map the area of the binocular field of vision that is involved in suppression in strabismus.

Previous reports suggest that suppression in esotropia differs from that in exotropia and that suppression is confined to one half of the retina.¹ Suppression scotomas have been demonstrated in the deviating eye, especially in esotropia.²

Our experiments do not support these concepts. Instead, they show that there is no difference between suppression in esotropia and in exotropia and that suppression is not confined to one half of the retina in either esotropia or exotropia. Our experiments

demonstrate the presence of a trigger mechanism for suppression. As a result of our experiments we have been able to look again at the literature on suppression and realise the differences between our findings and those of previous researchers result from the various testing methods used. A detailed description of the methods we used to explore and map the binocular field of vision in strabismic and nonstrabismic subjects, as well as our results, has already been published.³ The purpose of this paper is to collate our findings with those of previous authors to give a more complete picture of the area of the visual field of the deviating eye that is involved in suppression in strabismus.

Binocular field of vision

We have used the term 'binocular field of vision' throughout our investigations into suppression. The term is used to describe the total field of vision enjoyed by the subject with both eyes open while gazing steadily at one point. The term binocular field of vision does not necessarily imply bifoveal fixation.

With an Aimark perimeter it is possible to map the size of the binocular field of vision. These conditions are as close as possible to normal because the eyes are not dissociated in any way and nothing is placed between the patient and the test target. The patient maintains fixation on the central target of the perimeter while a 1 mm white spot is moved outward from the centre to the periphery along 8 meridians. The point at which the patient is no longer aware of the 1

Correspondence to Dr J. A. Pratt-Johnson, Children's Hospital, 4480 Oak Street, Vancouver, BC V6H 3V4, Canada.

mm spot is recorded. Strabismic patients fix throughout with their normally fixing eye.

The binocular field of vision in normal subjects corresponded to that described by many authors. There is a central area, common to both eyes, in which diplopia is avoided by fusion of images falling on corresponding points. On either side of this is an area which can only be seen by one eye and is called the monocular temporal crescent.

Using the Aimark perimeter the monocular temporal crescent of both eyes could easily be demonstrated in normal subjects. In strabismic patients it could be demonstrated only in the deviating eye. This is because normal subjects fix the central target on the perimeter bifoveally, whereas strabismic subjects fix the target only with the fovea of the nondeviating eye. The area of the monocular temporal crescent is determined by briefly occluding one eye and asking the patient if he can still see the peripheral target whilst maintaining straight ahead fixation. A strabismic patient would therefore be forced to assume fixation with his normally deviating eye at times and thus change the testing conditions.

The binocular field of vision in esotropia is narrower than in normals, as is the monocular temporal crescent of the deviating eye. In exotropia the binocular field of vision is wider than in normals, as is the monocular temporal crescent of the deviating eye. Swan⁴ has previously reported that the monocular temporal crescent of the deviating eye is not suppressed in strabismus. Indeed there is no need for it to be, since it has no correspondence with the fixing eye, nor does it receive any image of an object seen by the fixing eye. Patients with the monofixation syndrome⁵ have a binocular field of vision that is similar to that of normal subjects.

If a reliable strabismic patient indicated that he saw only one target in all areas of his binocular field of vision throughout the test, 3 possibilities existed. First, the image of the target fell within the monocular temporal crescent of vision of one or other eye. Secondly, the target image fell on the blind spot of either eye. Thirdly, the target fell on some other retinal area and was suppressed. Since only one image was seen throughout the test, all areas of the visual field, except the monocular temporal crescents and the blind spots, must have been suppressed. Therefore images falling on both nasal and temporal retinal areas must have been suppressed by this patient. This can be shown to occur in both esotropia and exotropia.

It has become the convention to refer to suppression in terms of retinal area involved—e.g., hemiretinal suppression. Since this convention seems to be firmly established and we shall be referring to various articles already published on this topic, we

shall follow the convention and refer to suppression of retinal areas rather than areas of the binocular field of vision.

Diplopia

Suppression is usually described as being developed in order to overcome confusion and diplopia caused by strabismus, but it is rare for a patient to complain of confusion or even to have noticed it initially. In a nonstrabismic subject the image of the same object normally falls on the fovea of each eye. If different images are presented to the foveas of each eye, for example by a haploscopic device, retinal rivalry results, and the 2 images are not perceived simultaneously; one or the other is suppressed. Similarly, in a strabismic subject the different images falling on the foveas of each eye are not perceived; the image falling on the fovea of the deviating eye is suppressed because, as in retinal rivalry the brain is unable to see simultaneously 2 different images on corresponding points. Thus it is diplopia and not confusion which is the most distressing phenomenon for the patient.

The Aimark perimeter may also be used to map the area of the binocular field of vision in which a subject is aware of diplopia. The subject can be either a normal made diplopic by the use of a base-down prism in front of one eye or someone with an acquired strabismus and diplopia. It is important to remember that what is being mapped on this occasion is the area of the binocular field of vision in which diplopia is appreciated and not the field of binocular single vision or binocular fixation, which maps the ability of the subject to maintain bifoveal fixation in the various directions of gaze. It can be shown that when diplopia is appreciated it involves all objects within the binocular field of vision at that time, except for those objects whose images fall on the blind spots or in the monocular temporal crescents. Thus both nasal and temporal retinal areas must be involved. Diplopia occurs wherever the visual fields of the 2 eyes overlap. Patients who acquire strabismus when they are visual adults see double of everything within that area of their binocular field of vision which is common to both eyes. They are naturally most keenly aware of diplopia of the main object of interest in their field of vision but, if questioned, will admit to also being aware of diplopia of surrounding objects, thus showing that both nasal and temporal areas of the retina are involved.

It would be logical to conclude that suppression in strabismus must occur in all the areas of the visual field in which diplopia occurs. Since it can be shown that diplopia occurs everywhere the visual fields of the 2 eyes overlap suppression would also have to occur everywhere the visual fields of the 2 eyes

overlap, thus involving both nasal and temporal retinal areas. If it did not do so but was only regional, as has previously been described, one would expect to find areas of the binocular field of vision in all strabismic patients where diplopia was experienced in the testing conditions previously discussed.

Experiments using dissociation by means of Polaroid filters have also shown that suppression involves both nasal and temporal retinal areas in esotropia and exotropia.⁶

Fusion

We further confirmed that both nasal and temporal retina is involved in suppression in all types of strabismus, by experiments using a Lees screen from which the Hess chart markings had been removed and fusion targets which were presented to known retinal areas. Although the method used involved dissociation of the eyes by a mirror and the subjects therefore were not really tested under normal conditions the results again showed suppression on both nasal and temporal retina in esotropia and in exotropia.

If fusion targets are used and are presented to a pair of corresponding points, one in each eye, suppression or fusion occurs. Responses are similar to those on a major amblyoscope except that with the Lees screen the area of the retina to which the images are presented can be determined by the researcher. In normal subjects fusion was elicited on all points tested. Subjects with the monofixation syndrome showed suppression only at the foveal/parafoveal area of the deviating eye with fusion on peripheral points. In all types of strabismus without fusion suppression was elicited on both nasal and temporal retinal areas. Only the temporal crescent, which has no correspondence with the fixing eye, was not suppressed in patients without fusion.

Suppression scotomas have previously been described using dissimilar targets—i.e., nonfusible targets and an adapted Lees screen.^{7,8} Different targets present different challenges to the visual system. If a patient with strabismus is tested by presenting an identical blank screen to each eye, there is no challenge to the visual system of either eye and so no need to suppress unless a test object is introduced. If the patient is made to fix a central spot on one screen with the dominant eye and then a test object is placed on the other screen so that it falls on a peripheral visual area of the deviating eye, there is still no need to suppress. This is because there is still no visual challenge and no possibility of diplopia. As soon as the target approaches the fovea of the deviating eye, suppression will occur, since there is now a conflict of interest from two dissimilar, non-

fusible targets falling on corresponding points. This has been explained by Linksz as a form of rivalry⁹ which prevents normals from seeing 'one thing in two places or two things in one place.' The object of main interest, that is the fixation object seen by the fovea of the dominant eye, prevails, and the image falling on the fovea of the deviating eye is suppressed. This explains why some previous experiments show a suppression scotoma localised to the general area of the fovea of the deviating eye.

Suppression scotomas have also been found at the 'diplopia point' of the deviating eye, which is that retinal point receiving the image of the object seen by the fovea of the fixing eye. This point would normally give rise to diplopia, hence the name 'diplopia point.' These scotomas have also been explained by Linksz as resulting from the same form of rivalry. In this case the second image, which is formed on an extrafoveal retinal area in the deviating eye, is less clear and is suppressed. The image falling on the fovea of the fixing eye dominates.

The difference between our findings and those previously described can be attributed to the different types of targets used and to their method of presentation.

We could find no evidence to support Jampolsky's concept of hemiretinal suppression in either esotropia or exotropia. Jampolsky¹⁰ found that, so long as the image of the fixation object in a deviating exotropic eye fell on temporal retina, suppression occurred, and that if the image was then made to cross the vertical hemiretinal line to fall on nasal retina (by the use of prisms) the patient became aware of diplopia. Our experiments confirm these findings but not his conclusions. He concluded that suppression is confined to one half of the retina in strabismus and said that, 'In exotropia the temporal retinal suppression involves the whole of the hemi-retina regardless of the degree of exotropia.' This seems to imply that even the monocular temporal crescent is involved, whereas both our work and that of Swan has shown that this area is not suppressed. If the binocular field of vision of an exotropic patient is explored on the Aimark perimeter, while the patient is aware of diplopia, it can be shown that both nasal and temporal areas are involved—i.e., diplopia occurs everywhere the visual fields of the 2 eyes overlap. Similarly, when suppression occurs, it is not hemiretinal but involves both nasal and temporal retina, only the monocular temporal crescents are not involved.

Trigger mechanism

We found evidence to support the presence of a trigger mechanism for suppression which operates on a hemiretinal basis in strabismus that has been

Fig. 1 The hemiretinal trigger mechanism. (A) Esotropia—suppression of right visual field where it overlaps that of the fixing left eye. (B) Same patient made exotropic—diplopia everywhere the visual fields overlap.

Fig. 2 The hemiretinal trigger mechanism. (A) Exotropia—suppression of right visual field where it overlaps that of the fixing left eye. (B) Same patient made esotropic—diplopia everywhere the visual fields overlap.

present from visual immaturity. Suppression is developed during visual immaturity to overcome diplopia caused by strabismus. The image of the fixation object always falls on the same half of the retina—i.e., nasal retina in esotropia (Fig. 1A) and temporal retina in exotropia (Fig. 2A). Suppression develops in response to this. So long as the image of the fixation object continues to fall on the same half of the retina of the deviating eye suppression will continue to occur—i.e., suppression will be triggered. The important point to remember, however, is that the resulting suppression is not confined to one half of the retina but involves both nasal and temporal retina. If the strabismus is overcorrected, either by prisms or by surgery, the image of the fixation object crosses the vertical retinal midline and now falls on the opposite half of the retina in the deviating eye (Figs. 1B, 2B). Diplopia is immediately triggered, and once again both halves of the retina are involved. This is a new situation. The patient has never before had to suppress under these circumstances. Of course if the patient is still visually immature, a new pattern of suppression can be developed in response to this new situation. If the patient is a visual adult, however, suppression cannot be developed, and diplopia will persist as long as the overcorrection persists. Only making the image of the fixation object recross the vertical retinal midline in the deviating eye, by prisms or further surgery, will allow the trigger mechanism to operate and restore suppression to both nasal and temporal retinal areas. This crossing of the vertical midline of the retina operates a trigger mechanism which determines on

the basis of past experience whether diplopia or suppression results.

The retinal midline divides temporal retina and one side of the brain from nasal retina and the opposite side of the brain. It is possible that this is the physiological basis for the hemiretinal trigger mechanism, since, as the image of the fixation object crosses the vertical retinal midline first, one side of the visual cortex and then the other is stimulated.

In other words, whenever the visual fields of the 2 eyes overlap in strabismus the patient will appreciate diplopia or he will suppress. This is an all or nothing phenomenon that involves both nasal and temporal retina and is not confined to just one half of the retina. The hemiretinal trigger mechanism determines whether suppression or diplopia occurs. The trigger is activated by the image of the fixation object crossing the vertical hemiretinal midline from one side of the retina to the other.

We do not dispute the results of tests described by other authors, only the interpretation of their results. We suggest that suppression is not confined to one half of the retina nor does it differ in esotropia and exotropia. Suppression scotomas in patients without fusion appear to be artefacts caused by the method of investigation used.

Strabismic suppression involves both nasal and temporal retina except in the monofixation syndrome, where the deviation is small enough to allow peripheral fusion to occur in the presence of a foveal/parafoveal suppression area in the deviating eye. Suppression is controlled by the hemiretinal trigger mechanism.

References

- 1 Jampolsky A. Characteristics of suppression in strabismus. *Arch Ophthalmol* 1954; **54**: 683–96.
- 2 Travers TaB. Suppression of vision in squint and its association with retinal correspondence and amblyopia. *Br J Ophthalmol* 1983; **22**: 577–604.
- 3 Pratt-Johnson JA, Tillson G, Pop A. Suppression in strabismus and the hemi-retinal trigger mechanism. *Arch Ophthalmol* 1983; **101**: 218–24.
- 4 Swan K. False projection in strabismus. Alleviations by anomalous retinal correspondence. *Arch Ophthalmol* 1965; **73**: 189–97.
- 5 Parks MM. The monofixation syndrome. *Trans Am Ophthalmol Soc* 1969; **67**: 609–57.
- 6 Pratt-Johnson JA, MacDonald AL. Binocular visual field in strabismus. *Can J Ophthalmol* 1976; **11**: 37–41.
- 7 Pratt-Johnson JA, Wee HS, Ellis S. Suppression associated with esotropia. *Can J Ophthalmol* 1967; **2**: 284–91.
- 8 Pratt-Johnson JA, Wee HS. Suppression associated with exotropia. *Can J Ophthalmol* 1969; **4**: 136–44.
- 9 Linksz A. *Physiology of the eye*. New York: Grune and Stratton, 1952: 2.
- 10 Jampolsky A. A simplified approach to strabismus diagnosis: in symposium on strabismus. *Trans New Orleans Acad Ophthalmol*. St Louis: Mosby, 1971.