Detection and management of squint

Aetiology and clinical features

Strabismus is always due to some defect in the binocular fixation reflexes that normally enable the 2 eyes to work together to perceive a single stereoscopic view of the visual panorama. This complex process quite frequently breaks down or fails to develop, accounting for the incidence of squint in some 3% of children. Abnormality of the sensory, motor, or central coordinating parts of the reflex may cause squint but only motor defects will limit the range of ocular movements.¹

An incomitant squint may be caused by nerve palsy or mechanical limitation of movements due to muscle fibrosis or orbital pathology. The angle of motor squint changes as the eyes move and they may still be parallel in certain directions of gaze, the patient adopting a compensatory head posture to make maximum use of this residual area of binocular single vision. As the head can be straightened passively this should not be confused with true torticollis.

If the vision of one or both eyes is poor there is little stimulus to achieve binocular single vision and a strabismus may develop. Such a sensory squint differs from motor strabismus in that full ocular movements are retained and the squint is concomitant—its angle remains the same in all directions of gaze. Because of strong accommodation most childhood sensory squints are convergent but a constant concomitant uniocular divergent squint in infancy is nearly always the result of poor vision.

Childhood squint is commonly caused by some abnormality of the central coordinating mechanisms. Concomitant strabismus may result from neurological deficit or more usually from refractive error. Many young children are hypermetropic, and as their eyes are short the images of fixated objects are focussed behind the retina. This defect can be overcome by accommodation but as this process is accompanied by convergence, especially on near fixation, a concomitant convergent squint may develop. Typically such a squint first appears after age 12 months, initially on near fixation only and when the child is tired. There is often a family history of squint and the squint becomes more constant and severe. Such a true squint is thus fairly easy to distinguish from apparent squint that is an optical illusion caused by epicanthic folds which by obscuring the sclera give the impression of a con-

vergent strabismus. This false squint is most marked in early infancy and becomes less noticeable as the face grows.

Young children rapidly overcome the diplopia and confusion caused by strabismus by suppressing the central vision of the deviating eye. If the squint is constant and uniocular this quickly proceeds to the development of amblyopia and vision remains poor even when the normally fixating eye is occluded. This process is irreversible after age 6–7 years. Similarly, recovery of normal binocular single vision after treatment is unlikely once a squint has been constantly present for more than a few months, especially if the onset was before the age of 2–3 years when the normal binocular reflexes have not been fully developed. Once a true strabismus develops it virtually never resolves spontaneously.

Identification and management

The most important aims of management, in order of priority, are:

- 1) To ensure that the squint, either motor or sensory, is not symptomatic of ocular or neurological disease.
- 2) To identify and treat any amblyopia before it becomes irreversible.
- 3) To promote or restore binocular function and if that is not possible to produce at least a cosmetically satisfactory appearance.

The important aspects of history taking, the importance of looking for external evidence of eye disease, for compensatory head postures, and epicanthic folds have been discussed. From age 3 years most children will cooperate in illiterate Snellen type vision tests such as the Sheridan-Gardiner STYCAR letter matching test or the 'E game'. These testing methods are more accurate if lines of capitals rather than single letters are used. Single letters are easier to identify and the presence of a moderate degree of amblyopia induces no identifiable difference in the visual acuity of the 2 eyes. In younger or less cooperative children reliance must be placed on simpler clinical tests. Does the child always fix an interesting visual target with the same eye and lose steady fixation altogether when that eye is covered? This is strong evidence of a unilateral visual defect.

The presence of manifest squint may be assessed very simply, although not entirely accurately, by

looking at the reflections of a fixation light on the cornea of each eye. If both eyes are fixing correctly, each small bright reflection will be centrally placed. With a convergent squint the reflection is displaced towards the temporal corneal margin and if it is right at the margin the angle of squint is about 45° . Both the direction and the angle of strabismus can therefore be roughly tested in infants using this quick method. Much more accurate but more difficult is the cover test. The eye suspected of squinting is observed while the child's attention is attracted by an interesting fixation target such as a mobile toy. The fixing eye is then covered with an occluder such as a piece of card; the other eye will have to move to take up fixation and the direction and degree of this movement is noted. To induce an accommodative convergent squint, the child must be stimulated to look intently at a small near target. It is as well to have a series of toys available and to substitute a new one each time attention is lost. The range of ocular movements is then noted in all the different directions of gaze and any variation in the angle of squint is ascertained by repeated cover testing in each position.

If a sensory squint is suspected, the pupillary light reflexes should be tested. These are normal if an eye has poor vision due to amblyopia, but defective if there is disease of the retina or optic nerve causing poor vision. Disease may be confirmed by thorough ophthalmoscopy with the pupils dilated using a short acting mydriatic such as cyclopentolate (0.5%) or tropicamide (1%).

Once the examiner is convinced that a true squint

is present, the child should be referred without delay to a specialist. As squints may be indicative of disease and may well require surgical correction, children with strabismus should never be referred to opticians. Most children will tolerate spectacles for refractive correction and eye patches for amblyopia treatment from the age of 18 months or earlier. Reluctance to do so is often due to the parents' lack of determination and they need to be convinced at the earliest stage of the essential nature of the treatment prescribed. Squint surgery is straightforward and can be carried out at any age. It often needs to be done in more than 1 stage and except in squints of late onset (after 3 years) is rarely fully curative.²

The temptation to delay referral comes from the difficulty with many young children of being really certain of the diagnosis. But although clinical medical officers must do their very best to learn to exclude apparent squints caused by epicanthic folds, if there is real doubt hospital referral should not be postponed. The possible dangers of delay far outweigh the inconvenience of occasionally unnecessary hospital examination.

References

- ¹ Parks MM. *Ocular motility and strabismus*. Hagerstown: Harper & Row, 1975.
- ² Harcourt B. Squint. In: Lock S, ed. *Problems of childhood*. London: British Medical Association, 1976:105-10.

R B HARCOURT
45 Park Square,
Leeds LS1 2NP