tion corrected by prisms for comfort. This rule of thumb is too dogmatic to be of value. In acquired deviations it is often necessary to give almost the whole dissociated measurement in prisms, but in long-standing deviations a minority require as much as two-thirds, and it is striking that most need half and some less than one-third of the dissociated prism cover test measurement.

It would seem, therefore, that prisms are an effective method of relieving symptoms, with advantages far outweighing their disadvantages. In this series they have been used with success in a number of conditions in addition to relatively straightforward heterophorias and acquired and congenital palsies, including patients with: (1) Manifest squint and diplopia in whom no sensory or motor adaptation has been possible, for example consecutive divergence. In these cases prisms have been used to put the image back on to a suppression area. (2) Unilateral or bilateral aphakia. (3) Anisomyopia of high degree (the heavy eye phenomenon). (4) Nystagmus in addition to heterophoria. (5) Diplopia following detachment surgery. (6) Certain orbital injuries. (7) Exophthalmic ophthalmoplegia.

This form of therapy is particularly suitable for the last condition. Patients must necessarily be kept under observation for a very long period before the condition is sufficiently static for surgery, and it is difficult to decide when this stage has been reached. Patients are attending, for example, who have been euthyroid for two or more years before presenting with diplopia.

As well as relieving diplopia and the discomfort from an abnormal head posture, prisms in this condition have other advantages: (1) There is ample evidence that these patients develop a large fusion range. By allowing binocular single vision, prisms help the development of this range, which might be prevented by the alternative of occlusion. The proof of this can be seen in a number of patients who eventually manage with less or no prism. (2) The limitation of movement in many cases is bilateral, and occlusion is therefore very difficult to tolerate, whereas a binocular field is invaluable.

Two further arguments for prisms can be advanced:

(1) Although six months is usually regarded as sufficient time lapse before operating on static acquired conditions, several patients in this series (excluding those with thyroid disease) have shown a delayed improvement occurring several years after the onset. Therefore, a longer period of conservative treatment may be advisable.

(2) Prisms may minimize or obviate the need for an abnormal head posture which is often advocated as a means of compensating for a deviation, but may itself cause discomfort and, more important, may eventually cause arthritic changes in elderly patients.

Binocular single vision is important and it is the aim of any form of treatment of squint to produce it: an individual's dependence upon it is increased if he is ill, as many of these patients are; if he is working, and needs binocular vision to perform or, more important, keep his job; if he is handicapped in other ways, e.g. elderly people, those with difficulty in walking, with low visual acuity, or a field loss, or with bilateral limitation of ocular movement.

Given that it needs co-operation on the part of the patient to make prisms a success, it is suggested that a large number should at least be given an opportunity to gain binocular vision by this means.

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Mr C T Hart
(*Royal Infirmary, Sheffield*)

Disturbances of Fusion following Head Injury

This report is based on a series of patients who have lost fusion or suffered disturbances of fusion after head injury of varying grades of severity; some of them have subsequently regained this function. The series is small but has been collected from the records of the orthoptic department of the Sheffield Royal Infirmary over the past ten years. Fusion loss is defined as failure to demonstrate the patient's ability to maintain a single fused image in any circumstances, i.e. on the major amblyoscope, with prisms, or in normal life in any position of gaze. All the patients who had lost fusion after injury complained of constant diplopia with overlapping of images when tested with prisms, or superimposition of images when tested with varying size fusion and stereoscopic slides on the major amblyoscope.

The varieties of trauma suffered by the patients have included: gross cerebral contusion associated (1) with fracture of the skull (7 cases) and (2) with closed head injury and hæmatoma formation (4 cases); whiplash injury (2 cases); electrical injury (1 case); and local ocular trauma (1 case). These groupings are only approximate, particularly as concerns the open and closed injuries – one patient with gross fracture of his frontal bones required orbital roof repair and excision of prolapsed forebrain amounting to a pre-frontal leucotomy.

The post-traumatic state of fusion has been divided into the following categories: (1) Complete loss of fusion with no recovery (6 cases). (2) Complete loss of fusion with full recovery, including stereopsis (5 cases). (3) Reduced fusion range with limited stereopsis (4 cases) (normal fusion range: horizontal 25Δ prism base out, 6Δ prism base in).

The methods of investigation for fusion in the orthoptic department have largely comprised the extensive use of prisms, the major amblyoscope with use of large and small fusion and stereoscopic slides, and the Wirt stereotest (Vectograph) when it appeared that some fusion was possible, i.e. categories (2) and (3) already referred to. Obstacles to eliciting fusion include torsion, field loss, nystagmus, pupillary inequality with possible resultant uniocular accommodative disparity, muscle palsy and suppression. None of these appears to act as an absolute barrier to fusion since the mechanism of peripheral or central fusion can compensate in the presence of intact central nervous system pathways.

It is assumed in the absence of evidence to the contrary that these patients possessed normal binocular single vision prior to their injuries. All the patients were fully co-operative when they attended for ophthalmic and orthoptic examination, the majority having been discharged from hospital for some weeks. The age given with each patient is that at first clinic attendance.

Case Histories

Case 1 C H aged 21. Road traffic accident, unconscious 2–3 days. RV 6/12, LV 6/6. X-rays: linear fracture right frontal bone. Right VII, VI and partial III nerve palsies. No field defect. No fusion.

Case 2 T W aged 21. Mining accident. RV 6/9, LV 6/24. Fracture frontal bones with repair of both orbital roofs and evacuation of prolapsed frontal lobe. Left ptosis due to severance of frontalis and levator. No fusion.

Case 3 S H aged 63. Injury to left eye with blunt object at work. Patient subsequently complained of constant horizontal diplopia. RV 6/9, LV 6/12. No fusion. ? Compensation neurosis.

Case 4 G C aged 26. Road traffic accident. RV 6/18, LV 6/6. Comminuted fracture right frontal sinus repaired. Right partial III nerve palsy with optic atrophy and marked field loss. RDS – corrected with surgery. Twelve months interval before fusion with full stereopsis regained although deterioration to RV 6/36.

Case 5 A N aged 16. Climbing accident. RV 6/5, LV 6/5. Fracture left parietal region. LDS with L/R. Manifest horizontal nystagmus with vertical nystagmus on elevation. Bilateral limitation of elevation and abduction. Three months interval before fusion regained.

Case 6 D N aged 19. Road traffic accident. RV 6/5, LV 6/4. Left temporal lobe hæmatoma. Right superonasal quadrantanopia. Right optic atrophy. Nine months interval before fusion regained.

The next patient differs from those previously described in that the loss of fusion occurred approximately one year after the cerebral trauma and was preceded by loss of convergence.

Case 7 J H aged 41. Road traffic accident. RV 6/5, LV 6/5. No fracture but prolonged retrograde amnesia with initially right hemiparesis. Bilateral IV nerve palsies. Dilated sluggish right pupil with defective accommodation. Fusion and stereopsis present. Ten months later the patient complained of constant horizontal diplopia. Momentary fusion was present with no range. One month after no fusion was demonstrable and he did not regain full fusion for two years.

Case 8 D C aged 37. Fork lift truck fell on patient. RV 6/5, LV 6/4. Vertical fissure fracture right frontal region. Right VI and VIII nerve damage. Markedly reduced fusion range. Stereotest: fly only.

Discussion

Disturbance of fusion after head injury has received very little attention in the ophthalmic literature, and this is surprising in view of the increasing numbers of head injuries due to traffic or industrial accidents. Taylor (1967) quotes a figure of 75,000 new post-traumatic cases each year. Wade (1965) described 7 patients who had lost fusion, noting that their intellect was impaired after severe head injury and postulating that fusion loss and intellectual impairment were related. This finding has not been confirmed in the present small series, judging from the response to tests and easy understanding of them, apart from one patient who suffered loss of part of his frontal lobes. Doden & Bunce (1965) reported on a series of 79 patients with cerebral trauma and found a reduction of the horizontal fusion range: in 24 patients there was eventual normalization of fusional amplitude after orthoptic treatment (28° Lyle); no loss of fusion was noted. In the same article Doden & Bunce surveyed the Continental literature on fusion abnormalities after head injury but the information obtained appears limited to short series of cases, and only included those with a diminished fusion range.

Anderson (1961) reported on 5 typical cases of loss of accommodation and convergence following whiplash injury to the cervical spine; loss of fusion following this injury does not appear to have been previously documented.

Dodwell & Engel (1963) in a paper entitled 'A Theory of Binocular Fusion' stated that the fusion process can be thought of as essentially dependent on the coding of information about the fine grain structure of the disparate stimuli, and the 'matching' of this information from the two eyes with the subsequent analysis or interpretation in terms of pattern and depth. It is this latter function which appears to be lost in the present cases and is presumably affected at higher cortical level.

Micropathological studies on cortical white matter obtained from 'concussed' rats has demonstrated the fracture of white fibres (Biggart 1936) similar to those seen in patients dying after head injury (Nevin 1964). The degree of cerebral injury suffered by the majority of patients in the present series can be considered to have produced gross abnormalities in local capillary blood flow, permeability and neuronal organization. Although no specific injury would appear to account for loss of fusion, it is reasonable to suppose that association tracts or commissural bundles are disrupted either permanently or, in the case of those patients who have subsequently regained fusion, temporarily. It is important to stress that in the present cases precise site of injury could not be determined, the cerebral insult being both severe and diffuse. Venables (1967) described the finding of a diminished fusion range in three different groups of minimally handicapped children in whom some damage to the central nervous system was presumed to be the underlying cause, the percentage with a reduced fusion range being far greater than would be expected in a normal group of children.

The greater recognition of this failure of fusion after head injury is important, both for a greater understanding of the fusion process itself, and for the patient, who may well be labelled as having a compensation neurosis and left permanently with occlusion, when in fact recovery may occur after a long interval.

Acknowledgments: I wish to thank Mr A Stanworth and Mr C A L Palmer for permission to use their cases.

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Mr C A L Palmer
(Royal Infirmary, Sheffield)

The Surgery of the Inferior Rectus Muscle [*Abstract*]

The anatomy of the inferior rectus muscle was briefly reviewed with special reference to the important fascial arrangements in the lower part of the orbit.

In the surgical management of superior oblique muscle palsy, adjustment of the over-acting contralateral inferior rectus muscle may be employed and the various procedures were reviewed (simple recession, marginal myotomy, and realignment of the insertion).

Five patients with superior oblique palsy in whom recession of the over-acting contralateral synergist, the inferior rectus, was followed by reversal of the diplopia and an appearance on the Hess chart of the induced superior oblique paresis on the operated side, were described. The possible explanations for this phenomenon were discussed.

In dysthyroid states, fibrous contracture of the inferior rectus muscle with downward deviation of the eye is a common finding. This phenomenon was discussed in relation to complex fascial arrangements in the lower part of the orbit. The surgical management of these dysthyroid hypotropias involves the demonstration of fibrotic contracture in the lower orbit by the traction test. If confirmed, the contracted inferior rectus muscle must be released either by dissection and recession or by free tenotomy.

The phenomenon of retraction of the lower eyelid after free tenotomy or recession of the inferior rectus muscle was discussed and attributed to the fascial connexion between the two structures. Cases of vertical deviation in dysthyroid states were described.

The following paper was also read:

Surgical Management: The A and V Phenomena

Mr A Stanworth

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