Traumatic third nerve palsy

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SUMMARY  Twenty patients with a traumatic third nerve palsy had sustained a closed head injury with prolonged loss of consciousness in a high-speed deceleration accident. Sixteen were male, and the average age was 25 years. Seven had skull or facial fractures, 15 damage to the anterior visual pathways, and 16 other permanent neurological damage. Nineteen developed the misdirection/regeneration syndrome. Thirteen had strabismus surgery, and an area of binocular single vision was enlarged or achieved in three.

Serious head injury in road traffic accidents is a major cause of death and morbidity in young adults. The third cranial nerve may be damaged either directly as a result of the injury, or indirectly due to compression from an expanding extradural or subdural haematoma. Recovery to complete normality is unusual, and the frequency of the development of the misdirection/regeneration syndrome makes attempted surgical correction difficult. Twenty cases of direct traumatic third nerve palsy were investigated to establish the severity of their head injury and permanent neurological deficit. The results of surgical and other management were assessed.

Subjects and methods

The patients were referred by ophthalmologists and neurosurgeons over a four-year period (1979–83) because of ocular motility problems following a traumatic third nerve palsy. They were 20 consecutive cases, first seen at times ranging from three days to several years after the accident. All had suffered a direct third nerve injury, substantiated by the time course of the clinical signs—immediate onset following trauma—and radiological exclusion of an expanding supratentorial lesion in equivocal cases.

The following information was recorded: (1) Age at time of trauma; (2) sex; (3) nature of accident; (4) length of loss of consciousness; (5) presence and type of skull or facial bone fracture; (6) the function of: (a) the ipsilateral fourth and sixth nerves; (b) the visual pathways, assessed by best corrected visual acuity (Snellen chart), colour vision (Ishihara chart),

aff erent pupillary defects, visual fields (Goldmann perimetry, augmented in certain cases by visual evoked potential recordings (VEPS)), optic disc pallor; (c) function of the remaining cranial nerves, and of the central and peripheral nervous system, including intellectual function; (7) the development of the misdirection/regeneration syndrome, and any recovery of binocular function; (8) surgical treatment.

Results

Direct traumatic third nerve palsy was shown to be an injury of young adults (Fig. 1) in car or motorcycle accidents. The majority of patients were male (16 males, 4 females). The two patients under 10 years of age were unrestrained front seat passengers in cars.

The types of accident were as follows: (1) Road traffic accident 19 (car 13, motorbike 6); (2) hang-gliding accident 1; total 20.

![Fig. 1 Age in years at time of injury.](http://bjo.bmj.com/)

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Eight patients sustained a skull or facial bone fracture, the details of which appear in Table 1. All were rendered unconscious by the accident for periods ranging from a matter of hours to a maximum of six weeks (Fig. 2).

All fractures were on the side of the third nerve palsy. Pretraumatic amnesia for hours or days was usual and in one case it extended to two years.

A diagnosis of traumatic optic neuropathy was made if the best corrected visual acuity was 6/9 or less in the presence of at least one confirmatory physical sign—reduced colour vision, afferent pupillary defect, visual field defect, or optic disc pallor—and provided there was no ocular or other explanation such as amblyopia for the poor vision. The diagnosis was made in two cases when the vision was 6/6, when two confirmatory physical signs were present. One patient was diagnosed by these criteria as having bilateral traumatic optic neuropathy. Table 2 shows the visual acuity and visual fields in the 15 cases in which the optic nerve ipsilateral to the third nerve palsy was damaged.

Visual field defects ranging from overall constriction (Fig. 3) to a central scotoma with or without

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Table 1  Skull or facial bone fracture

<table>
<thead>
<tr>
<th>Bone</th>
<th>Number</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maxilla</td>
<td>3</td>
<td>(1 had ipsilateral ethmoid fracture)</td>
</tr>
<tr>
<td>Zygoma</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>2</td>
<td>(1 extending into base of skull)</td>
</tr>
<tr>
<td>Base</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

Table 2  Visual acuity and visual fields in ipsilateral optic nerve lesions (15 cases)

<table>
<thead>
<tr>
<th>Description</th>
<th>Number</th>
<th>6/6</th>
<th>6/9</th>
<th>6/12</th>
<th>6/18</th>
<th>6/24</th>
<th>6/36</th>
<th>6/60 or less</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>6</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constricted</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central scotoma alone</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>—with bitemporal hemianopia</td>
<td>3</td>
<td></td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>—with quadrantanopia</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Fig. 2  Length of loss of consciousness in days following injury.

Fig. 3  Goldmann perimetry. Constricted visual field left eye: left IIIrd nerve palsy.
peripheral abnormalities were found. The six patients with normal fields and the three with generalised constriction had normal or relatively well preserved visual acuity. The vision was severely reduced in the others, three of whom had a bitemporal field defect indicating trauma to the chiasm (Fig. 4). One case of right third nerve palsy had a right upper quadrantanopia with bilateral optic atrophy, indicating left optic tract damage (Fig. 5).

There were six cases of ipsilateral fourth nerve palsy, associated in one case with a sixth nerve palsy on the same side: this patient had a facial palsy and anaesthetic cornea associated with basal skull fracture. One other case of ipsilateral sixth nerve palsy was diagnosed, and one patient had ipsilateral nerve deafness and anosmia (Table 3).

Long tract signs ranging from mild sensory loss to significant hemiparesis were found in five patients, all

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Fig. 4 Goldmann perimetry. Bitemporal field defect, right central scotoma: right IIIrd nerve palsy.

Fig. 5 Goldmann perimetry. Right upper quadrantanopia: right IIIrd nerve palsy.
Table 3 Cranial nerve palsies

<table>
<thead>
<tr>
<th>Cranial nerve</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior visual pathways</td>
<td>15</td>
</tr>
<tr>
<td>Troclear</td>
<td>6</td>
</tr>
<tr>
<td>Abducent</td>
<td>2</td>
</tr>
<tr>
<td>Facial</td>
<td>1</td>
</tr>
<tr>
<td>Auditory</td>
<td>1</td>
</tr>
<tr>
<td>Olfactory</td>
<td>1</td>
</tr>
</tbody>
</table>

of whom had anterior visual pathway damage, and two of whom had a skull or facial bone fracture. Post-traumatic epilepsy developed in three cases. A personality change characterised by euphoria, lack of inhibition, and poor short-term memory occurred in four cases. One of these patients became physically aggressive. A previously normally developed child whose head injury was associated with subarachnoid haemorrhage and fits has subsequently developed a hypopituitary syndrome. Neuroradiological investigation has revealed no other cause such as a space occupying lesion, and it is suggested that the trauma is responsible. He has an ipsilateral optic nerve lesion but no chiasmal defect (Table 4).

The third nerve misdirection/regeneration syndrome developed in 19 cases. All showed lid elevation on attempted use of the medial rectus and inferior rectus (pseudo von Graefe sign) and 13 had pupillary constriction on adduction or downgaze. Adduction or globe retraction on attempted upgaze was seen in four cases. In the remaining case partial recovery of normal third nerve function took place (Table 5).

Four patients recovered an area of binocular single vision in downgaze, and in two others fusion was demonstrable when the angle of squint was corrected with prisms. The remaining 14 had developed suppression of the image from the deviating eye and were not troubled by diplopia.

Thirteen patients have had strabismus surgery, five having had two or more operations. These were 14 procedures on the affected eye and eight on the contralateral eye (Table 6).

The area of binocular single vision was enlarged or transferred to a more useful position in two of the four patients in whom it was present preoperatively. (Surgery was not attempted in the other two cases.)

Table 4 Permanent neurological sequelae

<table>
<thead>
<tr>
<th>Description</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long tract signs (sensory or motor)</td>
<td>5</td>
</tr>
<tr>
<td>ipsilateral to third nerve lesion</td>
<td>3</td>
</tr>
<tr>
<td>contralateral</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>5</td>
</tr>
<tr>
<td>Post-traumatic epilepsy</td>
<td>3</td>
</tr>
<tr>
<td>Personality change</td>
<td>4</td>
</tr>
<tr>
<td>Pituitary/hypothalamic damage</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 5 Misdirection/regeneration syndrome

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lid elevation on adduction/downgaze</td>
<td>19</td>
</tr>
<tr>
<td>Pupillary constriction on adduction/downgaze</td>
<td>13</td>
</tr>
<tr>
<td>Adduction/retraction on upgaze</td>
<td>4</td>
</tr>
</tbody>
</table>

Table 6 Surgical procedures

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Ipsilateral ocular muscles</td>
<td></td>
</tr>
<tr>
<td>Horizontal recession and resection</td>
<td>5</td>
</tr>
<tr>
<td>Horizontal recession and resection and supero-</td>
<td>4</td>
</tr>
<tr>
<td>position of insertions</td>
<td></td>
</tr>
<tr>
<td>Vertical recession or resection</td>
<td>1</td>
</tr>
<tr>
<td>Superior oblique tendon transposition</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
</tr>
<tr>
<td>B. Contralateral ocular muscles</td>
<td></td>
</tr>
<tr>
<td>Horizontal recession and resection</td>
<td>2</td>
</tr>
<tr>
<td>Horizontal recession and resection and posterior fixation</td>
<td>2</td>
</tr>
<tr>
<td>Vertical recession or resection</td>
<td>3</td>
</tr>
<tr>
<td>Vertical recession and posterior fixation</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>8</td>
</tr>
<tr>
<td>C. Lid</td>
<td></td>
</tr>
<tr>
<td>Levator recession</td>
<td>3</td>
</tr>
<tr>
<td>Lateral tarsorrhaphy</td>
<td>1</td>
</tr>
<tr>
<td>D. Other</td>
<td></td>
</tr>
<tr>
<td>Faciomaxillary</td>
<td>4</td>
</tr>
</tbody>
</table>

An area of single vision in downgaze was achieved after two procedures in one of the two patients in whom fusion was demonstrated preoperatively; the other 10 patients were improved preoperatively (Table 7).

Discussion

The third nerve is damaged in fatal, high-speed closed head injury by either avulsion from the mesencephalon, primary contusion necrosis, or intra- and perineural haemorrhage in the subarachnoid space. Damage to the anterior visual pathway lesions, for instance from haemorrhage into the optic nerve sheath, and chiasmal damage may also be found. The severity of the head injuries reported in this study, indicated by the length of unconsciousness and permanent neurological deficits, indicates that

Table 7 Surgical results: 13 patients, 27 procedures

<table>
<thead>
<tr>
<th>Preoperative categories</th>
<th>No. of BSV expanded or achieved</th>
<th>Cosmetic improvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Area of BSV recovered</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Fusion demonstrated</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Suppression demonstrated</td>
<td>14</td>
<td>9</td>
</tr>
<tr>
<td>Totals</td>
<td>20</td>
<td>13</td>
</tr>
</tbody>
</table>

BSV=binocular single vision.
similar mechanisms may be responsible in these patients. The frequency of involvement of the ipsilateral fourth and sixth nerves, in the context of third and optic nerve lesions, suggests that the damage occurs at the anterior end of the middle cranial fossa in relation to the body and wings of the sphenoid bone. The decelerating force is transmitted to this region by the frontal, zygomatic, and maxillary bones, which take the initial impact and are frequently fractured (8/20 cases). The third, fourth, and sixth nerves are in close anatomical relationship at the anterior end of the cavernous sinus, and the impact stretches and distorts these structures, disrupting their delicate pial blood supply. The optic nerve lies above and medially and is particularly vulnerable to indirect trauma in the optic canal, where it is tethered to bone. The pathological correlate in fatal cases is normally haemorrhage into the nerve and its surrounding subarachnoid space. The chiasm is probably also damaged by this mechanism, branches of the anterior cerebral and anterior communicating arteries being sheared by the inertia of the cerebral hemispheres moving forwards at the moment of impact.

The severity of the head injuries in these patients is emphasised by the evidence of focal neurological damage outside the visual system, such as long-tract signs and post-traumatic epilepsy. Eight of the 20 patients were unconscious for more than one week, and four of these suffered personality change and intellectual deterioration probably attributable to factors complicating concussion, such as cerebral laceration, oedema, or raised intracranial pressure. The damage to the optic tract in one case and the hypothalamic-pituitary dysfunction in another may be due to these factors or secondary to the disruption of the blood supply.

The misdirection/regeneration syndrome was diagnosed in 19 cases. The lid sign developed in all, the pupil sign in 13/19 cases (see Table 5). It is difficult to ascribe these consistent physical signs to random axon regrowth, and it seems that the re-establishment of function of the traumatised third nerve is not adequately explained by misdirection of regenerating peripheral nerve fibres.6 The pupil sign, for example, would require an axon originally ending in the medial or inferior rectus to innervate the neuroectodermal smooth muscle of the iris, and such a neuromuscular junction would probably not be functional. A central mechanism seems more plausible, since retrograde chromatolysis follows axonal injury, and the organisation of the cell bodies and synapses of the third nerve nucleus must be disrupted by this. When third nerve recovery takes place, only about 50% of the axons regrow,9 and the central synaptic disruption may persist. Such an alternative hypothesis is supported by cases of primary misdirection/regenera-

tion syndrome occurring in the absence of acute third nerve palsy. The alternative label of acquired oculomotor synkinesis is therefore preferred.10

Severe closed head injury may be responsible even in the absence of a third, fourth, or sixth nerve palsy for loss of fusion. The mechanism is unknown but probably the result of diffuse upper midbrain neuronal damage.11 In patients with, in addition, a third nerve palsy and abnormal accommodation, visual field defects, particularly bitemporal hemianopia,12 make the recovery of fusion unusual. Four patients in this series nevertheless spontaneously recovered a small area of binocular single vision in downgaze, and two others had fusion when the angle of the squint was corrected. Table 8 suggests that these patients form an identifiable subgroup, having a less serious head injury assessed by length of loss of consciousness and little or no damage to the visual pathway.

These patients can be managed surgically with the aim of either enlarging the existing area of single vision and improving its position (achieved in 2/2 cases) or creating an area of single vision (achieved in 1/2 cases). The surgical techniques involved include the use of posterior fixation sutures on the contralateral eye to increase innervational drive to the yoke muscle13 and adjustable sutures, particularly in the correction of the vertical deviation. Ptosis surgery may also be required.

For most patients, however, the sensory fusion mechanism is disrupted. Any surgery in these patients should therefore be limited to the simplest procedure that gives a predictable cosmetic improvement. The horizontal and vertical deviations can be managed in one procedure by a maximal lateral rectus recession and medial rectus resection with transposition of the insertions to that of the superior rectus. Despite thereby bringing the blurred second image closer to that of the fixing eye, none of the eight cases in this study whose surgery was purely cosmetic developed postoperative diplopia.

The pupil in traumatic third nerve palsy shows denervation hypersensitivity14 and constricts in response to low-dose (0.1%) pilocarpine drops. Used
two or three times a day this improves the cosmetic appearance of the eye and counters glare. In the less seriously injured subgroup by mediating accommodation it may help in the re-establishment and strengthening of fusion in downgaze.

CONCLUSION
Patients who have closed head trauma severe enough to cause a direct third nerve palsy usually have multiple permanent neurological deficits. The anterior visual pathways, the other oculomotor nerves, the hypothalamic-pituitary axis, the long tracts, and higher centres may be involved. The acquired oculomotor synkinesis syndrome almost always develops, and the spontaneous recovery of binocular single vision is unusual. There is a subgroup of patients with a less severe head injury in whom surgery may be expected to achieve a functional result. For the majority the appearance can be improved.

References