avoid caval compression; (iii) avoid adrenaline in the local anaesthetic; (iv) because epidural analgesia may augment the response of antihypertensive therapy, modify the dosage of antihypertensive drugs before an epidural needle is inserted; (v) avoid epidural analgesia in the presence of coagulation defect; and (vi) avoid an epidural block for caesarean section if the degree of hypertension is moderate to severe — the block must extend up to T6 in order to provide pain relief during the operation, and this reduces the capacity of the patient’s system to react promptly and appropriately to an episode of sudden haemorrhage such as is frequently encountered at caesarean section.

CONCLUSION

The management outlined is of intensive antepartum and postpartum care of hypertensive pregnant patients. This involves prolonged hospitalization, and the financial costs are high. Significant savings are evident, however, when the cost of hospitalization is compared with the cost of a stay in a neonatal intensive care unit for a grossly premature or severely ill neonate — intensive neonatal care is much more expensive than the cost per day for a mother. It is apparent that in most circumstances the mother is not only the best incubator for the infant but the most cost-effective one as well!

REFERENCES


Direct Traumatic Third Nerve Palsy

N. B. SOLOMONS, D. J. SOLOMON, J. C. DE VILLIERS

SUMMARY

Traumatic third nerve palsy is an unusual condition. It is a serious prognostic feature, not for life but for permanent, serious neurological deficit. It can only be diagnosed with confidence if it is known to have been present from the moment of impact and if adequate investigation excludes a compressing haematoma.


Oculomotor palsy frequently follows head injury when space-demanding intracranial lesions such as intracerebral, subdural and extradural haematomas cause transtentorial herniation. Less frequently, direct injury to the third nerve anywhere along its course may occur. Traumatic mydriasis and intra-orbital injury to ocular muscles may simulate partial third nerve palsy and give rise to diagnostic confusion.

The mechanism by which indirect or compressive third nerve palsy is produced is now universally accepted as compression, displacement and deformity of the third nerve and brainstem by transtentorial herniation caused by a supratentorial expanding lesion.1-3 Little has been written about the incidence and mechanism of direct third nerve palsy.4 The present study, therefore, has been carried out to determine the incidence of primary traumatic third nerve palsy, its frequency in craniocerebral trauma, and the significance of the associated neurological state in the eventual outcome.

MATERIAL AND METHODS

The case summaries of all patients treated for head injury in the Department of Neurosurgery, Groote Schuur Hospital, Cape Town, from 1966 to 1977 were examined.
The records of these patients covered the period from admission to discharge from hospital.

In accepting a diagnosis of primary traumatic third nerve palsy, the following criteria were used: (i) the presence of a fixed, dilated pupil from the onset of injury; (ii) the presence of a space-occupying lesion excluded by carotid angiography, air encephalography or computed tomography (patients not neuroradiologically investigated were excluded from the study); (iii) an improving clinical state despite persistent third nerve palsy; and (iv) no evidence of traumatic mydriasis (regular pupils) or of orbital injury to the ocular muscles.

The following information was extracted from the records of patients considered to have direct traumatic third nerve paralysis: (i) the site of injury as judged by the external signs of trauma; (ii) on which side the third nerve palsy was present; (iii) other cranial nerve involvement; (iv) associated hemiplegia and which side was affected; (v) postural reactions of the patient; and (vi) the presence or absence of fractures on skull radiographs.

Unfortunately, owing to the inadequacy of many of the initial notes and the lack of follow-up reports, the precise duration of loss of consciousness and of persistent third nerve signs could not be recorded satisfactorily in every patient. However, it was possible to divide patients into those who suffered loss of consciousness for less than 1 day and those who were unconscious for periods varying from 2 days to several months.

**RESULTS**

Of the 2 100 head-injured patients who were considered, 26 were thought to have direct traumatic third nerve palsy according to the abovementioned criteria. Two hundred and forty-six patients had post-traumatic, extracerebral intracranial haematomas. There were 130 patients with acute subdural haemorrhage, of whom 80 had unilateral third nerve palsy; 116 extradural haematomas were associated with third nerve palsy in 47 instances.

**Clinical features.** In the 26 patients with direct traumatic third nerve palsy, the clinical features were as follows:

**Unconsciousness.** Fifteen of 21 patients in whom the period of loss of consciousness was recorded were unconscious for longer than 2 days. Of these, 11 remained unconscious for longer than 2 weeks, 3 did not lose consciousness, and 3 regained consciousness within 24 hours.

**Site of injury.** Fourteen cases were associated with injury to the ipsilateral frontal, temporal or parietal regions, 9 had diffuse head injury, and 3 had evidence of injury to the head on the side opposite to the third nerve palsy. Seven had evidence of injury to the orbit or eye in which the third nerve palsy occurred. Two had fractures of the orbital margin.

**Associated cranial nerve paralysis.** In only 3 patients other associated cranial nerve palsies were recorded. Two had facial palsy, 1 of which was associated with fractures of the frontal, zygomatic, orbital wall and maxilla. One patient also had paralysis of the fourth, fifth and sixth cranial nerves.

**Hemiplegia.** Six patients had hemiplegia, 4 on the same side as the third nerve palsy and 2 contralaterally.

**Radiological features.** Ten patients had fractures on skull radiographs. Seven had ipsilateral fractures (3 frontal, 1 parietal, 1 occipital and 2 of the antrum and orbital margin). Three had contralateral fractures (1 frontotemporal, 1 parietal and 1 parieto-occipital). Twelve had normal skull radiographs and in 4 there was no record of the radiological findings.

**Outcome.** Of the 26 patients, only 2 died as a result of the head injury; one after 4 months and the other after an unknown period of time.

**DISCUSSION**

**Incidence**

Our findings show that direct injury to the third nerve is uncommon (26 out of 2 100). The cause of a third nerve palsy after head trauma is much more likely to be compression, e.g. subdural haematoma (80 out of 130) or extradural haematoma (47 out of 116). The ratio of compressive to direct nerve paralysis was 5 : 1.

This incidence agrees with that reported by Memon and Paine, who found 12 instances of direct third nerve palsy in 1 102 patients with head injury as opposed to 48 cases of compressive third nerve palsy.

Turner reported 15 instances of oculomotor paralysis in a series of 1 550 cases of head injury, but it is not clear whether all were direct injuries. Hooper described oculomotor involvement in 12 out of approximately 500 patients with head injuries.

In series of patients with third nerve palsy who present to an ophthalmologist, the incidence of trauma as a cause is much more obtrusive, because all patients with traumatic third nerve paralysis, of direct or indirect type, will be represented.

**Mechanism**

The anatomy of the third nerve is important in understanding the mechanism of injury in which it is involved. The third nerve nuclei are situated in the midbrain at the level of the superior colliculi, rostral to the fourth nerve in the peri-aqueductal grey matter. Crossed, and mainly uncrossed, fibres course through the red nucleus and medial part of the substantia nigra, leaving the midbrain on the medial aspect of the cerebral peduncle in a series of rootlets invested in pia mater between the posterior cerebral and superior cerebellar arteries, lateral to the termination of the basilar artery. The nerve passes downwards, forwards and laterally beneath the posterior communicating artery in the subarachnoid space, which it leaves by piercing the dural fold between the free and fixed edges of the tentorium cerebelli lateral to the internal carotid artery. Entering the cavernous sinus it runs in the lateral wall, still lateral to the internal carotid artery, and leaves the cranial cavity through the superior orbital fissure where it divides into superior and inferior branches.

**Traumatic Mydriasis**

This follows a concussive injury of the globus and is...
characterized by a moderately but not maximally dilated, eccentric pupil, sometimes with absent but usually with diminished reactions to light and accommodation. The mechanism of these reactions is disputed and is probably varied. The frequent absence of pathological change in the eye suggests that the effect may be due to damage to the nerves in their passage through the ocular tissues by the pressure wave which traverses the eye. In other cases tears in the iris are visible or transillumination reveals ruptures in the sphincteric muscle. In 1947 Kilgore experimentally demonstrated in the monkey that after relatively slight injury cyclic changes, followed by replacement fibrosis of the muscle and subsequent atrophy, are frequent sequelae.

Site of Injury

There is surprisingly little information as to the site where direct third nerve injury usually occurs. Walsh and Hoyt believe that this happens at the point where the third nerve pierces the dura to enter the cavernous sinus. They also state that fractures with momentary displacement of bone fragments may lacerate the nerve. The nerve, however, bears a close bony relationship only to the margins of the orbital fissure and the posterior clinoid process, but the latter is hardly ever involved in a fracture. Stretching and distortion of the third nerve at the time of impact are probably the main causes of the nerve injury. The two oculomotor nerves project forward, laterally and slightly downwards in an open V formation that extends from the interpeduncular fossa to the two posterior clinoid processes. This configuration would predispose the third nerve to shear and stretch by a frontal blow, causing a line of force along its course. This is probably why bilateral direct third nerve injury is rarely observed clinically. Variations in size of the tentorial incisura may play a role in the pathogenesis of traumatic third nerve palsy. A large tentorial opening may allow free movement of the midbrain and adjacent structures attached to it at the time of impact. During transient downward displacement, the third nerve may impinge on the posterior clinoid process.

As regards the mechanism and the site of production of the third nerve palsy, there is little that our study can add. Only 3 of the patients had injury to the opposite side of the head and the rest had ipsilateral or diffuse head injury. The virtually constant presence of a frontal blow, causing a line of force along its course, is of some interest to consider the importance of orbital fractures. Seven of our 21 patients had local orbital injury, but no other nerves passing through the superior orbital fissure, except the third nerve, were involved. This suggests that localized involvement of the third nerve at this level is not very likely. The force from a blow to the orbit would be virtually directly transmitted backwards in line with the long axis of the orbit and the intracranial course of the ipsilateral third nerve, so that stretching of this nerve behind the cavernous sinus is very likely. Cross and Hooper found that orbital injury was frequently responsible for oculomotor damage and that the resultant paralysis may be partial and often associated with other orbital nerve injuries. The fact that there were 6 cases of hemiplegia, of which only 2 were contralateral, makes it rather unlikely that one is dealing with a traumatic Weber syndrome with damage to the third nerve fascicles in the brainstem. It is more likely that there is diffuse injury with associated stretching or distortion of the third nerve in these patients. Only in the 2 patients with crossed palsy could one reasonably suggest that the damage occurred in the upper brainstem, but this could not be proved.

The possibility remains that the injury may occur at the site where the nerve passes through the dura, and that during the time of impact the tension on the dural fold, through which it passes, may be such that the nerve is injured. It has been shown experimentally that the common, lateral crush injury, which produces longitudinal fractures of the petrous bone and is frequently associated with seventh nerve palsy, may cause such tension. Distortion of and tension on the nerve during its passage through the dura may be a more common cause of injury than has been thought previously, and would explain the association with diffuse head injury causing transient deformity of the skull at the time of impact. Dural tension may, of course, in particular instances be associated with the other mechanisms of nerve stretching, rotation and downward displacement of the brainstem.

It is likely that, at the time of impact, many factors are at work and the injury to the third nerve could occur either by stretching at the time of the initial impact, or by downward movement of the brainstem or rotational strain exerted on the brainstem. Such mechanisms also may involve the nerve or its branches in the orbital fissure or in the orbit.

The practical implications of this survey are clear-cut. It would be highly dangerous to diagnose primary third nerve injury on purely clinical grounds. A history of palsy from the moment of impact and of an improving clinical state would indicate that this may be the case, but such an accurate, reliable history is often lacking. For the patient's safety it would be better to perform an investigative procedure, preferably a non-invasive one like computed tomography, to exclude the presence of a compressive lesion. It is important to recognize a primary third nerve injury, because prognostically it indicates a serious injury with prolonged unconsciousness and permanent brain damage in more than 50% of instances. However, in a significant number of patients it is not followed by a fatal outcome.

REFERENCES