Br Ir Orthopt J 2004; **1:** 19–23

Traumatic oculomotor nerve palsy: a literature review

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Abstract

Aim: **To review traumatic oculomotor nerve (IIIN) palsy, looking at the prognosis, site of lesion and mechanism of recovery.**
Methods: **A literature review was undertaken in order to research opinions on the prognosis following a traumatic IIIN palsy. The nuclear, fascicular midbrain, subarachnoid, cavernous sinus and orbital portions were investigated and the prognosis discussed.**
Results: **Trauma affecting the nuclear region alone is rare, but IIIN palsy can occur indirectly as a result of haemorrhage, and spontaneous recovery may be expected. Direct nuclear palsy following trauma is not prone to recovery. The midbrain is highly vulnerable to damage caused by rootlet avulsion and recovery may be further limited by post-traumatic fusion deficiency. The cavernous sinus is particularly prone to damage but the prognosis is generally good, as is that for trauma affecting the region of the superior orbital fissure.**
Conclusion: **IIIN palsy as a result of trauma is not a common finding, and the extent of the trauma and the site of the lesion will, in part, determine the prognosis for recovery. The mechanism of recovery is by axonal regeneration, by a process of Wallerian degeneration and new cellular cord formation. Prognosis is unaffected by age. Consideration should be given to aetiology and the site of the traumatic lesion in the management of traumatic IIIN palsy.**

Key words: Oculomotor nerve, Trauma, Regeneration, Wallerian degeneration

Introduction

Oculomotor nerve (IIIN) palsy is a well-recognised clinical condition, which may be complete or partial.[1] Lesions producing nerve dysfunction following trauma may be located anywhere from the nerve nuclei to the termination of the nerve in the extraocular muscles within the orbit.[2]

The IIIN can be divided into five subsections: (1) oculomotor nerve nuclei, situated on either side of the midbrain tegmentum; (2) midbrain portion, consisting of the nerve fascicles running from the nucleus through the brainstem; (3) subarachnoid portion, where the nerve runs in close proximity to the posterior communicating artery; (4) cavernous sinus portion; and (5) orbital portion, where the nerve enters through the superior orbital fissure and will divide before or after entry into the superior and inferior divisions.[2]

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Methods

A MEDLINE search was carried out using key word combinations including oculomotor nerve palsy, IIIrd nerve, trauma and nerve regeneration. Information was also obtained from a range of scientific textbooks and Internet web sites.

Trauma to the oculomotor nerve

Trauma accounts for only around 6% of acquired IIIN palsies.[3] A comparison of all aetiologies of IIIN palsies carried out in 1964 showed that almost 72% occur in males and only 14% of these are complete.[4] Elston,[5] in 1984, found that the highest incidence of traumatic IIIN palsies occurred in patients between the ages of 10 and 20 years.

The vulnerability of the IIIN to trauma varies along its path and the site of the lesion will influence clinical signs and the prognosis for recovery.

Nuclear region

Lesions of the IIIN nucleus often produce bilateral defects in ocular motility, eyelid position or both owing to the unique anatomy. The levator muscles share a common central nucleus and therefore it is expected that a unilateral lesion will result in bilateral ptosis.[6]

Nerve fibres that innervate the superior rectus originate primarily from the contralateral subnucleus before traversing to the ipsilateral subnucleus, decussating within the oculomotor nuclear complex.[6] Therefore a nuclear lesion could cause ipsilateral weakness of the superior, medial and inferior recti and inferior oblique with weakness of the contralateral superior rectus if those traversing fibres are involved.[7]

If the nuclear lesion is rostral, pupillary involvement is likely and lid function may be spared.[7,8] With caudal lesions, bilateral ptosis may be the presenting sign or may even be an isolated finding.[7]

Trauma is highly unlikely to cause a discrete nuclear lesion without other surrounding damage because the nuclei lie in close anatomical relationship to other structures; therefore other neurological findings would be expected.[6,9]

Midbrain region

Harrison[10] believes that the midbrain is the most likely site of injury as the nerve fibres form closely packed bundles that are vulnerable to disruption by shearing or haemorrhages.

Traumatic brainstem injuries can be classed into two groups: primary and secondary. Primary injuries are where direct damage is caused at the moment of impact. This group is considered to have a poor prognosis for recovery. Secondary injuries are due to a mass lesion such as a haemorrhage following the trauma, and these have a better prognosis if the haemorrhage disperses.[11]

Primary brainstem injuries often result in avulsion of the cranial nerves. Avulsion of the nerve rootlets often occurs where they emerge from the surface of the brainstem and can occur without skull fracture. Heinze[12] believes that differential mass movement between the brainstem and its surrounding structures, relative to each other and the skull bones, may explain this pathogenesis in the absence of skull fracture. However, a more likely explanation is that of Mariak *et al.*,[13] who draw a relationship between the power of the impact and a downward shift of the brainstem resulting in the nerves becoming detached. Kruger *et al.*[14] reported that these injuries are not liable to spontaneous recovery. This is perhaps linked to the suggestion that the distance between the severed ends of the axons limits regeneration.[15] Avulsion should therefore be considered as a possible explanation for delayed recovery.

Hashimoto *et al.*[11] carried out a retrospective review of 21 patients with brainstem lesions following trauma. Five of these had just a single brainstem lesion, thought to be the result of the shearing mechanism in and around the brainstem very close to the tentorial edge. All the primary brainstem lesions were in the dorsal midbrain. Naokatsu *et al.*[16] agreed that the dorsal aspect of the midbrain is the most vulnerable to trauma.

Tiffin *et al.*[17] found that the IIIN had a high rate of recovery. Only one case showed no recovery, having a total palsy with pupillary signs of aberrant regeneration. Golnik *et al.*[15] suggest that when the pupil is affected recovery may occur more slowly; however, Tiffin *et al.*[17] suggest that pupil involvement does not affect the speed of recovery but is likely to be associated with lesions that have a slower recovery.

Keane[18] reported a patient with complete IIIN palsy with partial sparing of the pupil following a traumatic midbrain haemorrhage. This palsy showed a gradual improvement over a 6 month period, suggesting that palsies caused by haemorrhage have a good prognosis.

Another limiting factor of recovery following a midbrain IIIN palsy could be disruption of the fusion centres. Pratt-Johnson[19] postulates that a motor association area, controlling fusional amplitudes, exists in the midbrain.

Subarachnoid space

Of the nerves travelling between the brainstem and the superior orbital fissure, the IIIN is the shortest. Given its course above the tough posterior petroclinoid ligament, it is most susceptible to damage due to stretching when the brainstem is forced downwards.[13] Heinze[12] also reports focal softening in discrete axonal bundles in the proximal subarachnoid segment of the nerve.

Compression may also be a reason for nerve damage as a result of trauma-induced haematoma or oedema. Such injuries have a better prognosis, as it is more likely that the whole axon is not damaged.[20]

Herniation of the nerve prior to its entry into the cavernous sinus can result from a traumatic injury. Herniation generally produces a unilateral, widely dilated pupil which is fixed to light, recovery of which is likely to be dependent on the degree of damage to the pupillary fibres. This condition should not be confused with traumatic mydriasis.[8]

Secondary IIIN aberrant regeneration is a common occurrence with traumatic IIIN palsy, not specifically related to the subarachnoid segment but thought to have associations. Aberrant regeneration usually results in one or more of the following: elevation of the eyelid on downgaze, adduction on attempted upgaze, globe retraction on attempted down- or upgaze, and constriction of the pupil on adduction.[1,21]

Cavernous sinus portion

Trauma to the region of the cavernous sinus and petrous bone may result in a range of cranial nerve palsies. The third, fourth and sixth nerves are in close anatomical relationship at the anterior end of the cavernous sinus.[2] Impact to the frontal, zygomatic and maxillary bones can transmit a decelerating force, causing compression of the IIIN. Alternatively fracture of these bones could also lacerate the nerve.[5]

Ninety-five per cent of compressive IIIN palsies result in pupil involvement; however, if the sixth nerve is spared the pupil may also appear spared. This is because the sympathetic fibres also travel within the anterior cavernous sinus and if these are damaged in addition to the parasympathetic fibres of the third nerve the pupil will appear to be only partially affected.[2]

The cavernous region is thought to be particularly prone to penetrating intracranial trauma.[22] The optic nerve lies within the optic canal and is tethered to the bone above the IIIN.[2] This could be a useful diagnostic factor for the site of trauma if a patient presents with IIIN palsy and vision loss due to suspected optic nerve damage.

Muthu and Pritty[23] studied the prognosis of a patient with IIIN palsy with ophthalmoplegia caused by trauma in the region of the cavernous sinus. The palsy partially recovered over a 10 month period and the ptosis had nearly fully recovered during this time. Return of IIIN function was observed after only 5 days, but no improvement in vertical muscle movement was found over a period of 6 months. This was thought to be a result of contracture of the vertical recti due to aberrant regeneration of the axons.

Ing *et al.*[24] considered that most congenital IIIN palsies are of traumatic origin, caused by deformation of the soft skull of the fetus by the moulding forces during labour. The compression is thought to occur at some

point between the brainstem and the entry of the nerve into the cavernous sinus. No further literature was found to reinforce this idea.

Superior orbital fissure and orbit

According to Hooper,[25] IIIN lesions are usually in the orbit or the superior orbital fissure. In 7 of the 12 patients examined for his study, multiple orbital fractures were present. Memon and Paine[26] found that in the majority of traumatic IIIN palsies there were associated injuries of the orbit. Unfortunately they do not quantify this as a percentage.

Orbital localisation of IIIN disruption carries an excellent prognosis for prompt, spontaneous recovery without aberrant regeneration. Therefore surgical intervention should be delayed at least 1 year, and longer if continued slow but progressive recovery is apparent.[27]

Heinze[12] believes the portion of the nerve within the superior orbital fissure is at risk from intraneural haemorrhages. Rowe and Eariss[28] reported a good level of recovery in those patients with haemorrhages in the area of the superior orbital fissure. The recovery is good because these haemorrhages can be absorbed and the nerve can thus regain its function.

In the study by Mariak *et al.*,[13] lesions of the IIIN were accompanied by fractures of the skull base and/or orbit in each case. A relationship between local damage to the orbital structures and injury to the IIIN seems unlikely because in all cases the nerve was clearly torn off from the midbrain.

Orbital injuries may cause traumatic muscle rupture and extraocular muscle laceration. These can sometimes simulate nerve palsies and give rise to diagnostic confusion.[20]

Bilateral traumatic IIIN palsy

Kruger *et al.*[14] believe that severe facial trauma can result in bilateral orbital injury. Bilateral traumatic IIIN palsy is extremely rare (less than 1.1% of head traumas).[14] This is somewhat surprising, as midbrain damage (to the nerves as well as the nuclei) seems common. Solomons *et al.*[20] believe that a traumatic bilateral IIIN palsy is rarely seen because the damage usually occurs in the region of the clinoid processes due to shearing forces.

Axonal regeneration

In the central nervous system there is little or no repair of damage to the neurons, but the nerves of the peripheral system have more ability to regenerate.[29]

At around 6 months of age virtually all neurons lose their ability to undertake mitosis and therefore they cannot be replaced by daughter cells from other neurons.[30]

Immediately or soon after trauma, the severed or crushed axon begins the regeneration process, although Golnik and Miller[15] believe that in some cases resolution may not begin for many weeks.

The axon regenerates by a process of Wallerian degeneration and new cellular cord formation. The separated ends of the axon seal and swell, and the myelin sheath at the injury site disintegrates. Debris is phagocytosed by macrophages. This process of Wallerian degeneration spreads throughout the axon, fragmenting it. Nerve growth factor stimulates the Schwann cells and cell surface adhesion molecules encourage new axonal growth, which is guided into position by cellular cords.[30]

Golnik and Miller[15] state that recovery may occur faster when the paresis is incomplete. New axons will not grow if the site of injury is too large or the gap becomes filled with collagen fibres. The greater the distance between the severed ends, the lower the chance of recovery because adjacent tissues block growth by protruding into the gaps. Repair also depends on the cell body remaining intact and the Schwann cells remaining active.[30]

Post-traumatic axon growth is never the same as that prior to injury.[30] Elston[5] states that only 50% of axons regrow, but found that misdirection occurred in 95% of patients studied. Lepore and Glaser[31] disagree with this theory. They believe that when the nerve regenerates there are more fibres than were originally present and this commonly leads to misdirection.

Traumatic oculomotor palsy in childhood

Mudgil and Repka[32] studied the prognosis of IIIN palsy in children under the age of 8 years. Of 41 cases studied, 15 were the result of trauma. The authors looked specifically at visual acuity and sensorimotor outcome in each case. The trauma group showed the greatest proportion of patients with decreased visual acuity: 10 due to amblyopia and 5 due to optic neuropathy or cortical trauma.

A retrospective study followed up 20 of the 41 patients to assess recovery. Of all aetiologies, trauma showed the highest percentage of decreased vision at the last follow-up appointment. Of those patients who underwent surgery, none demonstrated stereopsis post-operatively. The authors found that motor and sensory outcomes were best in those cases that spontaneously resolved or when the underlying cause was successfully treated.

The article does not specify the exact mechanism of trauma for each patient, nor does it indicate where along the nerve pathway the trauma occurred. The article as a whole indicates that those patients with a congenital, rather than acquired IIIN palsy respond best to amblyopia therapy due to fewer complications.

Ing *et al.*[24] also studied the prognosis in children. Of the 54 cases studied, 31 were traumatic. They found complete recovery to be rare in their series of traumatic IIIN palsies.

To summarise, paediatric IIIN palsies are rare but trauma is a frequent cause. Both children and adults with traumatic oculomotor palsy have a poor prognosis for recovery.

Management

The rate of recovery of traumatic IIIN palsy is generally thought to be slow in comparison with other aetiologies.[15] Some investigators believe that regardless of the cause, maximum recovery is usually complete within a period of 1 year.[15,33]

The general trend in the pattern of recovery is that the medial rectus is likely to recover first. Rowe and Eariss[28] believe this is because convergent eye movements are relied on predominantly for daily tasks. Depression is also heavily relied on so the inferior rectus should also be expected to show recovery. The superior rectus and inferior oblique are least likely to show recovery as elevation is used least in daily life. No evidence could be found to support Rowe and Eariss's theory. It could be related to the management strategies proposed by El Mansouri *et al.*,[34] designed to improve sensorial potential by the means of exercise. They believe that these exercises contribute to muscle growth but unfortunately do not explain the mechanism for this.

The timing of surgery in a patient who originally had binocular single vision is a matter of dispute. A prolonged period of time is required to allow for stability before surgery is considered, but during this period the patient's potential for regaining binocular function may decrease. If the patient is denied binocular single vision for a period of 3 or more months, the ability to motor fuse may be lost.[15]

Recovery may take 12 months or more, although it is unlikely that a patient would be left for any longer than 12 months before surgery was considered.[35,36] Golnik and Miller[15] recommend that patients who show some initial signs of improvement should be stabilised conservatively with the use of prisms at regular intervals over several months before considering surgery.

Docherty and Pope[33] believe that the muscles of an eye with a manifest squint show irreversible changes if not corrected, although the mechanism for this is not explained. They suggest not leaving the patient longer than a year prior to surgical intervention.

Severe closed head injury may be responsible for loss of fusion even in the absence of IIIN palsy. Elston[5] states that in most patients in his study the sensory fusion mechanism was disrupted. This is described as post-traumatic fusion deficiency and can pose a problem for management.[37]

Conclusion

IIIN palsy as the result of trauma is not a common finding and the prognosis is generally limited. The extent of trauma is a prediction of whether the palsy will recover. The mechanism of recovery is axonal regeneration.

Trauma to the nuclei and brainstem exit site carry the poorest prognosis, with trauma to the site of the superior orbital fissure and orbit showing the best prognosis. Trauma to the nuclear region alone is extremely rare but can occur secondary to haemorrhage and may show spontaneous recovery. The midbrain is highly vulnerable to trauma. Nerve rootlet avulsion is common and not prone to recovery, as this is dependent on the distance between the ends of the severed axon. The cavernous sinus is particularly prone to damage but the prognosis is generally good. This is the suspected site of damage in congenital IIIN palsy. Trauma to the superior orbital fissure generally carries a good prognosis.

Aberrant regeneration and post-traumatic fusion deficiency are limiting factors for recovery.

It appears that childhood palsies have the same prognosis for physical motor recovery as adult palsies, but sensory adaptations such as the development of amblyopia severely limit the ability to regain sensorial potential.

Although the proposed management for all IIIN palsies is similar regardless of their aetiology or site of lesion, we should perhaps take these factors into consideration. It is known that there are discrete centres that control fusion which are commonly affected during trauma, but very few links have been made when investigating reasons for delayed recovery of a palsy. This is an area that requires further research to provide insight for the best management of traumatic IIIN palsies.

I would like to thank Mrs Claire MacIntosh for her helpful advice and encouragement.

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