Case Report

IPSILATERAL TRAUMATIC PARALYSIS OF THE Vth AND VIIth CRANIAL NERVES

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It is not uncommon for the VIIth cranial (or facial) nerve to be injured somewhere along its length in closed head injuries. Occasionally it happens that both VIIth nerves are damaged. Turner[1] in a series of 70 cases of VIIth nerve injury collected from 1,560 cases of closed head injury, found one case of bilateral injury and no case with an associated other cranial nerve injury. Storey and Love[2] reported an instance of traumatic bilateral abducent and facial nerve paralysis, and these authors were able to find one other case in the literature by Friedman and Merritt[3] who described 22 cranial nerve injuries in 19 head injuries; among these were 7 cases of facial nerve injury and one case of bilateral VIIth nerve trauma associated with damage to the left VIth cranial nerve.

It is the purpose of this paper to report, for its rarity, a case of ipsilateral traumatic paralysis of the Vth and VIIth cranial nerves with uncertain initial prognostic import and a consequent course of almost total recovery of function of the VIIth nerve and a complete absence of evidence of recovery of the injured Vth nerve. In the indexed literature since the published cases mentioned above, I have not been able to uncover another like it. However, Russell and Schiller,[4] in their very interesting and practical enquiry into the mechanics of crush fractures of the skull, describe 15 such cases of which their no. 8 had bilateral facial palsy and complete anaesthesia and masticatory paralysis on the right.

CASE HISTORY

The patient, who was minding a moulding machine used in the packaging industry, was struck a very severe blow on the right side of his face by a heavy steel cone which was a part of the machine. He was not rendered unconscious—a surprising fact in the light of the special examinations carried out later and the way in which he sustained his injuries. But he was shocked and in great pain. A vertical laceration 2 inches (5 cm.) long was present over the right cheek, roughly coincident with the anterior border of the parotid gland. This laceration laid the cheek open. Bleeding was profuse and blood covered the face and neck and filled the right earhole. The bleeding was staunched and the laceration sutured. Afterwards, when the blood had been removed from the right external auditory canal, it was observed that the canal refilled with blood. A fracture of the base of the skull was diagnosed, probably a fracture of the petrous portion of the temporal bone, but when the patient recovered from the shock some hours later, he became aware that he was unable to move the right side of his face. At first it was thought that this might be due to a missed traumatic interruption of the facial nerve on the face after the divisions of the nerve have emerged from the parotid gland. But evidence appeared that there was complete paralysis of the facial distribution of the nerve and closer examination and questioning disclosed also a complete paralysis of the trigeminal nerve on the same side. This was presumptive evidence that the site of the nerve injuries was within the skull.

Radiological Findings

The radiologist was asked to examine the patient's skull and comment on his findings. These can be summarized:

1. There is a fracture of the temporo-mandibular joint on the right side . . . the fracture-line passes into the glenoid fossa.
2. There is an impacted fracture of the neck of the condyle of the mandible on the right with slight loss of alignment and there is some rotational displacement of the condyle.
3. The petrous bone appears damaged in its medial aspect with a possible crack running vertically towards the base. In several of the views the medial aspect of the petrous bone shows definite loss of alignment, suggesting a fracture through the internal auditory canal region with slight rotational displacement. There is, also, a diffuse opacity over the whole base of the skull in the basal views on that side, so that the foramen lacerum and foramen ovale can barely be detected on this side.
4. There is also a slight crack fracture on the posterior aspect of the zygomatic arch and another at the junction of the zygoma and lateral margin of orbit and antrum. There appears to be a fracture through the lateral wall of the antrum. The involvement of the apical region of the petrous bone is, no doubt, the cause of the marked involvement of the trigeminal nerve.

Course

Two months after the injury an otorhinolaryngologist was asked for an opinion on the state of the right ear and its association with the VIIth nerve involvement. He thought there had been improvement in the muscular innervation of the right side of the face and that there was also improvement in the sense of taste 'but not to normal'. Voluntary movement was detectable in the muscles of the forehead, eyelids, and ala nasi, but there was no movement at the corner of the mouth on that side. Cochlear function was undisturbed, the tympanic membrane was intact and the patient could hear a faint whisper at about 1 foot (30 cm.) distance on both sides. The Rinne test was positive for 256 - 1,024 c.b.s. on each side. Vestibular function judged by caloric tests was normal and there was no postural vertigo. Audiograms showed slight right perceptive deafness. The otologist was also of the opinion that the longitudinal fracture of the petrous temporal bone was responsible for the VIIth nerve injury. Because the nerve was recovering he saw no indication for operation. The weakness at the corner of the mouth he was inclined to attribute to severance of the peripheral branch of the facial nerve supplying that part of the face. He could not say whether there would be improvement in the perceptive deafness still present at that time. Three months after his first examination, i.e. 5 months after the injury, the otologist was able to say 'apart from the weakness at the angle of the mouth, the facial paralysis has virtually cleared up. The right tympanic membrane shows some scarring but no perforation and he hears a whisper at 1 foot. A repeat audiogram on 15 July shows practically the same state of affairs indicated by the previous audiogram on 19 April.'

Another colleague, an ophthalmologist, was consulted initially about the management of the conjunctival and corneal anaesthesia of the right eye and he advised and carried out a tarsorraphy which the patient still tolerates after 2 years for there has been no return of sensation to that eye. The operation was done primarily to prevent *xerosis superficialis* which threatened and was maintained because of the danger to the eye inherent in its continuing anaesthesia.

Present status. Two years after the injury the patient has slight limitation of the range of mandibular movement and this is accountable to the injury to the right temporo-mandibular joint and not to defective function of the masseter muscle. Whereas for a period following the injury the patient dribbled saliva from his mouth, this does not now occur, which suggests that there has been some slight recovery in the trigeminal nerve. The patient volunteers that right-sided sensation of the tongue, buccal, and dental surface, is absent; salivation is normal but the right side of his nose continues 'to run' with a persistency that he finds embarrassing in that he cannot feel his nose 'running'.

DISCUSSION

A search through the indexed literature has not uncovered another case of *ipsilateral* traumatic paralysis of the Vth and VIIth cranial nerves. The 'case 8' of the series of 15 crush head injuries of Russell and Schiller[4] corresponds with the case described but had bilateral facial palsy and complete anaesthesia and masticatory paralysis on the right side. The VIth nerves were also involved. These 2 authors ascribe the noteworthy fact of the preservation of consciousness in their series

to the absence of acceleration concussion in the circumstances in which the injuries were suffered. Dr. Russell, in a personal communication to me, states that it is unheard of to get so much damage to the skull and cranial nerves, as in my case, from an acceleration type of injury and he is of opinion that the skull must have been crushed. Because of and despite Dr. Russell's most valued opinion I have not been able to confirm by further careful inquiry into the exact manner of production of the injury that this was *not* an acceleration type of injury. All the clinical and radiological evidence points to the nerve injuries as being intracranial and the result of a fracture of the petrous part of the temporal bone. The anatomical extent of the injury may be more serious than the radiological picture, for, as Russell and Schiller[4] pointed out in 5 of their 15 cases with clinical proof of fractures of the base of the skull, radiological confirmation was lacking.

Involvement of the VIIth nerve in a fracture of the temporal bone is not uncommon and the nerve injury may vary from a relatively minor contusion of the nerve, with recovery, to complete severance, which today invites surgical repair. But trauma to the Vth cranial nerve is rarely come by so that an injury to both nerves on one side must indeed be rare. However, the degree of recovery of the intrapetrous injury to the VIIth nerve was complete; this indicated no severance of nerve fibres and yet the Vth nerve has shown only the slightest recovery, if any, which points to a complete interruption of the sensory roots to the Gasserian ganglion, for the minor portion is intact in that the masseter, temporal and pterygoid muscles were functional. Nor was there evidence of functional impairment of the tensor tympani, tensor veli palatini, mylohyoid or digastric muscle.

Siting the Injury to the Vth Cranial Nerve

The Vth cranial nerve injury has interesting anatomical considerations. Since the portio minor or motor fibres from the motor nucleus of the Vth nerve at midlevel of pons pass through the foramen ovale towards their distribution which showed no clinical defect, the foramen ovale was unlikely to be in the line of fracture. The mandibular nerve leaving the skull through the foramen rotundum could be so involved and yet the ophthalmic division of the Vth nerve which does not have a course through bone immediately related to it, was injured. It could be assumed, however, for there is no certain evidence to support the statement, that there was some degree of separation of the anterior part of the petrous bone from the greater wing of the sphenoid, enough to damage Meckel's cave or, by stretching or tearing, the fibres in the ganglion or roots. That an injury which produced only temporary paralysis of the VIIth cranial nerve, now completely recovered, should also have produced an irrecoverable or permanent discontinuity in the largest of the cranial nerves in all its divisions (other than the motor portion) makes a curious finding. The actual anatomical separation of the motor root from the Gasserian ganglion suggests that the Vth nerve was injured proximal to the ganglion. I find myself concluding that all the sensory afferent fibres *after* leaving the Gasserian ganglion, were damaged before they reached the pons—*without injury to the motor root*. If the ophthalmic, maxillary, and mandibular nerve fibres of the Vth cranial nerve had all been severed just before their entry into the Gasserian ganglion then it would appear that the motor part which joins the mandibular division immediately outside the foramen ovale should, likewise, have sustained an injury of which there was no clinical sign.

Siting the Injury to the VIIth Cranial Nerve

If we disregard the associated trauma to the Vth cranial nerve, then the points taken in evidence to try and place exactly the site of injury in the VIIth cranial nerve are: the loss of the sensation of taste in the anterior two-thirds of the right half of the tongue, implies injury to the chorda tympani. Since the whole of the peripheral distribution of the VIIth nerve was affected, this fact places the most distal compatible position of the lesion *in the facial canal*. The patient did not complain of hyperacusis, so the injury was not likely to have been higher in the canal than the point of emergence from the VIIth nerve of the nerve to the stapedius muscle, and this precludes the genicular ganglion from involvement. A lesion even higher, at the internal auditory meatus, would have produced in all likelihood, deafness from an associated VIIIth cranial nerve injury, for this nerve is frequently damaged together with the facial nerve in fractures of the middle fossa of the skull. This implicates the internal auditory meatus, for the acoustic nerve is rather soft in texture, being without neurolemma, and it may be either torn across to produce a permanent deafness, or bruised, pressed upon by extravasated blood, when the deafness is temporary. This nerve is known also to be injured by violent blows on the head without a fracture of the skull, and deafness has arisen from loud explosions causing trauma to which its structure makes it more liable than any of the other cranial nerves. The question whether there was any impairment of hearing from a paralysis of the tensor tympani was investigated but the rupture of the tympanic membrane was an influence in the assessment. The audiogram performed later showed no real hearing loss, perhaps only a minimal degree of perceptive loss.

SUMMARY

This paper records an extremely rare case of ipsilateral traumatic paralysis of the Vth and VIIth cranial nerves. The difference in the end-results of the injury to the 2 nerves is described and the probable sites of the lesions in the nerves are discussed.

REFERENCES

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