TRAUMATIC BILATERAL ABDUCENT AND FACIAL PARALYSIS
WITH GOOD RESTORATION OF FUNCTION
A CASE REPORT

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Unilateral paralysis of the 6th or 7th cranial nerve is encountered from time to time in disease or injury. Bilateral weakness or paralysis of the 7th nerve is seen in myasthenia gravis, in association with polyneuritis following diphtheria, in polyneuritis of the so-called infectious type as well as in alcoholic B-avitaminosis. However, bilateral facial paralysis of traumatic origin is much less frequent as indicated by various reports. Among 71 cases of closed head injury, Turner reports bilateral facial involvement in 1; none had associated abducant injury. Friedman and Merritt reviewed the records of 430 patients with acute injuries of the head and found involvement of 22 cranial nerves in 19 cases (4 per cent of total). The facial nerve was injured in 7 cases, in 1 of them bilaterally and in association with damage to the left 6th nerve. Therefore when bilateral abducent and bilateral facial paralysis were found following trauma, the case was believed to warrant reporting.

CASE REPORT

A white female of 26 years was rendered unconscious by a blow to the left mandible during an automobile accident on 26 July 1947. There was a slight amount of bleeding from both external auditory canals, chiefly the right. Upon return of consciousness, 12 hours after injury, there was diffuse, severe headache and vomiting. These diminished progressively and ceased by the end of the 2nd day. Upon regaining consciousness, she complained of diplopia and it was noted that her facial expression did not change.

Roentgenograms of the mandible showed a fracture in the region of the left mental foramen with slight upward displacement of the proximal fragment. Views of the skull showed nothing clearly indicative of fracture.

Examination. She was a pleasant, intelligent and cooperative young woman of good development and satisfactory nutritional status. Her recent health included no illness or previous injury. A few small blood clots were found in the right external auditory canal. The neck was not stiff, and no ecchymosis or abrasions were found anywhere. Blood pressure was 116/58; pulse was 64, regular and of good volume; temperature was 99.2 F. Blood count, urinalysis and Kahn test were normal.

A careful neurologic survey (cranial nerves, spinal nerves, cerebellar functions, and autonomic system), made approximately 40 hours after injury, yielded the following pertinent data:

(1) There was bilateral internal strabismus and attempts to abduct either eye past the mid-point failed although the opposite internal rectus moved normally. Therefore, no impairment of conjugate deviation could be said to exist.

(2) The face was expressionless and there was total inability to close the eyes, smile or purse the lips. No thalamic or synkinetic effects could be elicited.

(3) Tests of taste, repeated upon separate days, indicated loss on the right side (anterior 3) but good preservation on the left.

(4) There was no impairment of hearing as tested by tuning fork, pocket watch, or spoken voice. Weber's test was not lateralized and conduction by bone was equally good on the two sides. There was no spontaneous nystagmus on movement of the eyes and no vertigo. Rotary tests yielded normal results in past-pointing and gait.
Subsequent Course. A week after injury, the patient was free of headache and other generalized symptoms of the head injury and was allowed to return to her home in another city.

At 7 weeks after injury (12 Sept. 1947), re-examination disclosed that the internal strabismus, previously bilateral, persisted only on the left and to a lesser degree than formerly. There was evident improvement in voluntary abduction of each eye but, as yet, maximal abduction was not possible. Hearing continued unimpaired for ordinary conversational tones and vestibular function was normal. Both conjunctivae were slightly hyperemic, the right more so. She volunteered that while she felt all the emotion associated with crying, no tears came from her eyes.

Facial function showed little if any improvement. She was still unable to close her eyes and there was no blinking. Taste could not be retested because of the wired mandible. Hearing seemed normal and all other neurologic functions were satisfactory.

At 9 weeks after injury she began to note impaired hearing and at 10 weeks (3 Oct. 1947) re-examination showed the same findings in the abducent and facial tests as at 7 weeks. The conjunctival hyperemia was less since use of a simple antiseptic-emollient locally. The mandible had been unwired and taste was beginning to return on the right.

At this time the consulting otologist, Dr. John B. Thompson, reported: "Examination of ears reveals both tympanic membranes retracted to a marked degree. Bone conduction is greater than air conduction bilaterally. Schwabach test is increased and Weber's test is lateralized to the right. Audiogram shows impaired hearing with loss of 60 decibels on right and 45 decibels on left in conversational range. Caloric tests on each side, with 10 cc. ice water, show normal labyrinthine reactions. Impression: Air-conductive deafness bilaterally, greater on right, due to hemorrhage in tympanic cavities and, at present, adhesions causing fixation of ossicles and retraction of tympanic membranes."

At 5 months (17 Dec. 1947), she reported that return of facial function commenced about 3 months following injury and shortly after the previous visit. This was first noted in the left cheek. Examination showed ability to close both eyes, wrinkle the forehead, to smile and to purse the lips, although these functions were somewhat weaker on the right. Particles of food frequently accumulated between the right gum and cheek and it was necessary to dislodge them digitally. No diplopia remained. Abducent function and taste were fully recovered. After weeks of eustachian insufflation hearing was somewhat better for conversational tones but no audiogram was made. Otherwise she was well and her morale was greatly improved.

At 13 months (1 Sept. 1948), her chief complaint was a lack of suppleness in the musculature of the right side of the face. It was still necessary, occasionally, to dislodge small particles of food from the right cheek. Examination indicated improved strength in both sides of the face with the right side still weaker. As she spoke, involuntary associated movements were noted as slight winking first on one side and then the other. Abducent function and taste were normal. Hearing was adequate for ordinary conversation but a watch, while audible at 11 inches on the left, was barely heard at 1 inch on the right. She remarked that since April 1948 tears had appeared under appropriate circumstances, but, thus far, they were freer on the stronger left side. This indicated that lacrimal function began to recover at about 9 months after injury and was essentially normal at a year.

Throughout convalescence, no treatment was given beyond hopeful encouragement and attention to nutrition and other general hygienic measures.

COMMENT

In our opinion, the most likely cause of the disability in this case was bilateral basal fracture of the skull. The aural hemorrhage on both sides makes this highly probable, notwithstanding the lack of satisfactory roentgenologic evidence. The bilateral abducent paralysis is accounted for by the fact that this nerve courses over the tip of the petrous portion of the temporal bone on its way to the optic fissure and