POSTERIOR INFERIOR CEREBELLAR ARTERY SYNDROME
FOLLOWING A FRACTURE OF THE CERVICAL VERTEBRA

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The syndrome of the posterior inferior cerebellar artery was described by Wallenberg\(^8\) in 1895, and subsequently recorded by many other authors.\(^4,5,7,9\) The symptom complex is usually typical and is characterized by a sudden onset of decreased pain and temperature sensitivity on the side of the body contralateral to the lesion and disturbance of these same modalities of sensation on the side of the face ipsilateral to the lesion. Generally there is no motor involvement except a slight transient weakness of the extremities on the ipsilateral side. There may be an ipsilateral Horner's syndrome, ipsilateral involvement of the 5th through the 12th cranial nerves, ataxia, and nystagmus. Loss of consciousness is unusual.

The usual cause of occlusion of a posterior inferior cerebellar artery is considered to be arteriosclerosis. Goldstein\(^2\) described 6 cases with this as the etiologic factor and there are many more similar reports in the literature. Davison and Spiegel\(^1\) presented 2 cases in which the occlusion was attributed to metastatic carcinoma, primary in the lung.

The purpose of this report is to present a case in which the syndrome occurred as a result of skeletal traction for a fractured cervical vertebra. No case with similar etiology was found in a review of the literature.

CASE REPORT

A 29-year-old robust police officer was admitted ambulant to the hospital on July 20, 1954, 18 hours after a diving accident. His only symptom was numbness of his right thumb. Physical and neurological examinations at this time revealed no abnormality except for spasm of the muscles of the neck. Roentgenograms of the cervical spine showed a fracture of the lateral mass of the 5th cervical vertebra on the right (Fig. 1). The 5th cervical was dislocated on the 6th cervical vertebra (Fig. 2).

Crutchfield tongs were applied and 5 pounds of traction was used. The patient did well for 4 days when he suddenly cried aloud because of bilateral facial pain. He also complained of a transient numbness of the right side of his body. Traction was removed and his complaints were immediately relieved. A few hours later skeletal traction was reapplied and the patient remained asymptomatic until the 8th of August. At this time he complained of a vague discomfort over the entire left side of his body accompanied by nausea and vomiting. At no time did he lose consciousness. Neurological examination at this time revealed a right Horner's syndrome, an absent right corneal reflex, an hypoesthesia over the right side of his face in the distribution of the 2nd and 3rd divisions of the trigeminal nerve and of the left side of his body from the 3rd cervical dermatome caudad, a rotary nystagmus, a right 6th nerve palsy, slurred speech, difficulty in swallowing, and a weakness of the right hand. The Crutchfield tongs were removed.

Three days later the neurological signs and symptoms began resolving. He could now talk and swallow without difficulty. The diplopia and the weakness of the right hand disappeared. Four weeks after this episode examination revealed a mild hypoesthesia on the left side of his
Figs. 1. and 2. Roentgenograms showing (left) fracture of lateral mass of the 5th cervical vertebra on the right and (right) forward displacement of the 5th on the 6th cervical vertebra.

body and on the right side of his face. The Horner's syndrome and the involvement of the other cranial nerves had disappeared. He was given a cervical fixation brace and was discharged from the hospital.

DISCUSSION

The symptoms presented by this patient correlate well with those of the syndrome of the posterior inferior cerebellar artery. It is believed that the traction caused compression of the vertebral artery with resultant ischemia of the area supplied by the posterior inferior cerebellar artery. Goodhart and Davison proved the vascular anatomy in this region and concluded there are many variations of the posterior inferior cerebellar and vertebral arteries. Ramsbottom and Stopford stated that the syndrome is easily recognized and the anatomical location of the lesion is usually well defined but that it is very difficult, if not impossible, to state with certainty which of these two vessels is involved. This patient's cervical fracture was on the same side as his thrombosis and in all probability the traction applied to his neck in some way decreased the circulation in the vertebral artery or stretched the posterior inferior cerebellar artery, thereby producing an occlusion. The use of traction must have been implicated in some manner since its removal reversed the acute symptoms on two occasions. Direct compression or trauma from the fracture to the right vertebral artery can be dismissed since one would expect symptoms almost immediately which this patient did not have. There was no evidence of any of the usual common etiological factors such as arteriosclerosis or metastases.