Complications of halo-pelvic traction

Case report

RODNEY A. ROZARIO, M.D., AND BENNETT M. STEIN, M.D.

Department of Neurosurgery, Tufts-New England Medical Center, Boston, Massachusetts

When halo-pelvic traction is applied at a rapid rate it may induce cranial nerve palsies. The sixth, ninth, and tenth cranial nerves appear to be the most vulnerable. A proposed etiology is the stretching of these nerves resulting in a compromised blood supply with a consequent temporary paralysis which usually improves within 8 to 10 weeks.

KEY WORDS • halo-pelvic traction • cranial nerve palsy

Neurological complications incidental to the utilization of halo-pelvic traction have been commented upon in the orthopedic literature, but have been described less often in neurological and neurosurgical literature. We should like, therefore, to alert our colleagues to this problem by reporting a recent case.

Case Report

This 12-year-old mentally retarded girl underwent halo-pelvic traction for correction of a severe and progressive scoliosis. On neurological examination before this treatment no cranial nerve abnormalities had been recorded. The child had been virtually mute for almost all of her life, however, and had been called autistic for 10 years because she had little appropriate interactional development and a measured intelligence quotient in the severely retarded range.

Following placement of the halo-pelvic apparatus and 3 days of gradually increasing traction, the child did well with no change in neurological status. On the fourth day, a sudden episode of respiratory distress occurred while she was being fed. This was treated with vigorous suctioning and partial decrease in amount of traction. A chest x-ray film revealed a right middle lobe pneumonia, which was felt to be secondary to aspiration. Treatment with antibiotics and corticosteroids was started. Two days later, bilateral abducens palsies were noted and a neurosurgical consultation was requested.

Examination revealed an alert but mute child unable to understand commands or cooperate in the examination. There was disconjugate gaze in the primary position; bilateral abducens palsy was demonstrated by pursuit testing and confirmed with cold caloric irrigation. Gag reflex was markedly depressed bilaterally, and suctioning, which had previously been distressing, was now tolerated. No sensory, motor, or reflex changes were noted in either upper or lower extremities compared to previous examinations. X-ray examination of the cervical spine at this time showed marked distraction of C-1 from the occipital condyles (Fig. 1).
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The halo-pelvic traction was discontinued, and a soft cervical collar applied. Over the course of the next 2 weeks, the pneumonia resolved, and the child was discharged from the hospital. She still experienced difficulty swallowing and the bilateral abducens palsies were still present. Dysphagia cleared gradually over the next 4 to 6 weeks. At the tenth week, there was definite evidence of lateral rectus recovery, although it was not yet back to normal. It is our impression that the aspiration pneumonia had resulted from ninth and tenth cranial nerve paresis that decreased the gag reflex, thus disturbing the swallowing mechanism.

Discussion

Halo-pelvic traction, when stressed, has a propensity to injure specific cranial nerves. O'Brien, et al., stressed the vulnerability of the sixth, ninth, tenth, and twelfth cranial nerves and postulated that their nearly vertical-to-oblique course subjected them to the greatest traction, and that therefore they were most prone to injury. Telfer, et al., reported a case of bilateral sixth cranial nerve palsy combined with dysphagia, dysarthria, nausea, and vomiting as a complication of halo-pelvic traction, and suggested that the cause was either brain-stem ischemia or distortion of the cranial nerves.

Our patient had paralysis of the sixth, ninth, and tenth cranial nerves without other signs of brain-stem involvement. We feel, therefore, that the primary pathology must lie outside the brain stem, most likely in the extra-axial course of the cranial nerves. Traction that produces increased intraneuronal tension and consequent compromise of the blood supply to the nerves appears to be a plausible explanation of these cranial nerve palsies.

Other factors that influence the development of cranial nerve palsies are the rate of application of traction and the total magnitude of the distracting force. MacEwen, et al., were of the opinion that the common denominator in the development of cranial nerve palsies was the rapid application of traction; this view is supported by the work of Sunderland, who pointed out that if a distracting force is applied gradually, a nerve may be stretched beyond the normal range of elasticity without any disturbance of function. The usual recommended rate of extension is 2 mm per day, and the limit of the total safe force was found to be 40 lb. Indications to stop traction were complaints of spasms, discomfort, or the emergence of neurological signs. Our patient, an autistic child, was unable to complain of discomfort, and thus a useful means of monitoring was unavailable.

In our case, the stretch indicated in Fig. 1 was reached over a period of 3 days. This, in all probability, resulted in an excessively rapid stretching of the nerves and would tend to support the observation of MacEwen, et al., that a rapid rate of distraction is an important factor in the development of cranial nerve palsies. With regard to the degree of distraction between C-1 and the occipital condyles seen in our patient, another possible complication should be mentioned. This is the possible development of avascular necrosis of the dens. Tredwell and O'Brien reported that this complication occurred in 13.9% of their cases of halo-pelvic distraction and proposed that the necrosis was consequent to tearing of the apical and alar ligaments, which Schatzker, et al., and Schiff and Parke have shown to be important avenues of the blood supply to the dens. The proximal one-third to one-fourth of the dens is the area most prone to necrosis. Other reported complications of halo-pelvic traction include brain abscess, brachial plexus injuries, superior mesenteric artery syndrome (cast syndrome), paraplegia, and displacement of the dens from the ring of C-1.
References


Address reprint requests to: Rodney A. Rozario, M.D., Department of Neurosurgery, Tufts-New England Medical Center, Boston, Massachusetts.