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# Traumatic Atlantooccipital Dislocation with Survival

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Traumatic atlantooccipital dislocation is generally considered incompatible with life. However, there have been isolated survivals from this injury, and a few patients initially have minimal neurologic deficits disproportionate to the gravity of their injury, a feature that has not been adequately stressed. The potentially catastrophic results of delayed therapy make early radiographic detection imperative. Marked retropharyngeal soft-tissue swelling, an abnormal basion-odontoid alignment, and posterior displacement of the atlas are diagnostic of anterior atlantooccipital dislocation. In the more uncommon posterior atlantooccipital dislocation an abnormal basion-odontoid alignment associated with marked soft-tissue swelling should suggest the correct diagnosis. Conventional tomography can be confirmatory.

Traumatic atlantooccipital dislocation has been generally accepted as a rare and fatal injury [1-3]; survival has been considered exceedingly infrequent. Although there have been scattered case reports of survival in the general literature, we could find only one previous report of such a case in the radiologic literature [4]. We saw two such cases during a recent 3 year period. It has not been appreciated that some of the patients surviving a traumatic atlantooccipital dislocation initially will be neurologically intact, nor have the radiographic findings been adequately stressed. The serious implications of overlooking such an injury make early radiographic detection imperative. We review the previous experience in the literature and describe our two patients who survived traumatic atlantooccipital dislocation.

## Case Reports

### Case 1

A 9-year-old boy involved in a bicycle-automobile accident was injured in the head, left hemithorax, and left thigh. He was unconscious but responsive to pain and was moving all four extremities. He had a left sixth cranial nerve palsy, a mild right hemiparesis, and bilateral positive Babinski reflexes. The left femur had a closed fracture. A lateral radiograph of the cervical spine (fig. 1A) revealed anterior atlantooccipital dislocation.

He was immobilized and placed in skeletal traction; a repeat lateral film (fig. 1B) showed distraction of the cranium from the spine. The traction was diminished and on subsequent radiographs (not shown) the dislocation was reduced.

Over the next 30 days, there was progressive recovery of consciousness; it became increasingly difficult to keep the boy immobilized. The left sixth cranial nerve palsy persisted but the right hemiparesis was resolving. The atlantooccipital dislocation remained reduced and appeared stable. The child was removed from traction, allowed to ambulate, and was subsequently discharged and did well. A follow-up visit 1 year after injury revealed a persistent left sixth cranial nerve palsy; the right hemiparesis had resolved.

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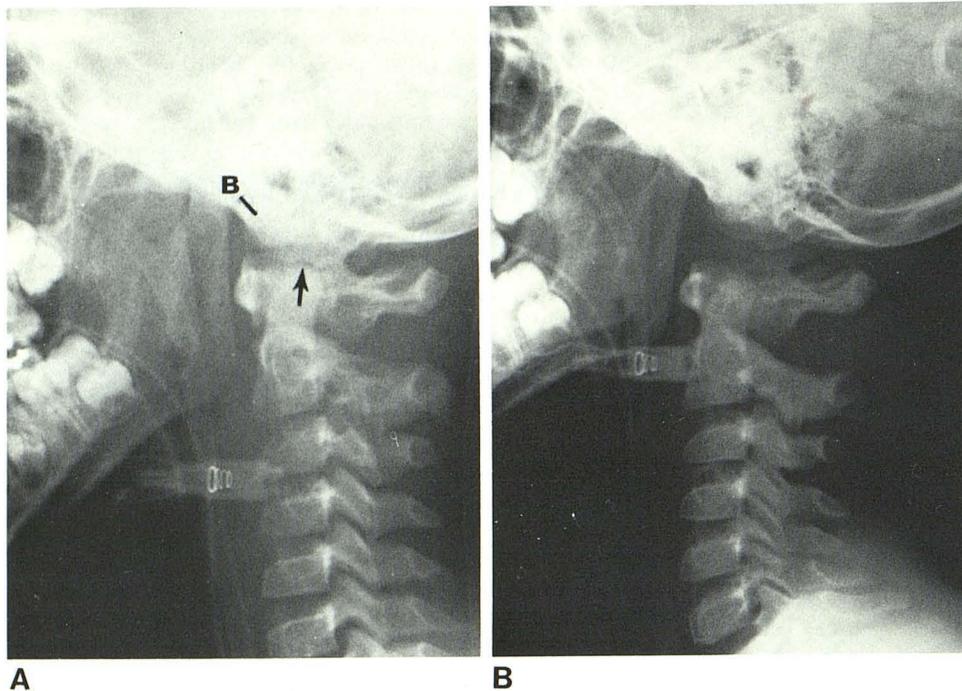


Fig. 1.—Case 1. A, Initial film. Forward displacement of basion (B) relative to tip of odontoid (arrow). B, After application of mild skeletal traction. Separation between cranium and atlas has increased.

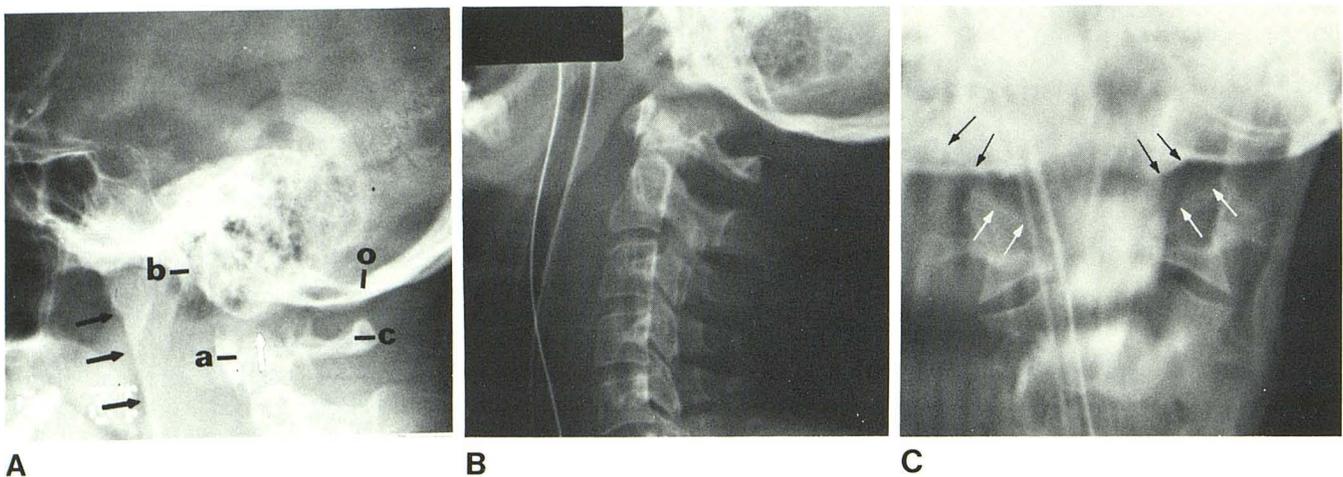


Fig. 2.—Case 2. A, Lateral skull film. Large retropharyngeal hematoma (black arrows). Forward displacement of basion (B) relative to tip of odontoid (white arrow). BC/OA ratio = 1.20. B, After application of mild skeletal traction. Increased separation between cranium and atlas. C, Anteroposterior

tomogram. Separation of occipital condyles (black arrows) from articular surfaces of atlas (white arrows) with right lateral subluxation. No fractures of cervical vertebral bodies.

#### Case 2

A 27-year-old man involved in an automobile accident was injured in the chest, left shoulder, and left side of his head and neck. He was ambulatory in the hospital with left sided neck and shoulder pain. Examination revealed absent left biceps and left wrist reflexes and diminished muscle strength in the left arm. Sensation in the arm was diminished in the C6 distribution and absent in the C3–C5 distribution. The patient was believed to have a left brachial plexus injury.

Radiography of the skull and cervical spine revealed marked prevertebral soft tissue swelling in the upper cervical spine and an anterior atlantooccipital dislocation best seen on the lateral skull radiograph (fig. 2A). Unfortunately, the patient was not immobilized. A chest radiograph revealed a fracture of the left clavicle and widening of the mediastinum. A thoracic aortogram done for evaluation of the mediastinum was normal.

Shortly after this the patient experienced a respiratory arrest. He was successfully resuscitated with endotracheal intubation and remained alert. Repeat examination revealed total quadriplegia; diaphragmatic breathing was intact. A repeat cervical spine radiograph (fig. 2B) and anteroposterior linear tomogram (fig. 2C) confirmed the atlantooccipital dislocation. He was placed in cervical traction for 1 month during which time his diaphragmatic breathing was lost and he became respirator dependent. One month after the initial injury he underwent an atlantooccipital fusion. He was subsequently discharged to a chronic care facility totally quadriplegic and respirator dependent.

#### Discussion

Survival of traumatic atlantooccipital dislocation is uncommon. In a thorough review of the literature we found 13

cases [5–13]; the addition of our two patients makes a total of 15 known cases of survival after traumatic atlantooccipital dislocation. While 13 of 15 of these patients had some initial neurologic impairment, two were completely normal neurologically at the time of presentation [5, 12]. Three of the 15 patients died within 48 hr after injury [6, 14], 12 patients had long term survival of their injury.

The total experience with traumatic atlantooccipital dislocation is small, but definite conclusions can be made concerning this injury. It is one of the more common fatal injuries of the cervical spine [15, 16]. In a review of 112 victims of trauma who succumbed at the scene of injury, Bucholz and Burkhead [15] discovered that 26 of them had a cervical spine injury. Of these 26 patients, nine (35%) had traumatic atlantooccipital dislocation; the next largest group was five (19%) with odontoid fracture. In a similar series of 312 patients of whom 98 had fatal cervical spine injuries, Alker et al. [16] found that 19 (19%) had atlantooccipital dislocation, although C2 fractures were more common in their series (30%). Although most traumatic atlantooccipital dislocations are immediately fatal, at least three patients have survived 48 hr [6, 14]; and 12 much longer. Fatalities are more common in children [15]; the 15 known survivors are not identified by gender or age.

The majority of patients are involved in either an automobile-pedestrian accident or some other type of automobile accident. The most frequent mechanism of injury appears to be an extreme hyperextension injury with a distraction force applied to the head, frequently associated with a deep submental laceration [4, 7, 10, 15]. A marked lateroflexion injury (as in our two cases) has also been implicated [4]. The injury results in rupture of the tectorial membrane and alar ligaments of the occipitoatlantoaxial joints [4] allowing forward dislocation of the cranium on the spine (one case of survival with posterior dislocation has also been reported [12]). There is almost always a large retropharyngeal hematoma, and air may be present if the posterior pharyngeal wall is lacerated [15].

While most patients surviving a traumatic atlantooccipital dislocation have some neurologic impairment, at least two of the patients were entirely normal at presentation [5, 12]; a normal neurologic examination should not be misinterpreted as evidence against a significant, potentially devastating injury. The most common neurologic abnormalities appear to be cranial nerve palsies, most likely due to avulsion of the nerve roots from the brainstem [10]. Cranial nerves VI and IX–XII have been reported to be involved; palsy of cranial nerve VI appears to be the most common [4, 9–11] (as in our case 1). There may be contusion of the brainstem and upper spinal cord as evidenced by transitory hypertension, respiratory depression, and cardiac arrhythmia [10–12]. Contusion, partial laceration, or complete transection of the spinal cord causes varying degrees of sensory and motor loss [6, 10, 15]. Diffuse spasticity, hemiparesis, quadraparesis with the arms more severely involved, and quadriplegia may be present depending on the site and level of injury [12]. If diaphragmatic respiration is lost the patient may be respirator-dependent as was the patient of Blackwood [6] and our case 2. Although some patients may be rendered permanently quadraplegic, most have a gradual

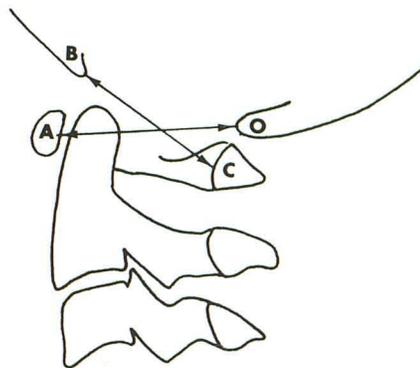


Fig. 3.—Normal  $\overline{BC}/\overline{OA}$  ratio. Ratio of distance between basion and anterior margin of posterior arch of first cervical vertebra ( $\overline{BC}$ ) and opisthion and posterior border of anterior arch of first cervical vertebra ( $\overline{OA}$ ) should normally be less than 1.0.

return of function; several patients showed complete resolution of their motor deficit [7, 9–11, 13]. However, persistence of cranial nerve palsies seems to be more common. Occlusion or stenosis of the vertebral arteries and anterior spinal artery has been demonstrated [7, 9] and may produce the syndrome of alternate hemiplegia with unilateral lower cranial nerve palsies and contralateral hemiparesis.

The serious implications of delayed recognition and treatment make it imperative that radiologists and other physicians dealing with patients with cervical spine trauma be familiar with the radiographic findings of atlantooccipital dislocation. A retropharyngeal hematoma (fig. 2A) should be the first clue. Normally the tip of the odontoid lies directly beneath the basion [2, 17]. While the distance between the basion and odontoid may be variable, significant anterior or posterior displacement of the odontoid from beneath the basion should be highly suggestive of atlantooccipital dislocation [2, 13] (figs. 1A and 2A).

Displacement of the atlas from the occipital condyles may also be seen. The ratio of the distance between the basion and the posterior arch of the atlas ( $\overline{BC}$ ) and the opisthion and the anterior arch of the atlas ( $\overline{OA}$ ) should normally be less than 1.0 [13] (fig. 3). The exact distance is not important; however, the ratio is important and is constant for all ages. A ratio  $\overline{BC}/\overline{OA}$  equal to or greater than 1.0 is diagnostic of anterior atlantooccipital dislocation [13]. In our case 2 the  $\overline{BC}/\overline{OA}$  ratio was 1.2 (fig. 2A). In the more uncommon posterior atlantooccipital dislocation the  $\overline{BC}/\overline{OA}$  ratio will be less than 1.0; however, marked soft tissue swelling and an abnormal basion-odontoid alignment will be present. With traction there will be separation of the cranium from the spine (figs. 1B and 2B). Air may be present in the soft tissues as a result of pharyngeal laceration. Tomography of the atlantooccipital region may better reveal the separation of the occipital condyles from the lateral masses of the atlas (fig. 2B). Arteriography may reveal stenosis or occlusion of the vertebral arteries [9].

The management of all patients surviving traumatic atlantooccipital dislocation should begin with immediate immobilization. Their respiratory distress should be treated, taking care not to move the head or neck during endotracheal intubation. While excessive skeletal traction may actually

worsen the deficits, mild skeletal traction is indicated and 1–2 kg of traction seems to be optimal [4, 9–11]. Immobilization of the head and spine in a halo brace or similar apparatus can aid in early mobilization of the patient. While Page et al. [7] believed that spontaneous fibrous fusion did not occur, our experience in case 1 and that of Farthing [5] suggests that, at least in children, spontaneous fibrous fusion is possible. This may not occur in adults and the experience in literature would indicate that surgical fusion of the atlantooccipital joint is necessary in most patients when their clinical status allows. Long term follow-up of these patients indicates a remarkable recovery of their deficits with several returning completely to normal.

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