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Neck Injuries: II. Atlantoaxial Dislocation—A Pathologic Study of 14 Traffic Fatalities

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ABSTRACT: C1-C2 vertebral dislocations have not been commonly recognized at autopsy. Among 66 subjects with neck injuries, drawn from a series of 155 traffic fatalities, were 14 with injuries at the level of the atlantoaxial motion segment, ranging in age from 8 months to 93 years. Thirteen had sprains or lacerations of the atlantoaxial facet joints, and one had a healed C1 fracture. Six of the 14 had odontoid fractures. None had transverse ligament lacerations. Injuries of the alar ligaments and the tectorial membrane were frequent. Only 3 subjects had subaxial cervical injury. All 14 had evidence of impact to the head or neck. Four had fractures of the mandible or facial bones, and 5 had skull fractures. Subdural and subarachnoid hemorrhages were found in 7 and 6, respectively. Brainstem lacerations were not uncommon, but only one had a pontomedullary laceration. Spinomedullary cord injuries occurred in 5. Acute neurogenic shock was the major mechanism of death in 9 of the 14, including 5 with major cardiovascular lacerations. Delayed effects of craniocerebral trauma accounted for the majority of the remainder. The biomechanical mechanisms are discussed.

KEYWORDS: pathology and biology, central nervous system, motor vehicle accident, craniocervical dislocation, atlantoaxial dislocation, neurogenic shock

Because the atlas and axis articulate by means of two atlantodental joints and two facet joints, atlantoaxial dislocations can take two forms. In the clinical literature, the term "atlantoaxial dislocation" almost always refers to atlantodental dislocation. Although physicians interested in neck injuries have radiologically recognized the atlantodental variety of atlantoaxial dislocations for years, C1-C2 dislocations are uncommonly recognized at autopsy. In both the clinical setting and the autopsy room, they may be missed because attention is diverted by severe head trauma [*1*]. The clinical diagnosis of the atlantodental type of C1-C2 dislocation generally depends on radiographic recognition of anterior or posterior displacement of the atlas with respect to the dens. In some cases a fracture of the odontoid process is implicated, and in some instances the dislocation is presumed to be due to laceration of the transverse ligament or laxity of the ligament in persons with connective tissue disorders or acute pharyngitis. Patients with these kinds of atlantoaxial dislocations are at risk of repeated dislocations, induced by trivial trauma, if surgical fusion is not performed [*2*]. Chronic atlantoaxial dislocations are classified by etiology as traumatic, spontaneous, and congenital [*3*].

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Dislocation of the atlantoaxial facet joints has been indirectly described in a few pathologic studies, but the author is aware of no clinical studies of C1-C2 facet dislocation. Lack of autopsy recognition probably stems from failure to autopsy traffic crash victims in many jurisdictions, failure to perform an adequate examination of the neck, or faulty classification of the injury. The latter two causes are generally the result of incomplete examination and of deficient knowledge of cervical anatomy. A less frequent reason is the erroneous assumption that cervical radiographs are a competent substitute for dissection of the neck.

Available clinical studies of atlantoaxial dislocations tend to represent the survivors rather than the fatalities. Lethal neck injuries generally have received increasing attention from biomechanically oriented researchers who use dummies, monkeys, and donated human cadavers as subjects. An adequate database of real lethal neck injuries does not exist, although some seminal postmortem studies have been done. This paper is one of a series of pathologic studies of real traffic fatalities, and provides descriptive data on lethal cervical dislocations at the C1-C2 motion segment.

Method

The injuries reported here are drawn from a series of 155 traffic fatalities autopsied by the author in the Commonwealth of Massachusetts between 1985 and 1987. The design of the study has been detailed previously [*4*]. Briefly, 66 subjects with neck injuries were assigned to hierarchal injury categories according to the most rostral facet joint injury. Fourteen subjects had neck injuries at the atlantoaxial motion segment. Examination of the neck was conducted by techniques described elsewhere [*5,6*]. An anterior approach was used with all cases, and a posterior dissection was done in 9 of the 14 autopsies. In three of the five autopsies with no posterior neck dissection, the extent of distraction (5 cm, 5 cm, and total decapitation) precluded the necessity of a posterior dissection. In the other two autopsies, the vertebral bodies up to C2 were removed by the anterior approach, and the alar ligaments, transverse ligament, and dens (odontoid process) were evaluated through the foramen magnum. Autopsy roentgenograms were not prepared.

Results

Crash Characteristics

Among the 155 traffic fatalities were 14 atlantoaxial dislocations. Three subjects were occupants of vehicles struck on the front. One of the three was an infant who was facing forward in a rear seat infant carrier designed to face the rear. Two persons were vehicular occupants in side impact collisions; one vehicle sustained ipsilateral (inboard) impact and the other sustained contralateral (outboard) impact. One subject was in a multiply impacted vehicle, one was in a rollover crash, two were cyclists, three were pedestrians, and one was a skateboardist. One victim was killed when a large truck crashed through her house, where she sat. Full ejection occurred with the victims of the rollover crash and the multiple impact crash, one of the two victims of side impact crashes, and none of the front impact victims (Table 1).

Victim Characteristics

The victims comprised three children, aged 8 months, 4 years, and 4 years; five young adults, ranging in age from 16 to 28 years; and six persons of advanced years, with an age range of 62 to 93 years. Four of the five young adults were male, whereas five of the six older adults were female. The heights of the vehicular occupants included 74 cm

TABLE 1—*Crash type and fractures of the mandible, face, and skull.*

Case	Type of Crash	Fracture			
		Mandible	Face	Vault	Base
1	front	+	−	−	−
2	front	−	−	−	−
3	front	−	−	−	−
4	side[a]	+	+	−	−
5	side	−	−	−	−
6	multiple[a]	−	−	−	+
7	rollover[a]	−	−	−	−
8	cyclist	−	−	−	+
9	cyclist	+	−	−	−
10	pedestrian	−	−	+	−
11	pedestrian	−	−	−	−
12	pedestrian	−	+	+	+
13	skateboardist	−	−	+	−
14	occupant of house	−	−	−	−

[a]Ejected.

(2 ft, 5 in.) for the infant, and ranged from 152 to 180 cm (5 ft, 0 in. to 5 ft, 11 in.) for the adults. The children's weights ranged from 11 to 21 kg (24 to 46 lb), the young adults' from 59 to 118 kg (130 to 260 lb), and the older adults' from 49 to 90 kg (108 to 198 lb). Ethanol was detected in the blood in two individuals. One had a concentration in the 0 to 0.05% range; the other was in the 0.15 to 0.20% range. The former subject rapidly succumbed to acute neurogenic shock. The latter died one day and seven hours after the crash from cerebral edema. Ethanol was tested for and not detected in nine cases, and was not tested for in three cases. Two of the three subjects not tested were children; the other was a five-month survivor.

Body Impact

When the data were analyzed by comparison of the sites of head impact with the sites of torso impact, no correlation could be made. The site of the impacts to the head were indicated by cutaneous and skeletal injuries of the face, the sides of the head, the back of the head, and the top of the head. The sites of the impacts to the torso were indicated by cutaneous and skeletal injuries and were distributed anteriorly, dextroposteriorly, and on the left and right. One subject had a direct impact to the side of the neck. An 85-year-old woman was fully decapitated in the wheelwell of a tractor/trailer rig after she was ejected from the front passenger seat of an automobile.

Pathological Findings

Thirteen subjects had sprains or lacerations of the atlantoaxial (C1-C2) facet joints or fractures of the odontoid process, to qualify as atlantoaxial dislocations in this series. None had a laceration of the transverse ligament. One person had healed fractures of the C1 ring, with no prior clinical or residual pathologic evidence of C1-C2 dislocation. Within this general definition, the atlantoaxial dislocations took a variety of forms.

A total of six had odontoid fractures. Three of these subjects had a fracture of the odontoid process, accompanied by full laceration of the C1-C2 facet joint capsules, the anterior ligament, and the tectorial membrane—and C1-C2 distraction—leaving the dens hanging loosely by the alar ligaments (Figs. 1 and 2). One person had an odontoid fracture and sprain of the alar ligaments. In two instances, the odontoid process fractures were

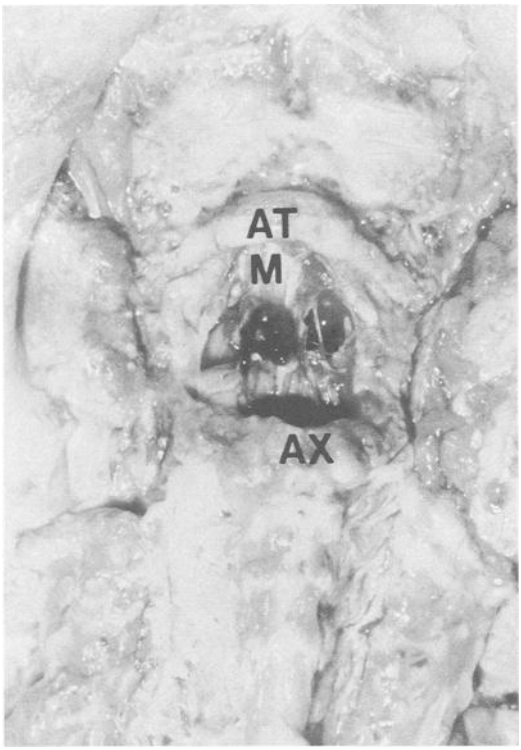

FIG. 1—*Atlantoaxial dislocation in an 8-month-old child, a posterior view of the vertebral column before laminectomy. The posterior arches of C1* (AT) *and C2* (AX) *are widely separated and the atlantoaxial membrane* (M) *is torn and bloody.*

not accompanied by disruptions of the alar ligaments, facet joint capsules, or dura mater. None of the odontoid fractures occurred in the young adults. Two of the three children had a fractured dens; the third had ligamentous sprains without lacerations. Four of the six older subjects had odontoid fractures. All the odontoid fractures were complete and displaced, and all were at the base of the process. There were no apical fractures.

Eight of the 14 subjects had no odontoid fracture. Three of these individuals had loosening of the dura mater and tectorial membrane over the odontoid process. One of these three had full lacerations of the alar ligaments and atlantoaxial joint capsules. Another had lacerations of both joint capsules and the left alar ligament, as well as a nondisplaced disruption of the C4-C5 disc. The third had sprain injuries of the atlantoaxial facet joints and had intact alar ligaments.

The remaining five of the eight subjects without odontoid fractures had no loosening of the dura mater. Four of these had various degrees of injuries of the alar ligaments and C1-C2 facet joint capsules, and one had healed fractures of the anterior and posterior rings of the atlas. In these four subjects with acute ligamentous derangement, the injuries comprised full laceration of the alar ligaments and C1-C2 facet joint capsules (one case), partial posterior laceration of the facet joint capsules with bilateral alar ligament sprains (one case), unilateral alar laceration and joint sprain (one case), and unilateral C1-C2 facet joint sprain with probable sprain of the apical odontoid ligament, as manifested by blood in the region of the ligament with no dural loosening. (The apical ligament is not

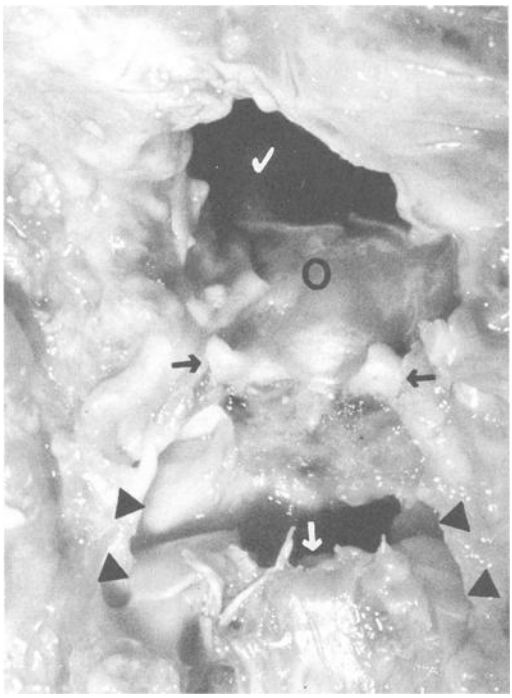

FIG. 2—*Atlantoaxial dislocation, in the same subject as Fig.* 1a, *a posterior view of the vertebral column after laminectomies for spinal canal exposure. The odontoid process is shown covered by the tectorial membrane* (0). *The transverse ligament has been divided* (black arrows). *Fracture through the base of odontoid process is shown with laceration of the distal tectorial membrane* (white arrow). *Exposed atlantoaxial facet joints* (arrowheads) *are shown. The foramen magnum is indicated by a check mark.*

readily identifiable as a discrete structure at autopsy.) The one subject without acute ligamentous derangement was a five-month survivor, with healed, clinically diagnosed fractures of the atlas. At autopsy, only fibrosis was evident and the fractures were not demonstrable. This injury was classified for this study as a probable Jefferson fracture.

Subaxial Neck Trauma

Cervical injuries below C2 were uncommon. One subject had an undisplaced C4-5 disc injury, and one had anterior ligament sprains over the C2-3 and C5-6 discs. The victim of wheelwell decapitation had a fracture of the C4 spinous process.

Craniofacial Injuries

Four of the 14 subjects had fractures of the mandible, facial bones, or both. Three of these had mandibular fractures and two had facial fractures. Three of the 14 subjects had cranial vault fractures and three had basilar skull fractures; a total of five had cranial fractures (Table 1). No correlation could be made between these fractures and other injury patterns.

Meningeal Hemorrhages

Subdural hemorrhages were present in seven of 14 subjects. Five were thin, one was a posterior fossa hematoma, and one was a thick hematoma of the spinal canal. Six of the 14 subjects had subarachnoid hemorrhages. One had a spinal epidural hematoma.

Injuries of the Brain and Spinal Cord

Three of 14 subjects had cerebral cortical contusions, two had cerebral lacerations, two had lacerations of the midbrain, one had a pontomedullary laceration, and five had injuries of the spinomedullary junction. The spinomedullary injuries comprised three lacerations, one contusion, and one healed injury manifested grossly as narrowing. The three spinomedullary lacerations occurred among the cases with odontoid fracture, alar laceration, and full distraction. The spinomedullary contusion involved one of the three instances of laceration of the dura mater and tectorial membrane over the odontoid process.

Survival Interval and Mechanisms of Death

The interval from the crash to the loss of vital signs was immediate in nine instances. These nine subjects had no vital signs when first responders arrived from seconds to minutes after the crashes. One of the nine subjects was an infant who had cardiac resuscitation and died with hypoxic encephalopathy one week later. In all nine subjects, acute neurogenic shock, as defined previously [*4*], was considered the primary mechanism responsible for the initial loss of vital signs.

One person survived a few minutes, only to succumb in the ambulance to cardiac tamponade from an intrapericardial laceration of the aorta. Another victim survived 22 h and succumbed to cerebral edema accompanying a subdural hematoma and adult respiratory distress syndrome. One person survived 31 h and died from cerebral edema consequent to probable diffuse axonal injury and a traumatic subarachnoid hemorrhage of the posterior fossa. A three-day survivor had brain contusions and cerebral edema. The subject with a healed old cord narrowing survived five months in a vegetative state, and then died in her sleep of presumed respiratory arrest. No evidence of septic, thromboembolic, or cardiovascular disease was found at autopsy.

Nonneurogenic Trauma

Six individuals had lacerations of the heart or aorta. In only one instance, that of the cardiac tamponade mentioned above, was this considered a significant contribution to the lethal sequence of physiologic derangements. Four of the other five subjects with cardiovascular lacerations had accumulations of blood in the pleural cavities or mediastinum totalling less than 200 mL for each person. These individuals had had external cardiac massage for intervals of 0, 0, 15, and 40 min. The fifth subject had blood collections of 2200 mL from lacerations of the heart and aorta, and 500 mL from lacerations of the liver and spleen. However, cardiopulmonary resuscitation efforts had been conducted for at least 35 min in this individual.

Four subjects had a flail chest, a traumatic pleuroperitoneal fistula, or a pneumothorax. Cardiopulmonary resuscitation had been attempted for 44 and 19 min, respectively, in the two subjects with flail chest and pneumothorax, for 15 min in the subject with a traumatic pleuroperitoneal fistula, and not at all in the subject with flail chest alone. In none of these subjects were deranged chest mechanisms thought to have contributed to the initial cessation of vital activity. However, the deranged chest mechanisms, especially

the pneumothoraces, may well have been relevant to efforts to provide efficient artificial ventilation.

Heart-Filling Status

The heart was filled in one subject with acute neurogenic shock and no cardiopulmonary resuscitation efforts, and in six subjects with prolonged therapy. In two subjects with acute neurogenic shock, and no significant hemorrhages, the heart was empty. One had an aortic laceration tamponaded by the mediastinum, and one had no vascular injuries. In four instances the heart was emptied by the combined effects of neurogenic shock and vascular laceration. One of these four was the case of decapitation mentioned above. For the subject with cardiac tamponade, the filling status of the heart was not noted.

Comment

The most striking finding in this series was the observation of a previously little-known injury, namely, dislocation of the atlantoaxial facet joints. Equally striking was the absence of the ligamentous atlantodental form of atlantoaxial dislocation, which is frequently reported in short-and long-term survivors. In this series there were no lacerations of the transverse ligament.

Unlike a companion series of occipitoatlantal dislocation victims, which comprised mostly young adult males [*4*], this series of atlantoaxial dislocations comprised a wide spectrum of ages and sexes. The victims generally were children, young adult men, or elderly women. The number of subjects is too small for statistical analysis, but one can speculate that the less-well-developed cervical musculature of women and children predisposes them to different sorts of injuries than those incurred by men.

Likewise, the relative sobriety of the victims of atlantoaxial dislocation, compared with the heavy intoxication of victims of occipitoatlantal dislocation reported in the companion study [*4*], may be a statistical quirk. However, the possible role of ethanol intoxication in promoting muscle relaxation, and therefore increased ligamentous loads and more severe forms of injury, cannot be discounted.

In this study, after excluding the subject with the healed C1 fractures, there were six odontoid fractures among 13 atlantoaxial dislocations. Thus, seven of the 13 dislocations were purely ligamentous. In contrast, one clinical study reported that only seven of 33 atlantoaxial dislocations were purely ligamentous injuries not involving fractures of the dens [*1*]. In that study, measurements of the space between the anterior ring of C1 and the dens led to the inferred conclusion that the transverse ligament was lacerated in the seven cases. In contrast, in the pathologic study presented here, no transverse ligament sprains or lacerations were identified. The different data can be reconciled by at least two explanations. One explanation would involve speculating that the transverse ligament can be radiologically sprained (stretched) without showing any blood extravasation. The other explanation would be that the spectrum of injuries is different in survivors than in those who rapidly succumb to injuries. I favor the latter hypothesis.

In this study, the odontoid fractures were found only among the children and the aged. This finding may relate to the lack of bony union of the component parts of the axis in children. In the elderly, the apparent predilection for bony instead of ligamentous failure of the atlantoaxial motion segment might be explained by osteoporosis of the dens, or perhaps by inelasticity of the alar ligaments.

Just as clinical series probably underestimate ligamentous injuries, this series probably underreports certain fractures which are not part of dislocations of motion segments. These would include compression fractures of vertebral bodies due to axial loading, facet

fractures, and undisplaced fractures of the anterior arch of C1, which is covered by dense ligamentous tissue.

No one type of crash was particularly associated with atlantoaxial dislocations. Likewise, no particular pattern of impacts to the head and torso emerged from this study. Only four of the 14 subjects had facial or mandibular fractures. Thus, the crash type, cutaneous injuries, and fractures did not provide clues to the biomechanical mode of neck failure.

Of interest was the low number—one—of victims with pontomedullary laceration, in comparison with a majority from a series of occipitoatlantal dislocations [*4*]. It may be that the modes of atlantoaxial failure involve distraction and extension to a lesser degree than in occipitoatlantal failure. The asymmetric involvement of the alar ligaments in many of these subjects may indicate that hyperrotation or lateral bending is involved. Beyond the foregoing speculations, no further comment is possible based on these data, other than to suggest that more than one type of mechanical load may produce the injuries described in this paper.

Five subjects had impact injuries of the spinomedullary junction of the neural axis, indicating some type of compromise of the diameter of the spinal canal at the time of impact. Whether this resulted from translation of C1 on C2, hyperrotation, or some other combination of failure modes is unknown.

In this series, like that of Davis [*7*], there were no lacerations of the transverse ligament. Of interest are experiments demonstrating that the transverse ligament is dense and strong in comparison with the alar ligaments, which stretch easily [*8*]. Previous studies of injuries of the craniocervical articulation [*9–14*] are not easily compared with the findings reported here because of their failure to describe injuries in terms of motion segments, failure to utilize available autopsy data in studies by radiologists, and failure to describe sprain injuries.

Mouradian et al. [*15*] commented on possible biomechanical explanations for odontoid fractures but came to no definitive conclusions other than an opinion that avulsion was not a reasonable mechanism. They pointed out that the lateral masses of the atlas might act as impactors on the dens, especially if the atlantodental joint is in rotation, but also speculated that the anterior ring of C1 or the transverse ligament might provide the impacts. In their clinical series, patients who could recall their accidents recounted occipital or lateral occipital head impacts.

In experimental studies, monkeys subjected to sudden deceleration while restrained in an accelerating sled had tension injuries of the cervicocranial articulation, including seven occipitoatlantal dislocations, two atlantoaxial dislocations, and three basilar skull fractures [*16*].

Compared with victims of occipitoatlantal dislocation, for whom a survival of 30 min is a long time [*4*], victims of atlantoaxial facet joint dislocation can occasionally survive for a few days to die of the effects of brain trauma. Victims of odontoid fracture and ligamentous atlantodental dislocation frequently survive, but may have delayed complications because of nonunion [*17*].

In most of the fatalities in this series, the mechanism of death was acute neurogenic shock. In all but one of the victims with major cardiovascular trauma, neurogenic shock was the overriding lethal mechanism. The remainder died from the delayed effects of craniocerebral trauma.

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