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# Neck Injuries: I. Occipitoatlantal Dislocation—A Pathologic Study of Twelve Traffic Fatalities

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**ABSTRACT:** Twelve of 155 persons killed in traffic crashes had occipitoatlantal dislocations. Nine were vehicular occupants, 2 were cyclists, and one was a pedestrian. The dislocations involved various combinations of lacerations of the alar ligaments, the occipitoatlantal joint capsules, the dura mater, the tectorial membrane, the rectus capitis muscles, and the suboccipital muscles. In 2 instances, an occipital condyle failed instead of the corresponding alar ligament, producing condyle fractures. Atlas ring fractures occurred in 3 instances. Axial and subaxial cervical trauma were uncommon. Facial or mandibular fractures occurred in a majority of cases, vault skull fractures were uncommon, and basilar fractures were absent. Pontomedullary brainstem lacerations occurred in 9 of the 12, and 4 had midbrain lacerations. The majority of the victims succumbed to acute neurogenic shock as the sole or the major mechanism of death. The biomechanical basis for occipitoatlantal dislocation is discussed, and the author suggests that distraction, in concert with variable combinations of extension, rotation, and posterior translation is responsible for occipitoatlantal dislocations.

**KEYWORDS:** pathology and biology, motor vehicle accidents, occipitoatlantal dislocations, musculoskeletal system, neck, spine, craniocervical dislocations, neurogenic shock

Because victims of occipitoatlantal dislocation so rarely survive any length of time [*1*], there are few clinical descriptions of this injury [*2–4*]. In contrast, this type of craniocervical dislocation is frequently found in victims of traffic accidents. However, the entity is mentioned only briefly in textbooks of forensic pathology, and the few extant pathologic descriptions of occipitoatlantal dislocation have been written by clinicians and radiologists [*5–12*]. Many forensic pathologists habitually lump together all high cervical derangements under a single heading such as "fracture of C1" or "occipitoatlantal dislocation," despite the fact that most such injuries are primarily dislocations and not fractures, and occipitoatlantal dislocation is but one of several types of craniocervical dislocation.

Although data on neck injuries are beginning to accumulate from biomechanically oriented studies on cadavers and dummies, corresponding baseline pathologic data from real crashes remain few and meager. This report provides retrospectively collected data for one type of injury, the occipitoatlantal dislocation.

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## Method

This study is part of a review of fatal traffic crashes investigated by the Office of the Chief Medical Examiner in Central Massachusetts during the two-year period July 1985 through July 1987. The study was limited to 157 traffic deaths in which the autopsy was performed by the author. In two cases, the file was incomplete; these cases were not further studied.

Among the 155 studied cases were 66 with trauma of the cervical spine or craniocervical articulation. The 66 neck injuries were classified as 12 occipitoatlantal dislocations, 14 atlantoaxial dislocations, 21 craniocervical articulation injuries without facet joint injury at the occipitoatlantal or atlantoaxial motion segments, and 19 subaxial injuries. The injuries were classified according to a hierarchial scheme, according to the most rostral injury. Classification as an occipitoatlantal dislocation required the finding of a sprain or laceration of an occipitoatlantal joint capsule and could, in addition, include alar ligament injuries, atlantoaxial sprains, and subaxial injuries. "Sprain" is defined for the purposes of this paper as a nonlacerating mechanical injury of a ligament, joint capsule, or muscle caused by overstretching. Sprains were manifested by blood extravasation in the injured tissue, sometimes accompanied by edema or overt lengthening of the tissue. "Laceration" is used for overt tearing injuries. Other neck injury classifications required the absence of occipitoatlantal dislocation.

In all the subjects, the anterior neck dissection was conducted as has been described elsewhere [*13*]. Posterior neck dissections were done in many, but not all, cases [*14*]. Postmortem roentgenography was not employed because of the tendency of rigor mortis to reduce dislocations [*13*] and raise the shoulders, and because the study was not prospective.

## Results

### *Crash Characteristics*

Twelve instances of occipitoatlantal dislocation were identified. Nine subjects were occupants of vehicles, two were cyclists, and one was a pedestrian. For the nine occupants, the impact sites to their vehicles were the front (2), the side (6), and atypical sites (1). The six subjects in side-impacted vehicles were all operators. Three of their vehicles sustained left impacts, and three sustained right impacts. The atypical impact involved front-to-back perforation of the cab of a pickup truck by a guardrail. One of the nine occupants in this series was fully ejected, one was partly ejected, and seven remained in the occupant space (Table 1). There was no evidence that any vehicle occupant used restraint belts. I participated in the scene investigation in two instances.

### *Victim Characteristics*

Nine were men and three were women. The ages of the victims ranged from 18 to 73 years, the body length from 160 to 191 cm (measured as 5 ft, 3 in. to 6 ft, 3 in.), and the body weight from 58 to 109 kg (measured as 128 to 239 lb). Nine of the twelve individuals with occipitoatlantal dislocation had detectable blood ethanol. Four of them had concentrations in the 0.20 to 0.25% range, two in the 0.15 to 0.20% range, and three in the 0.05 to 0.10% range.

### *Surface and Skeletal Injury Patterns*

Eleven of the twelve subjects had impacts to the head, manifested by cutaneous wounds or fractures of the mandible, face, or cranial vault. One subject had no evidence of impact

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 | side | − | − | − | − |
| 4 | side | + | + | + | − |
| 5 | side | − | − | + | − |
| 6 | side | − | − | + | − |
| 7 | side | + | − | − | − |
| 8 | side[a] | − | + | − | − |
| 9 | cyclist | + | − | − | − |
| 10 | cyclist | − | − | − | − |
| 11 | pedestrian | + | − | − | − |
| 12 | atypical[b] | − | − | − | − |

[a]Ejected.
[b]Partly Ejected.

to the head. He was a motorcyclist who had an impact to the neck, marked by contusion. There were no subjects without evidence of impact to either the head or the neck.

Injury clustering on the body was charted. No combinations of impacts to the head, neck, and torso were clearly correlated with occipitoatlantal dislocations. However, an absence of impacts to the top and back of the head, and to the back of the torso, was noted.

*Pathologic Description of Dislocation Injuries*

Five of the twelve dislocations involved complete laceration of both alar ligaments, laceration of both occipitoatlantal joint capsules, and laceration of the dura mater and the tectorial membrane overlying the odontoid peg. The prevertebral fascia was infiltrated with blood in all instances. The rectus capitis and suboccipital groups of muscles were sprained and sometimes lacerated. When the posterior neck musculature was examined, it often revealed sprains of the splenius capitis and semispinalis capitis muscles, or blood extravasation in the interspinous ligaments (Figs. 1 and 2).

Two of the twelve subjects had injury patterns similar to those of the five described above, including tectorial lacerations except that, instead of being lacerated, one alar ligament in each subject was intact and attached to a fractured and avulsed occipital condyle. Although separated from the occipital bone, the avulsed condyles remained attached to intact occipitoatlantal joints. One subject had avulsion of the left occipital condyle and the other had avulsion of the right occipital condyle. The latter subject also had sprains of the atlantoaxial joints.

One of the twelve subjects had an injury complex similar to the first five described above, with tectorial laceration, except that the left occipitoatlantal joint was partially rather than fully lacerated.

The four remaining subjects did not have laceration or loosening of the dura mater and tectorial membrane over the odontoid process. Three of these four dislocations involved fractures of the atlas. Two of these three had fractures of the anterior ring of C1 accompanying full laceration of the alar ligaments and joint capsules. One had a fracture of the right side of the posterior arch of C1, partial laceration of the occipitoatlantal joints, and full laceration of the alar ligaments. In one of these three cases, the spinal canal had a circular laceration of the dura mater; the dural sleeve was everted into the cranial cavity. The fourth of the remaining cases had a laceration of the right

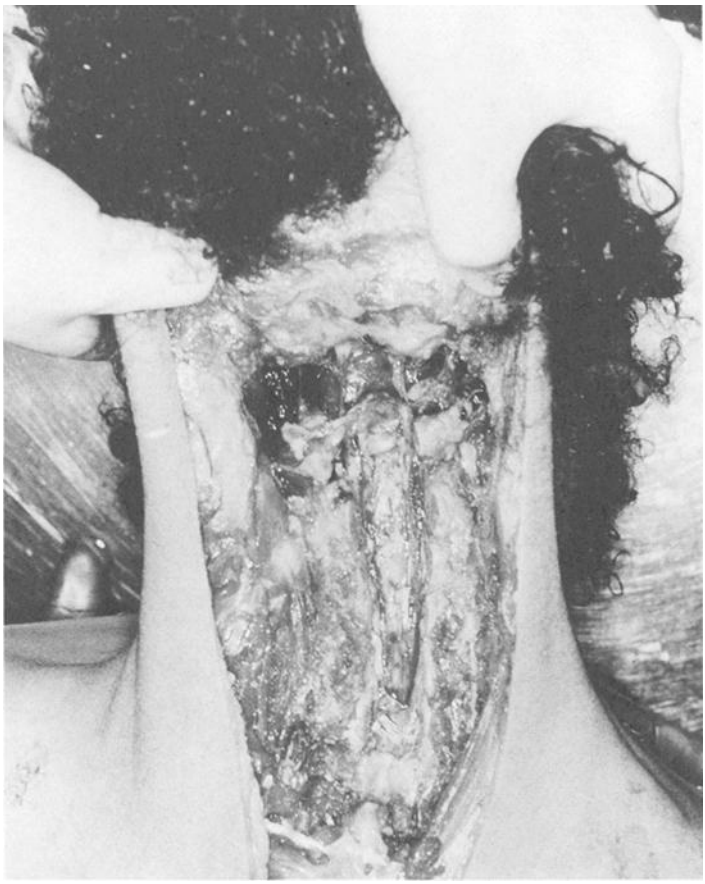

FIG. 1—*Posterior view of occipitoatlantal dislocation after serial laminectomies and exposure of the spinal canal. Note the separation between the skull and the atlantoaxial complex. The background of gray tissue visible through the distraction is the posterior wall of the pharynx.*

alar ligament, and sprains of the left alar ligament, both occipitoatlantal joints capsules, and the atlantoaxial joint capsules.

Axial and subaxial cervical trauma was uncommon. The anterior longitudinal ligament was subaxially lacerated in one person, one subject had interspinous blood extravasations, and one had a fracture of the C7 spinous process.

### *Cranial, Facial, and Mandibular Injuries*

Seven subjects had fractures of either the mandible, the facial bones, or both. Five of the seven had mandibular fractures, and three of the seven had facial fractures. One had both types of fractures (Table 1). No correlation was apparent with any other injury pattern.

Three of the twelve subjects had fractures of the cranial vault; two were on the right side, and one was on the left side. None of the twelve had basilar skull fractures (Table 1). The three vault fractures occurred among the eight subjects with lacerations of the tectorial membrane.

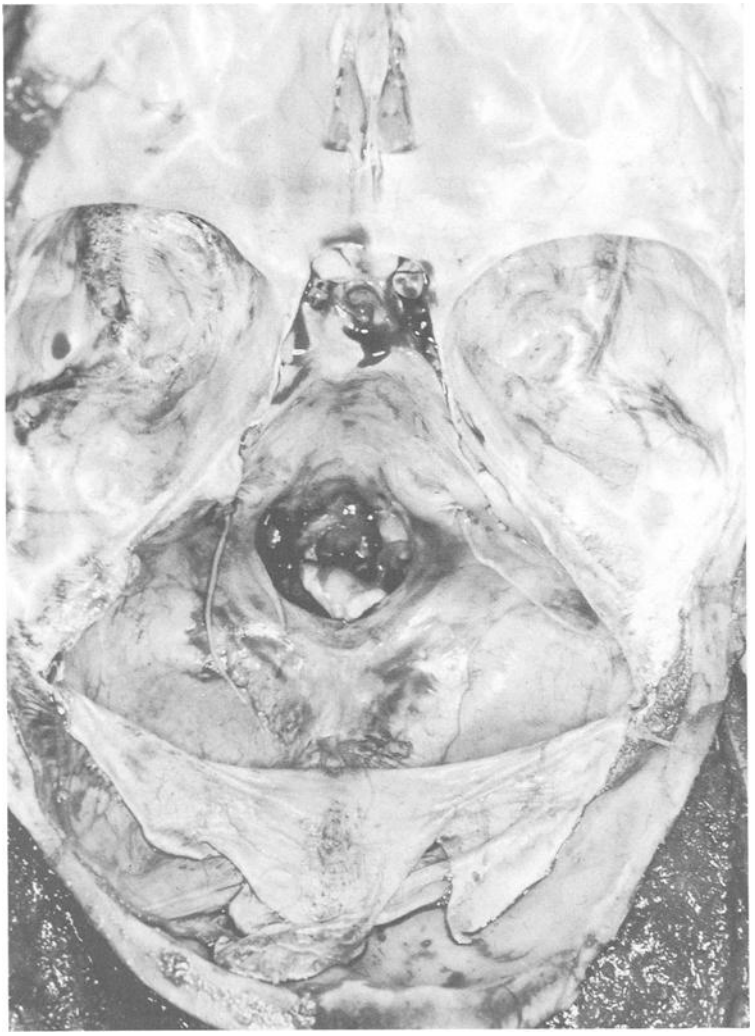

FIG. 2—*Same subject as Fig. 1a, the base of the skull, with a view of the foramen magnum. The rostral spinal cord has been severed from the medulla oblongata during removal of the brain and is pinched between the foramen magnum and the odontoid process. The juxtaposition of the dens and the foramen magnum is due to positioning of the head during autopsy. C = spinal cord. Large white arrow = torn edge of the dura and tectorial membrane. The small white arrow overlies the dens and points to left alar ligament stump.*

*Meningeal Hemorrhages*

Ten of the twelve subjects had subarachnoid hemorrhages. In eight of the ten, the hemorrhages were thin. Two were distending hematomas beneath the leptomeninges. One of these subarachnoid hematomas had spilled into the subdural space of the posterior fossa.

Two persons had subdural hemorrhages, one of which was localized to the posterior fossa.

*Injuries of the Brain and Spinal Cord*

Cerebral cortical contusions were found in four of the twelve subjects. The presence or absence of cortical contusions had no apparent correlation with other injury patterns. The midbrain was stretched and lacerated in four instances. In nine of the twelve subjects, the pontomedullary junction was lacerated, either partially or completely. In five instances, the region of the caudal medulla oblongata and rostral spinal cord was injured. Four of these five injuries were contused lacerations and one was a contusion manifested by softening without gross blood extravasation. All five spinomedullary wounds were found among the subjects with laceration of the adjacent tectorial membrane. The observations were made in situ during brain removal by the author in all instances.

*Mechanisms of Death*

In each case, based on the injuries, volumes of extravasated blood, and documented survival intervals, an assessment was made of which physiologic derangements importantly contributed to the cessation of vital signs, which were relevant to or exaggerated by artificial ventilation and circulation, and which were only potential mechanisms that were not contributory because of short survival of the subject.

Neurogenic shock was the most frequent lethal mechanism. Neurogenic shock is broadly defined here as any centrally mediated derangement of cardiovascular homeostasis which leads to a failure to circulate blood adequately. This definition includes ventricular cardiac arrhythmias and redistribution of blood in capacitance vessels, resulting in the empty heart often seen by trauma surgeons and forensic pathologists [*15*]. In only two of the twelve subjects were a pulse and respiration detectable by the first responders to the scene. In these two persons, spontaneous cardiovascular circulation occurred for 30 and 43 min, respectively. In the other ten subjects, no vital signs were ever detected. In one of these ten, the discovery of the scene was delayed and it could not be determined how long before discovery the vital signs had disappeared. In this case, resuscitation efforts were maintained for 54 min. In the other nine cases, the response time ranged from seconds to 5 min before the arrival of ambulance attendants. In five of the nine crashes, no resuscitation activity was undertaken at the scene. In the remaining four, resuscitation efforts were maintained from 20 min to 1 h and 26 min.

Of the twelve deaths, bleeding from lacerations of the heart or aorta was deemed to have been terminated by acute neurogenic shock in three instances and by the combined effects of neurogenic shock and a 100-mL cardiac tamponade in one instance. The volumes of the hemothoraces in the three subjects with cardiovascular bleeding terminated by neurogenic shutdown of cardiovascular function were 200, 200, and 800 mL. Artificial cardiovascular circulation was maintained by external cardiac massage for 0, 20, and 32 min, respectively, in these three persons and contributed an undetermined fraction of the two latter volumes of blood. Similarly, in one subject without pleural hemorrhage, a perisplenic peritoneal hemorrhage of 700 mL was found, corresponding to 54 min of cardiopulmonary resuscitation efforts following the delayed scene discovery noted above.

Two individuals survived several minutes with spontaneous cardiovascular function. One had no identified mechanism of death except neurogenic shock, possibly exacerbated by ethanol intoxication; the blood ethanol concentration was 0.24%. In the other subject, traumatic pleuroperitoneal fistula, pneumothorax, and cerebral edema probably contributed to the effects of neurogenic shock.

The filling status of the heart was generally noted in the cases accrued after 1986. Three subjects had filled hearts, two had empty hearts, one had an underfilled heart, one had a heart probably filled by fluid therapy during resuscitation efforts, and two

persons with lacerations of the heart or aorta had empty hearts. In three cases the filling status of the heart was not noted.

## Discussion

### *Pathologic Findings*

Previous pathologic studies [*6–8,10–12*] conducted by clinicians have been limited by overreliance on radiographs, the skimpiness of pathologic descriptions, a lack of crash data, a lack of description of body impact data, failure to autopsy all the subjects, or reliance on autopsy reports written by pathologists who failed to examine the neck adequately. Nevertheless, the pathologic findings from this study are in broad general agreement with those of previous studies.

*Biomechanics of Injury Causation*—In this study not only were the injuries delineated, but both the human and vehicular impact sites were known. Because the vehicles were inspected by the author in only a minority of instances, data on the interior impacting surfaces were not generally available in this study.

In many of these autopsies, the occipitoatlantal joints had considerable residual distraction. When examined, the broad muscles of the posterior neck compartment were sprained. These findings are consistent with distraction, that is, tensile stress, as the mechanism responsible for occipitoatlantal dislocation.

Although the skeletal and cutaneous injuries combined could not be correlated for the purpose of considering the biomechanics involved, the skeletal injuries when considered alone, were interesting. Seven of the twelve victims in this study had fractures of the facial bones or of the mandible, indicating that the impacts were anterior to the axis of the neck. All seven of these subjects were among the nine with pontomedullary lacerations. Pontomedullary lacerations have been associated with injuries attributed to hyperextension [*16–18*] and rotation [*17*].

Rotation as a mode of cervical injury has often been ignored in the clinical literature because it leaves no pathognomonic roentgenographic injuries. However, if the head is not constrained from rotation, there is no reason to believe the craniocervical articulation will not be rotated when the head strikes an object and the torso loads into the neck.

Since the spinomedullary junction has no anatomic changes as abrupt as those of the pontomedullary junction, one would not necessarily expect tensile mechanical failure of the neural axis immediately adjacent to the site of bony and ligamentous failure when the craniocervical articulation is distracted, hyperexended, or hyperrotated.

However, five of the victims had laceration or contusion of the spinomedullary junction or cervical spinal cord. All five had laceration of the tectorial membrane. The cord injuries can be reasonably attributed to cord impact from displacement of the skull with respect to the atlas. Given the evidence cited for facial impacts, such a displacement could involve posterior translation of the skull with respect to C1. On the other hand, radiologic studies of occipitoatlantal dislocation, and one recent pathologic study [*19*], most often describe anterior displacement of the skull, and thereby infer a flexion mode of injury. However, my experience suggests that the residual displacement may be a function of technical procedures because, during an autopsy, the residual displacement appears as anterior translation of the skull when the back of the head is supported by a block, and as posterior displacement when the shoulders are supported and the head extends passively.

The possibility that the tectorial membrane lacerations and spinomedullary injuries were caused by the dens bursting through the tectorial membrane—that is, by flexion, or by anterior displacement of the skull—cannot be excluded. However, this explanation

is inconsistent with the finding of pontomedullary lacerations and facial impacts. Thus, the results of this study do not support the commonly held but undocumented clinical notion that craniocervical derangements are caused by flexion.

It is my current hypothesis that most occipitoatlantal dislocations are probably produced by distraction in concert with variable combinations of extension, rotation, and posterior translation of the head. This is consistent with the findings of experimental studies [*20–23*].

*Fracture versus Dislocation*

The fractures of the occipital condyles in two cases in this study can be considered variants of alar ligament laceration, in which the bony insertions failed before the ligaments themselves.

The other fractures involved two instances of fractures of the anterior ring of C1 and one instance of fracture of the right posterior arch of C1. In none of the three was the tectorial membrane lacerated. All three involved lacerations of the pontomedullary junction, and in one case a sleeve of torn spinal dura was everted into the skull, so it is unlikely that these C1 fractures represented Jefferson fractures caused by axial compression.

It is evident that the injuries described in this paper are not primarily neck fractures. They are dislocations caused by lacerations through a crucial joint. Cervical dislocations are usually produced indirectly, by impact of the head during movement of the torso. Severe ligamentous derangement of the craniocervical articulation has been aptly described as internal decapitation.

*Mechanism of Death*—Clearly, the great majority of victims of occipitoatlantal dislocation succumb to acute neurogenic shock as the sole or major mechanism of death. In this study, the victims with aortic or cardiac lacerations died from their neck injuries before they could exsanguinate. Survival for several minutes can occur but is uncommon. Only two of the twelve subjects in this study had detectable vital signs after their crashes; both died within an hour. Rare instances of longer survival are to be found in the medical literature [*2*]. Experimental support exists for the role of neurogenic shock as a mechanism of death. Cervical spinal cord trauma in dogs produced cardiac arrhythmias as a consequence of disturbances in both sympathetic and parasympathetic outflow [*24*]. In monkeys cervical axial tension produced a decrease in evoked potentials followed by decreases in heart rate and blood pressure [*20*].

In this study some subjects had empty hearts and some did not. Taken together, these findings are consistent with the concept that neurogenic shock can be manifested by either cardiac arrhythmias, vascular collapse, or both.

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