Wound Hematoma After Anterior Cervical Spine Surgery: in vitro Study of the Pathophysiology of Airway Obstruction

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Abstract

Airway obstruction by wound hematoma is a serious adverse event associated with anterior cervical spine surgery. Although intrinsic airway edema is the most plausible pathophysiologic mechanism of obstruction, we hypothesized that extrinsic compression of the trachea by a hematoma can result in airway occlusion at an angle to the sagittal plane.

A silicone indenter and a servohydraulic test frame were used to apply pressure to the ventral neck of 7 human cadaveric specimens. Increasing pressure was applied in the anteroposterior (AP) and oblique planes until the trachea collapsed, as visualized with fluoroscopy. A paired *t* test was used to determine any statistically significant differences in maximum pressure or indenter displacement at tracheal occlusion between the 2 test modes.

Mean (SD) pressure required to cause complete tracheal collapse was 227.9 (54.8) mm Hg in the AP test mode and 135.6 (73.4) mm Hg in the oblique test mode. The difference was statistically significant (P = .004). Indenter displacement was significantly higher in the AP mode than in the oblique mode (P = .031).

The trachea can collapse from external force within a physiologic pressure range when pressure is applied in an oblique orientation. The mass effect of a wound hematoma appears to be a viable mechanism of airway occlusion.

Anterior cervical spine operations typically involve decompression of the neural elements and reconstruction of the vertebral column. The surgical approach is anterolateral and exploits the plane between the carotid sheath and the midline viscera. One of the most serious adverse events associated with anterior cervical spine surgery is postoperative airway compromise caused by a wound hematoma. The reported incidence of this complication of anterior cervical operations ranges from 0.2% to 1.9%.[1-3]

When a hematoma occupies space within the surgical wound, there are 2 potential pathophysiologic mechanisms by which airway obstruction can develop. The first is direct mechanical compression of the trachea leading to reduction in the cross-sectional area of the airway lumen. The second potential mechanism involves the development of intrinsic airway edema. In this situation, the mass effect of the hematoma impairs venous and lymphatic drainage from the head, leading to edema of the supraglottic structures.

The secondary edema mechanism of airway obstruction is theoretically more plausible than direct mechanical compression given the low pressure required to occlude the venous system and the relatively high pressure necessary to deform the trachea. We hypothesized that tracheal compression can play a role in airway obstruction after anterior cervical spine surgery. An in vitro model was designed to investigate whether extrinsic compression of the trachea can result in airway obstruction within a physiologic pressure range.

Materials and Methods

Seven fresh-frozen human cadaveric specimens were used for this study. Mean (SD) age was 67.0 (12.3) years. Each specimen included the occiput, cervical spine with intact surrounding soft tissues, and the proximal thorax to the level of the midsternum. Specimens were maintained in a freezer at -20°C until approximately 24 hours before testing. After thawing, each specimen was mounted in an MTS servohydraulic load frame (MTS Systems Corp, Eden Prairie, Minnesota). Radiopaque solution (Omnipaque; GE Healthcare, Buckinghamshire, United Kingdom) was manually applied to the inner tracheal surface through the distal aspect of the specimen. This dye coating was applied to track diameter changes during the testing process using a Philips BV29 fluoroscope (Philips Healthcare, Andover, Massachusetts).

A custom silicone indenter (Dragon Skin; Smooth-On Inc, Easton, Pennsylvania) was cast to facilitate circumferential application of pressure to the ventral neck. Compression was applied at 12.7 mm/min. Load and displacement data were col-

Authors' Disclosure Statement: The authors report no actual or potential conflict of interest in relation to this article.

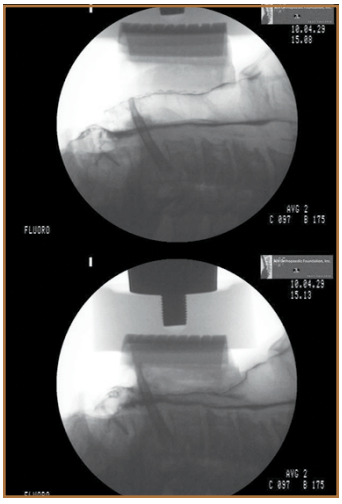

Figure 1. Anteroposterior test mode intact (top) and fully occluded (bottom).

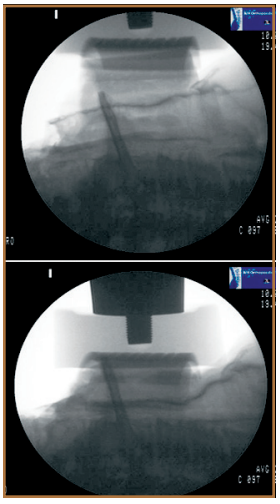

Figure 2. Oblique test mode intact (top) and fully occluded (bottom).

lected digitally at 25 Hz using MTS FlexTest SE controller. For each specimen, the width of each silicone indenter in contact with the neck was measured using Vernier calipers. Surface area during the test was then calculated in millimeters squared. Pressure was calculated as the recorded load divided by the surface area throughout the test: Pressure (Pa) = Load (N)/Surface Area (mm^2); 1 Pa = 0.00750 mm Hg. Pressure was determined from the load-cell feedback as the neck was compressed.

Two test modes were applied to each sample—direct anteroposterior (AP) compression followed by oblique compression. For the oblique test mode, the center of the indenter was oriented 45° from the sagittal and coronal planes. The custom indenter encompassed the region extending from the distal part of the cricoid cartilage to the proximal end of the sternal notch. This zone of indentation simulated the location of hematoma development after an anterior cervical spine procedure. The servohydraulic test frame was used to apply pressure in the AP and oblique planes until the trachea was fully compressed, as visualized with fluoroscopy (**Figures 1, 2**).

A paired *t* test was used to determine any statistically significant differences in maximum pressure or indenter compression at tracheal collapse between test modes. Statistical significance was set a priori at P = .05.

Table. **Pressure at Full Tracheal Occlusion in Each Specimen**

Test Mode, ID	Age, y	Measured Pressure, mm Hg	Maximum Compression, mm
Anteroposterior			
1	58	281.5	20
2	78	157.6	20
3	89	280.8	17.5
4	61	179.6	20
5	66	176.9	20
6	63	274.7	20
7	54	244.4	20
Mean (SD)	67	227.9 (54.8)	19.6
Oblique			
1	58	197.5	17.5
2	78	105.3	17.5
3	89	106.5	17.5
4	61	54.2	10
5	66	55.9	17.5
6	63	189.7	17.5
7	54	239.8	15
Mean (SD)	67	135.6 (73.4)	16.1

Results

Mean (SD) pressure required to cause complete tracheal collapse was 227.9 (54.8) mm Hg in the AP test mode and 135.6 (73.4) mm Hg in the oblique test mode. The difference was statistically significant (P = .004). The pressures for each specimen are listed in the **Table**.

Indenter displacement, the distance traveled by the indenter to achieve maximum tracheal collapse, was statistically significantly (P = .031) higher in the AP mode (mean, 19.6 mm; SD, 0.9 mm) than in the oblique mode (mean, 16.1 mm; SD, 2.8 mm). **Figures 1** and **2** depict the trachea in the intact and fully compressed states for both treatment groups.

During indentation, the trachea was visualized to displace away from the direction of compression in the oblique plane but not in the AP plane. Tracheal displacement could not be quantified with fluoroscopy.

Discussion

There are 2 potential pathophysiologic mechanisms by which a wound hematoma can reduce the cross-sectional area of the airway lumen after anterior cervical spine surgery. The first is direct mechanical compression. Caudal to the thyroid and cricoid cartilage, the trachea is theoretically vulnerable to extrinsic compressive force. The second potential mechanism involves impaired venous drainage from the head due to the mass effect of the hematoma. In this scenario, secondary edema develops within the supraglottic structures, the epiglottis, and the arytenoids.

The secondary edema mechanism of progressive airway obstruction is thought to be more plausible than direct mechanical compression given the low pressure required to occlude the venous systems in the neck compared to the relatively high pressure required to directly collapse the trachea. Carr and Benjamin[4] conducted an in vitro study and concluded that the maximum pressure achievable within a neck wound from a hematoma (maximum systolic blood pressure) was insufficient to cause airway obstruction by

external compression. Given the significant limitations in methodology, it may not be appropriate to extrapolate these in vitro results to the clinical setting of cervical spine surgery.

Specifically, the experimental model of Carr and Benjamin[4] does not adequately replicate the in vivo pathologic processes associated with bleeding after an anterior cervical spine operation. Explanted pig tracheal specimens were subjected to compressive force applied in the AP plane over an area of 1 cm^2. This direction and surface area of force application do not approximate the clinical situation.

Further, the morphology of the trachea is optimized to resist force applied in an AP plane because of the C-shape of its cartilaginous rings. In contrast, force applied at an angle to the sagittal plane is transferred along the open section of the cartilaginous ring structure. Tracheal resistance to a lateral or oblique force is more reliant on the posterior soft-tissue attachments of the cartilage segments. Thus, the airway in vivo may be more sensitive to pressure from the side.

Our hypothesis was that a wound hematoma can obstruct the airway after anterior cervical spine surgery by means of extrinsic tracheal compression. An experimental model was designed to reproduce the pathoanatomy of this postoperative complication. Fresh-frozen human cadaveric specimens were used. The model was based on the concept that extravasated blood collects in the potential space created by the surgical exposure and compresses the airway in an oblique direction. Force was applied to the trachea over a surface area and at an angle that simulates the deforming pressure produced by postoperative hemorrhage.

Because blood flows across a pressure gradient, the highest potential pressure that can be generated in the neck by bleeding equates to maximum systolic blood pressure, about 250 to 260 mm Hg.[5] In the present study, mean (SD) pressure required to cause complete tracheal collapse in the oblique test mode was 135.6 (73.4) mm Hg. The results suggest that the extrinsic tracheal compression caused by a hematoma can deform and obstruct the airway within a physiologic submaximal blood-pressure range.

Mean (SD) pressure required to cause complete tracheal compression in the AP test mode was 227.9 (54.8) mm Hg. In multiple specimens, a supraphysiologic pressure level was needed to occlude the trachea in the AP plane. Tracheal collapse occurred at a mean pressure that was statistically significantly lower in the oblique test mode than in the AP test mode. In addition, the indenter displacement needed to achieve tracheal collapse was significantly higher in the AP mode than in the oblique mode. These data support the theory that the trachea in vivo is more sensitive to pressure applied at an angle to the sagittal plane.

Mean age of cadaver specimens in this study was 67 years (range, 54-89 years). The advanced age of the specimens represents a limitation of this study, as the mechanical properties of human tracheal cartilage may vary with age. Rains and colleagues[6] studied the tensile stiffness of human tracheal cartilage rings in 10 specimens (age range, 17-81 years) and concluded that it increased significantly with age. Airway cartilage is anisotropic, and their study assessed tensile stiffness, which may or may not behave similarly in compression. However, age-related changes in the biomechanical properties and biochemical composition of airway cartilage may influence airway dynamics, and it is possible that the trachea may be more susceptible to collapse at lower pressures in younger, less stiff specimens. This is an interesting subject for further investigation.

In terms of clinical implications, we think that intrinsic edema is the most plausible pathophysiologic mechanism of airway obstruction in the setting of wound hematoma. Our results indicate that extrinsic tracheal compression is another viable mechanism of airway compromise and may occur in a subset of cases. As such, evacuation of the hematoma can be a lifesaving step in the emergency management of acute postoperative airway obstruction after anterior cervical spine surgery.

Conclusion

Postoperative airway compromise caused by a wound hematoma is a serious complication of anterior cervical spine surgery. The results of the present study indicate that external force can collapse the trachea within a physiologic pressure range. Extrinsic compression caused by the mass effect of extravasated blood in the surgical wound, therefore, appears to be a viable pathophysiologic mechanism of airway occlusion. Hematoma evacuation can be a lifesaving measure in the management of postoperative airway obstruction.

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Am J Orthop. 2013;42(6):E35-E37.

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