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# Airway-obstructing Supraglottic Edema Following Anesthesia with the Head Positioned in Forced Flexion

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Upper airway obstruction following endotracheal intubation and anesthesia, though infrequent, has been widely investigated and reported.[1–4] Of the reports in which the sites of the lesions are adequately documented, three describe supraglottic edema as the principal lesion.[2–4]

We report such a lesion which appeared following anesthesia in a young woman. In our investigation, we discovered a previously undescribed mechanism for this pathologic condition.

## Report of a Case

A 27-year-old white woman whose chief complaint was insidious hearing loss was scheduled for craniotomy and excision of a left acoustic neuroma. Past medical history was unremarkable, and the patient had no allergies. Results of physical examination were unremarkable with the exception of a marked "receding chin" (micrognathism). The operation was performed with halothane anesthesia. Oral intubation with placement of an 8-mm (I.D.) polyvinylchloride endotracheal tube§ was performed without difficulty. The patient was positioned in the right lateral position with her head acutely flexed. A radial arterial catheter was inserted intraoperatively. Ventilation was controlled. Duration of anesthesia was 11½ hours, and the trachea was extubated 45 minutes after termination of the operation.

The patient was quite hoarse postoperatively, and complained of a sore throat. In the recovery room a 3 × 2-cm red patch on the skin centered over the hyoid prominence was noticed (fig. 1). Inspiratory stridor and other signs of upper airway obstruction, prominent during sleep, were observed on the morning of the first postoperative day. A nasal airway was inserted and 40 per cent humidified oxygen delivered via Venturi mask. The $Pa_{CO_2}$ was consistently slightly elevated (41–47 torr) during the next day, while $Pa_{CO_2}$ values ranged from 77 to 154 torr.

Because of persistent signs of airway obstruction, upper airway examination with a fiberoptic bronchoscope was performed on the second postoperative day. This study revealed severe supraglottic edema involving both the epiglottis and the arytenoids, most marked on the left (fig. 2). The left arytenoid was edematous

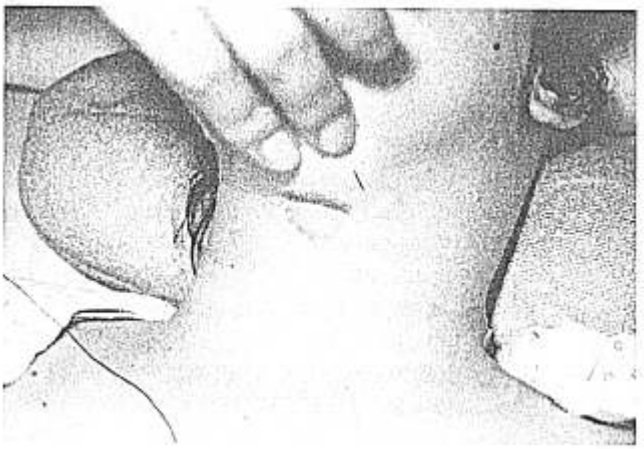

Fig. 1. Cutaneous lesion over the hyoid prominence resulting from prolonged tissue compression in the flexed-neck position.

to the extent that it created a ball-valve effect in the glottis, moving into and almost totally obstructing the airway during inspiration. Moderate pseudomembrane formation involving the same structures was present as well. The membrane was removed during the examination. Racemic epinephrine therapy was considered, but not instituted. Dexamethasone, 6 mg, *iv*, was given *q*. 4 *h*. as a part of the neurosurgical regimen postoperatively.

Approximately 48 hours following extubation of the trachea, ventricular irritability appeared, as evidenced by ten premature ventricular contractions per minute with frequent coupling. Marked upper airway obstruction, evidenced by inspiratory stridor, intercostal retractions, and marked tracheal tug, was present as the patient slept. With $F_{I_{O_2}}$ .20, $Pa_{O_2}$ and $Pa_{CO_2}$ were 48 and 39 torr, respectively, in an arterial blood sample that had been obtained several seconds after vigorous arousal, with consequent improved ventilation. Replacement of the oxygen mask returned $Pa_{O_2}$ to 120 torr, and the ventricular irritability disappeared. Subsequent therapy consisted only of administration of humidified $O_2$ by mask. The patient's clinical status and arterial blood gases were closely followed. There was no further hypoxic episode or ventricular arrhythmia.

The signs of upper airway obstruction gradually subsided, and reexamination with the fiberoptic instrument on the seventh postoperative day revealed almost total resolution of the previous pathologic condition. The cutaneous lesion had also largely subsided at this time, and only point tenderness immediately superior to the larynx remained.

The surgical procedure was performed two weeks later with the patient's head in a non-flexed position. Intubation of the trachea and the postoperative course were uncomplicated.

## Discussion

Most reports of upper airway tissue injuries following intubation of the trachea contain only specula-

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§ Manufactured by American Hospital Supply, implant-tested, gas-sterilized, with a high-volume, low-pressure cuff.

tion concerning their causes. Implicated as etiologic factors have been intubation-induced trauma, oversized endotracheal tubes,[5] glottic movement (*e.g.*, coughing or swallowing) during intubation,[1,4,5] operative procedures involving the neck, and long durations of endotracheal intubation. Many positions other than the supine have been identified statistically as risk factors for such airway injuries, including the prone position, and both flexion and extension of the head.[3,4] Lindholm, finally, pointed out that the posterior glottis and arytenoids absorb "fulcrum" forces of the imperfectly elastic endotracheal tube.[6]

The rather long duration of endotracheal intubation, as well as strong "fulcrum" forces generated in the flexed-neck position, were probably factors in the creation of the supraglottic insult in this patient. The cutaneous lesion and point tenderness directly external to this patient's supraglottic region, however, suggest an additional force in this tissue trauma. A trans–supraglottic pressure vector was created by gathered soft tissue underlying this patient's micrognathic mandible during acute flexion of the neck. This force vector is documented by a postoperative radiogram of the neck obtained with the patient in this position (fig. 3). Sustained intraoperative compression of the epiglottis and arytenoids against the endotracheal tube, with consequent tissue edema and eventual airway obstruction, resulted.

Interestingly, two of the three reports we located describing postoperative airway obstruction secondary to supraglottic edema also followed anesthesia with the head positioned in flexion. In one case, marked epiglottic and aryepiglottic edema occurred in a 25-year-old man whose trachea was intubated for four hours.[3] The second report described edema of the epiglottis, aryepiglottic folds, and arytenoids in a 3-year-old child whose trachea was intubated for "several" hours.[4] The causes of the edema in these two instances were undetermined. We are tempted to speculate that the mechanism we have suggested in this report may have been involved.

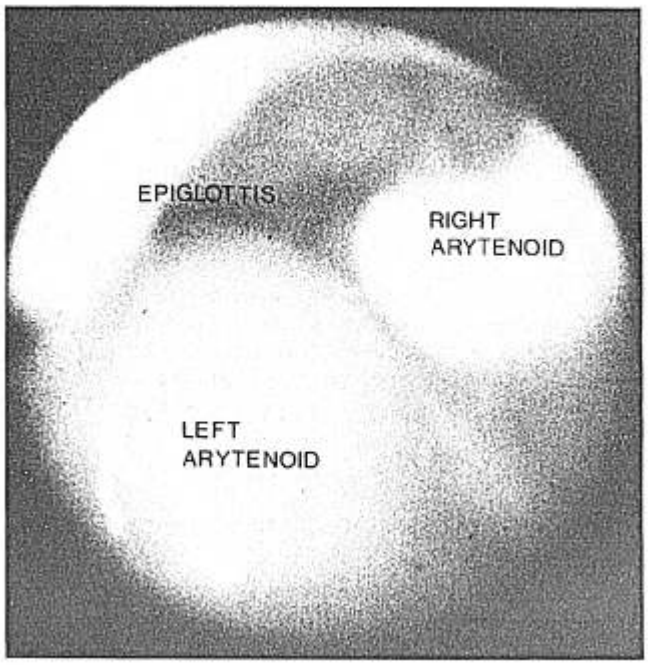


FIG. 2. Massively edematous supraglottic region during inspiration.

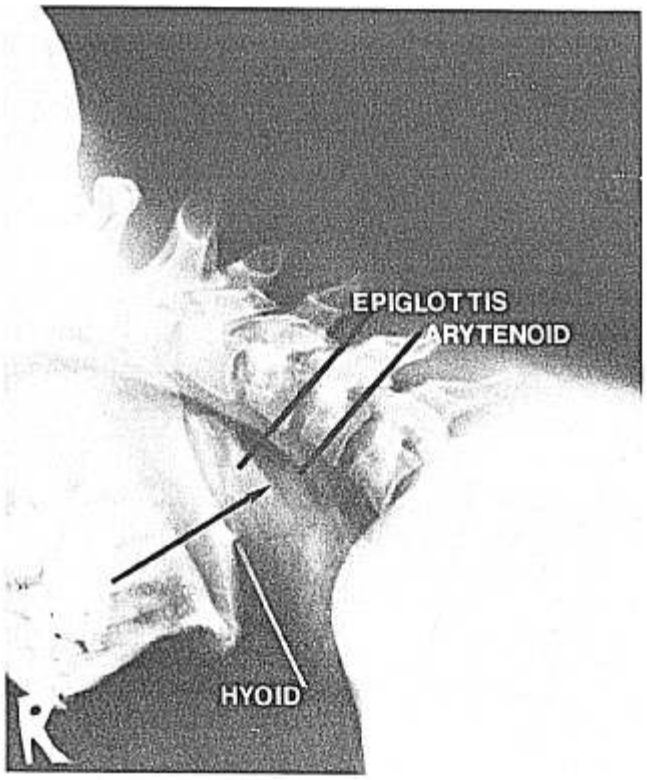


FIG. 3. Convalescent film of the airway with neck acutely flexed. The arrow indicates the compression force vector that transects the airway through the epiglottis and arytenoids.

In conclusion, we urge caution in positioning patients in a forceful flexed-neck position for operative procedures, as supraglottic compression and subsequent significant tissue edema may occur. This may be especially important in management of patients with micrognathism.

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# Airway Obstruction Following Surgical Manipulation of the Posterior Cranial Fossa, an Unusual Complication

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Removal of a tumor that necessitates neurosurgical manipulation with retraction in the posterior fossa may temporarily or permanently injure the delicate structures coursing through the posterior fossa. The following report describes an unusual cause of airway obstruction following extubation of the trachea in a patient who underwent subtotal removal of a tumor in the posterior cranial fossa.

## REPORT OF A CASE

A 71-year-old man was admitted for investigation of headaches, syncope, and episodic losses of consciousness of three months' duration. Results of physical examination were essentially within normal limits. Neurologic examination revealed no cranial nerve dysfunction. Tandem gait was ataxic. Cerebral angiogram and pneumoencephalogram revealed a midline posterior fossa mass in the region of the cerebellum, extending into the fourth ventricle and inferiorly into the foramen magnum. The patient was scheduled for exploration of the posterior cranial fossa and removal of the tumor.

The patient was premedicated with phenobarbital, 100 mg, and atropine sulfate, 0.4 mg, im, one hour prior to the anticipated time of operation. In the operating room, cannulations of a peripheral vein, right internal jugular vein, and left radial artery were performed using local anesthesia. Anesthesia was induced with thiopental sodium, 300 mg, iv, given in incremental doses, and endotracheal intubation was facilitated with pancuronium, 8 mg, iv. Anesthesia was maintained with 60 per cent nitrous oxide in oxygen, supplemented by fentanyl citrate as needed. Throughout the operation, the electrocardiogram, direct arterial and central venous pressures, urinary output, esophageal temperature, and heart tones (through an esophageal stethoscope) were monitored. A Doppler ultrasonic detector was appropriately positioned on the anterior chest wall to detect air embolism. The sitting position was achieved without difficulty. Arterial blood *p*H and blood gases were monitored as needed throughout the operation, and $Pa_{CO_2}$ was maintained around 30 torr. The entire intraoperative course was uneventful. The operation consisted of subtotal removal through a suboccipital craniectomy of an ependymoma arising from the floor of the fourth ventricle.

Upon conclusion of the surgical procedure, the patient was ventilated with oxygen for 5 min, and the residual neuromuscular block was reversed with atropine, 2 mg, and prostigmine, 5 mg, iv. Within 5 min, the patient was awake and responded appropriately to verbal commands, but was tolerating the presence of the endotracheal tube. Tidal volume was about 525 ml, with a forced vital capacity of 1,800 ml and a respiratory rate of 12–15/min. Nerve stimulation revealed normal responses to single and tetanic stimuli, with no posttetanic potentiation. The patient had good hand grasp and was able to perform a sustained head lift for more than 15 sec. Since the patient met all the criteria for extubation of the trachea, the endotracheal tube and oral cavity were suctioned and the trachea was extubated. Stimulation of oropharynx by sucitioning did not provoke any gag or cough reflex. Immediately following extubation, upper airway obstruction with marked inspiratory stridor occurred. The obstruction remained in spite of forward subluxation of the mandible. Laryngoscopy revealed a normal-sized tongue and epiglottis. The vocal cords and the movements of vocal cords during inspiration and expiration appeared normal. The patient could not protrude the tongue on command, and did not cough or gag when the laryngoscope was placed in the mouth. The trachea was reintubated.

Postoperatively the patient did not regain either the motor function of the tongue or the gag and cough reflexes. Impairment of salivary secretion by the parotid gland was not evaluated. Clinically there was no injury to the vagus nerve. On the sixth postoperative day, a tracheostomy was performed, since the reflexes had not returned.

## DISCUSSION

Laryngoscopy and visualization of the upper airway and normal movement of vocal cords in this patient excluded macroglossia, glottic edema, recurrent laryngeal nerve palsy, and external compression of the airway as the cause of the airway obstruction following extubation. Normal neuromuscular re-

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