

compromise and deterioration of vital signs were alleviated by discontinuing nitrous oxide and, in some instances, by tapping the pneumothorax.⁶

Tension pneumothorax as a complication of cannulation of the internal jugular vein is rare, and represents a serious risk to the anesthetized patient. Early identification by physical examination and prompt treatment by catheterization of the pleural space are essential. The contribution of nitrous oxide to the tension component of pneumothorax has been documented, and this anesthetic should be discontinued when pneumothorax is suspected.

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Acute Laryngeal Edema 24 Hours after Passage of a Nasogastric Tube

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Complications associated with the use of nasogastric tubes are, for the most part, infrequent, and mostly self-limited, such as slight epistaxis and abrasion of the pharyngeal mucosa upon insertion. Isolated instances of more serious complications have included laryngeal obstruction, ulceration and stricture of the esophagus, and perforation of the stomach or intestine.¹ Laryngeal injury, although usually associated with prolonged nasogastric intubation, has also been reported to occur after intervals as short as four to six days.² Presented is a case of severe laryngeal edema with airway obstruction which began 24 hours after passage of a nasogastric tube.

REPORT OF A CASE

A 74-year-old white man was admitted to the hospital with a six-day history of melena. With the exception of a duodenal ulcer 20 years prior to admission, his health had been good. There was no history of any recent upper respiratory tract infection. Physical examination was unremarkable; vital signs were stable. Chest x-ray was clear; admission hematocrit was 25 per cent. A no. 18 Fr Salem sump nasogastric tube³ was passed atraumatically through the nose, yielding small amounts of old and fresh blood. The tube was placed to suction at -170 cm H₂O pressure. Twenty-four hours after admission the patient complained of pain in the throat radiating to the left ear. The temperature was 101 F. Examination of the throat, ears and lungs disclosed no abnormality. During the next 12 hours mild inspiratory stridor and increasing restlessness developed. The nasogastric tube was removed 36 hours after admission. Within a few hours after removal of the tube, inspiratory and expiratory stridor, shortness of breath, and a hoarse, raspy voice were evident; respiratory rate had increased to 28 breaths/min. Arterial blood-gas values (room air) were P_O₂ 85 torr, P_{CO}₂ 33 torr, pH 7.43. Indirect laryngoscopy revealed mild edema of the uvula with severe edema of the arytenoid mucosa and markedly limited ability to abduct the vocal cords. Sputum culture taken at the time of the initial fever (24 hours)

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grew *Hemophilus influenzae*. A diagnosis of acute supraglottitis was made, and the patient was treated with dexamethasone (8 mg, iv), furosemide (20 mg, iv), ampicillin (500 mg, iv, every 6 hours), and humidified oxygen administered by face mask. He was transferred to the Respiratory Intensive Care Unit for observation. Inspiratory and expiratory stridor increased and were treated with racemic epinephrine (0.5 ml 2.5 per cent solution diluted with 3 ml physiologic saline solution) administered via a face-mask nebulizer.² Arterial blood-gas values (40 per cent oxygen by face mask) were P_{aO_2} 160 torr, P_{aCO_2} 32 torr, pH 7.46. Seven hours after the patient's admission to the Respiratory Intensive Care Unit in spite of decreased inspiratory stridor, increased tachypnea to 32/min and somnolence were found. Arterial blood-gas values (40 per cent oxygen by face mask) were P_{aO_2} 215 torr, P_{aCO_2} 50 torr, pH 7.35. Indirect laryngoscopy revealed an increase in the supraglottic edema involving the arytenoid cartilages and posterior aryepiglottic folds, with only slight edema of the epiglottis. The vocal cords were easily visualized but had essentially no abducting motion. The patient was taken to the operating room, anesthetized with halothane and oxygen, and the trachea intubated under direct vision with a Portex nasotracheal tube, 7.5 mm ID and 10.0 mm OD.

Following the procedure the patient appeared comfortable and more alert, breathing spontaneously without evidence of airway obstruction. Steroid therapy, ampicillin, and racemic epinephrine therapy were continued. The following day (96 hours after admission), indirect laryngoscopy showed decreased edema of the uvula and arytenoid cartilages. On the third day of intubation the patient was returned to the operating room, where direct laryngoscopy was repeated using deep halothane-oxygen anesthesia. The epiglottis was normal, with very slight edema of the arytenoid cartilages and mild erythema and inflammation of the true vocal cords. Despite limited motion of the left cord, the patient appeared able to maintain a patent airway, and the decision to extubate the trachea was made. The patient subsequently tolerated extubation well, maintaining his airway without difficulty. Steroid therapy was tapered off and discontinued 24 hours after extubation, and the patient was discharged from the Respiratory Intensive Care Unit, with normal arterial blood gases, 48 hours after extubation. The remainder of the hospital course was uneventful, with no recurrence of gastrointestinal bleeding.

DISCUSSION

Laryngeal edema associated with the use of nasogastric tubes most often results from mechanical irritation of the posterior esophageal wall and mucosa of the cricoid cartilage by motion of the tube. At this point, the cricopharyngeus muscle acts to keep the

cricoid cartilage pressed firmly against the cervical spine in order to keep the esophageal lumen closed. Mucosal irritation is greater when the nasogastric tube lies directly in the midline, i.e., at the point of greatest pressure, rather than laterally, where the effect of the cricopharyngeus muscle is less pronounced. Motion of a malpositioned tube in this area leads to inflammation of laryngeal tissue, especially the cricoid cartilage and arytenoid cartilage, which in turn predisposes to edema, infection, ulceration, and eventually stenosis. Less common causes include inflammation secondary to infection or acute cricothyroid arthritis. This patient did have *H. influenzae* identified by sputum cultures, and perhaps a prior upper respiratory tract infection initiated the sequence of events. However, any infection present could also have resulted from prior ulceration of mucosal surfaces, making it difficult to ascertain which preceded the other. Acute arthritis usually does not respond to treatment as quickly as in this case.⁴

This patient's course was unusual in that symptoms of upper airway obstruction developed rapidly, within 24 hours after placement of the nasogastric tube. A possibly significant difference between this case and those previously reported is in the type of tube used. Previous reports all involve the use of soft, flexible rubber Levine tubes. In this case, a no. 18 Fr Salem sump tube made from virgin polyvinylchloride was used. This tube is considerably stiffer than conventional Levine tubes and hence, movement of the firmer surface of the sump tube against the cricoid and esophageal mucosa may well have been the decisive factor in the sequence of events.

A case of acute laryngeal edema occurring 24 hours after passage of a nasogastric tube has been presented. Etiology has been discussed, with mention of the possible influence of the sump tube on the rapidity of development of symptoms. The possibility of laryngeal edema and upper airway obstruction must be kept in mind in the management of all patients who have nasogastric tubes in place, especially those with sump tubes, as symptoms and signs may be present earlier than in patients with rubber tubes.

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Renal Failure Following Enflurane Anesthesia

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Enflurane (2-chloro-1,1,2-trifluoroethyl-difluoromethyl ether; Ethrane, Ohio Medical Products) has rapidly become very popular, and is the inhalation agent most frequently used at this hospital. Enflurane is biotransformed in part to inorganic fluoride ion.¹ Fluoride ion-induced nephrotoxicity has been established as a cause of the vasopressin-resistant polyuric renal failure occasionally seen following exposure to significant doses of methoxyflurane.² Postanesthetic renal failure in a patient who received enflurane led to measurement of serum fluoride in a search for a possible etiologic factor.

REPORT OF A CASE

A 66-year-old white man (65 inches tall, weight 61 kg) was admitted to the hospital for an elective ileal-loop urinary diversion as first-stage treatment for a carcinoma of the bladder, diagnosed six weeks previously by biopsy during cystoscopy using

enflurane anesthesia. A rectal carcinoma had been excised by anteroposterior resection three years prior to admission. The patient had angina pectoris and a documented previous myocardial infarction, and was subject to paroxysmal atrial fibrillation. Medications were nitroglycerin and digoxin. Preoperative blood urea nitrogen (BUN) was 23 mg/100 ml; serum creatinine was 1.4 mg/100 ml, urinalysis showed 1+ protein by dipstick, with a few leukocytes evident microscopically. Preoperative blood pressure was 140/80 torr.

The patient had a six-hour operation with an uneventful anesthetic course for the creation of the ileal loop. Anesthesia was induced with thiopental, 200 mg, and succinylcholine, 100mg, and was maintained with 66 per cent N₂O, 33 per cent O₂, and enflurane at an average concentration of 1 per cent for the six hours, and a total of 5 mg pancuronium. Intraoperatively, the lowest blood pressure was 110/65 torr and the lowest arterial blood P_{ao} was 123 torr. Immediately following the procedure urinary output was greater than 40 ml per hour.

As shown in table 1, urinary output exceeded fluid intake on the first postanesthetic day, and the patient lost 1.2 kg in body weight. The patient became virtually anuric on the second postanesthetic day. Urinary sodium was 106 mEq/l, potassium 6 mEq/l. Dipstick urinalysis showed pH 7, 3+ protein, and no glucose, and many erythrocytes were evident microscopically. Blood was drawn for determination of serum inorganic fluoride ion. An intravenous pyelogram showed no evidence of obstructive uropathy or extravasation. Furosemide in three doses totaling 130 mg had no effect. On the third postanesthetic day, the patient was anuric. A single dose of furosemide, 300 mg, had no effect. A contrast study of the ileal loop failed to show reflux from the loop into the ureters. Urinary output spontaneously returned the following day, then gradually increased. BUN and creatinine improved as shown in table 1. At the time of the patient's

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