

Tension Pneumothorax Following Internal Jugular Cannulation and General Anesthesia

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Percutaneous catheterization of the internal jugular vein for central venous pressure (CVP) monitoring is used routinely in the operating room.¹ Of the complications attending internal jugular cannulation, tension pneumothorax during general anesthesia is probably one of the most serious.² Tension pneumothorax associated with internal jugular cannulation has not been previously reported.

REPORT OF A CASE

A partially dehydrated, 64-year-old man was scheduled for exploratory celiotomy. Additional history and physical examination were noncontributory, and results of all laboratory studies were within normal limits. Prior to induction of general anesthesia, we decided to catheterize the right internal jugular vein to monitor CVP and to administer hyperalimentation fluids postoperatively. Initially, the vein was approached anteriorly 1 cm above the clavicle (central or supraclavicular approach) with a 22-gauge 1.5-inch guide needle, without success. The approach then was changed to one higher in the anterior neck at the junction of the sternal and clavicular heads of the sternocleidomastoid muscle (anterior approach). A 17-gauge, 8-inch catheter was passed into the internal jugular vein through a 2-inch Bardic needle. There was blood return, and the CVP fluctuated with respiration at 0-1 cm H₂O. The patient was without complaint throughout this procedure; however, following placement of the CVP cannula, he became anxious and restless. Induction of anesthesia was begun after 5 minutes of preoxygenation. *d*-Tubocurarine, 6 mg, thiopental, 200 mg, and succinylcholine, 120 mg, were administered iv; vital signs were stable. Despite preoxygenation and efficient intubation, cyanosis about the lips and nail beds was seen. The endotracheal tube cuff was inflated. Manual ventilation proved difficult, and peak inspiratory airway pressures of 35-40 cm H₂O were

necessary to produce sufficient gas exchange. Wheezing was heard through an esophageal stethoscope. Auscultation of the chest revealed bilateral wheezing, but chest percussion appeared to be equal on both sides. Enflurane, 1 per cent, with oxygen at 6 l/min was used for maintenance of anesthesia. After the midline abdominal incision, nitrous oxide, 3 l/min, and oxygen, 3 l/min, were administered. Over the next 5 minutes wheezing increased and peak inspiratory airway pressure necessary for gas exchange approached 55 cm H₂O. Mean arterial pressure declined from 80 to 50 torr; CVP increased from 3-4 to 18 cm H₂O. Enflurane and nitrous oxide were discontinued. The right chest now was found to be tympanitic, with absent breath sounds. A #14 Jelco catheter was placed percutaneously between the second and third ribs anteriorly on the right, without an audible rush of air. The catheter was left open to room air and, within several minutes, the wheezing disappeared, along with the high peak inspiratory airway pressures. Vital signs returned towards preanesthesia values, and administration of nitrous oxide and enflurane was resumed.

DISCUSSION

The preinduction attempt at cannulation of the internal jugular vein near the clavicle probably caused the pneumothorax.² Cyanosis following preoxygenation, the appearance of wheezing, and increased airway pressures were the early signs.¹ We speculate that the pneumothorax may have been enlarged and stabilized during positive-pressure ventilation. Vital signs and chest examination were normal until nitrous oxide was administered. Nitrous oxide can enlarge a pneumothorax; in dogs, pneumothoraces of 300 ml doubled in volume within 10 minutes of breathing 75 per cent nitrous oxide.³ Thus, inhalation of nitrous oxide probably enlarged the pneumothorax enough to impair both ventilation and circulation. Hunter first described this phenomenon, which he found in tuberculosis patients who had chronic, artificial pneumothoraces as part of their therapy. During anesthesia, they received positive-pressure ventilation and nitrous oxide. Respiratory

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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 tube§ 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_{O₂} 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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