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Tension Pneumoperitoneum—A Cause of Ventilatory Obstruction

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Tension pneumoperitoneum is an unusual, but life-threatening cause of respiratory inadequacy. Though of obvious significance, pneumoperitoneum has not been discussed in the anesthetic literature. A case is described in which the use of intermittent positive-pressure breathing and preintubation manual ventilation by mask precipitated an increase in intra-abdominal pressure making ventilation almost totally impossible. Rapid abdominal decompression brought about instant relief.

REPORT OF A CASE

A 27-year-old mentally retarded, obese (height 174 cm, weight 111 kg) man suffered a 6-cm stab wound in the left mid-clavicular line over the sixth and seventh ribs. Except for some local wound tenderness, physical examination was entirely negative. Arterial blood pressure was 126/80 torr, pulse rate 100/min, and respiratory rate 20/min. The left lung was fully expanded with no subdiaphragmatic free air on chest roentgenogram. A nasogastric tube was passed; gastric aspirate was negative for blood and a left subclavian line was inserted. Eight hours later, decreased breath sounds were heard over the lower lobe of the left lung. With the negative chest roentgenogram and the belief that the pain was causing splinting, intermittent positive-pressure breathing (IPPB) was ordered in the mistaken anticipation of improving ventilation. Following this the patient complained of a bloated sensation. A second IPPB treatment was in progress five hours later when the patient complained of dyspnea and pain in the left shoulder and neck. A large quantity of free air under the diaphragm was observed on a chest roentgenogram. Antibiotic therapy was started and immediate surgery scheduled. Atropine, 0.4 mg, was then administered intramuscularly.

On arrival in the operating room a few minutes later, the patient complained of further dyspnea and had difficulty lying flat. The true degree of abdominal distention was difficult to estimate because of his marked obesity. Anesthesia was induced by the intravenous administration of thiopental, 300 mg, followed

by succinylcholine, 40 mg, to facilitate endotracheal intubation. Marked difficulty in ventilating the patient via mask was experienced. Following intubation, ventilation was controlled, but despite high inspiratory pressures, (up to 60 cm H₂O) maximum tidal volume was only 125 ml. Fiberoptic esophagogastrosocopy performed at this time was negative. With pronounced cyanosis present, the abdomen was rapidly opened and the ensuing hiss, caused by the escape of a large quantity of free air, confirmed the diagnosis of tension pneumoperitoneum. The tidal volume promptly increased to 700 ml with an inspiratory pressure of 25 cm H₂O; the cyanosis then disappeared. A small laceration was found high on the greater curvature of the stomach which was closed with a few sutures. No other injury was discovered. The post-operative course was uneventful and he was discharged a week later.

DISCUSSION

Pneumoperitoneum usually constitutes an urgent surgical emergency.¹ The etiology may be either from intra-abdominal causes such as a ruptured viscus² or from intrathoracic causes when air from pulmonary alveoli rupture first into the pleural space and then into the peritoneum via the retroperitoneal area along the esophagus and great vessels.³

Continued leakage from a ruptured viscus leads to the development of tension pneumoperitoneum. This effectively splints the diaphragm leading to severe dyspnea,⁴ or, as in our case, an almost total inability to manually inflate the lungs. Rapid decompression by trocar and cannula (or quick surgical incision if the patient is already in the operating room) can be as lifesaving as it would be in the case of pneumothorax.

In this patient the sequence of events appears to have begun with a small laceration of the stomach caused by the initial stab wound but without abdominal symptoms. The stomach remained decompressed by a nasogastric tube until IPPB was instituted. The IPPB probably forced air down the esophagus as well as into the trachea and despite the presence of a nasogastric tube, air now leaked through the gastric perforation into the abdominal cavity causing symptoms which indicated the need for surgical exploration. The patient's obesity masked the degree of pneumoperitoneum, while the manual ventilation prior to endotracheal intubation pushed more gas into the stomach and peritoneal cavity, increasing the tension to a point where the diaphragm became fixed.

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In retrospect our method of inducing anesthesia with thiopental and succinylcholine might not have been the ideal. However, the degree of splinting from the pneumoperitoneum and thus the difficulty in ventilating the patient was not anticipated. The patient was restless, mentally retarded, obese, and dyspneic, though not cyanosed at this time. Both inhalation induction and awake endotracheal intubation were considered and rejected as being too difficult and time consuming. Paralysis with rapid intubation of the trachea was the immediate goal and did, in fact, allow immediate surgical intervention.

If pneumoperitoneum is suspected, endotracheal intubation, awake or asleep, without prior artificial ventilation is indicated. If there is respiratory embar-

assment in association with pneumoperitoneum, abdominal decompression by trocar and cannula should be performed before induction of anesthesia.

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Intraocular Pressure after Transurethral Prostatic Surgery

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A problem associated with transurethral resection of the prostate gland (TUR P) is acute water intoxication.^{1,2,3} Visual disturbances have been attributed to cerebral edema resulting from this. These latter cases involved one patient with blurred vision,⁴ another seeing only a faint glow,⁵ and another who thought he was going blind.⁶ No intraocular pressure (IOP) measurements were made in these three cases.

Two patients recently complained of transient blindness following TUR P at the University of Nebraska Medical Center. This blindness resolved with correction of the hyponatremia.

We questioned whether transient blindness might be due to an increase in IOP from water absorption rather than cerebral edema. The purpose of this in-

vestigation was to study changes in IOP that may be related to the absorption of the non-electrolyte irrigating fluid employed in patients undergoing TUR P.

MATERIALS AND METHODS

Twenty-two patients between the ages of 54 and 81 years (mean age: 67) were scheduled for a TUR P. Approval for the study was granted by the Committee on Human Investigation at the University of Nebraska Medical Center. Preoperative informed consent was obtained from each patient.

Preoperative serum electrolyte determinations (Na^+ , K^+ , Cl^- , $p\text{H}$, CO_2) were obtained. Pre- and post-operative visual acuity was examined with a Rosenbaum pocket vision screener. Drug histories were obtained to note any drugs which might change the patient's IOP. None of the patients were premedicated.

Seven of the twenty-two patients had general anesthesia. No muscle relaxants were used. Two were anesthetized with thiopental/narcotic/ $\text{N}_2\text{O}/\text{O}_2$ technique, three with halothane/ $\text{N}_2\text{O}/\text{O}_2$ and one with enflurane/ $\text{N}_2\text{O}/\text{O}_2$. The remaining patients received subarachnoid or epidural block. Schiøtz tonometry was performed bilaterally approximately 20 min after induction of general anesthesia and after fixation of the block in patients with conduction anesthesia.

Intravenous fluid therapy consisted of 5 per cent

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