

Pulmonary complications following endotracheal intubation for anesthesia in breech extraction

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Abstract. A 28-year-old, healthy pregnant patient developed bilateral pneumothorax, subcutaneous emphysema, pneumomediastinum, pneumoretroperitoneum and pneumoperitoneum following endotracheal intubation and manual ventilation during general anesthesia for breech extraction. It is likely that positive-pressure ventilation was the cause for this very rare combination of complications. Early recognition and treatment may prevent such a catastrophe.

Key words: Pneumothorax – Pneumomediastinum – Pneumoretroperitoneum – Pneumoperitoneum – Subcutaneous emphysema – Breech extraction

Anesthesia with endotracheal intubation generally has an acceptably low incidence of complications. Although various authors have reported barotrauma following endotracheal intubation, the combination of subcutaneous emphysema, pneumothorax, pneumomediastinum, pneumoretroperitoneum and pneumoperitoneum is extremely rare [1,2]. The relative rarity of pulmonary barotrauma in anesthesia is partly due to the difficulty in increasing airway pressure above 50 cm H₂O by bag pressure alone [3]. We report an unusual combination of complications which occurred despite the presence of a reservoir bag. The possible mechanism of its development is discussed.

Case report

A 28-year-old was admitted to the maternity ward in her 39th pregnancy week with twins, due to premature rupture of membranes. Her past history was unrevealing. Her previous three pregnancies were normal with term deliveries. On admission, no abnormal finding was detected on examination. The head of the first

twin was engaged in the pelvis, fetal heart sounds were normal and the cervix was dilated to 4.5 cm. Ultrasonography showed the second twin to be in a breech presentation. Continuous analgesia was given with 10 ml of 0.5% bupivacaine at the L₁₋₂ level. Following 5 h of labor the cervix was completely dilated and a 2800 g male with 9/10 Apgar score was delivered. After amniotomy of the remaining sac, the diagnosis of the footling presentation was established. A trial extraction failed as the second leg could not be reached in the contracted uterus. General anesthesia was then immediately induced: An uneventful endotracheal intubation was performed with a soft-cuffed tube (8 mm ID) after 250 mg sodium thiopentone and 100 mg succinylcholine iv were injected. The second baby, a 3050 g female with 1/9 Apgar score was extracted by its feet.

The mother was connected to the manual mode of a Brompton-Manley ventilator, which was set to deliver 9 l/min fresh gas flow, immediately after endotracheal intubation. The first few manual ventilations were associated with increased resistance, abdominal distension and severe cyanosis. The endotracheal tube was checked and found patent and positioned correctly. Breath sounds were absent on auscultation and hypertympanic sounds were produced on percussion. Subcutaneous emphysema was felt all over the trunk and neck. A tension pneumothorax was suspected. Assisted ventilation was discontinued and the patient was allowed to breathe 100% oxygen spontaneously. Immediate chest and abdominal X-ray revealed bilateral pneumothorax with more than 50% collapse of both lungs (Fig. 1), pneumomediastinum (Figs. 1 and 2), pneumoretroperitoneum (Fig. 2), subcutaneous emphysema (Fig. 2) and pneumoperitoneum (Fig. 2). Bilateral chest drains were inserted immediately and connected to an underwater seal with continuous suction. The patient improved immediately. A chest X-

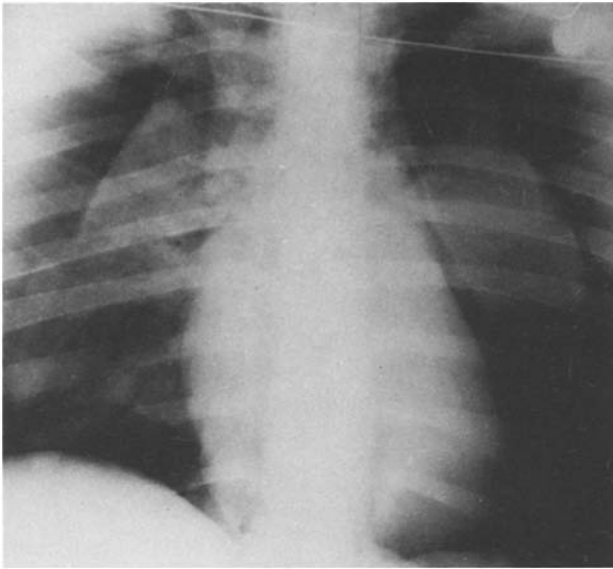


Fig. 1. Chest X-ray demonstrates pneumothorax and pneumomediastinum. The air passage to the peritoneum is clearly shown as demarcated lines of air on both sides of the thoracic and abdominal aorta

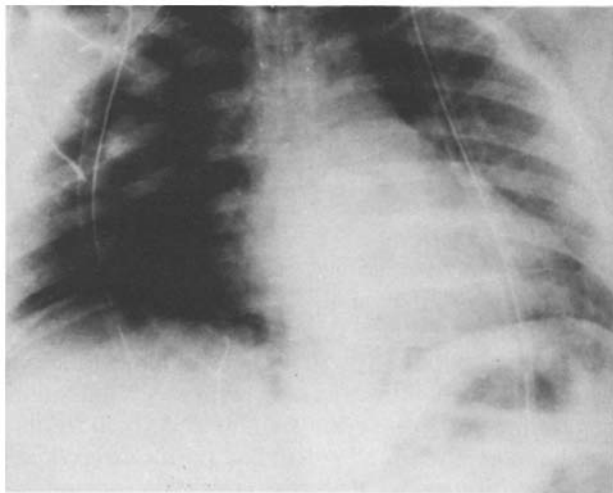


Fig. 2. Chest X-ray demonstrating surgical emphysema, pneumoretroperitoneum and pneumoperitoneum. The air passage to the peritoneum is clearly shown as demarcated lines of air on both sides of the thoracic and abdominal aorta

ray performed shortly afterwards showed complete reexpansion of both lungs (Fig. 2). At this time the patient was transferred to the ICU. A few hours later blood gases were normal and she was extubated. Her subsequent course was uneventful. Twenty-four hours later the chest drains were removed.

Discussion

Mediastinal emphysema in pregnancy is uncommon. It is known to complicate the second stage of labor and has been described as a rare complication of hyperemesis in early pregnancy, following general anesthesia for Cesarean section, after persistent coughing in pregnancy, or spontaneously in healthy people at rest. The mechanism is thought to be alveolar rupture caused by an acute increase in mean airway pressure, with a leakage of air along the vascular sheaths to the hilum and into the mediastinum. Other known causes of this pathology can be tracheal, esophageal or gastric rupture following instrumentation or trauma. It may also occur as a complication of bronchial asthma, pneumonia, or following inhalation of foreign bodies into the bronchial tree.

Spontaneous mediastinal emphysema is usually self-limiting. However, when its cause is tension pneumothorax the mediastinal pressure increases, causing cardiac decompensation and circulatory failure. In the latter case, the pneumothorax must be drained with an underwater seal. When mediastinal emphysema occurs in pregnancy, vacuum extraction or forceps delivery is recommended to minimize an increase in intrathoracic pressure during bearing down.

The further development of pneumoperitoneum is less easily explained. It has been shown that pneumoperitoneum when associated with pneumothorax or pneumomediastinum is more likely to be secondary to barotrauma itself. Other causes of this complication are rupture or perforation of a viscus which demand surgical exploration, pneumatosis intestinalis and a passage of air through the female genital tract following knee-chest postpartum exercises. It is assumed that since intra-abdominal pressure exceeds intrathoracic pressure by an average of 20 to 30 cm H₂O during both inspiration and expiration, simple (not tension) pneumothorax, with or without subcutaneous emphysema, should not be associated with pneumoperitoneum. However, positive-pressure ventilation with pneumothorax increases the risk of air dissection into the peritoneal cavity. This mechanism, leading to the combined barotrauma in our case, explains the clinical and roentgenologic presentations of this complication, i.e., tension pneumothorax with dissection of air to the mediastinum (Fig. 1) and subcutaneous tissue, and through the retroperitoneum (Fig. 2) into the peritoneal cavity (Figs. 2). The mechanism suggested here is in keeping with the experimental model in cats [4] in which interstitial emphysema developed with intratracheal pressure over 40 cm H₂O, pneumoperitoneum occurred at pressures over 50 cm H₂O, and subcutaneous emphysema and pneumoperitoneum were observed at pressures over 60 cm H₂O. This

mechanism is similar to that described by Rosen [5] in which air from ruptured alveoli dissects along the vessel sheaths into the mediastinum; as the pressure increases, dissection extends both into the pleural space and along the great thoracic vessels and esophagus to the retroperitoneum, rupturing the peritoneal cavity.

The dilemma in the above-described patient was whether she needed an explorative laparotomy to rule out a ruptured viscus. We preferred to observe the patient carefully as the cause of barotrauma was artificial ventilation.

This case illustrates a rare, life-threatening complication of endotracheal intubation and manual mechanical ventilation with a reservoir bag. A high index of suspicion and knowledge of the pathophysiology involved in endotracheal intubation and manual ventilation are necessary, concomitant with an immediate diagnosis and correct decision in order to save the patient's life.

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Book review

J. W. Sear (Guest Editor). Clinics in Anaesthesiology, Volume 2, Number 1. Philadelphia: W. B. Saunders 1984. £ 12.50

This timely book fills an important void in the current anaesthetic/intensive care literature. It brings up to date all the information concerning intravenous anaesthetic/sedative agents and the subject has been discussed in depth under two headings: Clinical Pharmacology and The Practice and Applied Pharmacology. The first part of the book opens with a succinct and fascinating historical account of the development of the intravenous agents and includes the comment that the word 'barbiturate' is derived from a combination of Barbara and Urea. This chapter is followed by a comprehensive account of the pharmacokinetics and pharmacodynamics of the barbiturates with some interesting information on the response of patients with renal failure. The current clinical status of ketamine has been well outlined in a chapter devoted to this agent and this has been followed by an article on the benzodiazepines and nonbarbiturate hypnotic drugs used for TIVA. This is a specially useful section as it discusses the pros and cons of the currently available agents with particular reference to the potential offered by di-isopropyl phenol in 10% intralipid. The chapter on hypnotic infusions used in regional anaesthesia and the intensive care unit is also particularly useful. It gives an excellent critique of the current state of the art although althesin had not been withdrawn at the time of writing. The next two chapters are concerned with detailed discussions about the use of narcotic analgesics and include particular reference to fentanyl. One of the important conclusions is that fentanyl infusion rates must be geared to the pharmacodynamic effects in the individual patient rather than to a given plasma concentration. The first section of the book ends with an interesting chapter on recovery from anaesthesia and includes an exploration of the problem of appropriate tests for assessment. Emphasis is placed on

the importance of speed and reliability of recovery in this era of day-case surgery and developing short hospital stay patient facilities. The applied pharmacology portion of the book opens with an enlightening account of the evaluation of intravenous anaesthetic agents in volunteers in Flinders Medical Centre, Australia. This article includes interesting tables showing details of the information sheets recommended for use by volunteers. A most comprehensive chapter follows on Adverse Reactions to Intravenous Anaesthetic Agents. It is particularly well organised, helpful and interesting to read. The cardiovascular and ventilatory effects of intravenous anaesthetics are next neatly discussed under two headings: the cardiovascular effects of single doses and the haemodynamic effects of continuous infusions. The section in this chapter discussing the baro reflexes during induction is particularly interesting as it points to the increase in heart rate being the result of release of vagal tone rather than increased sympathetic activity. An erudite chapter follows which discusses the concept of minimum infusion rate (MIR) as an index of equipotency for intravenous agents. MIR is the intravenous equivalent index of the minimum alveolar concentration (MAC) for the inhalational agents. This is an important attempt to try to establish criteria for prescribing optimum dosage regimens. An interesting chapter on monitoring anaesthesia concludes this volume. This covers the monitoring of drug uptake and broad aspects of patient monitoring which include the unusual dimension of lower oesophageal activity as well as the role of EMG and EEG recordings. This is an important monograph which should be read by all those concerned with the practice of anaesthesia and intensive care. As the book indicates the field of intravenous anaesthesiology is in a state of flux and as yet there is no perfect agent. The editor is to be congratulated on compiling a volume which summarises the present position.

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