Bilateral Tension Pneumothorax Following Induction of Anesthesia in Two Patients with Chronic Obstructive Airway Disease

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Tension pneumothorax during anesthesia is an unusual complication. The following case reports of patients with chronic obstructive pulmonary disease (COPD) illustrate the rapidity of onset and catastrophic events related to the development of tension pneumothorax.

REPORT OF TWO CASES

Patient 1. A 52-year-old woman with a history of asthma since the age of 14 years was scheduled for anterior vaginal-wall repair and a Marshall-Marchetti procedure. The patient weighed 140 pounds. She appeared well-nourished, with fair-to-good chest expansion and prolonged expiratory wheezes in both lung fields. Chest x-ray showed generalized emphysema. The EKG was normal and blood pressure was 110/60 mm Hg, pulse 80 beats/min and regular. Medications included chlordiazepoxide, Tedral, and a barbiturate h.s.; prednisone and an Isuprel Mistometer were utilized to control the asthmatic condition.

Preanesthetic medication included propiomazine, 25 mg, and atropine, 0.5 mg, im, 50 minutes prior to induction of anesthesia. Mildly sedated, the patient arrived in the operating room with slight prolongation of expiration and wheezing. Blood pressure was 150/100 mm Hg. At 9:50 AM, anesthesia was induced with thiopental, 100 mg iv, followed by 60 mg succinylcholine chloride. The patient was gently ventilated without difficulty with a mixture of 3 liters each of nitrous oxide–oxygen and 3 per cent halothane. The vocal cords were visualized easily, and the trachea and cords sprayed with 4 per cent lidocaine. At this time the patient coughed, but intubation was performed without difficulty with a #38 French cuffed oral endotracheal tube coated with 5 per cent lidocaine ointment. The cuff was inflated with 3 ml of air and ventilation was continued with the same anesthetic mixture, with an airway pressure of 20 cm H_2O. Prolongation of expiration was noted during auscultation of the chest when the endotracheal tube was checked for position. At 10:05 AM, the operation commenced. Blood pressure was normal; halothane concentration was 0.5 per cent. The chest became more difficult to inflate, and at 10:10 AM the surgeon remarked that the blood was dark. Oxygen (100 per cent) was administered, and at 10:15 AM there was pronounced expiratory wheezing, the patient became more cyanotic, and the operation was discontinued. At 10:17 AM, with peak airway pressures of 40 to 60 cm H_2O, there was no apparent chest movement, and no sounds could be heard on auscultation of the chest. At 10:20 AM, carotid pulsations ceased and external cardiac massage was begun. An attempt to accomplish intracardiac injection of epinephrine revealed the presence of air in the chest, and a diagnosis of tension pneumothorax was made. The needle was left *in situ;* by 10:23 AM, the patient was ashen gray, pupils were dilated, and subcutaneous emphysema had spread over the entire chest, into the neck and down into the abdominal wall. The EKG, by now applied, confirmed the presence of ventricular fibrillation; attempts to defibrillate were unsuccessful. A left thoracotomy incision revealed a greatly inflated left lung with air leaking from several small holes. Air was present in the pericardium, and needling of the heart indicated that it too was filled with air. Air could also be aspirated from an intravenous cutdown in the right arm. All further attempts at resuscitation failed. The patient was pronounced dead at 11:00 AM. Figure 1 is a photograph of the heart at autopsy, showing air bubbles in the left circumflex artery.

Patient 2. A 60-year-old woman was admitted with a mass lesion of the cecum and a right hemicolectomy was proposed. She had a history of heavy cigarette smoking for 20 years, with orthopnea, dyspnea on exertion, and morning sputum production for at least five years prior to the onset of her present illness. On physical examination, she was emaciated, weighing 90 pounds. Expiratory wheezes were heard throughout the chest. A tender mass was palpable in the right upper quadrant. Chest x-ray showed fibrous scarring of both lung apices with pleural thickening of the right costophrenic sulcus. Vital capacity was 1.1 liters; total lung capacity was 4.1 liters; functional residual capacity 3.2 liters; residual volume 2.7 liters; and FEV$_1$ 68 per cent. There was no improvement after bronchodilator therapy, and results of other laboratory studies were normal.

Preanesthetic medication was diphenhydramine, 50 mg, and atropine, 0.4 mg, im, an hour prior to induction. Blood pressure was 120/70 mm Hg

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and pulse rate 80/min. Anesthesia was induced with thiopental, 250 mg, iv, and the patient gently ventilated by mask with nitrous oxide–oxygen, 2 liters each per minute, without difficulty. Succinylcholine, 80 mg, was administered and the trachea intubated with a #36 French cuffed tube after topical application of lidocaine. Ventilation was manually controlled and anesthesia was maintained with the same anesthetic mixture. *d*-Tubocurarine, 6 mg, was given iv and the EKG leads connected; the tracing showed normal sinus rhythm. Five minutes after intubation the blood pressure was unobtainable, heart sounds were muffled, and heart rate had increased to 120/min. Breath sounds were difficult to hear. The patient was cyanotic, and there was marked impedance to ventilation. The abdomen appeared distended. The endotracheal tube was quickly removed and an attempt made to ventilate by mask with 100 per cent oxygen. It was impossible to inflate the chest, and the patient was reintubated with a #34 French cuffed tube. Cyanosis had visibly increased and there were no heart sounds. The chest remained impossible to ventilate and the EKG now showed complexes of low voltage. Auscultation revealed coarse rhonchi throughout the chest. Subcutaneous crepitation was detected on the lower left anterior chest wall. A presumptive diagnosis of pneumothorax was made. No other area of subcutaneous gas was detected at this time. A needle with a syringe attached was inserted into the left chest and air aspirated. A left chest tube was inserted and connected to an under-water seal. Immediately the patient's color improved, the chest was easier to inflate, and within 5 minutes a blood pressure of 120/70 mm Hg was obtained. A portable chest x-ray taken after the insertion of the left chest tube revealed bilateral pneumothorax, and a chest tube was then inserted on the right side. The patient regained consciousness soon after insertion of the left chest tube and was taken to the Recovery Room breathing spontaneously through the endotracheal tube. Within 30 minutes the trachea was extubated; at that time the patient was fully conscious and oriented. Neurologic examination disclosed no abnormalities. There was full expansion of both lungs by the second postanesthetic day, and the right chest tube was removed on the fifth day, with the left being removed a day later. Ten days after initial anesthetization she underwent uneventful right hemicolectomy, with continuous lumbar epidural anesthesia.

Discussion

Both victims of severe bilateral tension pneumothorax during anesthesia had severe obstructive-restrictive pulmonary disease with the clinical diagnosis of chronic bronchitis and emphysema. Smooth inductions were followed by uneventful tracheal intubations. Both patients were then in extreme difficulty within minutes. Could the foreign body in the trachea have created a series of events leading to bronchospasm?[1] Both patients were noted to have "asthmatic" breathing at the time of difficulty, both received nitrous oxide–oxygen in a 50:50 mixture with halothane, and both experienced periods of pronounced circulatory depression during the period of respiratory distress.

In 1937, Macklin[2] showed that vigorous positive airway pressure produced by blowing air into a local region of the lung created alveolar hyperinflation, frequently followed by pneumothorax. In 1955, Dundee[3] reported a case of tension pneumothorax during induction of anesthesia and related its cause to a hole in the esophagus caused by recent esophagoscopy and high mask-inflation pressure. In 1960,[4] a classic paper described the four major etiologic pathways for pneumothorax. Additionally, a series of six cases of "mediastinal emphysema," during anesthesia was reported in 1958.[5] Pulmonary blood vessels lie in connective tissue sheaths and shorten and elongate with expiration and inspiration. Gas under high pressure external to these vessels (coming originally from alveoli which have burst) enters these sheaths of connective tissue and may rupture into thin-walled blood vessels, creating a gas embolus to the right heart (patient 1).

Other causes of pneumothorax reported during anesthesia include operations on the neck where negative pressure is pronounced,[6] faulty anesthesia equipment leading to accidental high airway pressure,[7] recent supraclavicular brachial plexus block,[8] and high airway pressure during induction.[9] There are at least two reports[10, 11] of bilateral tension pneumothorax during anesthesia in patients having chronic obstructive airway disease, as did both patients reported here. The likelihood of coexistence of intrinsic lung disease and alveolar rupture, with pneumothorax, must be recognized.

The danger of nitrous oxide in a closed space such as a pneumothorax has been described.[12] Air placed in the pleural space doubles in volume in 10 minutes if nitrous oxide is breathed. Eger and Saidman[12] theo-

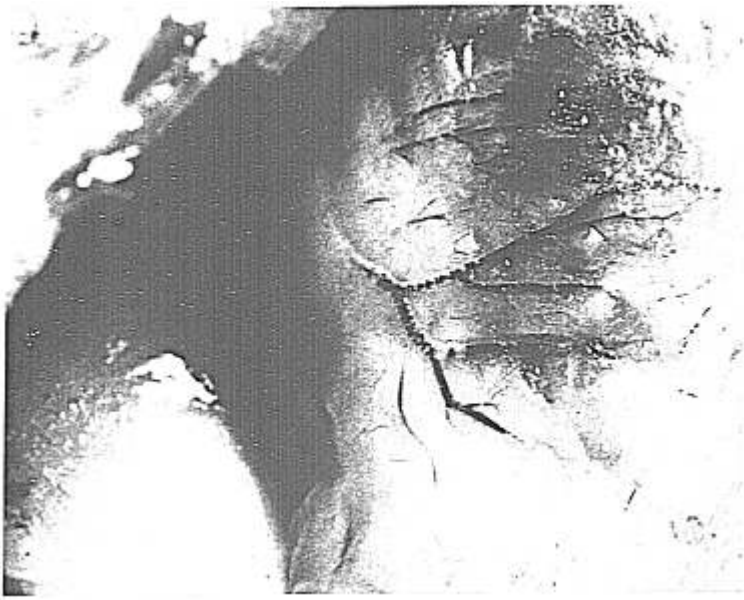

FIG. 1. The heart of Patient 1 *in situ* at autopsy, showing gas bubbles in the left circumflex artery. (From Werner U. Spitz and Russell S. Fisher, *Medicolegal Investigation of Death*, 1972, Courtesy of Charles C Thomas, Publisher, Springfield, Illinois.)

rized that pleural gas space expands more rapidly than other closed spaces (*e.g.*, bowel), owing in part to the greater blood flow to the pleura and in part to direct diffusion of nitrous oxide from alveoli beneath pleural surfaces. A tension pneumothorax provides direct access from airway to pleural space for any highly soluble gas such as nitrous oxide. It has also been demonstrated that oxygen is absorbed four times faster than air.[13] Since both patients described received 50 per cent nitrous oxide and cannot be assumed to have been completely denitrogenated, potential conditions for this closed-space volume-expansion situation existed.

The occurrence of a wheeze as a diagnostic clue during the establishment of tension pneumothorax was mentioned in 1955[6] and emphasized by Hamilton and Moyers[10] in 1966 and by Rastogi and Wright in 1969.[9] The apparent mechanism is bronchiolar compression resulting from the tension pneumothorax; this may delude the anesthetist into thinking that pure bronchospasm is the cause of the difficulty in breathing. This, in turn, may eventually result in misdirection of attempted therapy.

SUMMARY

Two cases of bilateral tension pneumothorax in patients with chronic obstructive airway disease are reported. Both patients came to operation apparently relatively asymptomatic and developed difficulty immediately after induction of anesthesia and tracheal intubation. Patient 1 died, but Patient 2 survived because of rapid diagnosis and removal of the gas. The available literature on tension pneumo-

thorax occurring during anesthesia is reviewed. Additionally, it has been found that a wheeze may occur as a diagnostic clue during the initiation of tension pneumothorax. However, since this wheeze may lead to a misdiagnosis of bronchospasm, the treatment of which may include raising the airway pressure, it may be dangerously misinterpreted.

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References

1. Gold MI, Helrich M: Pulmonary mechanics during anesthesia. V: Status asthmaticus. Anesthesiology 32:422–428, 1970
2. Macklin CC: Pneumothorax with massive collapse from experimental local overinflation of the lung substance. Can Med Assoc J 36: 414–420, 1937
3. Dundee JW: Tension pneumothorax during the induction of anaesthesia. Anaesthesia 10:74–75, 1955
4. Martin JT, Patrick RT: Pneumothorax: Its significance to the anesthesiologist. Anesth Analg (Cleve) 39:420–429, 1960
5. Nicholas JN: Mediastinal emphysema. Br J Anaesth 30:63–76, 1958
6. Schweizer O: Complications of anesthesia during radical surgery about the head and the neck. Anesthesiology 16:927–938, 1955
7. Gravenstein J: Pneumothorax and extensive emphysema after high intratracheal pressure in anesthetization. JAMA 171:158–160, 1959
8. Christian MS, Munson CS, Hamilton WK: Pneumothorax following induction of anesthesia. JAMA 209:1710–1711, 1969
9. Rastogi PN, Wright JE: Bilateral tension pneumothorax under anaesthesia. Anaesthesia 24:249–252, 1969
10. Hamilton WK, Moyers J: Pneumothorax during surgery. JAMA 198:187–188, 1966
11. MacKenzie AI, Patterson WD: Bilateral tension pneumothorax occurring during operation. Br J Anaesth 43:987–990, 1971
12. Eger EI II, Saidman LJ: Hazards of nitrous oxide anesthesia in bowel obstruction and pneumothorax. Anesthesiology 26:61–66, 1965
13. Northfield TC: Oxygen therapy for spontaneous pneumothorax. Br Med J 4:86–88, 1971

Anesthetic Hazards of Cold Urticaria

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Cold urticaria is an unusual disease characteristically manifested by relatively innocuous cutaneous lesions which develop on exposure to cold. Its recognition is essential, however, as it may be lethal under certain conditions.[1]

Report of a Case

A 25-year-old woman was admitted to Portsmouth Naval Hospital with symptoms of abdominal pain which had begun upon insertion of an intrauterine contraceptive device several days earlier. Roentgenograms of the abdomen showed the device to be intraperitoneal. A celiotomy was scheduled on an urgent basis.

Preanesthetic history and physical examination by both gynecologist and anesthesiologist disclosed no abnormalities except mild pain elicited by lower abdominal palpation. The patient specifically denied having any allergies.

Preoperative medication consisted of meperidine, 75 mg, diazepam, 5 mg, and atropine, 0.4 mg, administered intramuscularly an hour prior to induction of anesthesia.

Upon arrival in the surgical suite, an intravenous drip was begun, utilizing a 16-gauge polypropylene cannula, and 5 per cent dextrose in Ringer's lactate solution was administered briskly. Soon a painful erythematous wheal was noticed along the course of the cephalic vein and its tributary in which the cannula was placed. The solution, its container, and administration equipment were examined, but no gross abnormality was found. Similar equipment and solutions had been used for other patients that day without incident.

The entire iv set-up was immediately removed. The patient was requestioned and again denied having any allergies, including cutaneous reactions to contact with foreign materials. Using a 19-gauge metal needle and a 5 per cent dextrose solution from a different manufacturer, another infusion was begun in the other arm. While this was being done, the patient related that as long as she could remember, when exposed to cold, she would develop "hives," over the exposed area only. Meanwhile, a similar urticarial streak was developing along the course of the vein being used

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