

# Tracheal laceration with massive subcutaneous emphysema: a rare complication of endotracheal intubation

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**ABSTRACT** Our patient sustained a laceration of the membranous portion of the trachea associated with massive subcutaneous emphysema after an apparently uneventful intubation. The patient was successfully operated on within seven hours. The importance of prompt diagnosis and treatment in the event of this rare complication and the different causative factors are discussed.

Tracheal laceration is a rare complication of endotracheal intubation, which usually occurs during the act of intubation, or as a result of an immediate complication once the tube is in place (Blanc and Tremblay, 1974).

Published reports of the possible complications during the first two periods of intubation are numerous (McGovern *et al*, 1971; Blanc and Tremblay, 1974). The overall incidence of complications related to the act of intubation is unknown. In a recent study by Taryle *et al* (1978) 55% of patients being intubated in an emergency room suffered at least one complication during the procedure.

Laceration of the trachea has been reported as a result of repeated attempts at a difficult intubation, especially if a stylet is used (Schild *et al*, 1976). Other causative factors reported are tracheal abnormalities (Thompson and Read, 1968) and overinflation or rupture of the cuff of an endotracheal tube (Couniot and Santy, 1955; Elman *et al*, 1955; Törnvall *et al*, 1971), but tracheal laceration can occur in apparently uneventful intubation (Kumar *et al*, 1977).

## Case report

An 84-year-old woman with a long history of arteriosclerotic heart disease, diabetes, and hypertensive cardiovascular disease was brought to the

emergency ward. She was unconscious, cyanotic, and in obvious respiratory distress with blood-tinged sputum in the mouth.

She had blood pressure of 140/90 mmHg, pulse rate of 150/min, temperature of 37.2°C, spontaneous respiratory rate of 34/min with shallow breathing, and jugular venous distension. Bilateral diffuse coarse crackles with scattered wheezing were present throughout the lungs. Heart sounds were distant and unclear. The abdomen was soft, and bowel sounds were present. Hands and feet showed peripheral cyanosis and there was pitting oedema of the ankles.

Treatment with oxygen, intravenous frusemide, morphine sulfate, aminophylline, and the use of rotating tourniquets proved unsuccessful. The patient showed no evidence of clinical improvement, and the respirations became shallower. An electrocardiogram showed sinus tachycardia and a complete left bundle-branch block. A chest radiograph showed bilateral diffuse infiltrates with no evidence of cardiomegaly. Arterial blood gases when breathing 10 l/min of oxygen were as follows: Pao<sub>2</sub> 66 mmHg (9 kPa), Paco<sub>2</sub> 63 mmHg (8.5 kPa), and pH 6.93. After administration of diazepam, 5 mg intravenously, nasotracheal intubation was attempted with a Shiley low-pressure-cuff tube with a 6 mm inside diameter (Shiley Laboratories Inc, Irvine, California 92713). A minimal amount of bleeding resulted, and the

tube could not be introduced further than a few millimetres into the nasal passage. The naso-tracheal approach was therefore abandoned.

The patient was intubated with a similar tube without the use of a stylet and with no difficulty. The cuff had been tested and was intact. About 20 ml of air was used to inflate the cuff. On auscultation, breath sounds were heard throughout both lungs. Immediately after intubation a moderate amount of serosanguineous fluid drained from the endotracheal tube, which required almost continuous suction. About ten minutes after intubation while the patient was being manually ventilated a small leak was noticed around the endotracheal tube. At this point the pilot balloon was not turgid, so the cuff was deflated and then reinflated with 20–25 ml of air. No hissing around the tube was heard and breath sounds were heard throughout both lungs.

The patient was placed on a volume-cycled respirator at the following settings:  $\text{FIO}_2$  100%, tidal volume 1000 ml, positive end expiratory pressure (PEEP) of 5 cm  $\text{H}_2\text{O}$ , and IMV of 12 breaths a minute. Arterial blood gases obtained on the respirator at the above mentioned settings half an hour after mechanical ventilation had begun were as follows:  $\text{Pao}_2$  82 mmHg (11 kPa),  $\text{Paco}_2$  26 mmHg (3.5 kPa), and pH 7.36.

About 25 minutes after starting mechanical ventilation subcutaneous emphysema was noted on both sides of the neck, extending to the upper chest, face, and periorbital region. A chest radiograph showed that the endotracheal tube was above the carina, there was no pneumothorax, and subcutaneous emphysema was confirmed.

The PEEP was discontinued, and a tracheostomy was performed. A fiberoptic bronchoscopy via the tracheostomy tube showed an extensive laceration of the membranous portion of the trachea (figure).

A right thoracotomy confirmed the presence of a laceration extending from the upper thoracic trachea to the level of the carina. A closed airway system was established below the level of the tear, and the laceration was closed with silk sutures. A leak-proof closure was obtained despite difficulty caused by the poor quality of the tissue. Some of the mediastinal fat pad and areolar tissue was used to reinforce the suture line. The chest wall was closed in the usual manner. Two drainage tubes were inserted and attached to an underwater seal. A small air leak persisted.

The patient's respiratory state improved. Arterial blood gases obtained two hours after the operation when breathing 100% oxygen were as follows:  $\text{Pao}_2$  413 mmHg (55.1 kPa),  $\text{Paco}_2$  38 mmHg

(5.4 kPa), and pH 7.23. Penicillin was given because of the possibility of mediastinitis.

During the next 24 hours the patient's cardiac output remained low, and she had severe lactic acidosis. Despite all efforts to reverse this condition she suffered a fatal episode of bradycardia, despite several attempts at resuscitation. Request for necropsy was refused. At the time of her death the subcutaneous emphysema had dramatically improved.

## Discussion

Tracheal laceration as a direct complication of endotracheal intubation is rare (Stauffer and Olsen, 1978). Rupture of the trachea usually follows trauma to the chest and neck in patients injured in motor vehicle accidents (Hood and Sloan, 1959; Wilson *et al*, 1977). It can also occur as a result of a complication during the induction of anaesthesia (Törnvall *et al*, 1971), for instance, anaesthetic explosions, an increase in airway pressure during the anaesthesia caused by a closed expiratory valve, or opening the oxygen-flush valve while the expiratory valve is closed. Bronchoscopy, especially with the rigid instrument, may lead in inexperienced hands to tracheal rupture or laceration. Nach and Rothman (1943) describe instances in which rupture occurred when the head was suddenly thrown back during a severe coughing spell in an attempt to expel a foreign body. We are aware of only six previous cases where tracheal laceration could be attributed to endotracheal intubation (Couniot and Santy, 1955; Elman *et al*, 1955; Thompson and Read, 1968; Törnvall *et al*, 1971; Schild *et al*, 1976; Kumar *et al*, 1977).

There is no question that the intubation must be blamed for this injury, since our patient had not previously experienced any trauma and had evidence of massive subcutaneous emphysema before bronchoscopy and induction of anaesthesia.

What mechanism caused the tracheal injury? Possibly the tip of the endotracheal tube punctured the thin membranous portion of the trachea. In our case, however, this portion of the trachea, although thin, was not redundant, nor was the trachea bowed as in Thompson's case (Thompson and Read, 1968). A stylet was not used, and there was no difficulty in exposing the larynx. The endotracheal tube passed into the trachea without resistance. The endotracheal tube used in this case has potential dangers, which have been described previously by Clark (1965). The possibility of excessive inflation and rupture of the cuff exists since the pilot balloon in this prototype of endotracheal tube does not always reflect the degree

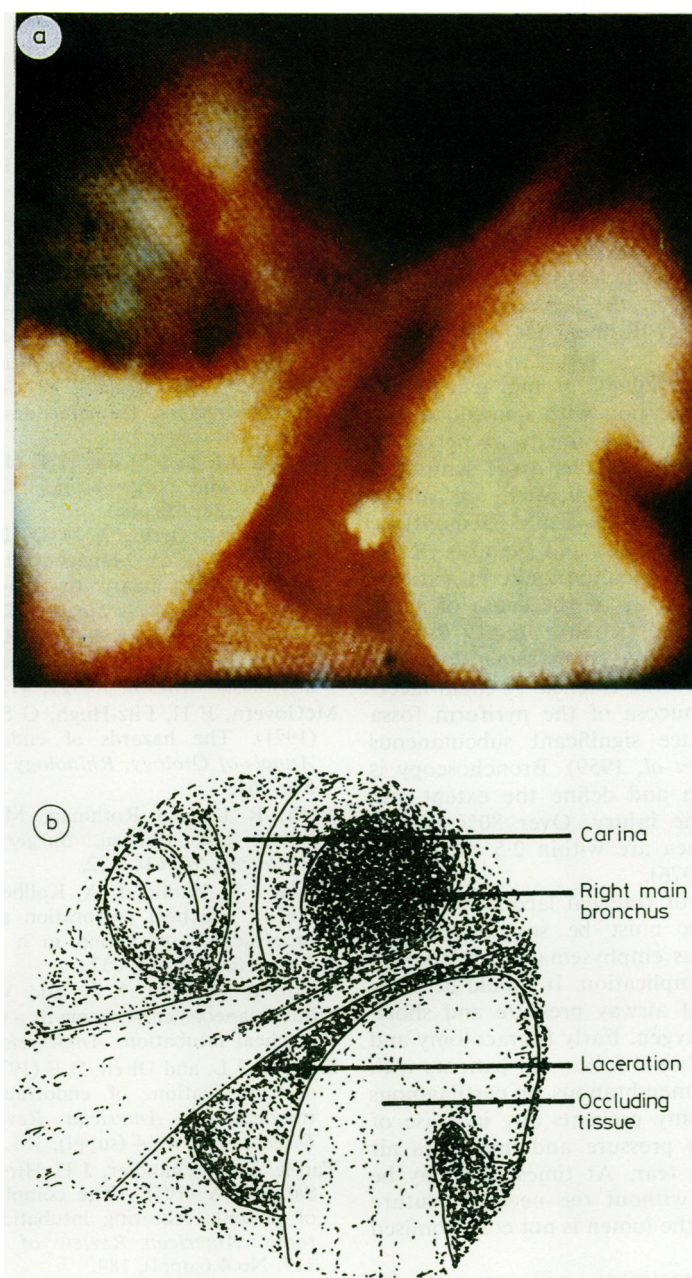


Fig 1 (a)—Photograph obtained during fiberoptic bronchoscopy showing tracheal laceration. (b) Artist's rendering of actual photograph.

of inflation of the tube cuff (Clark, 1965). The cuff acting as a distending force can cause tracheal rupture (Couniot and Santy, 1955; Elman *et al*, 1955; Thompson and Read, 1968; Törnvall *et al*, 1971). The normal trachea has been shown to withstand distension by the endotracheal cuffs, but in disease states this does not always hold true and the membranous portion of the trachea may yield. Further studies are necessary before we have a full understanding of the mechanisms concerned in these forms of tracheal injuries.

We think that in our patient the cuff acted as the distending force that caused the tracheal laceration. Possibly after the laceration occurred the cuff developed a small linear tear that caused the air leak.

Regardless of the aetiology, prompt recognition of this serious complication with immediate and accurate diagnosis and management are necessary if the patient is to survive. The usual symptoms that would herald this complication are subcutaneous and mediastinal emphysema, haemoptysis, and in severe cases dyspnoea and cyanosis (Kirsh *et al*, 1976). Tension pneumothorax in patients with tracheal injuries may be the cause of acute cardiorespiratory failure (Kirsh *et al*, 1976). The presence of subcutaneous emphysema does not always imply injury to the trachea. A small laceration through the mucosa of the pyriform fossa is enough to produce significant subcutaneous emphysema (Smith *et al*, 1959). Bronchoscopy is necessary to confirm and define the extent and exact location of the injury. Over 80% of the injuries to the trachea are within 2.5 cm of the carina (Kirsh *et al*, 1976).

The management of tracheal laceration is surgical. Pneumothorax must be sought for and treated. Subcutaneous emphysema in itself is not a life-threatening complication. It should be managed by reduction of airway pressure and short-term use of 100% oxygen. Early thoracotomy and closure of the tear should follow. In patients with a small tear of the membranous or cartilaginous portion a tracheostomy prevents any increase of intraluminal positive pressure and minimises air leakage through the tear. At times this may be sufficient treatment without the need for suture repair provided that the lumen is not compromised (Kirsh *et al*, 1976).

Early surgical treatment will prevent the potentially lethal complications of mediastinitis (Zimmerman *et al*, 1974) and tracheal stricture (Hood and Sloan, 1959). Excellent results can be expected in more than 90% of patients treated by early repair (Kirsh *et al*, 1976).

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The February 1979 issue of *Thorax* contains an article by Rubenstein, I, Baum, G L, Kalter, Y, Panzner, Y, Lieberman, Y, and Bubis, J J, entitled, "Resectional surgery in the treatment of primary carcinoma of the lungs with mediastinal lymph node metastasis." The material in this article is entirely contained in an article by the same authors entitled, "The influence of cell type and lymph node metastases on survival of patients with carcinoma of the lung undergoing thoracotomy" that was published in the February 1979 issue of the *American Review of Respiratory Disease*. The tables, when extracted, are similar and there is only a moderate degree of paraphrasing in parts of the text. Contributors are reminded that papers are submitted on the understanding that they are subject to editorial revision, and that they have not been and will not be substantially reproduced in whole, or in part, in any other journal.

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