# Anaesthetic management of tracheobronchial disruption during oesophagectomy

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## **Abstract**

Although tracheobronchial injuries occur rarely during oesophagectomy, the outcome of such injuries is mostly unfavourable. We report the case of a 50-year-old female, American Society of Anesthesiologists (ASA) class 1, who suffered a tracheobronchial injury during transthoracic oesophagectomy. The defect was repaired with an intercostal muscle flap but tracheobronchial disruption occurred again on extubation. As a result, she developed a profuse air leak postoperatively, through the bilateral thoracic and abdominal drains. A second surgical procedure using a single-lumen endotracheal tube was undertaken. During the procedure the patient deteriorated, owing to an increase in the tracheal rent, which resulted in a severe impairment of ventilation. This crisis was initially managed through advancement of the endotracheal tube into the left main bronchus. Subsequently, oxygenation and ventilation of both lungs was achieved by intubating both the main bronchi with microlaryngeal tubes, with the patient in the left lateral position.

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#### Introduction

Oesophagectomy has a high morbidity and mortality, despite advances in surgical technique and intensive care management. Tracheobronchial disruption is a rare, but the most feared, complication of transthoracic oesophagectomy (TTO). Early recognition and diagnosis are the keystones to management. We report the anaesthetic management of tracheobronchial disruption during TTO.

#### Case report

A 50-year-old non-smoker, nondiabetic, normotensive female was investigated for dysphagia and diagnosed with a middle-third oesophageal carcinoma. She was scheduled for a TTO under general anaesthesia.

During the preanaesthetic check-up, no history was revealed to suggest any medical illness. Her weight was 50 kg, height 154 cm, pulse 74/minute, blood pressure 134/82 mmHg and systemic examination revealed no abnormality. The full blood count, urine analysis, blood sugar, serum electrolytes, blood urea and serum

creatinine, pulmonary function tests, chest radiograph and electrocardiogram were within normal limits.

She was premedicated with alprazolam 0.5 mg and ranitidine 150 mg, the night before surgery and two hours preceding surgery. Standard monitors were attached prior to induction: heart rate 94/minute, blood pressure 124/86 mmHg and room-air saturation of peripheral oxygen (SpO<sub>2</sub>) 97%. An 18 G intravenous access line with Ringer's lactate was established. An epidural catheter was placed at the T9-10 interspace to aid postoperative analgesia. General anaesthesia was induced after intravenous glycopyrrolate 0.2 mg, using pentazocine 30 mg, propofol 120 mg and vecuronium 5 mg. After three minutes a leftsided 35 Fr double-lumen tube (DLT; Rusch, Germany) was inserted without any difficulty and correct placement confirmed by auscultation (a fibre-optic bronchoscope was unavailable). Urinary catheterisation and right subclavian venous and radial arterial cannulations were performed and the patient was placed in the lateral thoracotomy position.



Anaesthesia was maintained using halothane 0.5-1%. During dissection of the oesophagus, blood-stained fluid was seen in the clamped tracheal lumen of the DLT. This was found to be the result of an injury in the trachea at the level of the carina and was subsequently repaired with an intercostal muscle flap. Tracheal integrity was checked after the repair. After closure of the thoracotomy incision, the patient was placed supine. There was no difficulty in ventilation at that time. A right chest drain was inserted, showing minimal leakage. After completion of surgery, which lasted nine hours, the trachea was extubated.

Upon removal of the DLT, a suture was found attached to the tip of the bronchial lumen. Suspecting a tracheobronchial injury, the patient was reintubated with a cuffed orotracheal 7.5 mm polyvinyl chloride (PVC) tube. A left-sided chest tube was also inserted and the patient was transferred to the intensive care unit (ICU) for elective postoperative ventilation. She rapidly developed progressive subcutaneous emphysema, and massive bilateral bubbling was observed in the chest drains. Ventilation being inadequate, the ventilator settings were adjusted to a tidal volume of 200 ml and a rate of 30/minute, with a fraction of inspired oxygen (FiO<sub>2</sub>) value of 100%. SpO<sub>2</sub> improved to 97-98% and heart rate settled at around 130-140/minute with a blood pressure of 100/60 mmHg.

Arterial blood gas analysis after six hours revealed the following: pH 7.20, partial pressure of carbon dioxide (pCO<sub>2</sub>) 48.5 mmHg, partial pressure of oxygen (pO<sub>2</sub>) 102.1 mmHg, bicarbonate (HCO<sub>2</sub>) 14.6 mmol/l, base excess (BE) -12.8 and arterial oxygen saturation (SaO<sub>2</sub>) 97.4%. Other laboratory investigations were normal, but subcutaneous emphysema had progressed to the facial region and bubbling also appeared in the abdominal drain.

It was then decided to re-explore the surgical site. Anaesthesia was induced and maintained with the alreadyexisting single-lumen tube in place. The carinal rent was explored in the left lateral position. During exploration the rent increased, leading to total inadequate ventilation. There was a dramatic fall in SpO, and heart rate decreased to 30/minute. This was recognised as a life-threatening situation, and the endotracheal tube was pushed into the left main bronchus, improving saturation and correcting the bradycardia. The unilateral ventilating tube was replaced with two microlaryngeal tubes (MLTs), with the patient in the left lateral position. A 5.0 mm PVC MLT was surgically guided into the right main bronchus under direct vision, along with a 4.0 mm MLT in the left main bronchus. Ventilation was achieved using a DLT Y-connector. The carinal rent was resealed using a stomach serosal flap.

The patient was transferred back to ICU and ventilated with bilateral endobronchial tubes for 48 hours. Being small in size, the tubes became blocked and were replaced with a 7.0 mm PVC endotracheal tube on the third postoperative day. There was minimal leakage from the chest drains and ventilation was adequate, but the patient developed sepsis and passed away as a result of septic shock on the fifth postoperative day.

### **Discussion**

latrogenic tracheal tear is a rare complication that may result in an immediate inability to ventilate the patient. The incidence of tracheal tears during trans-hiatal oesophagectomy is less than one per cent in experienced hands, but there are insufficient data regarding TTO.1,2 Immediate presentation may include pneumothorax, pneumomediastinum, tracheobronchial haemorrhage, subcutaneous emphysema or difficulty during positive-pressure ventilation. Later lifethreatening respiratory complications such as atelectasis and pneumonia may occur because of hypoventilation and aspiration of blood through the tear. These patients may require prolonged ventilatory support in ICU.3,4

Tracheobronchial disruption is suspected perioperatively if a large amount of air is escaping through the operative field and/or there is a significant decrease in airway pressure or sudden changes in capnography readings.<sup>3,4</sup> In our patient the tracheal tear was suspected initially when a collection of blood was observed in the clamped tracheal lumen of the DLT, and it was confirmed when a large air leak was witnessed on ventilation through the tracheal lumen. During repair of this tear, a suture was inadvertently placed through the DLT, resulting in tracheobronchial disruption once again upon extubation.

Tracheal tears may be either membranous or transmural. Small tears can be managed conservatively, whereas transmural tears, extending more than 1 cm, need surgical repair.4 Various ventilatory approaches are described for repair of tracheobronchial injury, including spontaneous ventilation, high-frequency jet ventilation, high-frequency positive-pressure ventilation, cardiopulmonary bypass, insertion of a tube into the open trachea distal to the area of resection in addition to the use of a DLT, and standard orotracheal intubation.5,6 Other modalities, including a modified Ring, Adair and Elwyn (RAE) tube, modified Foley's catheter and rubber tubes cut off beyond the cuff, have also been reported for lower airway repair.7

Early extubation is always the primary goal in tracheal surgery.3 Keeping this in mind we extubated our patient on completion of surgery, but because of the tracheobronchial disruption after extubation, she had to be re-intubated



and was managed with assisted spontaneous ventilation utilising low tidal volumes with accompanying low airway pressures, until it was decided to repair the tear surgically. Since the tear was at the level of the carina, neither a DLT, nor tracheostomy, nor inflation of a cuff beyond the tracheal tear could be helpful. In this scenario, where there was a need to ventilate the lungs separately by the orotracheal route, achieving adequate tube length was a concern. We used MLTs with the desired length, but there were shortcomings. Occlusion of the right upper lobe bronchus hampered cuff inflation. Interestingly, Roy et al reported the insertion of MLTs in the sitting position using a fibre-optic bronchoscope, ventilating both lungs separately with two ventilators in a patient with a tracheogastric fistula.8

#### Conclusion

Tracheobronchial disruption is a rare but potentially grave complication of TTO, requiring a team approach and planning. We propose that specialised smaller-sized long tubes, with smaller cuffs, could be of help in this lifethreatening situation.

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