TRACHEOESOPHAGEAL FISTULA (TOF) — BLIND POUCH INTUBATION: A CASE REPORT

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SUMMARY

A case of Tracheoesophageal Fistula (TOF) was presented where the blind upper esophageal pouch was mistakenly intubated; in spite of this, adequate lung ventilation was possible for more than one hour. This was only noticed by the surgeon upon incision of the lower end of the pouch.

CASE HISTORY

The patient was a one-day-old male, born 38 weeks by dates but weighing only 1.56 kg. Failure to insert an orogastric tube beyond the 5 cm mark alerted the paediatrician to the possibility of Tracheoesophageal Fistula (TOF) and this was subsequently confirmed radiologically.

Immediate nursing care included low continuous suction through a Replogle tube inserted nasally into the blind upper oesophageal pouch and hourly flushing of the pouch with 1 ml of normal saline. He was placed in a cot at a temperature of 32-34°C and inspired oxygen concentration of 30%. Serum biochemistry was normal.

Anaesthesia was induced with halothane, the trachea was intubated by a Registrar with a size 3 non-cuffed Portex tube nasally and the lungs ventilated with a mixture of nitrous oxide and oxygen in a ratio of 2:1 using Ayre’s T-piece. No relaxant was given as breathing was easily controlled manually with halothane and fentanyl. Air entry was equal on both sides and chest expansion was satisfactory provided the neck was not unduly flexed. The tube was tightly secured to avoid displacement as the operation was to be done on the left lateral position.

Temperature control was facilitated by the use of a head stockinette, wrapping the body and limbs with gauze and the use of a heated-water warming blanket. Rectal temperature was kept at 36-37°C.

The Dinamap(R) and S & W(R) oscilloscope were used to monitor blood pressure and ECG respectively.

The TOF was approached via a right thoracotomy incision and the fistula was easily identified as joining the lower end of the trachea to the lower oesophageal end. Up till after the ligation of the fistula there were two short episodes of bradycardia when heart rate slowed down to between 70-90 beats per minute. This was attributed to inadvertent manipulation of the vagus nerve which was in close proximity. However, enrichment of the inspired oxygen tension and slight extension of the neck to allow better lung ventilation corrected the bradycardia without the use of atropine.

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After ligation of the fistula the next step was to anastomose the defect in the continuity of the oesophagus. When the lower end of the blind upper pouch was incised, a large air leak was observed with sudden loss of resistance to manual ventilation and chest expansion became minimal. The surgeon noted the presence of two tubes in the oesophageal pouch; one was the tip of the Replogle tube while the other was indeed the tip of the endotracheal tube.

Laryngoscopy confirmed the misplacement of the endotracheal tube and a new tube was used to intubate the trachea orally, this being done in the left lateral position so as not to upset the sterility of the surgical field. The patient thus suffered a period of bradycardia of as low as 50 beats per minute for about three minutes, but this reverted to normal spontaneously after administration of oxygen via the correctly placed endotracheal tube.

Surgery was subsequently uneventful. Inter­costal nerve block for postoperative pain relief was done under direct vision using a total of 3 ml of 0.25% Bupivacaine with adrenaline. Postoperatively, controlled ventilation was electively instituted overnight as the patient was noticed to have abnormal twitchings of his limbs and we could not rule out the possibility of cerebral oedema following intraoperative hypoxaemia. However, the twitchings subsided after calcium chloride therapy. The trachea was extubated the following morning in the absence of any evidence of pulmonary aspiration and blood gases being normal.

DISCUSSION

The anatomical classification of oesophageal atresia (EA) and tracheoesophageal fistula (TOF) is shown in Fig. 1. The abnormality in this patient is of Type C. As reported by Stothert et al,3 this accounted for 88.4% of total patient population. It was all the more surprising that the abnormality in this patient was not of type B, D or even E as it would have been easy to explain how we managed to ventilate the lungs had the ETT been in the oesophagus from the start.

![Fig. 1 Anatomical Classification of Oesophageal Atresia (EA) and Tracheoesophageal Fistula (TOF). Percentage incidence is shown in parenthesis.](image-url)
It had not occurred to us to check the position of the first ETT as chest expansion was adequate. We therefore could not discount the possibility that the ETT had been dislodged from its original position in the trachea. However, the following series of events gave convincing evidence that it was indeed the upper oesophageal pouch which was originally intubated.

The Registrar who intubated the 'trachea' had expressed doubts about the position of the tube but was convinced when chest expansion and air entry were satisfactory. Flexion of the neck with concomittant airway blockade by the tongue and soft tissues in the floor of the mouth caused decreased air entry with consequent hypoxaemia and bradycardia. Extension of the neck improved air entry. We had noticed that there was intermittent high pressure air leak from the corners of the mouth but had not attached much importance to this initially. The upper oesophageal pouch was 3 cm long and the tip of the ETT was noticed at its blind end. Had the ETT been in the trachea the tip would also have been the same distance from the larynx. It was unlikely that the act of extending the neck had managed to dislodge this length of ETT out of the trachea with it being securely anchored at the nostril. Lung expansion was satisfactory until the time of incision of the lower end of the upper oesophageal pouch when sudden loss of resistance and the presence of two catheter tips alerted us to this possibility. Moreover we were unable to inflate the lungs immediately thereafter.

It could be argued that with the ETT misplaced, the patient was actually breathing on his own. However, it was unlikely that his own spontaneous breathing alone had been adequate in this situation, taking into consideration the nature of the operation.
and the restriction of lung expansion. Furthermore, the use of fentanyl would have depressed his respiratory centre. Therefore it was reasonable to believe that we had actually managed to ventilate the lungs even with the ETT in the blind upper oesophageal pouch.

The following explanation is offered for the events. Manual compression of the reservoir bag actually pressurised the pharynx; the same effect that one would get by ventilating with a mask. In this case, one nostril was occupied by the Replogle tube while the other contained the ETT. The mouth was closed by two pieces of sticky tape slung around the jaw to fix the ETT securely. Thus, with all avenues for air escape closed, the path of least resistance was down the trachea when the airway was clear. This set-up was only disrupted when the upper oesophageal pouch was opened.

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