Clinical Reports

Hypoxaemia produced by an oesophageal stethoscope: a case report

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A seven-month-old child with complex cyanotic heart disease desaturated dramatically following induction of anaesthesia. While a degree of hypoxaemia would have been acceptable in this infant, pulse oximetry detected an abrupt desaturation prompting the anaesthetist to consider other less common causes of cyanosis. This episode of desaturation subsided with the removal of a 12 french oesophageal stethoscope which had been inserted following induction. Further attempts to re-insert this oesophageal probe led to repeated episodes of desaturation. The most likely cause of this desaturation was a reduction in pulmonary blood flow due to compression by the oesophageal probe of an aorto-pulmonary collateral posterior to the oesophagus.

Heart sounds and ventilation are usually monitored with an oesophageal stethoscope during anaesthesia and surgery for the palliation of complex cyanotic lesions in children. This monitor is considered to be safe, non-invasive and rarely associated with complications. Most reported complications involving the use of an oesophageal stethoscope resulting in hypoxaemia are due to interference with ventilation rather than alterations in pulmonary blood flow.^{1,2} The following case report describes an episode of hypoxaemia which occurred after

insertion of an oesophageal stethoscope. The hypoxaemia was most likely due to a reduction in pulmonary blood flow.

Case report

A seven-month-old female infant weighing 5.6 kg presented for palliative cardiac surgery. On examination the infant was mildly cyanotic with a respiratory rate of 54/min, systolic blood pressure was 114 mmHg and heart rate was 129 bpm. The chest x-ray demonstrated cardiomegaly and increased pulmonary blood flow. Arterial blood gases were: pH 7.40, PCO₂ 51 mmHg, PO₂ 49 mmHg, HCO₃⁻ 31.

Cardiac catheterization demonstrated a ventricular septal defect, a large collateral from the descending aorta to the main pulmonary artery, pulmonary atresia, and confluent pulmonary arteries. The aortic pressure was 85/30 mmHg, the right ventricular pressure was 93/5 mmHg and the large collateral was 60/25 mmHg. Oxygen saturation was 81 per cent in the aorta and 55 per cent in the right ventricle. The infant had failed to thrive despite aggressive medical management with digoxin and diuretics.

The planned surgical procedure was ligation of the aortic collateral to decrease the elevated pulmonary blood flow and reduce the risk development of pulmonary vascular occlusive disease. Because it was felt that ligation of the aortic collateral would critically reduce the pulmonary blood flow it was decided to establish a left Blalock-Taussig shunt prior to ligation of the collateral vessel to maintain pulmonary blood flow.

The patient was premedicated with atropine 0.1 mg IM 1/2 hour prior to surgery. On arrival in the operating room, the following monitors were applied; blood pressure cuff, doppler pulse meter, electrocardiogram, precordial stethoscope, and a pulse oximeter (right thumb). Following three minutes of pre-oxygenation, anaesthesia was induced with halothane by mask in increasing concentrations to a maximum of 2 per cent in 100 per cent oxygen.

Key words

ANAESTHESIA: paediatric, equipment; HEART DEFECTS: congenital; LUNG: hypoxaemia.

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FIGURE 1 Pulse oximetry record showing desaturation following insertion (i) of the oesophageal stethoscope and subsequent improvement following removal (r). The dashed line indicates oxygen saturation, the solid line represents heart rate.

After induction, a 20-gauge intravenous cannula was inserted in the left saphenous vein. Fentanyl $30 \mu\text{g}\cdot\text{kg}^{-1}$ and pancuronium $0.2 \text{ mg}\cdot\text{kg}^{-1}\text{mg/kg}$ were administered intravenously. The trachea was intubated with a 4.0 mm internal diameter endotracheal tube. Anaesthesia was maintained with isoflurane 0.25–0.5 per cent in oxygen, and a second dose of fentanyl $20 \mu\text{g}\cdot\text{kg}^{-1}$ was administered intravenously. Oxygen saturation remained stable at 96–98 per cent.

Shortly after intubation a 12 french oesophageal stethoscope with a nasopharyngeal temperature probe taped to the distal end was inserted into the oesophagus. Within 60 seconds of insertion, the arterial oxygen saturation decreased from 96 to 78 per cent. This decrease in saturation was not associated with changes in respiratory variables, although the right arm systolic arterial pressure increased 40 torr following insertion of the stethoscope. The oesophageal stethoscope was removed immediately and an abrupt increase in oxygen saturation to post-induction levels of 96 per cent occurred. The patient was then positioned in the right lateral position (the desired position for a left posterolateral thoracotomy) and the oesophageal stethoscope was again inserted. The arterial saturation again decreased, this time to 88 per cent and recovered as before with removal of the oesophageal stethoscope (Figure 1).

When the left lung was retracted to expose the pulmonary collaterals, the surgeons noted a large collateral arising from the pulmonary artery passing anterior to the oesophagus to end in the left lower lobe. A second collateral was identified arising from the descending aorta and passing behind the oesophagus to join the pulmonary artery. A left Blalock-Taussig shunt was fashioned with a 5.0 mm gortex graft. The collateral to the left lower lobe was ligated. When a ligature was placed around the large aorto-pulmonary vessel, the oxygen saturation fell to 70 per cent. The proximal end of the Blalock-Taussig was revised in order to improve pulmonary blood flow. A 3.0

FIGURE 2 Illustration of the anatomy of the central circulation adapted from the infant's cardiac catheterization record. Note the relationship of the aorto-pulmonary collateral to the oesophagus.

mm band was then placed around the large aorto-pulmonary collateral, in order to reduce pulmonary blood flow. At the conclusion of surgery the patient was transferred to the intensive care unit ventilated with 100 per cent oxygen. The oxygen saturation at that time was 80 per cent.

Discussion

This case report demonstrates that insertion of an oesophageal stethoscope in a patient with complex cyanotic heart disease may cause arterial oxygen desaturation. We attributed the desaturation to a reduction in pulmonary blood flow through a collateral. The exact site of pulmonary blood flow obstruction remains unclear but was likely due to obstruction by the stethoscope of the posterior aorto-pulmonary collateral (Figure 2). This vessel was immediately adjacent to the oesophagus and the arterial saturation decreased significantly when this vessel was temporarily occluded during surgery.

Oesophageal stethoscopes have been associated with hypoxaemia during anaesthesia. However, these episodes were generally due to some form of airway obstruction rather than vascular obstruction as described in this case.^{1,2} Pickard reported hypoxaemia after insertion of an

oesophageal stethoscope into the trachea.¹ The authors advocated a heightened awareness to the possibility of misplacement of oesophageal probes. Incomplete airway obstruction was the result of insertion of an oesophageal stethoscope, temperature probe and a nasogastric tube, in an infant with a double aortic arch.² In such patients, oesophageal monitors can compress the trachea below the endotracheal tube leading to partial airway obstruction. In our case report, although hypoxaemia occurred after insertion of the oesophageal stethoscope, it was not accompanied by any increase in airway pressure.

Had we been unable to monitor this patient's oxygen saturation with pulse oximetry, this episode of hypoxaemia might have gone unnoticed. It is apparent from our experience and cases reported elsewhere^{3,4} that monitoring oxygen saturation with pulse oximetry is essential during anaesthesia and surgery for palliation of congenital heart lesions.

The term "collateral artery" refers to a vessel connecting the aorta with the pulmonary segment. Survival often depends on the continued patency of these collaterals. This was the situation with our patient as any attempt to occlude this large vessel, even after the establishment of the Blalock-Taussig shunt, was accompanied by a precipitous decrease in oxygen saturation. The pressure within collateral vessels is usually less than that in the aorta because of stenotic lesions at their points of origin on the aorta.⁵ Such narrowing is desirable to decrease the pressure (which is otherwise equal to the systemic arterial pressure) and the flow through the collateral vessels to values more comparable with normal pulmonary artery pressure and flow. This natural decrease in pressure and flow will delay (but not arrest) the development of pulmonary vascular occlusive disease. Such a low pressure, however, predisposes collateral vessels to external compression by a foreign body such as an oesophageal stethoscope. In spite of these stenotic lesions the collateral artery of our patient was supplying too much pulmonary blood flow at too high a pressure making our patient at risk for development of pulmonary vascular occlusive disease and compromising future definitive repair. Since our patient would not tolerate ligation of this collateral even after establishment of a Blalock-Taussig shunt the final solution was to band this vessel to decrease pulmonary blood flow and pressure to acceptable levels.

In this case, we were confronted with a patient with complex cyanotic heart disease who progressively desaturated after induction of anaesthesia. To date, we are not aware that the insertion of an oesophageal probe is a cause of desaturation that the anaesthetist considers in his differential diagnosis. We conclude that oesophageal probes may cause arterial desaturation in patients with

large collateral pulmonary arteries, but that such occurrences are rare.

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Résumé

Un enfant âgé de sept mois présentant une malformation cardiaque congénitale cyanogène a présenté une désaturation dramatique après l'induction de l'anesthésie. Alors qu'un degré d'hypoxémie aurait pu être acceptable chez cet enfant le saturimètre a détecté une chute rapide de la saturation incitant l'anesthésiste à considérer d'autres causes moins fréquentes de la cyanose. Cet épisode de saturation s'est corrigé par l'extraction d'un stéthoscope oesophagien calibre 12 french qui était inséré après l'induction. D'autres tentatives de ré-insertion de ce stéthoscope oesophagien a amené une répétition des épisodes de désaturation. La cause la plus probable de désaturation serait la réduction du flot sanguin pulmonaire par une compression par le probe oesophagien d'une collatérale aoro-pulmonaire postérieure à l'oesophage.