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Recognising a reversed A-V shunt under anaesthesia in a neonate: the value of combined SpO2 PETCO2 and trends

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Key points

A reducing SpO2 combined with a rising PETCO2 may indicate a reversed cardiac shunt.

Abstract

Reversal of shunt under anaesthesia in children undergoing non cardiac surgery has been previously described. However, there is no prescribed formula for anaesthetising children with congenital heart disease for non-cardiac surgery. This case report demonstrates the reversal of left-to-right shunt flow through a persistent fetal circulation in a preterm neonate undergoing general anaesthesia for non-cardiac surgery.

Keywords: Cardiac septal defects, neonate, anaesthesia.

Case report

A 33 week premature 1.4 kg twin baby born by elective caesarean was found to have an isolated oesophageal atresia with no fistula. On day 1 he was scheduled for an urgent open gastrosotmy and was self-ventilating on room air with a peripheral SpO_2 of 96%. An echocardiogram had demonstrated concordant atria and ventricles with a patent ductus arteriosus, patent foramen ovale and tricuspid regurgitation.

Gaseous induction of anaesthesia was performed with a mixture of Sevoflurane in Oxygen. Muscle relaxation was achieved with atracurium and he was intubated with a 2.5 mm uncuffed oral endotracheal tube. Anaesthesia was maintained with 3% Sevoflurane in an equal oxygen air mixture. The ventilator was set to pressure control 14/4 cmH_2O, and rate of 30 per minute with an inspiratory time of 0.8 seconds. The $P_{ET}CO_2$ was approximately 3.0 and the waveform was appropriate. Pain control was achieved with 1mcg/kg fentanyl iv.

During surgical manipulation there was a reduction in SpO_2 and no change in the ventilation parameters to explain this. A concurrent rise in $P_{ET}CO_2$ from a stable level at 3.0 kPa and 4.5 kPa also occurred. This can be observed in the trend graph (Figure 1). The non-invasive blood pressure measured 48/34mmHg and remained stable throughout.

However, with additional intravenous fluid replacement the SpO_2 recovered to acceptable limits (SpO_2 88-95%)[1] within a short period of time. The recovery of SpO_2 was mirrored by the stabilisation and later a reduction in $P_{ET}CO_2$. Post-operative analgesia was achieved with 0.25% Bupivicaine local wound infiltration and Paracetamol iv. The neonate was successfully extubated at the end of surgery. He was self-ventilating in 30% oxygen and had a SpO_2 of 98-100% in recovery.

Discussion

Reversal of shunt under anaesthesia in children undergoing non cardiac surgery has been previously described[2] . However, there is no prescribed formula for anaesthetising children with congenital heart disease for non-cardiac surgery[3]. Reversal of shunt under anaesthesia may also occur in normal term neonates via a persistent fetal circulation in their first few days of

life. In the instance of a premature neonate, the risk of anaesthetic complications is further increased[4].

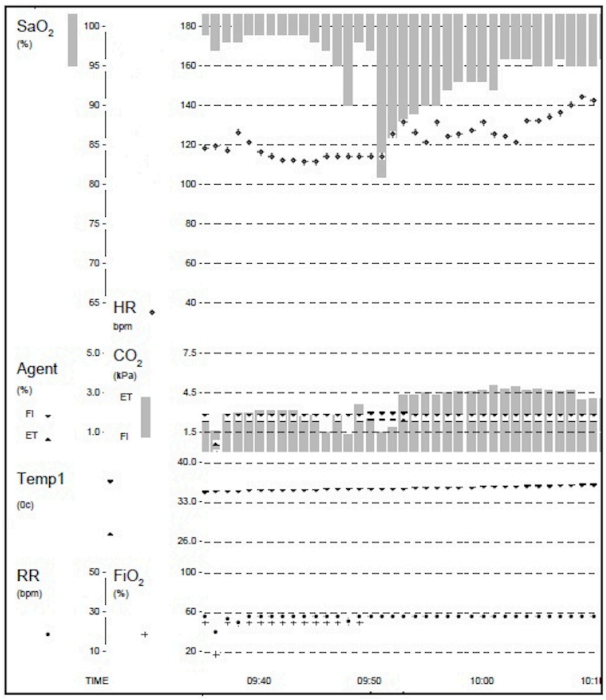

Figure 1. A trend graph showing a concurrent rise in $P_{ET}CO_2$ associated with lowering of SpO_2 and its subsequent gradual recovery

This case report demonstrates the reversal of left-to-right shunt flow through a persistent fetal circulation in a preterm neonate undergoing general anaesthesia for non-cardiac surgery.

There are many recognised factors known to affect the direction of shunt flow and the degree of shunting. Adequate oxygenation, CO_2 elimination and hydration are considered good practice to prevent an increase in pulmonary vascular resistance (PVR) and subsequent reversal of shunt. The manipulation of systemic vascular resistance (SVR) through vasopressors (phenylephrine) or pulmonary vascular resistance (PVR) through vasodilators (inhaled nitric oxide, prostacyclin) may be used to minimise the clinical effects of reversed shunts. Acute right-to-left atrial shunt is an important cause of profound hypoxia. The proportion of shunting between high-pressure systems like the great arteries or ventricles is dependent upon the size of the defect and the pressure gradient. This latter variable is determined by the ratio of PVR to SVR[5]. In this context, any factor that reverses the PVR:SVR ratio or alters the compliance of the ventricles can lead to a change in the proportion and direction of shunt flow[6]. This is particularly pertinent in a preterm neonate with shunting between high pressure arteries and ventricles and low pressure atria, via both a PDA and and PFO.

As was observed in our case, the reduction in SpO_2 in association with a rise in $P_{ET}CO_2$ could be explained by a reversal of shunt. In a scenario of concordant atria and ventricles with a PFO and PDA, under normal pressures, there would be a left-to-right shunt both at the PFO and PDA. Our findings are supported by observations made in the past, particularly in children with right-to-left shunting in whom $P_{ET}CO_2$ readings underestimated $PaCO_2$ and the discrepancy was greater in the presence of hypoxemia[7] .

The left-to-right shunt is defined as an anatomical communication between the systemic and pulmonary circulations that allows shunting of better saturated (systemic) blood to the less saturated (pulmonary) circuit. This means some blood draining from the pulmonary circulation, (both via PFO and PDA) will re-enter the pulmonary circulation without going through the systemic circulation. As a result there would be a 'pulmonary re-circulation' (see Figure 2). Thus, the CO_2 in systemic *venous* blood entering the pulmonary circulation via the right atrium will always be diluted by the re-circulating pulmonary venous blood leading to a lower CO_2 that will be reflected as a low $P_{ET}CO_2$. Therefore, we propose that, when the left-to-right shunts reverses, either at PFO or PDA level or both, the blood in the right atrium and blood in the pulmonary arteries (systemic venous blood) will enter the systemic circulation. Thus, a state of 'systemic re-circulation' will begin (see Figure 2). With a 'systemic re-circulation', CO_2 excretion via lung will diminish and as a result, CO_2 will rise in systemic blood. When this

blood enters lungs, it will be reflected by a rise in $P_{ET}CO_2$.

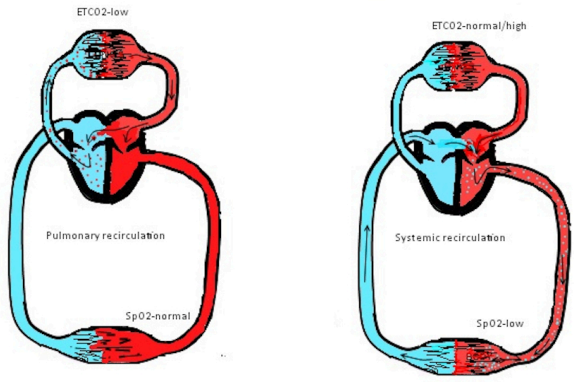

Figure 2. A sketch diagram of 'pulmonary re-circulation' and 'systemic re-circulation'

Therefore, L-R shunting at PFO and PDA level with 'a pulmonary re-circulation' will show a lower $P_{ET}CO_2$ as we observed in our patient at the beginning of anaesthesia. With reversed shunting, i.e. R-L shunting, a 'systemic recirculation' will ensue, increasing the $P_{ET}CO_2$. Therefore, a lowering of SpO_2 combined with a rise in $P_{ET}CO_2$, in the absence of any changes in ventilation can be interpreted as a situation of 'reversed shunt' as shown in our trend graph (Figure 1). This is relevant as the ability to objectively demonstrate the degree of shunting under anaesthesia in a neonate of 1.4 Kg is difficult and cumbersome.

'Pulmonary re-circulation' and 'Systemic re-circulation' are not terms that have been previously defined in the literature. In this context, a simple definition would be appropriate. The re-entry of pulmonary venous blood to pulmonary circulation without going through the systemic vascular bed is termed 'pulmonary re-circulation' whereas the converse, i.e. the systemic venous blood re-entering the systemic circulation without going through the pulmonary vascular bed is termed 'systemic re-circulation'. A reversal of flow in a patent ductus arteriosus would also fit this definition.

With reversal of a left-to-right shunt, less CO_2 reaches the pulmonary circulation, and its excretion rate will fall in a mechanically ventilated baby. This will lead to a rise in arterial CO_2, which will be reflected as a rise in $P_{ET}CO_2$ in a steady state.

Conclusions

Improving SpO_2 can be considered to reflect a reduced right-to-left shunt, and hence trend graphs of combined SpO_2 and $P_{ET}CO_2$ monitoring may be a good tool to assess shunt status in this scenario. Furthermore, both these parameters are continuous and non invasive measurements easily applicable in a neonate.

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