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End tidal carbon dioxide (ETCO2) and peripheral pulse oximetry (SpO2) trends and right-to-left shunting in neonates undergoing non cardiac surgery under general anesthesia

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Keypoints

In general, a reversed intra-cardiac shunt under general anesthesia leads to a reduced peripheral oxygen saturation combined with a rise in arterial CO2 tension (PaCO2). This is usually reflected as a rise in ETCO2. Therefore a reduction in SpO2 and a rise in ETCO2 noted under general anesthesia in neonates can be considered due to a reversed shunt, when other causes such as airway obstruction are excluded. Above clinical features may not be applicable if the reversed shunt is large and it substantially reduces pulmonary blood flow. In this situation a lowering of ETCO2 and SpO2 may be noted with a rise in PaCO2. Use of vasopressors to increase systemic vascular resistance seems an important aspect of treatment.

Abstract

Pulse-oximetry and capnography monitoring has improved the safety of anaesthesia. Using two case-reports we illustrate how trends in combined and concurrent pulse oximetry and capnography can assist diagnosis and management of acute right-to-left shunts in neonates undergoing non-cardiac surgery under general anaesthesia.

In both cases, rapid desaturation was the dominant sign resulting from right-to-left blood flow at the level of the patent ductus arteriosus and/or functionally opened foramen ovale (reversing the preoperative left-to-right shunt). The concurrent hypercarbia detected in arterial blood gas was significant but possibly not evident in $ETCO_2$ because only a small proportion of systemic venous blood reached the pulmonary circulation for gas exchange.

Treatment methods included improving oxygenation by increasing inspiratory oxygen concentration (FiO_2), volume expansion to improve pulmonary blood flow, reduction of CO_2 by hyperventilation and increasing the dopamine infusion dose in one infant to augment systemic vascular resistance.

In the event of a reversed left to right shunt, pulmonary arterial blood CO_2 ($PaCO_2$) rises as a result of systemic recirculation. However, a rise in $ETCO_2$ depends on the remaining pulmonary blood flow. For example, if the reversed shunt is large (i.e. increasing right to left shunt), then pulmonary blood flow is reduced and the CO_2 delivered to the lungs per unit time becomes inadequate to reflect it in expired breaths as $ETCO_2$. Therefore, $ETCO_2$ can be low in a suddenly reversed shunt state. Poor pulmonary blood flow leads to a low CO_2 elimination in expired air resulting in a low $ETCO_2$ record.

Keywords: Neonate, reversed intra-cardiac shunt, capnography, peripheral oxygen saturation, general anaesthesia.

Introduction

Several decades ago, the first sign of hypoxia under anaesthesia was cyanosis [1]. With the use of pulse-oximetry, hypoxia was recognisable before the onset of cyanosis. This led to a step-change in the safety of anaesthesia [2]. Capnography provided an additional safeguard by detecting airway issues before the onset of desaturation [3].

Interpretation of concurrent pulse oximetry and capnography further enhanced the detection and understanding of complex clinical scenarios: for example partial airway obstruction, pulmonary embolism, and oesophageal intubation [4] [5].

A rise in end-tidal carbon dioxide ($ETCO_2$), with concurrent desaturation was recently described in reversal of intra-cardiac shunt [6]. In this report, we describe a sudden intraoperative reduction in $ETCO_2$ with the reversal of intra-cardiac shunts in two neonates undergoing non-cardiac surgery.

In addition, we discuss the role of $ETCO_2$ and peripheral pulse oximetry (SpO_2) in the clinical diagnosis, potential intraoperative triggers, and immediate treatments of this life-threatening scenario.

Case report 1

A 6-day old, 640g neonate, born at 25-week gestation and ventilated since birth required an urgent laparotomy for a suspected intestinal perforation. She was on positive pressure ventilation (FiO_2 40%, respiratory rate 55 per minute, peak inspiratory pressure (PIP) 20cm H_2O and positive end expiratory pressure -PEEP 5cm H_2O). She was also requiring inotropic support with both dopamine and dobutamine each at 10 mcg/kg/hr. There was a right-sided pneumothorax with an intercostal drain in-situ. An Echocardiogram demonstrated a patent ductus arteriosus with normal cardiac anatomy.

Anaesthesia was induced and maintained with sevoflurane in oxygen and air, together with fentanyl and atracurium boluses, and continued inotropic support. During surgery, an episode of significant desaturation was noted immediately following a transfusion of 10mls of packed red cells. This was associated with a concurrent reduction of $ETCO_2$ (Figure 1).

Air embolism was considered unlikely as no air bubbles were seen in the intravenous access lines. An arterial blood gas showed a mixed respiratory acidosis (pH 7.11, $PaCO_2$ 10.9 kPa, PaO_2 7.7 kPa, BE -4.9 and Standard HCO_3 20.9 mmol/l) with a concurrent end-tidal $ETCO_2$ of 2.8 kPa. The blood transfusion was stopped, dopamine increased to 20mcg/kg/min and the inspired oxygen concentration was increased to 97%. The patient's oxygen saturations improved dramatically, and simultaneously the $PaCO_2$ returned towards normalcy (Figure 1 - Case 1).

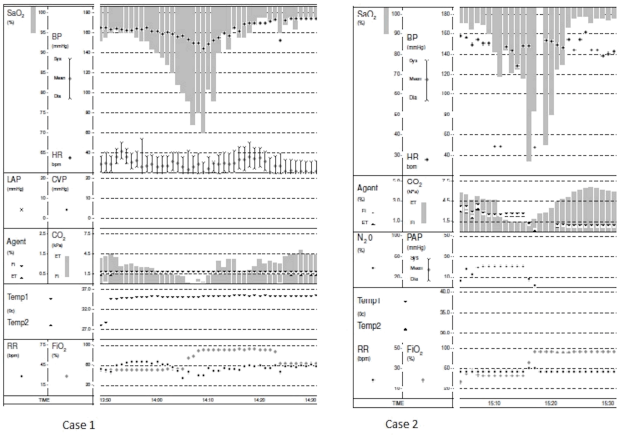

Figure 1. Anaesthetic trend charts showing reducing $ETCO_2$ and SpO_2 followed by gradual recovery of oxygenation associated with a subsequent $ETCO_2$ overshoot with ongoing rectifying measures.

Case report 2

A 38-week neonate was delivered by elective caesarean section for urgent surgery of an antenatally diagnosed meningo-myelocele. At 2 hours of age she was in the operating theatre self-ventilating room air whilst recording a variable peripheral SpO2 of 85-95%. She was not noted to have any other congenital abnormalities. The heart was reported normal in the antenatal scans.

Anaesthesia was induced with sevoflurane in oxygen and air, together with fentanyl and atracurium boluses for intubation. Immediately after induction, a reduction of $ETCO_2$ was noted with an associated reduction in SpO_2. A bolus of fluid (20mls of Hartmann's) was given

and FiO_2 was increased to 100%. The SpO_2 recovered and $ETCO_2$ also increased (Figure 1- Case 2). An arterial blood gas at this time showed a pH 7.21, $PaCO_2$ 8.8 kPa, PaO_2 26.7 kPa, BE -3.0 and a concurrent end-tidal (ET) CO_2 of 4.5 kPa.

Both neonates returned to neonatal intensive care in stable condition. They both made good clinical progress following surgery.

Discussion

Although a rise in $ETCO_2$ has been described in reversal of left-to-right cardiac shunts [6], in this report we conversely observe a reduction of $ETCO_2$.

We discuss possible explanations for a large discrepancy between $ETCO_2$ and $PaCO_2$, and triggering mechanism that led to the reversal of shunts. We also explain the rationale for rectifying measures undertaken, both physiological and pharmacological in these two neonates presenting with sudden intra-operative right-to-left-shunts.

The role of ET CO_2 and $PaCO_2$

The arterial-alveolar tension gradient for CO_2 has been investigated in normal new-born infants and in sick neonates as early as in 1962. Normal infants had virtually no PCO_2 gradient from pulmonary capillary to alveolus, whereas in sick infants an average difference observed was 13.9 mm Hg. This gradient for PCO_2 was considered to be a consequence of increased alveolar i.e. physiological dead space, largely due to poor perfusion of ventilated alveoli. In severely ill infants more than 60% of ventilated alveoli appear to be under-perfused [7]. What we have demonstrated in this report is a comparable situation of a larger dead space created by reduced pulmonary blood flow following a reversed left to right shunt.

Low pulmonary blood flow during right-to-left shunting is recognized. For example, cardiac catheterization data in neonates with transitional circulation has shown sluggish radio opaque dye movement in the pulmonary circulation when there is right-to-left intra-cardiac shunting via the foramen ovale and ductus arteriosus [8]. Since blood gases during cardiac catheterisation was masked with therapeutic interventions such as bicarbonate therapy, interpretation of $ETCO_2$ had been difficult in the past [8]. This was not so in our cases as no interventions were made that could directly interfere with the interpretation of blood gases.

In both our cases the $ETCO_2$ was markedly lower than $PaCO_2$ in association with a lower SpO_2. Our interpretation was that the systemic desaturation was due to reversal of blood flow at the level of the patent ductus arteriosus and/or concomitant right to left flows across a functionally open foramen ovale, the so-called transitional circulation. The desaturation and hypercarbia was related to relative separation of the two circulations with systemic recirculation of increasingly hypoxic and hypercarbic blood (Figure 2).

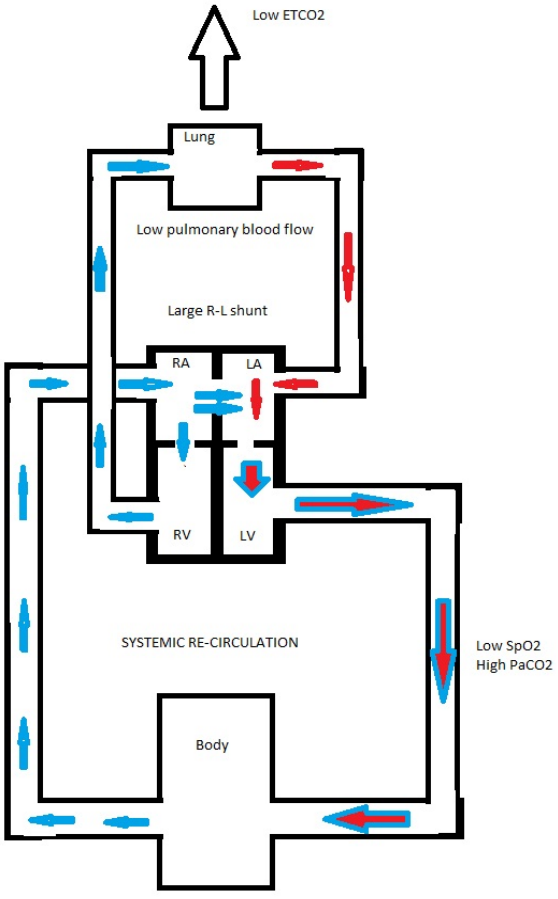

Figure 2. A schematic diagram of 'systemic re-circulation' leading to hypercarbia and desaturation. Lower $ETCO_2$ was due to proportionality low pulmonary blood flow.

A rise in $ETCO_2$ and a reduction in SpO_2 with reversal of L-R shunts in neonates results from accumulation of CO_2 in the systemic circulation due to newly established 'systemic re-circulation' following the reversal of the left to right shunt [6]. In our cases a high arterial CO_2 and a reduced CO_2 was detected in the expired gas. This is because only a small proportion of the systemic venous blood was able to reach the pulmonary circulation for gas exchange. Thus, a reversed left to right shunt can lead to a spiral decline with worsening hypoxia and rising pulmonary vascular resistance, diverting more blood away from the lungs.

The reduction in $ETCO_2$ was possibly related to pulmonary blood flow that was proportionately too low to establish adequate gas exchange. We propose that a reduction of $ETCO_2$ can also be a presenting feature in a reversed left to right shunt situation mimicking pulmonary embolism.

During the recovery phase, with the improvements in oxygenation, a rise in $ETCO_2$ was noted before it would plateau to pre-event levels. This is because with a large left to right shunt reversal, there is a build-up of CO_2 in the systemic circulation that would be released to alveolar gas with the re-establishment of the pulmonary circulation. The diagnosis of a reversed shunt using the above clinical features is swift and this will help clinicians to ameliorate such a situation before serious decompensation occurs.

In literature, end-tidal CO_2 monitoring in neonatal intensive care is described as less precise than transcutaneous monitoring. Therefore, it was considered only useful for trending [9]. It was not a substitute for direct $PaCO_2$ analyses in preterm infants [10] or in severe chronic pulmonary disease [11]. This discrepancy between $PaCO_2$ or transcutaneous CO_2 and $ETCO_2$ can be explained by variable shunt flows that may have co-existed in these neonates that have not been taken into account in the above investigations. However, the value of capnography trending in neonatal anaesthesia is overwhelming as demonstrated in our cases. For this purpose, sampling methods i.e. distal side stream vs mainstream is no bar [12].

We have described in these case reports how intracardiac shunting direction change creates a major gap between $ETCO_2$ and $PaCO_2$. We have also shown how we can use the widening $PaCO_2$-$ETCO_2$ gap resulting from a rise in $PaCO_2$ and a fall in $ETCO_2$ especially associated with a reducing SpO_2 to suspect a reversal of a shunt or re-establishment of a foetal circulation. Since $PaCO_2$-$ETCO_2$ gap has been shown to be consistently reliable in the same individual [13], $PaCO_2$-$ETCO_2$ gap may have a value for gauging the proportion of intracardiac shunting in the neonatal intensive care set-up where direct measurement of shunt flow by Doppler is not practical with the currently available instruments.

Potential triggers of sudden intraoperative right-to-left shunts

What we have demonstrated in our cases is a situation mimicking persistent foetal circulation under anaesthesia. The triggers we observed in one patient was blood transfusion and anaesthetic induction agents in the other.

Why can shunting suddenly be reversed under general anaesthesia? One known mechanism is lowering of systemic vascular resistance (SVR) leading to lowered systemic blood pressure and reversed pressure gradient at shunt level. This was the most likely mechanism in case 2. In case 1, however, the reversed shunt status was triggered by a blood transfusion whilst the patient was also on vasopressors. The most plausible explanation is a rise in pulmonary vascular resistance (PVR) reversing the pressure gradient shunt level. This mimics persistent foetal circulation in neonatal intensive care.

Ten millilitres of blood constituted 15-20% of blood volume in this neonate. This could have introduced a significant change in HbF: HbA ratio leading to a situation of poor oxygen release due to two reasons. Firstly, HbA is inherently poor in release of oxygen at tissue level with small changes on oxygen tension when compared to HbF. Its known that HbA increases right shift

of the oxygen dissociation curve, resulting in **less** affinity of oxygen in HbA, thus facilitating oxygen release and oxygenation in the pulmonary circulation. It also shown that in extremely premature neonates (< 27 weeks) the mean P_{50} prior and after transfusion of 26.9 mL/kg of packed red cells was 18.5±0.8 and 21.0±1 mm Hg (*P*=.0003) while the mean percentage of HbF was 92.9±1.1 and 42.6±5.7%, respectively [14]. In maternal blood P_{50} was estimated as 30.1mm Hg whereas in the normal new-born it was 22.9 mm Hg. P_{50} rises with addition of 2,3 DPG in vivo [15].

However, HbF with an extreme left shift oxygen dissociation curve, is designed to capture oxygen at a very low level in the placenta and release a large amount in foetal tissue even with a small reduction in oxygen tension. It is this capability for a large volume oxygen release that would be lacking when HbF is suddenly proportionately replaced with HbA in the sick neonate. Secondly stored blood with depleted 2, 3 DPG, further reduce ability for HbA to release oxygen, due to a left shift of the oxygen-haemoglobin dissociation curve. This sudden change of oxygen dissociation curve mechanics could have led to a rapid decline in amount of oxygen available at tissue level, in particular at the pulmonary circulation leading to pulmonary vasoconstriction and a rise in pulmonary vascular resistance (PVR) despite HbA increasing the P_{50}. This could have triggered the reversed shunt by altering pressure gradients at shunt level.

Management in neonates with sudden intraoperative right-to-left shunts

Intraoperative sudden left-to-right shunts respond commonly to volume and increase in systemic vascular resistance [16] [17]. Our treatment strategies were similar, both physiological and pharmacological. The physiological methods included hyperventilation, high oxygen therapy and volume expansion. This was to promote pulmonary vasodilatation and systemic vasoconstriction. On the other hand, the use of systemic vascular constrictor pharmacologically was useful to rapidly reduce a reversed shunt to a normal left to right shunt. These manoeuvres reduce right to left shunting by altering the pressure gradients between left and right heart chambers. Dopamine was useful in one infant. In situations of transitional circulation, dopamine infusion has been shown to be successful therapy. It mitigates myocardial dysfunction associated with transitional circulation [18]. It may have also increased SVR although was not reported [18].

In case 1, the situation was remedied by stopping the blood transfusion (the probable trigger), improving oxygenation, and the increase in the inotrope/vasopressor dose. These manoeuvres were expected to reduce the pulmonary circulation pressure and also increase systemic vascular resistance and reduce the reversed pressure gradients at the foramen ovale or patent ductus. This promoted the re-establishment of pulmonary blood flow leading to recovery of $ETCO_2$ and improved oxygenation (Figure 1).

In our case 2, desaturation responded well to fluids and 100% oxygenation. These manoeuvres would have reduced pulmonary vascular resistance and increased systemic vascular pressure improving the situation by reducing the reversed shunt flow at PFO or PDA.

Conclusions

We have demonstrated how the circulation in neonates can become easily unbalanced during non-cardiac anaesthesia by induction of general anaesthesia or by a blood transfusion, resulting in the lowering of SVR by anaesthetics agents, and a rise in PVR, respectively. The clinical consequences of an acute right-to-left shunt intraoperatively can be devastating. We have shown how this can be diagnosed and managed with relatively simple non-invasive capnography and pulse oximetry monitoring.

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References

1. Hanning CD. "He looks a little blue down this end". Monitoring oxygenation during anaesthesia. British Journal of Anaesthesia 1985;57:359-60.
2. Gaba DM. Anaesthesiology as a model for patient safety in health care. BMJ;2000:320:785-8.
3. Eichhorn JH. Prevention of intraoperative anesthesia accidents and related severe injury through safety monitoring. Anesthesiology 1989; 70:572-7.
4. Linko K, Paloheimo M, Tammisto T. Capnography for detection of accidental oesophageal intubation. Acta Anaesthesiologica Scandinavica 1983;27: 199-202.
5. Verschuren F et al. Volumetric capnography as a bedside monitoring of thrombolysis in major pulmonary embolism. Intensive Care Medicine 2004;30:2129-32.
6. Goonasekara C, Goodman J, Kurup M. Recognising a reversed A-V shunt under anaesthesia in a neonate: the value of combined SpO2 pETCo2 and trends. Pediatric Anesthesia and Critical Care Journal 2014;2:31-33.
7. Nelson NM et al. Pulmonary function in the newborn infant. II. Perfusion--estimation by analysis of the arterial-alveolar carbon dioxide difference. Pediatrics 1962;30:975-89.
8. Drummond WH, Peckham GJ, Fox WW. The clinical profile of the newborn with persistent pulmonary hypertension. Observations in 19 affected neonates. Clinical Pediatrics 1977;16:335-41.
9. Rozycki HJ et al. Mainstream end-tidal carbon dioxide monitoring in the neonatal intensive care unit. Pediatrics 1998;101:648-53.
10. Aliwalas LL et al. Agreement of carbon dioxide levels measured by arterial, transcutaneous and end tidal methods in preterm infants < or = 28 weeks gestation. Journal of Perinatology 2005;25:26-9.
11. Watkins AM, Weindling AM. Monitoring of end tidal CO2 in neonatal intensive care. Archives of Disease in Childhood 1987;62:837-9.
12. McEvedy, B.A., et al., End-tidal carbon dioxide measurements in critically ill neonates: a comparison of side-stream and mainstream capnometers. Canadian Journal of Anaesthesia 1990;37:322-6.
13. Goonasekera CD et al. Arterial and end-tidal carbon dioxide difference in pediatric intensive care. Indian Journal of Critical Care Medicine 2014;18:711-5.
14. De Halleux V et al. The effect of blood transfusion on the hemoglobin oxygen dissociation curve of very early preterm infants during the first week of life. Seminars in Perinatology 2002; 26:411-5.
15. Bauer C et al. Factors governing the oxygen affinity of human adult and foetal blood. Respiration Physiology 1969; 7:271-7.
16. Mohindra, R, Beebe DS, Belani KG. Anaesthetic management of patients with congenital heart disease presenting for non-cardiac surgery. Annals of Cardiac Anaesthesia 2002; 5:15-24.
17. Menghraj SJ. Anaesthetic considerations in children with congenital heart disease undergoing non-cardiac surgery. Indian Journal of Anaesthesia 2012; 56:491-5.
18. Fiddler GI et al. Dopamine infusion for the treatment of myocardial dysfunction associated with a persistent transitional circulation. Archives of Disease in Childhood 1980; 55:194-8.