

# Unexpected Increase in Arterial to End-Tidal CO<sub>2</sub> Gradient in a Child Undergoing Embolization of MAPCAs

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## Abstract

A 17-month-old infant with multiple aorto-pulmonary collateral arteries (MAPCAs) and pulmonary hypertension presented for diagnostic catheterization. On the day of the procedure, the infant was asymptomatic with oxygen saturation in the 90's on 1.0 L/min O<sub>2</sub> nasal cannula. His parents denied any recent illness. During the procedure, one coil was inadvertently embolized into the right lung resulting in markedly increased pulmonary artery pressures. The Pa-etCO<sub>2</sub> gradient increased to 25 mmHg from a baseline of 2 mmHg. Therapy was initiated to reduce the PaCO<sub>2</sub>. The patient could not be weaned from mechanical ventilation due to elevated PA pressures.

## Keywords

Multiple Aorto-Pulmonary Collateral Arteries (MAPCAs), Pa-etCO<sub>2</sub> Gradient, Ventilation-Perfusion (V/Q) Relationship

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## 1. Introduction

The difference [gradient] between the partial pressure of carbon dioxide in arterial blood (PaCO<sub>2</sub>) from that measured at the end of expiration (etCO<sub>2</sub>) is a result of the relationship between ventilation (V) and perfusion (Q) of the lung. There is normally a gradient of 2 - 5 mm Hg [1]. When lung perfusion is impaired, the V/Q will be mismatched and cause the gradient to increase. The PaCO<sub>2</sub> therefore, will be much higher than expected based on the etCO<sub>2</sub>. The consequence of this is a potentially unrecognized hypercarbia and respiratory acidosis. Written consent has been obtained to submit this case for publication.

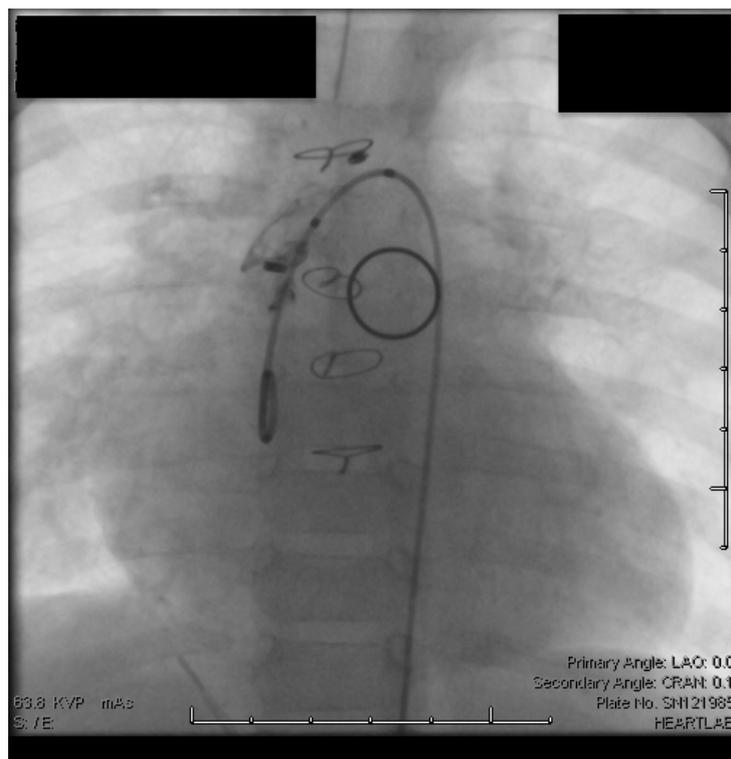
## 2. Case Presentation

A 17-month-old about 9.4 kg male presented for diagnostic cardiac catheterization. The infant was born with Tetralogy of Fallot, moderate bilateral pulmonary artery hypoplasia, and multiple aorto-pulmonary collateral arteries (MAPCAs). A Blalock-Taussig shunt, ligation of single aortic to pulmonary artery (PA) collateral, surgical placement of RA to focalized PA conduit, and creation of fenestrated VSD were performed at six months of age.

On this admission, he had significant PA hypertension and RV systolic dysfunction. His TTE demonstrated mild RV dilation with mildly depressed systolic function. Estimated RV systolic pressure was 75 mmHg. Pre-operatively, his medication includes aspirin, digoxin, enalapril, furosemide, spironolactone and sildenafil. Cardiac catheterization was initially planned to only evaluate the response of PA pressures to sildenafil, which was begun 2 weeks earlier.

On the day of the procedure, the infant was asymptomatic. His O<sub>2</sub> saturation was in the 90's on a baseline of 1.0 L/min of O<sub>2</sub> via a nasal cannula. His parents denied any recent fever or illness. Physical examination revealed no respiratory distress, wheezing, or cyanosis.

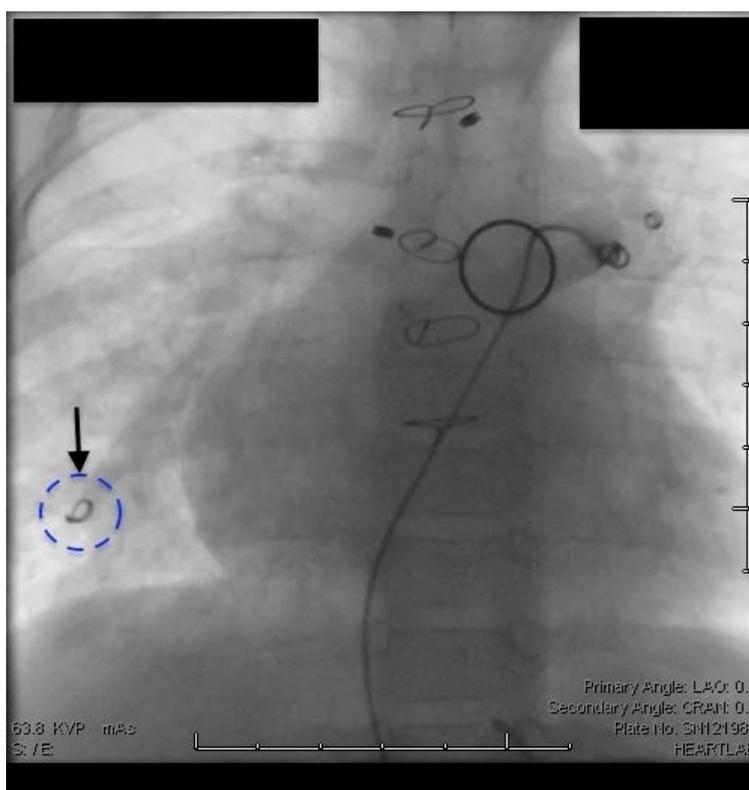
Induction of general anesthesia was achieved with inhalation of sevoflurane in 100% O<sub>2</sub>. A 22 g PIV was established, 25 mcg Fentanyl and 10 mg rocuronium were administered IV, and the airway was easily secured with a 4.5-cuffed ETT. Endotracheal tube position was confirmed with equal bilateral breath sounds, positive etCO<sub>2</sub> and fluoroscopy by the cardiologist (**Figure 1**). General anesthesia was maintained with sevoflurane (1MAC). PCV mode was used with a PIP of



**Figure 1.** Cardiac image before coil embolization.

15 cm H<sub>2</sub>O, RR of 20/min and F<sub>I</sub>O<sub>2</sub> of 0.21. With these ventilator settings, a V<sub>T</sub> of 100 ml was delivered and the etCO<sub>2</sub> was 33 mm Hg. The initial ABG values were: pH 7.45, PaCO<sub>2</sub> 35 mmHg, PaO<sub>2</sub> 50 mmHg, and HCO<sub>3</sub><sup>-</sup> 24 mEq. The Pa-etCO<sub>2</sub> gradient was 2 mm Hg.

During the procedure, multiple coils were placed in collaterals from the aorta to the left lung. One of the coils was inadvertently embolized into the right lung (**Figure 2**). Co-incident with the coil embolizing to the right lung, PA pressures markedly increased. An ABG at that time demonstrated a pH 7.29, PaCO<sub>2</sub> 62 mmHg, PaO<sub>2</sub> 423 mmHg, and HCO<sub>3</sub> 30 mEq. The etCO<sub>2</sub> at that time was 37 mmHg with a Pa-etCO<sub>2</sub> gradient of 25 mmHg. To reduce the PaCO<sub>2</sub>, hyperventilation was initiated with PIP of 22 cm H<sub>2</sub>O and RR of 24/min. A PaCO<sub>2</sub> within the normal range was only attained when the etCO<sub>2</sub> was reduced to 22 mmHg (**Table 1**).



**Figure 2.** Cardiac image after coil embolization. The circle depicts the coil in the right lung.

**Table 1.** PaCO<sub>2</sub>, end tidal CO<sub>2</sub> and Pa-etCO<sub>2</sub> gradient in mmHg in relation to the coil embolization. Note the gradient difference 30 minutes before the embolization and immediately after.

	Induction Base line (90 min before the events)	30 minutes before the event	Immediately after coil embolization	After hyperventilation
PaCO <sub>2</sub> (mmHg)	35	42	62	36
End tidal CO <sub>2</sub> (mmHg)	33	37	37	22
Pa-etCO <sub>2</sub> gradient (mmHg)	2	5	25	14

At the end of the procedure, the patient could not be weaned from mechanical ventilation. Despite the use of nitric oxide, PA pressures and the PaCO<sub>2</sub>-etCO<sub>2</sub> gradient remained elevated. The decision was made to keep the ETT in place, and the infant was transported to the PICU for further management. Transthoracic echocardiography performed immediately after the procedure demonstrated moderate RV dilation with moderately depressed systolic function and paradoxical inter-ventricular septal motion. RV systolic pressure was supra-systemic with estimates of 123 mmHg. Further management of right heart failure and pulmonary hypertension was initiated with a combination of diuretics, oxygen, and a milrinone infusion.

### 3. Discussion

Based on the complexity of the patient's medical condition and the nature of the procedure, this case report highlights the significance of a sudden intraoperative increase in the Pa-etCO<sub>2</sub> gradient. This should alert the anesthesiologist to the possibility of a pulmonary blood flow incident occurred. According to Nunn and Hill [2], the Pa-etCO<sub>2</sub> is  $4.6 \pm 2.5$  mm Hg in healthy, anesthetized patients. Because end-expiratory gas is a mixture from both well and poorly perfused alveoli, end-tidal PCO<sub>2</sub> is usually less than arterial PCO<sub>2</sub>. The Pa-etCO<sub>2</sub> gradient can be used as an index of alveolar dead space in most cases. In this case, embolization of a coil to the right lung acutely decreased the perfusion (Q) in the ventilation-perfusion (V/Q) relationship, while the ventilation (V) remained unchanged. This results in increased dead space ventilation. Using the etCO<sub>2</sub> alone to predict the PaCO<sub>2</sub> in such cases can lead to underestimation of the PaCO<sub>2</sub>.

Baraka *et al.* 1994 reports that in ASA class 1 or 2 patients during laparoscopy, no significant change in the PaCO<sub>2</sub>-etCO<sub>2</sub> gradient after carbon dioxide insufflation. They conclude that end-tidal capnography can be used as noninvasive techniques for monitoring arterial carbon dioxide elimination during laparoscopic surgery in healthy patients [3]. The end-tidal PCO<sub>2</sub> increases hand-in-hand with the arterial PCO<sub>2</sub> during laparoscopic surgery [4]. Thus, end-tidal PCO<sub>2</sub> accurately reflects the changes of the arterial PCO<sub>2</sub> during laparoscopy. Patients with severe cardiopulmonary disease can develop significant hypercarbia associated with an increased PaCO<sub>2</sub>-etCO<sub>2</sub> gradient [5]. In these patients, the end-tidal PCO<sub>2</sub> is not a satisfactory index of PaCO<sub>2</sub> measurement [6].

The reliability of using end-tidal PCO<sub>2</sub> as an indicator for arterial PCO<sub>2</sub> is highly controversial. Some studies have found that the gradient is highly variable and PetCO<sub>2</sub> cannot provide a reliable reflection of PaCO<sub>2</sub>, as in patients during craniotomies [7], and in multisystem trauma patients [8].

### 4. Conclusion

Using the etCO<sub>2</sub> alone to predict the PaCO<sub>2</sub> during an embolization procedure is not ideal. This case emphasizes the importance of intraoperative communication between the anesthesiologist and the cardiologist.

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