

A Persistently High End-Tidal Carbon Dioxide Value: Can This Be Spurious?

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Abstract

End-tidal carbon dioxide (EtCO₂) monitoring has now become the standard of care not only during anesthesia but also in intensive care units for patients on mechanical ventilation, emergency department, and pre-hospital settings to confirm and monitor the correct placement of endotracheal tube. It is a non-invasive and continuous method of measuring exhaled carbon dioxide (CO₂). Continuous waveform capnography measures EtCO₂ and monitors ventilation. EtCO₂ often correlates with partial pressure of carbon dioxide in arterial blood (PaCO₂) and is a reliable indicator of PaCO₂. A rise in EtCO₂ often implies increased production of CO₂ or decreased excretion (rebreathing, decrease ventilation) of CO₂. We report an unusual case where the monitor malfunction per se lead to spuriously increased EtCO₂ values without any clinical cause and did not correlate with PaCO₂, thereby re-emphasizing that various monitors must always be interpreted in correlation with clinical observation.

Keywords

- ▶ end-tidal carbon dioxide
- ▶ monitor malfunction
- ▶ anesthesia
- ▶ capnography

Introduction

End-tidal carbon dioxide (EtCO₂) monitoring is considered standard of care during anesthesia, in mechanically ventilated patients in intensive care unit and in pre-hospital settings to confirm correct placement of endotracheal tube (ETT).¹ Apart from the advantages of being a continuous and non-invasive monitor of exhaled carbon dioxide (CO₂), it is also a reliable indicator of partial pressure of CO₂ (PaCO₂) in arterial blood and is often used as its surrogate.² Changes in EtCO₂ and PaCO₂ normally parallel each other and the difference between the two is approximately 3.6 to 4.6 mm Hg in healthy awake patients.³ We report a case where EtCO₂ values were disproportionately high and did not correlate with PaCO₂.

Case Presentation

A 62-year-old female patient weighing 59 kg was diagnosed with L5–S1 listhesis and was planned for surgery. She was a known hypertensive, controlled on amlodipine (5 mg daily) for last 7 years. On pre-anesthetic check-up, she

had bilateral wheeze and was optimized with corticosteroid, and bronchodilator nebulization for 1 week before surgery. Her preoperative investigations including pulmonary function tests were within normal limits. In the operating room, peripheral intravenous access was established, and routine standard monitoring was started (electrocardiography, non-invasive blood pressure, pulse-oximeter, and sidestream capnography). After preoxygenation with 100% oxygen for 3 minutes, general anesthesia was induced with intravenous fentanyl 120 µg and propofol 140 mg. Rocuronium 50 mg was given to facilitate tracheal intubation, and the patient was ventilated with bag and mask for 3 minutes. While manually ventilating the patient, the multichannel monitor displayed a high EtCO₂ value (70–80 mm Hg, ▶**Fig. 1A**). Suspecting this to be due to inadequate mask ventilation, trachea was intubated and ETT position was confirmed by capnography and auscultation. Anesthesia was maintained with sevoflurane in O₂:N₂O (40:60) mixture. The EtCO₂ value remained high (90–100 mm Hg, ▶**Fig. 1B**) and kept rising constantly. Despite all measures to decrease EtCO₂ including increasing minute ventilation (up to 13 L/min), reconfirming ETT position, increasing fresh gas flow, use of fresh CO₂ absorbent,



Fig. 1 End-tidal carbon dioxide (EtCO₂) values displayed over the monitors. (A) Initial high EtCO₂ values; (B, C) gradually increasing EtCO₂ readings despite all efforts to correct it; (D) EtCO₂ value after changing the monitor.

change of sampling tube and manual ventilation, EtCO₂ value kept increasing gradually and reached up to 113 mm Hg (►Fig. 1C). An arterial blood gas (ABG) analysis was performed which showed a PaCO₂ value of 28 mm Hg with all other parameters within normal range, thus, indicating a probability of monitor malfunction and spuriously raised EtCO₂. The monitor was then replaced with a new monitor, which showed an EtCO₂ value of 26 mm Hg with same ventilator settings (►Fig. 1D).

Discussion

End-tidal carbon dioxide is the real-time monitoring of exhaled CO₂ over time that can be measured using either mainstream or sidestream capnometers with few inherent advantages and disadvantages of each type. EtCO₂ is often used as a surrogate for PaCO₂ in mechanically ventilated patients. Changes in EtCO₂ normally parallel changes in PaCO₂. However, there are several clinical situations where EtCO₂ values can be raised or decreased.

Sensor windows of mainstream capnometers or sampling line of sidestream capnometers are both prone to clogging by secretions, aerosols, or water droplets. Erroneous EtCO₂ readings, such as sudden drop in EtCO₂ due to obstruction of sampling tube leading to incorrect interpretation of venous air embolism or large pulmonary embolus, have been reported in literature.⁴ Simple maneuvers, such as purging the sampling line with a syringe to dislodge water droplets in the tubing, replacing the sensor, or sampling tube can

correct any artefactual changes in EtCO₂ and avoid further unnecessary interventions. At low sampling rates with long sampling catheters, the expired CO₂ gas is subjected to axial mixing and dispersion during its transit through the catheter, leading to underestimation of measured CO₂ value.⁵ Even a broken Male Luer connector of the sampling tube⁶ and placement of a jet nebulizer between the mainstream capnometer and ETT⁷ have been reported to cause reduced EtCO₂ values.

On the other hand, causes of high EtCO₂ can be broadly classified as those due to decreased excretion and due to increased production and iatrogenic causes.⁸ Decreased excretion can be due to increased inspired CO₂, decreased ventilation, or increased dead space. Problems with anesthesia machine, such as exhausted soda lime, faulty inspiratory or expiratory valve, and channeling through soda lime can increase inspired CO₂ causing high EtCO₂. Inadequate ventilation can occur due to leaks in the circuit, bronchospasm, endobronchial intubation, and partial airway obstruction due to kink or mucous plug. Increased production of CO₂ due to fever, shivering, convulsions, hyperthyroidism, burns, malignant hyperthermia (MH), and rarely, pheochromocytoma and transfusion reaction can cause high EtCO₂. Iatrogenic causes include CO₂ insufflation during laparoscopy, tourniquet release, and infusion of sodium bicarbonate. Further, false or spurious increase in EtCO₂ can occur due to monitor malfunction or calibration error. In our patient, inspired CO₂ values were within normal limits (fractional inspired [FiCO₂]: 1–2), CO₂ absorbent was fresh, and there were no leaks in the circuit. Expiratory unidirectional valve malfunction such as

valve breakage⁹ or misplaced metal retainer¹⁰ can result in raised EtCO₂ values due to rebreathing of exhaled air and manifest as increase in both inspired and expired segments of EtCO₂ waveform. However, in our case inspired CO₂ values were always within normal limits. Channeling is difficult to detect but is treated by replacement of soda lime. Hypoventilation was an unlikely cause in our patient as expired tidal volume and blood oxygen saturation were normal. On auscultation, bilateral air entry was equal ruling out endobronchial intubation. Airway pressure was normal, chest was clear, suctioning of the ETT was clear, and there was no delayed upstroke in EtCO₂ waveform; so, bronchospasm and partial ETT obstruction seemed unlikely. Our patient's temperature was normal, ruling out fever as a possible cause. It is not possible to detect clinical seizures or shivering in a paralyzed and mechanically ventilated patient. Burns are self-evident; transfusion reaction was not possible in this case since we did not transfuse any blood products. Malignant hyperthermia (MH) was another possibility in our case since we used sevoflurane for maintenance of anesthesia. We did a blood gas analysis to see if PaCO₂ values correlated with EtCO₂, but it was not increased. The only findings in favor of a diagnosis of MH were rapidly rising EtCO₂ values, use of sevoflurane, and tachycardia. There was no respiratory acidosis on blood gas analysis, PaCO₂ was not raised, temperature was normal, and there was no change in urine color; so, MH was unlikely. A wide difference in EtCO₂ and PaCO₂ levels in ABG prompted us to think in terms of spurious increase in EtCO₂. We changed the monitor. The new monitor showed EtCO₂ value of 26 mm Hg, thus, confirming that the cause of high EtCO₂ in this case was a fault with the monitor.

In healthy subjects, EtCO₂ and PaCO₂ generally correlate well. PaCO₂ values are generally 2 to 5 mm Hg above EtCO₂ values. However, in conditions of increased physiological dead space, the correlation between end-tidal and arterial CO₂ is poor. With increased physiological dead space, there is mixing of gas from poorly perfused areas of lung with that from well-perfused areas, resulting in a lower EtCO₂ relative to PaCO₂.¹¹ In our case, though the gradient was widened, the change was in the reverse direction, EtCO₂ being much higher relative to PaCO₂. An increase in physiological dead space could not have explained this discrepancy. A fault with the monitor or a calibration error was the only possible cause.

The manufacturing company of the monitor was contacted to detect and rectify the fault in the monitor. The service engineers tested and recalibrated the equipment. However, discussion with service engineer did not yield anything specific. Subsequent use of the monitor after recalibration displayed correct EtCO₂ values, which correlated with PaCO₂ values.

Conclusion

Our case highlights that monitor malfunction per se may sometimes result in fallacious EtCO₂ readings and it should always be seen as a possibility if no other cause is found before undertaking extensive interventions. It re-emphasizes the fact that various monitors used under anesthesia must always be interpreted in correlation with clinical observation.

Conflict of Interest

None declared.

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