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Arterial-to-End-Tidal CO₂ Gradient Direction as a Differential Diagnostic Clue During Sudden Hypercapnia in Thoracoscopic Surgery with Capnothorax: A Report of 2 Cases

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Case series

Patients: Male, 62-year-old • Male, 77-year-old

Final Diagnosis: A partial injury to the left main bronchus • severe systemic CO₂ loading

Symptoms: Abrupt end-tidal CO₂ surges during thoracoscopic surgery with capnothorax

Clinical Procedure: —

Specialty: Anesthesiology

Objective: Diagnostic / therapeutic accidents

Background: During minimally invasive thoracic surgery with intrathoracic carbon dioxide (CO₂) insufflation, an abrupt rise in end-tidal CO₂ (EtCO₂) alone cannot distinguish true hypercapnia from artifactual or exogenous CO₂ contamination. We report 2 contrasting cases in which the direction of the arterial-to-end-tidal CO₂ gradient, Δ(PaCO₂-EtCO₂), guided differential diagnosis.

Case Reports: Case 1: A 62-year-old man undergoing robot-assisted right lower lobectomy developed an abrupt EtCO₂ rise from 40 to 130 mm Hg immediately after CO₂ insufflation, with severe hypotension and bradycardia. Arterial blood gas (ABG) analysis showed pH 6.79, PaCO₂ 228.0 mm Hg, and Δ(PaCO₂-EtCO₂) +98.0 mm Hg, suggesting a possible contribution of low cardiac output to the widened gradient. Insufflation was stopped, 2-lung ventilation resumed, and surgery completed without re-insufflation. Case 2: A 77-year-old man undergoing thoracoscopic subtotal esophagectomy developed an EtCO₂ rise from 54 to 99 mm Hg while hemodynamically stable. After excluding circuit and sampling-line artifacts, ABG analysis showed pH 7.21, PaCO₂ 72.5 mm Hg, and Δ(PaCO₂-EtCO₂) -26.5 mm Hg, suggesting exogenous CO₂ contamination of expiratory gas rather than systemic retention alone. Surgical exploration confirmed a partial injury to the left main bronchus.

Conclusions: During abrupt EtCO₂ surges under 1-lung ventilation with capnothorax, the direction of Δ(PaCO₂-EtCO₂) on prompt ABG analysis may help prioritize the differential diagnosis between systemic retention and exogenous contamination, provided hemodynamics and circuit integrity are concurrently assessed. A markedly positive gradient during shock may indicate systemic CO₂ loading with reduced pulmonary perfusion, whereas a negative gradient after artifact exclusion suggests pleuro-airway communication or peri-device leakage. This hypothesis-generating approach remains unvalidated as a standalone diagnostic tool.

Keywords: One-Lung Ventilation • Hypercapnia

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Introduction

In minimally invasive thoroscopic surgeries, 1-lung ventilation (OLV) is routinely employed to provide an adequate operative field, and the concurrent use of intrathoracic carbon dioxide (CO₂) insufflation has become increasingly common, particularly in robot-assisted procedures [1-3]. These maneuvers, however, alter gas exchange, pulmonary mechanics, and hemodynamics [3,4].

During capnothorax, hypercapnia may reflect true systemic CO₂ loading, and in selected cases, intravascular CO₂ entrainment [4-6] or exogenous contamination of sampled gas through thoraco-airway communication [7]. Accordingly, the EtCO₂ value alone may be etiologically ambiguous, and arterial blood gas (ABG) correlation is essential [8,9].

The arterial-to-end-tidal CO₂ gradient, defined here as $\Delta(\text{PaCO}_2 - \text{EtCO}_2) = \text{PaCO}_2 - \text{EtCO}_2$, is usually positive [10,11]. A widened positive gradient indicates that dead space or ventilation-perfusion mismatch limits the elimination of exhaled CO₂ [10,11]. Conversely, capnographic values may be spuriously elevated when exogenous CO₂ contaminates sampled gas or when measurement artifacts affect inspired or expired CO₂ analysis; therefore, an abrupt EtCO₂ surge during capnothorax should not be interpreted from the EtCO₂ value alone [4,8,12]. Previous studies have separately described systemic CO₂ accumulation during capnothorax [4] and capnographic distortion from exogenous CO₂ contamination or measurement artifacts [5,12,13]. The underlying mechanisms and the negative-gradient phenomenon are therefore not themselves novel. The incremental contribution of this cautionary paired case report is a side-by-side contextual comparison of opposite-direction $\Delta(\text{PaCO}_2 - \text{EtCO}_2)$ patterns within the same operative setting—thoroscopic surgery with capnothorax during OLV. In addition, the report emphasizes that gradient direction should be

interpreted together with simultaneous hemodynamics, capnogram morphology, and circuit-integrity assessment rather than as a standalone diagnostic rule.

Case Reports

Written informed consent was obtained from both patients for the publication of this case report and the accompanying images.

Case 1

A 62-year-old man (height, 178 cm; weight, 51 kg) had a history of pleuritis and esophageal achalasia. During treatment for pleuritis at another hospital, computed tomography revealed esophageal wall thickening. Further evaluation led to a diagnosis of esophageal cancer with invasion of the right lung, and the patient was referred to our hospital for surgical treatment. Robot-assisted right lower lobectomy, subtotal esophagectomy, and esophagostomy in the prone position were planned under combined general and epidural anesthesia.

After placing an epidural catheter at the T8/9 interspace, anesthesia was induced with propofol 80 mg, remifentanyl 0.2 µg/kg/min, and rocuronium 50 mg. Endotracheal intubation was performed with an 8.0-mm internal diameter nerve integrity monitoring tube (NIM TriVantage EMG tube; Medtronic, Minneapolis, MN, USA), and a bronchial blocker was positioned in the right bronchus. Anesthesia was maintained with sevoflurane (1.2%-2.0%) and remifentanyl (0.1-0.25 µg/kg/min). Before commencing surgery, OLV was initiated, and ABG values were acceptable (Table 1). Thoroscopic inspection revealed extensive adhesions between the right lung and parietal pleura; thus, adhesiolysis was completed before intrathoracic CO₂ insufflation.

Table 1. Serial arterial blood gas analyses in Case 1.

Time point	FiO ₂	pH	PaCO ₂ (mm Hg)	PaO ₂ (mm Hg)	HCO ₃ ⁻ (mmol/L)	Base excess (mmol/L)	SaO ₂ (%)	Lactate (mmol/L)
After induction of anesthesia (A1)	0.5	7.36	48.1	280	26.6	1.4	99.0	0.4
During OLV (control, A2)	1.0	7.38	44.1	353	26.1	1.1	99.7	0.5
At the time of EtCO ₂ elevation (A3)	1.0	6.79	228.0	182	32.4	-9.5	97.3	0.5
After correction of elevated CO ₂ (20 min, A4)	1.0	7.22	71.1	393	28.0	-0.4	99.4	0.6
At the end of surgery	0.6	7.41	35.0	280	22.0	-1.7	99.6	6.2

OLV, 1-lung ventilation; EtCO₂, end-tidal carbon dioxide; FiO₂, fraction of inspired oxygen; PaCO₂, arterial partial pressure of carbon dioxide; PaO₂, arterial partial pressure of oxygen; HCO₃⁻, bicarbonate; SaO₂, arterial oxygen saturation. A1–A4 in this table correspond to the time points labeled A1–A4 in Figure 1.

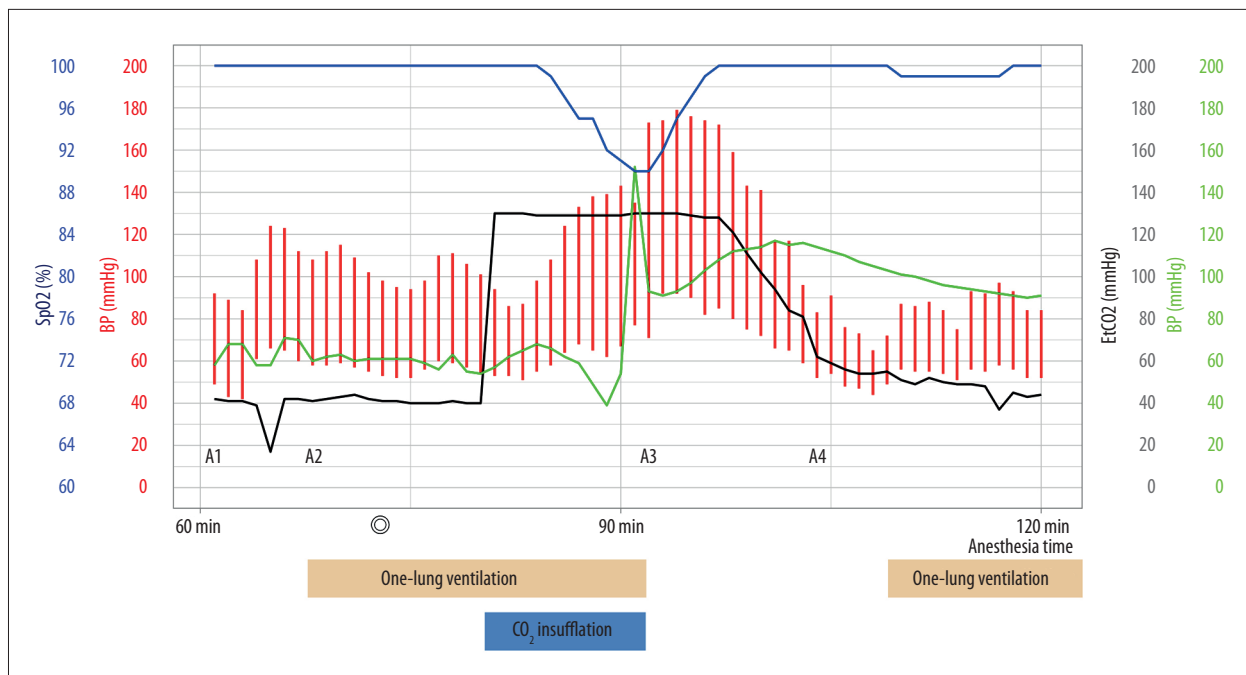


Figure 1. Intraoperative anesthesia record of Case 1. Anesthesia was maintained with sevoflurane and remifentanyl. Immediately after the start of intrathoracic CO₂ insufflation, EtCO₂ increased abruptly from 40 to 130 mm Hg, and arterial pressure decreased from 110/59 to 82/49 mm Hg. Hypercapnia improved after cessation of CO₂ insufflation, restoration of 2-lung ventilation, and hemodynamic support. The operation was completed without further insufflation. © Indicates the start of surgery. SpO₂, pulse oximeter oxygen saturation; BP, blood pressure; EtCO₂, end-tidal carbon dioxide; HR, heart rate.

Immediately after intrathoracic CO₂ insufflation was started at 8 mm Hg, EtCO₂ rose abruptly from 40 to 130 mm Hg within 1 min, and invasive arterial pressure fell from 110/59 to 82/49 mm Hg (Figure 1). A phenylephrine bolus (0.2 mg) followed by continuous infusion (1.0 mg/h) was administered, CO₂ insufflation was stopped immediately, and OLV was converted to 2-lung ventilation (TLV) with 100% oxygen. ABG analysis performed at that time showed severe respiratory acidosis and hypercapnia (pH 6.79, PaCO₂ 228.0 mm Hg), with a markedly widened positive $\Delta(\text{PaCO}_2\text{-EtCO}_2)$ of +98.0 mm Hg (Table 1).

Clinical reasoning at this stage focused on whether the EtCO₂ surge represented true systemic hypercapnia/intravascular CO₂ loading, or a spurious capnography value. The markedly positive $\Delta(\text{PaCO}_2\text{-EtCO}_2)$, severe acidosis, and contemporaneous hypotension favored true systemic CO₂ loading, with impaired pulmonary perfusion or low cardiac output contributing to the widened gradient. Isolated exogenous contamination of the sampled gas was considered less likely. Because brisk vascular CO₂ absorption after adhesiolysis and/or CO₂ entrainment could not be excluded, the team maintained cessation of insufflation, continued TLV with 100% oxygen, supported the circulation, and avoided re-insufflation.

During hypercapnia correction, the heart rate decreased from 59 to 39 beats/min with a transient decrease in peripheral

oxygen saturation, and atropine 0.5 mg was administered intravenously. After correcting hypercapnia (Table 1, Figure 1), surgery was completed without further CO₂ insufflation, and no additional major intraoperative event occurred.

Case 2

A 77-year-old man (height, 165 cm; weight, 54 kg) had a history of hypertension and cerebral infarction without residual deficit. He initially presented with progressive dysphagia after several months of increasing belching and was diagnosed with esophageal cancer after a detailed evaluation. Thoracoscopic subtotal esophagectomy under combined general and epidural anesthesia was scheduled.

After placement of an epidural catheter at the T9/10 interspace, anesthesia was induced with propofol 70 mg, fentanyl 200 μg , and rocuronium 40 mg. Anesthesia was maintained with sevoflurane (1.2%-1.5%) and remifentanyl (0.1-0.2 $\mu\text{g/kg/min}$). Endotracheal intubation was performed with an 8.0-mm internal diameter NIM TriVantage EMG tube, and a bronchial blocker was positioned in the right main bronchus. OLV was established before the start of surgery, and baseline ABG values were acceptable (Table 2).

Table 2. Serial arterial blood gas analyses in Case 2.

Time point	FiO ₂	pH	PaCO ₂ (mm Hg)	PaO ₂ (mm Hg)	HCO ₃ ⁻ (mmol/L)	Base excess (mmol/L)	SaO ₂ (%)	Lactate (mmol/L)
After induction of anesthesia	1.0	7.44	38.7	511	26.4	2.2	100	0.8
During OLV (control, A5)	1.0	7.33	50.2	138	25.7	0.1	99.0	0.6
At the time of EtCO ₂ elevation (A6)	1.0	7.21	72.5	137	27.6	-0.8	97.6	0.4
End of surgery	0.8	7.41	35.0	229	21.7	-2.1	99.9	3.2

OLV, 1-lung ventilation; EtCO₂, end-tidal carbon dioxide; FiO₂, fraction of inspired oxygen; PaCO₂, arterial partial pressure of carbon dioxide; PaO₂, arterial partial pressure of oxygen; HCO₃⁻, bicarbonate; SaO₂, arterial oxygen saturation. A5 and A6 in this table correspond to the time points labeled A5 and A6 in Figure 2.

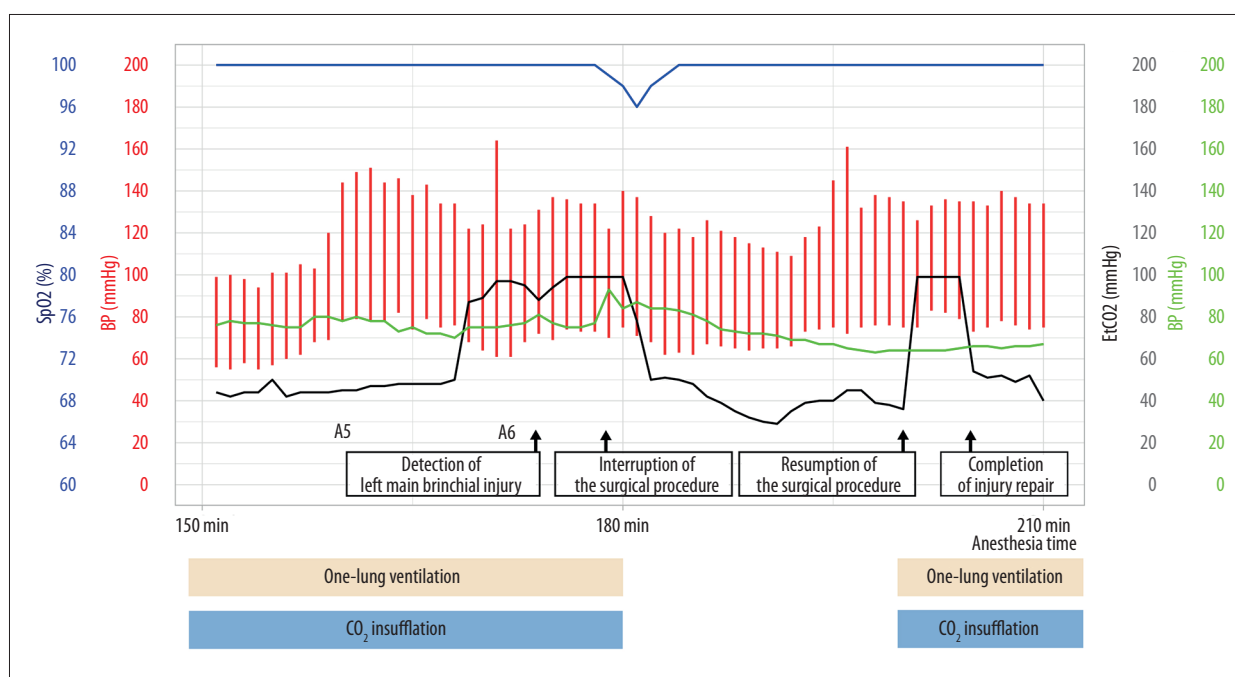


Figure 2. Intraoperative anesthesia record of Case 2. Anesthesia was maintained with sevoflurane and remifentanyl. EtCO₂ increased abruptly from 54 to 99 mm Hg during thoracoscopic manipulation. As the ABG-derived gradient was negative, exogenous CO₂ contamination of sampled gas was suspected and a left main bronchial injury was subsequently identified and repaired. SpO₂, pulse oximeter oxygen saturation; BP, blood pressure; EtCO₂, end-tidal carbon dioxide; HR, heart rate; ABG, arterial blood gas.

Ninety minutes after the initiation of intrathoracic CO₂ insufflation, EtCO₂ increased abruptly from 54 to 99 mm Hg during thoracoscopic manipulation. CO₂ insufflation was stopped immediately, and ventilation was converted from OLV to TLV with 100% oxygen, and the patient remained hemodynamically stable (Figure 2).

The anesthesia team first ruled out common artifactual causes, including sampling-line moisture or obstruction and circuit disconnection, before proceeding to bronchoscopy. Sampling-line leakage, loose circuit connections, inappropriate ventilator

settings, and obvious malposition or cuff leakage of the lung-isolation device were also assessed; no obvious circuit or sampling-line malfunction was identified.

After excluding these common circuit and sampling-line artifacts, ABG analysis obtained at that time exhibited pH 7.21 and PaCO₂ 72.5 mm Hg, yielding a negative $\Delta(\text{PaCO}_2 - \text{EtCO}_2)$ of -26.5 mm Hg (Table 2). As this pattern was inconsistent with isolated systemic CO₂ retention, exogenous CO₂ contamination of the sampled expiratory gas through a thoraco-airway communication or peri-device leakage was suspected. Bronchoscopy

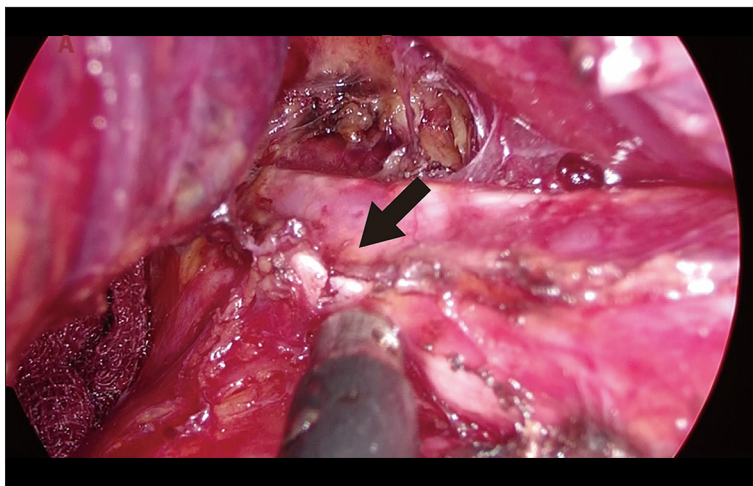


Figure 3. Intraoperative findings demonstrating injury to the left main bronchus. A sudden increase in EtCO₂ prompted concern for thoraco-airway communication, and injury to the left main bronchus was confirmed intraoperatively (black arrow).

was then performed to evaluate the airway, followed by surgical exploration of the operative field, which revealed a partial injury to the left main bronchus (**Figure 3**). After repair of the bronchial injury, the operation proceeded uneventfully.

Discussion

The incremental contribution of these paired cases is the side-by-side demonstration that the direction of $\Delta(\text{PaCO}_2\text{-EtCO}_2)$ may help generate hypotheses about true systemic CO₂ loading versus exogenous contamination of sampled gas when EtCO₂ rises abruptly during thoracoscopic surgery with capnothorax. The interpretive approach is meant to generate hypotheses and prompt concurrent hemodynamic, airway, circuit, capnogram, and surgical assessment; it should not replace comprehensive assessment and should not be viewed as a validated diagnostic tool.

In Case 1, the combination of profound acidosis, PaCO₂ 228.0 mm Hg, a markedly widened positive $\Delta(\text{PaCO}_2\text{-EtCO}_2)$ (+98.0 mm Hg), and immediate hemodynamic instability strongly indicated true extreme systemic CO₂ loading rather than an isolated capnography artifact.

Several mechanisms may have contributed to these conditions. First, the temporal relationship with insufflation immediately after extensive adhesiolysis suggests brisk vascular uptake from a large raw pleural surface and exposed venous channels. Second, a component of intravascular CO₂ entrapment or microembolism cannot be excluded. Clinically significant CO₂ embolism may occur either from direct intravascular insufflation or from gas entering injured vessels, and transesophageal echocardiography is a well-established diagnostic tool for confirming this [6]. As transesophageal echocardiography and anterior chest wall Doppler imaging were not performed in this case, it was not possible to definitively distinguish between

pure absorptive load and hemodynamic collapse caused by embolism. Therefore, regarding Case 1, we could only conclude that the event represented severe systemic CO₂ overload, possibly accompanied by hemodynamic collapse.

Low cardiac output and its effect on the PaCO₂-EtCO₂ gradient require separate emphasis. OLV and capnothorax can increase physiologic dead space and worsen ventilation-perfusion mismatch [4,8]. Moreover, reduced pulmonary blood flow during hypotension can further lower EtCO₂ relative to PaCO₂ by increasing the contribution of underperfused alveoli. Classic physiologic work demonstrated that cardiopulmonary hemodynamics can alter the arterial-to-alveolar/end-tidal CO₂ relationship [14]. Thus, the markedly positive gradient in Case 1 should not be attributed solely to CO₂ entry into the bloodstream; the concurrent severe hypotension and bradycardia likely contributed to the widened gradient. This reasoning explains why immediate cessation of insufflation, restoration of TLV, ventilatory support, and hemodynamic stabilization were prioritized.

In Case 2, the direction of the gradient was interpreted only after common equipment-related artifacts had been checked. Increased dead space, hypoventilation, or progressive systemic CO₂ absorption would usually maintain PaCO₂ at or above EtCO₂; therefore, these mechanisms alone do not explain the negative $\Delta(\text{PaCO}_2\text{-EtCO}_2)$. Rather, after exclusion of obvious circuit and sampling-line problems, a negative gradient suggested that the measured EtCO₂ had been spuriously augmented by exogenous CO₂ entering the sampled gas. In the thoracoscopic setting, this can occur through pleuro-airway communication caused by lung or bronchial injury or through leakage around a lung-isolation device [7]. Circuit-related re-breathing, sampling-line leakage or contamination, and other equipment-related artifacts remain important competing explanations and must be assessed before attributing the finding to bronchial injury [5,13,15].

Table 3. Focused narrative review of previously reported thoracoscopic/capnothorax cases with abrupt EtCO₂ elevation due to exogenous CO₂ contamination, pleuro-airway communication, or peri-device leakage.

Authors (year)	Procedure/setting	CO ₂ insufflation	Peak EtCO ₂ finding	PaCO ₂ /gradient	Waveform or ancillary clue	Most likely mechanism category/key intervention
Biles et al (1994) [7]	Thoracoscopy (VATS) with OLV	Yes (11-12 mm Hg)	Sudden EtCO ₂ increase (to 70-108 mm Hg)	PaCO ₂ 44 mm Hg; negative gradient	NR	Exogenous pleural CO ₂ contamination detected by the capnometer was suspected; a structural airway injury was not clearly documented
Amin et al (2002) [16]	Thoracoscopy (VATS)	Yes (6 mm Hg)	Sudden EtCO ₂ rise after a small lung tissue tear (from 80 to 94 mm Hg)	PaCO ₂ 42.75 mm Hg; negative gradient	Triggered by lung tear	Pleuro-airway communication after parenchymal injury was suspected; insufflation was stopped and ventilation was adjusted
Karnik et al (2017) [17]	Thoracoscopic excision of a left bronchogenic cyst	Yes (5 mm Hg)	EtCO ₂ increased after insufflation (113-115 mm Hg)	PaCO ₂ 45.6 mm Hg; negative gradient	NR	Likely exogenous CO ₂ contamination through an occult pleuro-airway communication; supportive management
Ravi et al (2020) [18]	Robotic thoracic surgery with OLV	Yes (8 mm Hg)	Marked EtCO ₂ elevation (114 mm Hg) with near-normal PaCO ₂	PaCO ₂ 45 mm Hg; negative gradient	EtCO ₂ decreased after lowering capnothorax pressure and increasing PEEP	CO ₂ leakage from capnothorax into the airway through an open bronchus; lowering capnothorax pressure and increasing PEEP reversed the pressure gradient across the open bronchus and proximal bronchial cuff, reducing CO ₂ entry into expired gas
Boyle et al (2023) [19]	Robotic lobectomy with OLV	Yes (10 mm Hg)	EtCO ₂ rose from the mid-30s to the mid-80s	ABG not obtained	Abnormal waveform morphology normalized after additional bronchial-cuff inflation	Open bronchial lumen with insufflation CO ₂ seeping around the bronchial cuff; cuff inflation resolved the spurious EtCO ₂ rise
Gourav et al (2025) [20]	Thoracoscopic esophagectomy	Yes (8-12 cm H ₂ O)	EtCO ₂ exceeded monitor scale (> 99 mm Hg)	PaCO ₂ 48 mm Hg; negative gradient	Leak around the tube noted during repair	Confirmed bronchial rent with direct thoraco-airway communication; insufflation was stopped and the injury was surgically repaired

EtCO₂, end-tidal carbon dioxide; PaCO₂, arterial partial pressure of carbon dioxide; ABG, arterial blood gas; OLV, 1-lung ventilation; PEEP, positive end-expiratory pressure; VATS, video-assisted thoracic surgery. NR indicates information not reported in the source article. This focused comparative table spans a mechanistic spectrum—from exogenous pleural CO₂ contamination without proven structural airway injury to confirmed bronchial injury and peri-device leakage around a lung-isolation device. Related publications outside the prespecified thoracoscopic/capnothorax focus (for example, mediastinoscopic pneumomediastinum with tracheal injury) informed the Discussion, but were not included in this table.

To contextualize these cases, we performed a focused narrative review to identify published case reports describing abrupt intraoperative EtCO₂ elevation and/or reversal of the PaCO₂-EtCO₂ gradient during thoracoscopic surgery (video-assisted, robot-assisted, or thoracoscopic esophageal procedures) with intrathoracic CO₂ insufflation. PubMed (MEDLINE) was searched from inception to 1 February 2026 using the following keyword combination: ((thoroscop*[tiab] OR thorascopic[tiab] OR VATS[tiab] OR “video-assisted thoracic”[tiab] OR “robot-assisted”[tiab] OR robotic[tiab]) AND (EtCO2[tiab] OR “end-tidal”[tiab] OR capnograph*[tiab]) AND (capnothorax[tiab] OR insufflation[tiab] OR “carbon dioxide”[tiab] OR CO2[tiab])) AND (case reports[pt] OR letter[pt] OR “case report”[ti]) AND english[la] NOT (laparoscop*[tiab] OR pneumoperitoneum[tiab]). Google Scholar was additionally searched to capture case reports and citations not yet indexed in PubMed. The signals of interest were abrupt or sudden EtCO₂ elevation, abnormal capnography, elevated inspired CO₂, or a reversed (negative) PaCO₂-EtCO₂ gradient, and the mechanisms of interest were true systemic CO₂ loading or intravascular entrainment, pleuro-airway communication due to lung, tracheal, or bronchial injury, and peri-device leakage around a lung-isolation device. We included English-language case reports or small case series describing thoracoscopic surgery with intrathoracic CO₂ insufflation and abrupt intraoperative EtCO₂ elevation and/or a negative PaCO₂-EtCO₂ gradient, with sufficient perioperative detail to infer mechanism and management. We excluded non-thoracic procedures; mediastinoscopic or purely pneumomediastinal procedures outside the prespecified thoracoscopic/capnothorax focus; reports without an abrupt intraoperative EtCO₂ change; purely physiologic cohort studies used only for background; narrative reviews without index cases; and duplicate records. Titles and abstracts were screened for relevance to this prespecified focus, and potentially eligible full texts were then reviewed for operative setting, mechanism relevance, and adequacy of perioperative detail.

Nine publications were identified, of which 3 were excluded during screening as duplicate records or publications outside the prespecified thoracoscopic/capnothorax focus. The 5 additional eligible reports are cited here [16-20]; together with the previously cited report by Biles et al [7]. **Table 3** summarizes 6 mechanism-relevant reports (Biles et al, 1994 [7]; Amin et al, 2002 [16]; Karnik et al, 2017 [17]; Ravi et al, 2020 [18]; Boyle et al, 2023 [19]; and Gourav et al, 2025 [20]). These previous reports have similarly noted negative gradients or spuriously high EtCO₂ values in the context of bronchial injury, pleuro-airway communication, or peri-device leakage [7,16-20]. Related reports outside the prespecified thoracoscopic/capnothorax focus (for example, mediastinoscopic pneumomediastinum with tracheal injury) informed the interpretation of capnographic findings but were not included in **Table 3**.

This report has several important limitations. It describes only 2 patients from a single institution, and the observations are hypothesis-generating rather than clinically validated. In Case 1, cardiac output, pulmonary artery pressure, and continuous echocardiographic evidence of intravascular gas were not measured; therefore, the relative contributions of systemic CO₂ loading, impaired pulmonary perfusion, and possible CO₂ embolism cannot be separated definitively. In Case 2, high-resolution capnogram images, inspired CO₂ tracings, and a standardized checklist documenting each equipment check were not archived, even though common circuit and sampling-line problems were assessed clinically. In both cases, ABG samples were obtained promptly after the abrupt EtCO₂ elevations, but were not strictly simultaneous, and rapid changes in ventilation or hemodynamics could have influenced the calculated gradient. Finally, the accompanying literature review was focused and narrative rather than systematic; it is presented to improve transparency regarding the contextual literature and should not be interpreted as a Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA)-compliant systematic review.

Conclusions

In conclusion, these 2 cases demonstrate that during a sudden increase in EtCO₂ levels in thoracoscopic capnothorax, the $\Delta(\text{PaCO}_2 - \text{EtCO}_2)$ gradient should be evaluated alongside simultaneous hemodynamic data and checks of circuit integrity. A significantly positive gradient in the context of shock should raise concern for reduced pulmonary perfusion in addition to CO₂ loading, whereas a negative gradient, after ruling out artifacts, suggests the entry of exogenous CO₂ into sampled gas. Clinicians should consider the gradient as 1 piece of contextual data rather than a standalone diagnostic rule.

Department and Institution Where Work Was Done

Kindai University Hospital, Osaka, Japan.

Ethics Approval And Consent To Participate

Under our institutional policy, which follows the Japanese Ethical Guidelines for Medical and Biological Research Involving Human Subjects, jointly issued by the Ministry of Education, Culture, Sports, Science and Technology, the Ministry of Health, Labor and Welfare, and the Ministry of Economy, Trade and Industry, ethics committee approval was not required for this case report.

Consent for Publication

Written informed consent was obtained from both patients for the publication of this case report and the accompanying images.

Availability of Data and Materials

All data supporting the findings of this case report are included in this article.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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