THE commonly used method for monitoring end-tidal carbon dioxide tension (PetCO2) during two-lung ventilation via a double-lumen endotracheal tube (DLT) involves sampling of mixed expired gases of both lungs. This approach may not always help to localize a pathophysiologic defect in an individual lung. Dual capnography circumvents this limitation by allowing carbon dioxide PetCO2 to be continuously monitored from each lumen of DLT during two-lung ventilation.1,2 The current report describes a situation wherein dual capnography helped us to detect a critical perfusion defect in the putatively normal left lung of a patient scheduled for right pneumonectomy.

**Case Report**

A 71-yr-old, 80kg man with a mesothelioma of the right lung was scheduled to undergo elective right extrapleural pneumonectomy. A ventilation/perfusion (V/Q) scan performed 10 days before surgery showed a normal left lung and a reduced, but matched, perfusion and ventilation defect in the right lung (% of total pulmonary activity: left lung V = 93%; Q = 88%; right lung V = 7%; Q = 12%). Two days before surgery, the patient was admitted to the hospital because of progressive dyspnea. Chest radiography showed complete opacification of the right hemithorax and a leftward shift of the mediastinum. Arterial blood gases with the patient breathing room air showed arterial oxygen tension (Pao2) = 71 mmHg, pH = 7.46, PacO2 = 41 mmHg. Hematocrit concentration was 24% (versus 37% three days earlier). A right-sided hemothorax was suspected and was presumed to have resulted from tumor necrosis and bleeding. The surgeons opted to perform an urgent right-side thoracotomy and pneumonectomy. Oxygen saturation (SpO2) at arrival in the operating room ranged from 93-96% while breathing oxygen 8 l/min via face mask. General anesthesia was induced with etomidate, followed by succinylcholine, and maintained with isoflurane. A single-lumen endotracheal tube was placed to facilitate fiberoptic bronchoscopy (FOB) and then replaced with a Rusch 59 left-sided DLT (Willy Rusch AG, Kernan, Germany).

The patient was placed in the left lateral position and proper DLT position was verified via FOB. The patient was ventilated with a tidal volume of 800 ml and a respiratory rate of 10 breaths/min (fractional inspired oxygen tension [FiO2] = 100%). PetCO2 was 28 mmHg.

As left-lung ventilation was initiated for the operative procedure, SpO2 decreased to 82%. Bilateral ventilation was resumed, and SpO2 increased to 98%. The FOB showed the DLT to be in proper position. With reinitiation of left-lung ventilation, SpO2 again decreased. Five centimeters of water continuous positive airway pressure applied to the upper lung increased SpO2, to approximately 90%. The PetCO2 during left-lung ventilation was 22 mmHg. After 7 min of left-lung ventilation, Pao2 = 68 mmHg, pH = 7.25, and PacO2 = 68 mmHg. Three minutes after returning to two-lung ventilation, PetCO2 was 28 mmHg, Pao2 was 256 mmHg, pH was 7.26, and PacO2 was 65 mmHg.

The persistence of the large PacO2–PetCO2 gradient and hypoxemia encountered during left-lung ventilation prompted us to estimate the alveolar dead space of each lung. Because the patient was unable to tolerate left-lung ventilation, we used a dual-capnography setup (see fig. 1) to measure PetCO2 values from the individual lungs without interrupting two-lung ventilation. PetCO2 of the right lung (47 mmHg) was unexpectedly higher than that of the left lung (22 mmHg). The capnograms of individual lungs were normal in shape and differed only in height of the alveolar plateau (phase III). The PacO2–PetCO2 gradient was more than two-fold greater in the left than in the right lung (43 versus 18 mmHg; table 1). The surgeons drained the right-side hemothorax (2.5 l blood), but the PetCO2 and PacO2–PetCO2 values of each lung remained unchanged, and hypoxemia again occurred during left-lung ventilation. Therefore, the operation was limited to right-side pleurectomy.

The patient was transferred to the intensive care unit with a single-lumen endotracheal tube in place. Respiratory acidosis persisted; the patient progressively became hypoxemic (Pao2 = 62 mmHg, pH = 7.27, PacO2 = 60 mmHg, FiO2 = 100%, minute ventilation = 9 l/min), and pharmacologic therapy was required for hypotension. A transsthoracic echocardiogram showed an echocardiographic density in the left pulmonary artery (PA). Pulmonary angiography confirmed the presence of a
large thrombus in the left PA with subtotal occlusions of multiple vessels in the left upper lobe and lingula. The interventional radiologists removed the large clots via aspiration, which resulted in an immediate increase in oxygen saturation and improved hemodynamic stability. A venacaval filter was inserted into the infrarenal portion of inferior vena cava. During the next 24 h, the respiratory acidosis resolved and hemodynamic support was withdrawn.

We reevaluated the PaCO₂-PetCO₂ gradient of the left lung. Sequential PetCO₂ samples were obtained from each lung via the suction port of an adult fiberoptic bronchoscope placed approximately 1 cm from the origin of each upper-lobe bronchus. The PaCO₂-PetCO₂ gradient was 5 mmHg in the left lung and 10 mmHg in the right lung (table 1). The patient made an uneventful recovery after embolectomy and was discharged home 2 weeks later.

**Discussion**

Dual capnography allows carbon dioxide waveforms to be obtained from individual lungs without interrupting two-lung ventilation. It has been suggested that such capnographic data can be used to confirm correct DLT placement and to detect malposition, unilateral obstruction, or accidental displacement of a DLT. Our case report suggests that dual capnography may also be useful for the rapid estimation of alveolar dead-space ventilation of an individual lung in situations in which ventilation or oxygenation is impaired.

The differential diagnosis of a large PaCO₂-PetCO₂ gradient (37 mmHg) during two-lung ventilation includes obstructive airway diseases and disorders associated with major reductions of pulmonary blood flow, such as low cardiac output states (hypovolemia, pericardial effusion, tumor-related obstruction of venous return), pulmonary vascular compression (tumor, large hemothorax), and thromboembolism of pulmonary arteries. We excluded the former cause, because our patient did not have clinical or capnographic evidence of airway obstruction. We also excluded low cardiac output as a cause of pulmonary hypoperfusion because a low-output state would have produced bilateral pulmonary hypoperfusion accompanied by similar increases in the PaCO₂-PetCO₂ gradients of the two lungs. Because hypoxemia during one-lung ventilation is not an infrequent occurrence, our conclusion that a major impairment of perfusion to the left lung existed was based primarily on the marked unilateral differences in carbon dioxide gradients (left > right). It was initially unclear, however, whether the impairment was caused by extrinsic compression (tumor) or internal obstruction (thromboembolism) of the left pulmonary artery. In either case, an
intraoperative transesophageal echocardiography could have helped to make the definitive diagnosis.

As with dual capnography, the standard method of \( P_{\text{ET}}\text{CO}_2 \) sampling via a DLT (capnograms of individual lung obtained via interruption of ventilation to contralateral lung) can also reveal the type of problem encountered in our patient. However, dual capnography was our preferred approach, because it allows one to assess the V/Q status of each lung in individuals who cannot tolerate one-lung ventilation long enough to reach a new steady state. Also, the critical perfusion defect in our patient could have been diagnosed on the basis of the low \( P_{\text{ET}}\text{CO}_2 \) of the left lung compared to that of the right, without measurement of \( P_{\text{a}}\text{CO}_2 \). By computing the \( P_{\text{a}}\text{CO}_2 - P_{\text{ET}}\text{CO}_2 \) gradient, however, we were able to obtain a more direct estimation of the alveolar dead space of each lung.

In the absence of a DLT, we used an FOB to obtain \( P_{\text{ET}}\text{CO}_2 \) samples from each lung. Our technique allowed us to detect a rapid decrease in the alveolar dead-space ventilation of the left lung after aspiration of large thromboemboli from the left PA. We acknowledge that there are no published comparisons of our approach and standard dual capnographic techniques. Nonetheless, this method, at least theoretically, may be useful to determine the \( P_{\text{ET}}\text{CO}_2 \) of expired gases from each lung. It is possible that bronchial sampling via FOB from one lung could be contaminated with expired gases from the contralateral lung, particularly when expired flow rate decreases to less than 150 ml/min (sampling flow rate of capnograph). Such contamination, however, seems unlikely in our case because the capnograms from each lung had normal shapes and differing heights of the alveolar plateau.

In conclusion, dual capnography helped to detect pulmonary emboli and to assess the efficacy of embolectomy therapy. Based on our findings, we suggest that dual capnography may be useful in the evaluation of ventilation or perfusion abnormalities associated with a variety of pulmonary diseases.

References