Hypothesis: We hypothesized that late pulmonary dead space fraction (Fdlate) would be a useful tool to screen for pulmonary embolism (PE) in a group of surgical patients, including patients who required mechanical ventilation and patients with adult respiratory distress syndrome.

Design: We prospectively calculated Fdlate in patients with suspected PE who underwent pulmonary angiography.

Setting: University-based, level I trauma center.

Main Outcome Measure: Ability of Fdlate to identify patients with PE.

Results: Twelve patients had 14 angiograms for suspected PE. The Fdlate was 0.12 or above in all 5 patients who had PE; 4 required mechanical ventilation. The Fdlate values were below 0.12 in 8 of 9 patients without PE. Four patients had adult respiratory distress syndrome. The Fdlate had 100% sensitivity and 89% specificity for the detection of PE.

Conclusions: The Fdlate is a valuable tool for bedside screening of PE in surgical patients. We were able to accurately detect all PEs.

Arch Surg. 1999;134:869-875

Pulmonary embolism (PE) presents the clinician with a difficult diagnostic dilemma. Pulmonary embolism occurs relatively infrequently, in about 1% to 2% of trauma patients, and it is fatal in about 25% to 50% of these cases. Initial signs and symptoms are varied and nonspecific. The consequence of failing to diagnose or delaying the correct diagnosis of PE may be death. Because the treatment of PE, anticoagulation, can be associated with substantial risks and even death, overdiagnosis is similarly problematic. Unfortunately, the currently available diagnostic studies, perfusion ventilation scan and pulmonary angiogram, are cumbersome, invasive, and expensive. The transportation of patients who required mechanical ventilation for either of these diagnostic studies is further associated with major risks. Because of these facts, a bedside noninvasive screening test that could accurately exclude the diagnosis of PE in patients without PE and identify patients with a high likelihood of PE is urgently needed.

Several investigators have described methods to detect PE at the bedside,* but these methods have been plagued with inadequate sensitivity and specificity, particularly in patients with underlying pulmonary disease. In contrast, Eriksson et al were able to differentiate normal patients, patients with PE, and patients with coexisting abnormal pulmonary function (chronic obstructive pulmonary disease [COPD] or interstitial lung disease) by means of a value, late pulmonary dead space fraction (Fdlate), calculated from the carbon dioxide (CO₂) expirogram. A CO₂ expirogram is different from the capnogram waveform familiar to most clinicians. The capnogram waveform, obtained from end-tidal CO₂ monitors, displays CO₂ vs time. Conversely, the CO₂ expirogram displays the CO₂ concentration of an exhaled breath vs the volume of a single exhaled breath (Figure 1). The end-tidal CO₂ is the CO₂ value at the completion of an exhaled breath. The CO₂ expirogram is divided into 3 phases. Phase I represents the volume of the exhaled airway breath, which is essentially CO₂ free; phase II represents the transition between exhalation of gas from the airway and alveoli; and phase III represents the exhaled gas from the alveoli. Eriksson et al found that the CO₂ value of the extrapolated phase III, fit to a logarithmic curve, reached PACO₂ levels at an equivalent exhaled volume of 15% of total lung...
capacity (TLC) (measured from the start of phase II) in both healthy subjects and patients with obstructive or interstitial pulmonary disease (Figure 2 and Figure 3). In patients with PE, however, the CO₂ value of the extrapolated phase III curve fails to reach arterial CO₂ levels (Figure 4). This “late dead space fraction,” Fₐ₀late, is calculated by means of the formula where Fₐ₀CO₂ is the arterial fractional CO₂ and F₁₅%TLC CO₂ is the fractional CO₂ value at an exhaled volume of 15% of the TLC:

\[ F_{\text{late}} = \frac{(F_{\text{aCO₂}} - F_{15\%\text{TLC CO₂}})}{F_{\text{aCO₂}}} \]

Ericksson et al found that patients with PE had an Fₐ₀late of 0.12 and above; healthy subjects and patients with COPD or interstitial disease had Fₐ₀late values less than 0.12. However, they studied predominantly medical patients, the majority of whom did not require mechanical ventilation. We wished to evaluate the utility of the Fₐ₀late in...
the bedside detection of PE in surgical patients, including patients who required mechanical ventilation and patients with adult respiratory distress syndrome (ARDS).

Eleven of the 12 patients enrolled in our study were victims of trauma. The mechanisms of injury were motorcycle collisions (5 patients), motor vehicle crashes (3 patients), assault (1 patient), a fall (1 patient), and a gunshot wound (1 patient). The injuries of the trauma patients are shown in Table 1. The remaining patient required emergency operation for a perforated ileum resulting from cocaine intoxication. The group consisted of 9 males and 3 females; the median age was 34 years (range, 12-61 years). At the time of evaluation, 10 patients required mechanical ventilation, 4 patients had ARDS, and 1 patient had COPD.

Twelve patients underwent 14 pulmonary angiograms for 14 separate episodes of suspected PE. Indications for pulmonary angiogram included acute hypoxia or desaturation (10 patients), tachypnea (8), increased oxygen requirement by means of a ventilator (4), chest pain (3), shock (1), and electrocardiogram changes (1). (Some patients had more than 1 reason to suspect a pulmonary embolism.) Five patients had pulmonary angiograms diagnostic of PE; 4 of these patients required mechanical ventilation. Their Fdlate values were 0.12 and above (0.12-0.21) (Figure 6). Nine patients had angiograms negative for PE. Eight of these patients had Fdlate values below 0.12 (−0.22 to 0.08) (Figure 6). One patient had an Fdlate greater than 0.12 (0.25) but a negative angiogram. Although Fdlate was calculated at the time PE was suspected, the patient underwent angiography 18 hours later. This patient was treated with long-term anticoagulation for clinically suspected PE. Four of the 9 patients had ARDS; all had negative angiograms and an Fdlate less than 0.12. Overall, the Fdlate had 100% sensitivity and 89% specificity for the detection of PE with the use of a cutoff of 0.12.

Physiological dead space, alveolar dead space fraction, end-tidal P\textsubscript{CO\textsubscript{2}}, and arterial end-tidal P\textsubscript{CO\textsubscript{2}} gradient are shown in Table 2 for patients with pulmonary angiograms positive and negative for PE. The Fdlate, physiological dead space, alveolar dead space fraction, arterial P\textsubscript{CO\textsubscript{2}}, and arterial to end-tidal P\textsubscript{CO\textsubscript{2}} gradient were significantly different between the 2 groups (P<.05, t test). The end-tidal CO\textsubscript{2} value was not significantly different in patients with pulmonary angiograms diagnostic of PE.
compared with the end-tidal CO\textsubscript{2} value in patients with angiograms negative for PE.

A readily available and portable screening tool for the bedside examination of patients with suspected PE is desperately needed. Although PE is relatively uncommon after trauma, its prompt diagnosis is a formidable task for the clinician. Physical findings are varied and nonspecific.\textsuperscript{3} Furthermore, assessment of arterial blood gases,\textsuperscript{11} calculation of alveolar arterial oxygen gradients,\textsuperscript{10,11} and evaluation of the chest radiograph or electrocardiogram are neither sensitive nor specific.\textsuperscript{3} In fact, in nearly one half of patients, sudden death is the initial sign of PE.\textsuperscript{3} A number of investigators have described various bedside techniques to screen for PE, including measurement of respiratory dead space\textsuperscript{6,9} and measurement of either arterial to end-tidal CO\textsubscript{2} gradient\textsuperscript{4,5,7} or alveolar dead space fraction\textsuperscript{8,9} with and without forced expiration.\textsuperscript{3,5-8} These studies have been limited because of difficulties in differentiating patients with PE from those with underlying lung disease alone.\textsuperscript{9} Eriksson et al,\textsuperscript{12} however, found that Fd\textsubscript{late} calculation from the CO\textsubscript{2} expirogram adequately differentiated these patient groups. All of these studies, including that by Eriksson et al, evaluated chiefly medical patients or studied large numbers of spontaneously breathing or ambulatory patients. We undertook this study to evaluate Fd\textsubscript{late} as a screening tool to detect PE in surgical patients, including those who required mechanical ventilation or had ARDS.

We found Fd\textsubscript{late} to be a valuable tool for bedside screening for PE in surgical patients including those who required mechanical ventilation and those who had ARDS. We were able to identify all of the patients with PE. Because of recent advances in technology, both the CO\textsubscript{2} expirogram and the Fd\textsubscript{late} value are readily determined at bedside.\textsuperscript{13,14} Both the accuracy and sensitivity were good, 93% and 100%, respectively. Only 1 patient was incorrectly classified.

Pulmonary embolism increases physiological dead space or, more specifically, alveolar dead space. Burki\textsuperscript{6} studied the value of the measurement of physiological dead space in the detection of pulmonary embolism. He found a physiological dead space of greater than 0.40 to have 100% sensitivity and 55% specificity for the detection of PE. The use of spirometry improved the specificity; however, it was not readily performed at the bedside. In contrast, Eriksson et al\textsuperscript{12} also found a significant overlap in the physiological dead space values in patients with PE, obstructive lung disease, and interstitial lung disease, and in a group of patients with negative pulmonary angiograms. Two of the patients with documented PEs in our study had a physiological dead space of less than 0.40. Using a physiological dead space of 0.40 as a threshold for diagnosing PE in our group of surgical patients resulted in a sensitivity of only 60%, a specificity of 100%, a negative predictive value of 81%, and a positive predictive value of 100%. On the basis of these data, we believe that physiological dead space has inadequate sensitivity to be used as a screening tool for PE.

Alternatively, several investigators have evaluated the utility of end-tidal CO\textsubscript{2} to screen for PE.\textsuperscript{4,5} Pulmonary embolism results in an increased arterial to end-tidal CO\textsubscript{2} gradient.\textsuperscript{6,17} Using this simple gradient, Robins et al\textsuperscript{17} found that 7 of 8 patients with PE had a gradient exceeding 5 mm Hg. Three healthy patients had values less than 5 mm Hg. However, the utility of this test is limited in clinical practice, as patients with underlying pulmonary disease other than PE also have an increased ar-

<table>
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<tr>
<th>Patient No.</th>
<th>Fd\textsubscript{late}</th>
<th>Physiological Dead Space</th>
<th>Alveolar Dead Space Fraction</th>
<th>Arterial to End-Tidal PCO\textsubscript{2} Gradient</th>
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</thead>
<tbody>
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</tr>
<tr>
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<td>0.34†</td>
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</tr>
<tr>
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<td>0.18</td>
<td>0.18</td>
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</tr>
</tbody>
</table>

**Table 2. Physiological Dead Space, Alveolar Dead Space Fraction, End-Tidal PCO\textsubscript{2}, and Arterial End-Tidal PCO\textsubscript{2} Gradient**

*Fd\textsubscript{late} indicates late pulmonary dead space fraction.
†P<.05, t test (positive vs negative pulmonary angiogram).
arterial end-tidal PCO2 gradient. In our group of surgical patients, a gradient of 5 mm Hg would have resulted in a sensitivity of 80%, a specificity of 78%, a positive predictive value of 67%, and a negative predictive value of 88%.

Inspection of the CO2 expirogram demonstrates that the end-tidal CO2 is highly dependent on the exhaled tidal volume, particularly in the presence of a steeply sloping phase III (Figure 3). To improve the discrimination of the arterial to end-tidal CO2 gradient, Hatle and Rokseth1 studied the effect of a forced expiration. They found that patients with PE maintained a large gradient despite an increase in exhaled breath volume. Conversely, in 19 of 21 patients with COPD, they noted “normalization” of the gradient with forced exhalation. They did not declare a threshold value for the diagnosis of PE. Chopin et al8 also used forced exhalation to detect PE in a group of 34 patients with COPD with acute respiratory failure. In place of the arterial to end-tidal CO2 gradient, they used a derived index, R ([(PaCO2 − E TCO2)/PaCO2] × 100) (an estimate of alveolar dead space2) or alveolar dead space fraction21 assuming end-tidal CO2 equals average alveolar CO220. They suggested an R value with maximal exhalation of greater than 5% as diagnostic of PE. In their patient group, their technique yielded a sensitivity of 100% for the detection of PE, although a specificity of only 65%. In a group of ambulatory emergency department patients screened for PE, Kline et al5 used a cutoff equivalent to an R of 20% and found the test to have a sensitivity of 88% and a specificity of 94%.

However, use of forced exhalation depends on active patient participation. Patients with postoperative pain may be unable to achieve an adequate exhalation, and this may limit the utility of the test. Further, exhaled volume may vary greatly among patients or within the same patient during the postoperative recovery. In contrast, calculation of Fd late from the CO2 expirogram does not require a forced exhalation. We instructed spontaneously breathing patients to breathe normally; patients who required mechanical ventilation did not have alteration of their ventilator settings. Phase III of the CO2 expirogram was extrapolated to an exhaled volume equivalent to 15% of predicted TLC (based on patient sex and size) and Fd late was calculated. Thus, use of Fd late compensates for size differences between patients as well as correcting for inability to deeply exhale. Further, physiological dead space, alveolar dead space and dead space fraction of carbon dioxide elimination are readily determined from the CO2 expirogram.

Although Fd late calculation is promising as a screening test for PE, several questions remain about the use of Fd late in surgical patients. First, the amount of thrombus during PE necessary to elevate Fd late above 0.12 is unclear. Carroll22 found detectable changes in the end-tidal CO2 with inflation of the balloon of a pulmonary catheter to 1 mL. Second, to date, inadequate data are present to define the “normal” Fd late in surgical patients who required mechanical ventilation. Third, decreased pulmonary blood flow increases physiological dead space and may also increase the Fd late. Further studies to determine the range of Fd late values in patients who required mechanical ventilation or hypotensive patients and to evaluate the sensitivity of Fd late to PE load are warranted.

**CONCLUSIONS**

Monitoring the CO2 expirogram and determining Fd late appear promising as bedside methods for the detection of PE. The Fd late was sensitive, easy to perform at the bedside, noninvasive, and functional on all surgical patients, and filled a critical need for the detection of PE. The CO2 expirogram offers several advantages and provides additional information to that obtained from the capnogram or monitoring of end-tidal CO2 alone. Further studies of the utility of the CO2 expirogram and Fd late as a screening tool for PE in critically ill surgical patients are warranted.

**REFERENCES**

Discussion

Kenneth Waxman, MD, Santa Barbara, Calif: Injured and postoperative patients in whom we suspect pulmonary embolism are often critically ill. There may be considerable risk in moving them to a radiology department for diagnostic tests. Further, the time delays in obtaining a radiological diagnosis can be prolonged in patients who may need urgent treatment. Clearly, for injured and postoperative patients, there are increased risks to empirically initiating heparin or thrombolytic therapy. For all of these reasons, a bedside test to detect pulmonary embolism has great potential importance. For those of us who may not have thought much lately (or maybe ever) about capnography, let me first review the concept of utilizing expired PCO2 data to detect pulmonary embolism. An embolism that occludes blood flow through the pulmonary artery increases physiological dead space. This occurs because alveoli in the portions of the lung without pulmonary arterial flow will not exchange gas, and thus alveolar PCO2 will not equal arterial PCO2. Hence, exhaled PCO2 measured with capnography will not reach arterial PCO2 during exhalation. A gradient between end-tidal PCO2 and arterial PCO2 thus results. An arterial to end-tidal PCO2 gradient of more than 5 mm Hg has been suggested for a number of years now as a diagnostic test for pulmonary embolism. But this has not proven reliable, particularly in patients with COPD. In my experience there also often is a gradient in ventilated postoperative and trauma patients, which may result from ventilation-perfusion mismatch due to microcirculatory abnormality. For this reason the method utilized in the present study is different than routine end-tidal PCO2 measurements, although it is closely related. The technique of this study assigns exhaled volume rather than time on the x-axis. This allows a computer to analyze expired PCO2, as a function of the amount of exhaled gas. Previous research has shown that in individuals without pulmonary embolism, expired gas approaches the CO2 concentration of arterial blood after 13% of the total lung capacity. The late dead space fraction utilized in the present study is defined as the difference between arterial PCO2 and the projected exhaled CO2 concentration after 15% of exhalation, expressed as a fraction. It is very much like the shunt fraction that we know for perfusion abnormalities. This fraction should normally be zero, that is, after 15% of exhalation there should be no further dead space exhalation.

In Dr. Anderson’s paper, the 5 patients with pulmonary embolism had late dead space fractions of 12% to 21%, whereas patients without pulmonary embolism had late dead space fractions of −22% to 8%. The bedside technique was thus highly accurate in this series in predicting which patients had pulmonary embolism, utilizing a cutoff of 0.12. I have a number of questions for the authors about their work. (1) First, why were pulmonary angiograms utilized rather than spiral computed tomographic (CT) scans in your study? Is there a role for ventilation-perfusion scan in the portions of the lung without pulmonary arterial flow will not exchange gas, and thus alveolar PCO2 will not equal arterial PCO2. Hence, exhaled PCO2 measured with capnography will not reach arterial PCO2 during exhalation. A gradient between end-tidal PCO2 and arterial PCO2 thus results. An arterial to end-tidal PCO2 gradient of more than 5 mm Hg has been suggested for a number of years now as a diagnostic test for pulmonary embolism. But this has not proven reliable, particularly in patients with COPD. In my experience there also often is a gradient in ventilated postoperative and trauma patients, which may result from ventilation-perfusion mismatch due to microcirculatory abnormality. For this reason the method utilized in the present study is different than routine end-tidal PCO2 measurements, although it is closely related. The technique of this study assigns exhaled volume rather than time on the x-axis. This allows a computer to analyze expired PCO2, as a function of the amount of exhaled gas. Previous research has shown that in individuals without pulmonary embolism, expired gas approaches the CO2 concentration of arterial blood after 13% of the total lung capacity. The late dead space fraction utilized in the present study is defined as the difference between arterial PCO2 and the projected exhaled CO2 concentration after 15% of exhalation, expressed as a fraction. It is very much like the shunt fraction that we know for perfusion abnormalities. This fraction should normally be zero, that is, after 15% of exhalation there should be no further dead space exhalation.

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Would the dead space fraction be sensitive enough to detect small pulmonary embolisms? The answer to that remains to be seen. Obviously, pulmonary embolism is a scale. There could be something as small as a few platelet aggregates going up. Obviously we wouldn't detect that, and a saddle embolism is at the other end of the spectrum. Where does this dichotomize? I don't have an answer to that.

Do you have data on how long the dead space fraction remains abnormal after a pulmonary embolism. We can't exactly pinpoint the moment of pulmonary embolism occurrence. We have done sequential measurements in at least 1 patient and saw that this measurement seems to return to close to normal around 10 hours after a major pulmonary embolism. Interestingly, in doing these serially during thrombolytic therapy, we find that it returns to normal more rapidly. So it does seem to return and do so at a reasonably rapid pace.

How reliable would this technique be in other situations that increase physiological dead space, such as trauma or ARDS? This also gets at Dr Gentilello's question. I appreciate his making that comment. Eriksson showed in COPD that it is actually quite useful. In our patients it appears to be useful in trauma, which has some increased ventilation-perfusion mismatching, as you see with COPD. The issue of what is ARDS may be as relevant as how effective this is in ARDS? One of my mentors, Dr Blaisdell, has convinced me that a substantial part of ARDS is based on actual thrombotic material embolizing to the lungs, which, after all, is what a pulmonary embolism is. It turns out that none of our patients had both ARDS and PE, and so we can't truly answer that question. I will be interested to see when the first concurrent set of diagnoses occurs to see how this works. The goal of this test is not so much to make the diagnosis of pulmonary embolism as it is to exclude it. If we can exclude it, I think we have a useful test.

Are we relying on this test to make the diagnosis of PE, or do we use it to rule out PE, which I would hope we could do? The answer is no, not yet. We have a larger trial ongoing to further validate our current findings, and once we finish that, then we will consider moving on with this as a primary test to exclude the diagnosis of pulmonary embolism.

Dr Atik, thank you for your question about thromboprophylaxis. Yes, these patients did undergo routine thromboprophylaxis. Our routine prophylaxis includes heparin, and specifically we use unfractionated heparin, given in a dose to bump a partial thromboplastin time about 5 seconds. In patients who have absolute contraindications to heparin thromboprophylaxis, we will use sequential compression device stockings for those patients who are at low risk for PE, and we will use caval filters if the patient is at extremely high risk of PE.

Dr Gentilello, hopefully I answered the question on the ARDS and getting to the question of what happens when you have a very low cardiac output. After all, that also results in areas of the lung that have a substantial ventilation-perfusion mismatch. That is an excellent question. The answer is, we don't have data yet on a substantial number of patients who were in absolute shock to determine whether the test will pick up the pulmonary embolisms. More to come on that.

Dr Demetriades, thank you for your questions. On the question of chest trauma, yes, this study appears to do reasonably well in patients with significant chest trauma. And what does it take to do this particular test? The computer system and the machine to actually measure the expired CO₂ is about the size of a pulse oximeter. We were doing the subsequent statistical analyses and curve diagrams on a computer offline, so that is obviously a bigger instrument. But I think as this gains acceptance, it will basically be yet another pack about the size of a small propack, so maybe about 8 inches by 8 inches, that can be just plugged inline to the ventilator with little trouble.

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**Announcement**

The Archives of Surgery will give priority review and early publication to seminal works. This policy will include basic science advancements in surgery and critically performed clinical research.