**REFERENCE**


**V/Q Inhomogeneity Causes Increased Physiologic Dead Space**

To the Editor:

It is easy to understand how ventilation of an unperfused area of lung, as in pulmonary embolism, will cause an increase of physiologic dead space. What is harder to understand, is not generally known, and is far more important is that inhomogeneity of V/Q ratios in the lung will cause an increase of the physiologic dead space. This is due to the hyperbolic relationship between alveolar PaCO₂ (PaCO₂) and alveolar ventilation (V̅ₐ) (Fig 1). In this example, reduction of PaCO₂ from 40 to 20 mm Hg requires increase of V̅ₐ from 5.2 to 10.5 L/min, whereas increase of PaCO₂ by a similar amount, from 40 to 60 mm Hg, results from much smaller decrease of V̅ₐ. Thus, if low V/Q units are present, compensation to produce a normal arterial PaCO₂ will require a much greater increase of ventilation in some areas of the lung than the decrease that occurred in others.

Why does this cause an increase of the physiologic dead space? This is evident from the Bohr equation:

\[ V̅_D = \frac{\text{PaCO}_2 - \text{PaCO}_2}{\text{PaCO}_2} \]  

To keep PaCO₂ normal when V/Q inhomogeneity develops, total ventilation must be increased. At a constant metabolic rate, expired CO₂ tension (PeCO₂) must then fall so that there will be increase of the dead space (V̅Ｄ).

The result of this relationship is that V/Q inhomogeneity requires increase of total ventilation for maintenance of a normal arterial PaCO₂. Patients with COPD are hypercapnic despite increased total ventilation because of increase of V̅D/VT.

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**Endobronchial Streptokinase to Dissolve a Right Mainstem Clot**

To the Editor:

The successful use of endobronchial streptokinase to dissolve an endobronchial blood clot has previously been reported.¹ We report another such case. A 51-year-old man with chronic obstructive lung disease was transferred to our hospital because of a six-month history of worsening cough, fever and shortness of breath. Admission chest x-ray examination revealed several cavities in the left apex and a diffuse infiltrate involving most of the left lung. Intubation and mechanical ventilation became necessary shortly after admission. The diagnosis of cavitary histoplasmosis was established by bronchoscopic smear and culture test results. No endobronchial lesions were seen on initial bronchoscopic examination. On the sixth hospital day, massive hemoptysis ensued approximately 10 min after endotracheal suctioning. This lasted only briefly, but the patient became markedly distressed and breath sounds were decreased over the right lung. Chest x-ray examination showed complete right lung collapse and the PaO₂ dropped to 40 mm Hg on 100 percent FIO₂. Bronchoscopic examination revealed a dense clot completely occluding the proximal right main stem bronchus. Attempts to dislodge this clot with suction, saline solution lavage, forceps and Coudé catheter manipulation were unsuccessful despite repeated efforts. Rigid bronchoscopic study was considered but not attempted. Streptokinase was then directed over the clot through the bronchoscope. A total of 60,000 units in 60 ml saline solution was injected over the clot in 10 ml aliquots. Following repeated installations of streptokinase, much of the clot was dislodged and removed through the ET tube. Further inspection then revealed a small clot adherent to the horizontal carina, partially obscuring the orifices to the right middle lobe and right lower lobe. This was assumed to represent the source of bleeding. Oxygenation and chest x-ray film showed progressive improvement, and the patient was eventually extubated and discharged.

Endobronchial blood clot cast are not a common cause of massive atelectasis,² yet many bronchoscopists have occasionally encountered this problem. We agree with Cole and Grossmen¹ that mechanical removal should be attempted initially, but that endobronchial streptokinase lysing may be successful when such attempts fail. The underlying cause of hemoptysis is likely an important factor. Optimal streptokinase dosage has not been determined. Use of a small plastic catheter to direct the streptokinase directly onto the clot may be of benefit, for we observed that much of what we instilled passed into the left mainstem branches without effect.

**REFERENCES**


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**Figure 1**

- **Graph** showing the relationship between V̅₂̅ (V̅₂̅) and PaCO₂ (PaCO₂) with various points illustrating the effect on V̅ₐ for different PaCO₂ levels.