Although the bibliography for Kirilloff’s review was extensive, one very important paper was not cited; the report by Reines et al documents that chest percussion might actually be contraindicated in another category of patients. Reines studied fifty consecutive children between the ages of three months and nine years who had undergone cardiac surgery for congenital heart disease. The children were randomized into two groups. The first group (CPT) received routine post-operative therapy comprised of deep breathing/suctioning/coughing/postural drainage/vibration/percussion. The second group (NCPT) received deep breathing/suctioning/coughing without postural drainage/vibration/percussion. Response to therapy was evaluated by a radiologist who was unaware of the treatment group to which each patient was assigned. CPT was associated with significantly more frequent and more severe atelectasis than NCPT. The authors speculated that the reason why CPT might actually be harmful is that the procedure causes pain which subsequently triggers splinting and reductions in functional residual capacity. Another notable finding of this study was the observation that atelectasis was not significantly associated with temperature spikes. Thus, the knee-jerk institution of respiratory therapy (with or without chest physiotherapy) secondary to the observation of a spiking temperature was not indicated in these patients. Thus, although the study has not been performed in adults, clinicians should remain skeptical regarding the routine use of respiratory therapy in open heart patients and especially skeptical with regard to the use of chest percussion in this group in the presence or absence of fever.

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REFERENCE

To the Editor:

We appreciate the comments of Mr. Demers regarding our article. The article cited in his letter provides excellent support for the need to objectively evaluate the effects of chest physical therapy, rather than to assume benefits. Further, the letter provides strong support for judicious use of this treatment from a highly-respected member of the respiratory therapy profession.

Our article focused on the effects of chest physical therapy in adults because this is the population served in our Division. We are delighted to highlight additional evidence which supports the need to identify specific indications for use of this treatment. The argument that “even if chest physical therapy might not add to patient therapy, at least it does not detract” is encountered frequently but, as noted by Mr. Demers, is not necessarily correct.

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Loss of Fiberoptic Laser Ferrule

To the Editor:

I found the recent report of the “Loss of Fiberoptic Laser Tip” by Doctor Mehta (Chest 1985; 88:798) particularly timely since I recently experienced a similar complication. In view of the structure of the quartz fiber system used with the Nd-YAG laser, this may be an underreported complication of the procedure.

A 72-year-old white man was referred because of total atelectasis of the left lung. He had enjoyed good health until 1979, when he was found to have an adenocarcinoma of the rectum. This was resected and he did well until 1982, when a small left lung mass was found. A diagnosis of adenocarcinoma was made by percutaneous thoracic needle aspiration. He received intermittent chemotherapy with 5-FU and did well until 1984, when he developed a left-sided pneumothorax. This was treated with a McSwain dart, but the lung failed to completely reexpand. The McSwain dart was removed and further evaluation was not performed at that time. Over the ensuing months, he developed progressively worsening dyspnea and malaise. A follow-up chest x-ray examination revealed total atelectasis of the left lung and bronchoscopic examination revealed total obstruction of the left mainstem bronchus with tumor. He then underwent Nd-YAG laser photoresection using a fiberoptic bronchoscope and local anesthesia. The bronchial lumen was recanalized using energy levels of 40 watts with 0.5 sec duration. The fiber was frequently removed for cleaning and inspection during the procedure. A total of 12,150 jouls was delivered during the procedure. Near completion of the procedure, the fiber was removed for cleaning and it was noted that the metal ferrule was missing from the tip of the laser fiber. The bronchoscope was reintroduced and the bronchi carefully searched for the metal ferrule, but it could not be found. The bronchoscope was next examined and the ferrule was found lodged in the proximal aspiration port. It was then easily extracted from the bronchoscope and replaced on the tip of the laser fiber. There had been no fires at the tip of the laser fiber during the procedure and the patient suffered no ill effects.

I assume by the description of Doctor Mehta’s case report that he is referring to the loss of the metal ferrule from the tip of the laser fiber when he refers to loss of the fiber tip, as happened in the preceding case report. Although the metal ferrule is small, its retention in a bronchus could cause problems over the long run, particularly if the procedure is being performed for a benign lesion. Potential complications could include granuloma formation around the ferrule, atelectasis and postobstructive pneumonia, as well as hemoptysis. It is also frustrating to try to replace the metal ferrule in order to complete the procedure.

The function of the metal ferrule is to hold the fiber tip in the center of the gas stream which is blown continually through the catheter containing the laser fiber. This helps keep the fiber tip clean as well as clearing the working surface of smoke and debris. Potential problems with this system were first alluded to by Hetzel,1 who pointed out that tissue or mucous can adhere to the fiber tip and result in absorption of energy from the emerging light, raising the tip to very high temperatures. This results in the expansion of the metal ferrule, as well as breakdown in the glue holding the ferrule. This may then result in difficulty withdrawing the catheter system from the bronchoscope, or even dislodgement or loss of the metal ferrule from the tip of the laser fiber.

I certainly agree with Doctor Mehta that the best way to avoid this complication is to keep the laser fiber tip meticulously clean. This can best be accomplished by keeping the laser fiber tip at least 0.5 cm from the working surface, as well as frequently removing the laser fiber for inspection, cleaning and cooling of the tip. Finally, if resistance is met in trying to withdraw the catheter system from the bronchoscope, additional time should be given for the tip to cool before applying further traction on the laser catheter system. If this becomes a frequently experienced problem, then perhaps further modification in the catheter system will be necessary.

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CHEST / 99 / 6 / JUNE, 1986 903
\[ \dot{V}/Q \text{ Inhomogeneity Causes Increased Physiologic Dead Space} \]

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 \( P_{CO_2} \) (PaCO\(_2\)) and alveolar ventilation (\( \dot{V}_A \)) (Fig 1). In this example, reduction of PaCO\(_2\) from 40 to 20 mm Hg requires increase of \( \dot{V}_A \) from 5.2 to 10.5 L/min, whereas increase of PaCO\(_2\) by a similar amount, from 40 to 60 mm Hg, results from much smaller decrease of \( \dot{V}_A \). Thus, if low \( V/Q \) units are present, compensation to produce a normal arterial PaCO\(_2\) 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:

\[ V_D = \frac{P_{CO_2} - PaCO_2}{PaCO_2} \times \dot{V}_A \]

To keep PaCO\(_2\) normal when \( V/Q \) inhomogeneity develops, total ventilation must be increased. At a constant metabolic rate, expired CO\(_2\) tension (PeCO\(_2\)) must then fall so that there will be increase of the dead space (Vd).

The result of this relationship is that \( V/Q \) inhomogeneity requires increase of total ventilation for maintenance of a normal arterial PaCO\(_2\). Patients with COPD are hypercapnic despite increased total ventilation because of increase of Vd/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 PaO\(_2\) dropped to 40 mm Hg on 100 percent FiO\(_2\). 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 Coude 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 casts are not a common cause of massive atelectasis,\(^2\) yet many bronchoscopists have occasionally encountered this problem. We agree with Cole and Grossmen\(^3\) 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.

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REFERENCES


\[ \text{FIGURE 1} \]

Communications to the Editor