PEEP and "Reverse Mismatch"*
A Case Where Less PEEP Is Best


A V/Q lung scan was obtained in a patient with LLL collapse who was receiving IPPV and PEEP. This revealed absent ventilation and hyperperfusion to the collapsed lobe. After a reduction in PEEP from 12 to 5 cm H2O, a repeat V/Q scan showed a more even distribution of pulmonary perfusion. Arterial hypoxemia improved.

(Chest 1991; 99:1034-36)

Despite continuing controversy regarding the most appropriate level of PEEP,1 it remains widely held that PEEP is useful to patients with acute respiratory failure;2 however, it is recognized that PEEP may adversely affect pulmonary gas exchange in some patients with asymmetric pulmonary disease,3-4 independent of its well-known adverse effects of reducing cardiac output and predisposing to barotrauma. We present a case which highlights the likely mechanism for this phenomenon.

CASE REPORT

A 65-year-old man was admitted to the intensive care unit for elective ventilation after repair of a hepatic artery aneurysm. After surgery, there was decreased spontaneous ventilatory effort (due to narcotic analgesics, obesity, and obstructive sleep apnea), and IPPV was continued. Gas exchange was adequate, with FIO2 of 0.4 and PEEP of 5 cm H2O. The pH was 7.31, PaO2 was 84 mm Hg, and PaCO2 was 42 mm Hg. The CXR remained clear.

On the fourth postoperative day the endotracheal tube became dislodged, and aspiration was witnessed. Examination of the chest revealed bronchial breath sounds at the left base. Arterial blood gas analysis immediately after stabilization revealed pH of 7.27, PaO2 of 108 mm Hg, and PaCO2 of 50 mm Hg, with FIO2 of 1.0, respiration rate of 10/min, tidal volume of 1,000 ml, and PEEP of 8 cm H2O. The CXR showed patchy opacification of the entire left lung field (Fig 1). Pulmonary gas exchange remained severely impaired for the following 72 hours. The SaO2 was maintained at 95 percent with FIO2 of 0.8 to 0.9 and PEEP of 12 cm H2O. Three days after the aspiration, the CXR had cleared, apart from persisting LLL collapse. Because pulmonary embolism could not be excluded, a V/Q lung scan was obtained in multiple projections using a mobile gamma camera. Technetium-99m-labelled pentetic acid (diethylenetramine pentacetic acid; DTPA) aerosol was used to assess ventilation. Pulmonary perfusion was subsequently assessed using technetium-99m macroaggregated albumin. This examination revealed absent ventilation to the LLL, in association with abnormally increased perfusion to the LLL (Fig 2).

The PEEP was decreased to 5 cm H2O without deterioration in SaO2.

A repeat V/Q lung scan two days later revealed persistent loss of ventilation in the LLL, which coincided with failure of expansion of the LLL on the CXR (Fig 3), but the previously demonstrated LLL hyperperfusion was no longer apparent (Fig 2).

Pulmonary gas exchange remained adequate with FIO2 of 0.5 and PEEP of 5 cm H2O (PaO2, 105 mm Hg; PaCO2, 49 mm Hg). Fiberoptic bronchoscopy resulted in full expansion of the collapsed LLL. Thereafter, the SaO2 remained satisfactory, spontaneous ventilatory effort improved, and the patient was extubated on the 12th postoperative day. His subsequent recovery was uneventful.

DISCUSSION

This case demonstrates that in the presence of lobar collapse consolidation, IPPV with PEEP may be associated with luxury perfusion (shunt) to the collapsed lobe. Specifically, hyperperfusion to the entire LLL was demonstrated with LLL collapse and IPPV and PEEP of 12 cm H2O. Moreover, after a decrease in PEEP from 12 to 5 cm H2O, a repeat perfusion lung study showed more uniform distribution of pulmonary blood flow to both lungs, despite persisting lobar collapse and absence of ventilation. There was an associated improvement in pulmonary gas exchange.

The phenomenon of "reverse mismatch" or hyperperfusion to a collapsed lobe with PEEP has been described previously5 (PEEP of 17.5 and 8 cm H2O, respectively). In either case was the effect of a reduction in PEEP documented; however, a patient with diffuse infiltration of the right lung showed an increase in PaO2 when PEEP was decreased from 15 cm H2O to zero.6 Assessment of pulmonary blood flow distribution using xenon-133 revealed a concomitant increase in fractional perfusion to the normal lung from 33 percent to 50 percent. Distribution of ventilation was not measured.

Our case strongly supports the proposed mechanism6 for the paradoxical decrease in SaO2 with PEEP in patients with asymmetric pulmonary disease. Tidal volume is preferentially distributed to areas of high compliance. This is demonstrated in our patient by ventilation to all areas except the collapsed LLL. The IPPV and PEEP therefore increase alveolar pressure in the compliant areas of lung, resulting in compression of the microvasculature and diversion of blood flow away from well-ventilated areas to the less compliant or consolidated area. As PEEP cannot affect alveolar pressure in the nonventilated LLL, perfusion to this area is increased, resulting in true right-to-left shunt, manifested as an area of hyperperfusion on the perfusion lung scan. Usually, lobar consolidation is associated with decreased

![Figure 1. Semisupine CXR. Right lung field is clear. There is patchy opacification of entire left lung field with dense opacity of LLL. Patent bronchi are seen within LLL.](http://journal.publications.chestnet.org/pdffileover.ashx?url=/data/journals/chest/21627/ on 06/14/2017)
perfusion to the collapsed lobe, seen on a V/Q lung scan as a "matched defect;" however, in dogs with LLL collapse, whole-lung PEEP (10 cm H₂O) increased LLL perfusion and overcame the effects of hypoxic vasoconstriction.

Likewise, we propose that the reduction in PEEP in our patient from 12 to 5 cm H₂O resulted in lower alveolar pressures in the normal lung, allowing better perfusion to well-ventilated areas and decreased diversion of blood flow to the still collapsed LLL. This is supported by the more uniform distribution of pulmonary blood flow on repeat perfusion scan.

Regional pulmonary blood flow is affected by gravity, and in the supine position, blood flow is diverted to posterior dependent regions, which include the LLL. Thus the supine posture may have accentuated the LLL hyperperfusion observed in this patient, although it cannot explain the more even distribution of perfusion observed after PEEP was reduced.

Fluid loading reverses the fall in cardiac output due to PEEP. Whether it would have improved pulmonary gas exchange in this patient is unclear; however, in dogs with asymmetric pulmonary edema treated with PEEP a 37 percent increase in cardiac output with nitroprusside did not improve intrapulmonary shunt.

The adverse effect of PEEP on V/Q mismatch described in this case report does not argue against the use of PEEP in hypoxic respiratory failure, even with unilateral disease. In a study of PEEP in patients with both diffuse and asymmetric pulmonary disease, PEEP at 9 and 20 cm H₂O produced a progressive increase in PaO₂ in patients with diffuse pulmonary infiltrates. In patients with asymmetric pulmonary disease, PEEP at 9 cm H₂O resulted in an increased PaO₂, and only at PEEP of 20 cm H₂O did the PaO₂ fall. Moreover, in an animal model of lobar collapse, PEEP of 10 cm H₂O increased perfusion to the collapsed lobe by 70 percent but showed a variable effect on PaO₂.

In conclusion, the phenomenon of reverse V/Q mismatch should be considered when there is refractory hypoxemia with PEEP in asymmetric pulmonary disease. This can be readily assessed with a radionuclide V/Q scan and interpretation of the study with knowledge of the clinical setting and radiologic changes. In some patients a reduction in the level of PEEP may result in improved pulmonary gas exchange; however, this should not preclude aggressive measures to reexpand the collapsed lung.

ACKNOWLEDGMENT: We thank Dr. K. Sherbon, Director of the Department of Diagnostic Radiology, Royal Prince Alfred Hospital, for his help in the interpretation of CXRs.

REFERENCES


Figure 2. Radionuclide lung scans (only posterior views shown). A (top left): Aerosol ventilation study shows absent ventilation in LLL. In addition, there is minor inhomogenous uptake in both lungs and some central airways deposition consistent with chronic airways disease. B (top right): Initial perfusion study shows marked increase in perfusion in nonventilated LLL. There is also mild matching inhomogeneous perfusion to both lungs. On repeat study, following reduction in PEEP, ventilation study was unchanged. C (bottom right): Repeat perfusion study showed resolution of hyperperfusion pattern.

Figure 3. Supine CXR. There has been some clearing of left lung field, but LLL remains densely consolidated (arrows indicate position of ETT and CVP lines).
Intrapleural Tetracycline for Spontaneous Pneumothorax in Acquired Immunodeficiency Syndrome*

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Spontaneous pneumothorax is occurring in patients with the acquired immunodeficiency syndrome and Pneumocystis carinii infection with increasing frequency. These patients are typically poor surgical candidates. Conservative management using tetracycline sclerosis was performed with good results in a patient with acquired immunodeficiency syndrome and recurrent pneumothorax.

(Chest 1991; 99:1036-37)

Spontaneous pneumothorax is becoming an increasingly more common occurrence in patients with acquired immunodeficiency syndrome and Pneumocystis carinii-related lung disease. This relates to the nature of the lung tissue destruction caused by P. carinii infection, namely: the formation of pneumatoceles and cutaneous bullous lesions. Not only does this place patients at risk for spontaneous pneumothorax, but also it increases the risk of recurrence.

In our experience these occurrences of pneumothorax tend to be more problematic in that prolonged air leaks from chest tubes are quite common and surgical risk is generally considered to be high in this group of patients. We therefore propose a more conservative approach in the treatment of this condition and present a case managed successfully using tetracycline pleurodesis.

CASE REPORT

A 40-year-old homosexual white man was diagnosed with acquired immunodeficiency syndrome in November 1986. He had cutaneous Kaposi's sarcoma, anal herpes and several episodes of P. carinii pneumonia. He presented to Saint Vincent's Hospital on April 23, 1987, with his fifth right-sided spontaneous pneumothorax. His chest roentgenogram revealed a 100 percent pneumothorax (Fig. 1).

His first two episodes of pneumothorax occurred in 1976 and 1978. The third occurred in December 1986, at which time he presented with P. carinii pneumonia documented by cytologic studies. The fourth pneumothorax occurred in February 1987 when he also developed a Proteus pneumonia. Each episode involved a 100 percent right-sided pneumothorax and was managed with tube thoracostomy. Surgical intervention had been recommended but was refused by the patient.

On the most recent admission for the fifth episode, tube thoracostomy was once again performed. Again, the patient refused surgery. A conservative treatment using tetracycline sclerosis was then offered and accepted.

On hospital day 3 when the air leak from the chest tube ceased, tetracycline pleurodesis was performed in the following manner: 1 gram of tetracycline was dissolved in 80 ml of saline solution and 20 ml of 2 percent lidocaine. This solution was then instilled into the pleural cavity via the chest tube. The chest tube was clamped for 2 h, then placed to underwater seal. The chest tube was removed after 24 h. Full lung expansion was documented by chest roentgenogram (Fig 2).

The patient's lung remained expanded, and he was discharged.