

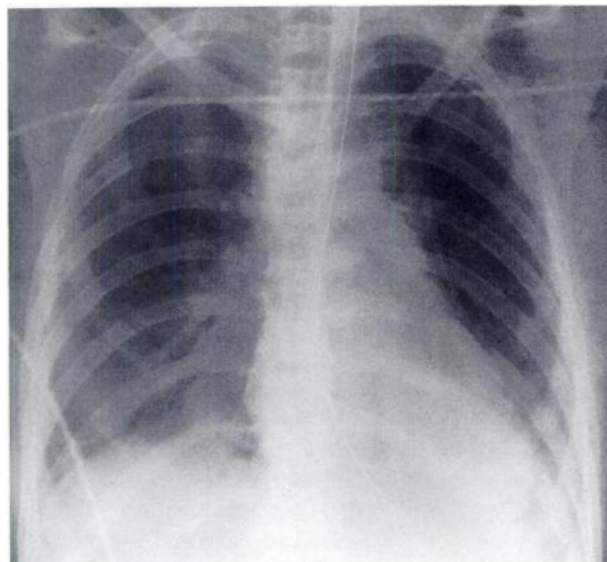
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### Hyperperfusion of a Lower-Lobe Pneumonia by Positive Pressure Ventilatory Support

**TO THE EDITOR:** Kim and Heyman (1) recently discussed an unusual scintigraphic pattern of ventilatory/perfusion (V/Q) mismatch in a 4-mo-old infant on positive end-expiratory pressure (PEEP) mechanical ventilatory support. Blood flow was redistributed away from the well-ventilated upper lungs to the underventilated atelectatic lower lobes. They attributed this redistribution directly to PEEP, which expands ventilated lung and increases its resistance to blood flow. These effects are most pronounced in the upper lungs where perfusion pressures normally exceed alveolar pressures only during the peak of the cardiac cycle. Normally, the vascular resistance of the lower lungs is similarly increased by PEEP and the degree of redistribution is limited. However, a large area of poorly ventilated lung that cannot expand may act as a low resistance vascular shunt and allow a significant redistribution of blood flow to occur. Regions of lung that are atelectatic, consolidated, or occluded by an endobronchial obstruction may, thus, become inappropriately hyperperfused with PEEP and appear as an area of reverse V/Q mismatch on perfusion/ventilation scintigraphy with  $Q > V$ . If a large region of lung is involved, an extensive right-to-left shunt may occur resulting in a profound deterioration of respiratory status characterized by a marked hypoxemia and a large Arterial-alveolar oxygen partial pressure gradient (A-a gradient). In this event, the upper lungs will generally be left significantly underperfused by the resulting redistribution of blood flow and may appear on scintigraphy as broad areas of V/Q mismatch with  $V > Q$ .

Regions of reverse V/Q mismatch, per se, are not altogether uncommon and have been previously attributed to a variety of factors including metabolic alkalosis, pulmonary venous and arterial hypertension, paralysis or impairment of the respiratory center, inhalational anesthetics, nitrate vasodilators, mucous plugs, atelectasis, and even lung disease such as COPD, pneumonia and asthma (2-6). However, the association with PEEP described by Kim and Heyman (1) has



**FIGURE 1**  
Chest radiograph findings include ventilatory apparatus, endotracheal and nasogastric tubes, and subcutaneous emphysema at the left shoulder/upper thorax. Neither the descending aorta or left hemidiaphragm are visualized, and infiltrates are seen near the left base, consistent with a diagnosis of left lower lobe pneumonia.

only been previously reported in one adult case (7) and one other infant case (8). Nuclear medicine physicians should be familiar with this pattern of V/Q mismatch and its significance since pulmonary scintigraphic studies may be ordered for ventilatory-dependent patients to rule out pulmonary thromboembolism (PE) as a cause of respiratory deterioration. Fur-



**FIGURE 2**  
Left posterior oblique perfusion image following the administration of [ $^{99m}\text{Tc}$ ]MAA through a patent i.v. catheter in the distal right arm. Clinically, the arm appeared unremarkable without evidence of thrombophlebitis or venous occlusion and the unusual degree of retained venous activity may reflect endothelial damage from prior i.v. medications. The entire left lower lobe appears markedly hyperperfused. Both lungs show some redistribution of activity away from the upper portions toward the bases.

thermore, in many institutions, ventilation scans cannot be performed on ventilator-dependent patients. To implicate PEEP-induced shunting then as a cause of respiratory deterioration, a large region of atelectasis or consolidation on chest radiograph must be found that is relatively hyperperfused on scintigraphy. To increase awareness of possible deleterious PEEP effects when regional lung disease is present and to confirm the generality of these limited previous reports, we offer a recent adult case from our institution.

A previously healthy 18-yr-old female became lethargic after a motor vehicle accident and was intubated before arriving at the hospital. Following surgical evacuation of a left temporoparietal subdural hematoma, she remained comatose on ventilatory support. After several days, chest radiographs showed evidence of increasing left lower-lobe atelectasis and consolidation. Her blood gases progressively deteriorated with her fraction of inspired oxygen ( $FiO_2$ ) continuously increased to maintain adequate blood oxygenation. On the third day of hospitalization, PEEP was raised to 10 cm  $H_2O$ , and the  $FiO_2$  was raised to 70% with arterial blood gas results pH 7.41,  $pCO_2$  33 Torr,  $PO_2$  56 torr, and an A-a gradient of ~400 Torr. A portable chest radiograph (Fig. 1) at that time shows the degree of left lower-lobe involvement.

Since the patient was at high risk, a perfusion scan was ordered to rule out PE as the cause of hypoxemia and increased A-a gradient. No evidence of PE was found, but the entire left lower lobe appeared markedly hyperperfused and there was some redistribution of perfusion away from the upper portions of both lungs (Fig. 2). After discussions with the pulmonary consultation, PEEP was lowered to 2.5 cm  $H_2O$  with a subsequent improvement in respiratory status. A left lower-lobe pneumonia was confirmed by identification of *E. Coli* in sputum cultures, and serial chest radiographs showed improvement after several days on appropriate i.v. antibiotic coverage.

Apparently, the pneumonia effectively prevented regional alveolar expansion under PEEP. The vascular resistance of the left lower lobe then remained inappropriately low and the patient's respiratory status deteriorated as blood was shunted through this large underventilated area. This condition subsequently corrected as PEEP was lowered and the pneumonia was treated.

Although ordered to rule out PE, perfusion scintigraphy played an important role in diagnosing an alternative correctable underlying cause of respiratory deterioration. This case confirms the generality of findings previously reported by Kim and Heyman (1) and others (7,8) when PEEP-induced regional shunting occurs; namely, the appearance of a marked reverse V/Q defect at the site of the compromised lower lung and some broad V/Q mismatch involving both upper lungs. Besides PE, then, the possibility of PEEP-induced abnormalities should also be considered when ventilatory-dependent patients undergo pulmonary scintigraphy.

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**REPLY:** We are pleased to see the case of relative hyperperfusion caused by PEEP to the area of lobar pneumonia reported by Wegener, and that his findings are essentially in agreement with findings described by us and others (1-3).

Some points need further clarification:

1. In the first paragraph of the above letter, it appears that Wegener mistook a part of our discussion. The statement "Alveolar pressure exceeds pulmonary arterial and venous pressure in the uppermost part of the upright lung, resulting in the collapse of the capillaries, and therefore blood flow occurring only at the peaks of the pulsatile pressure wave in this zone (4-6)" was made by us in order to explain physiologic uneven regional distribution of pulmonary perfusion in normal persons (upper lung < lower lung) (7-9), so that the understanding of the mechanism of diminished perfusion in normally ventilated (therefore more inflated iatrogenically by PEEP), nonatelectatic upper and middle lobes in our case, could be facilitated. We did not intend to say that PEEP effect was more pronounced normally in the upper lungs.
2. If we are not mistaken, Wegener implies in the last sentence of the first paragraph of his letter that inappropriate hyperperfusion in the atelectatic or consolidated lobe due to low vascular resistance is the primary event, and hypoperfusion in the remainder of the lung is the result of a steal phenomenon.

Alveolar pressure in areas of poorly ventilated lung that cannot expand would be lower than usual, which may result in lower vascular resistance and increased blood flow. On the other hand, vascular resistance in this area is increased secondary to a local hypoxic reflex (10,11). The fact that perfusion is generally diminished on scintigram to a variable degree in areas of poorly ventilated lung (atelectasis or pneumonia), indicates that the degree of increased vascular resistance due to a hypoxic reflex generally exceeds that of decreased vascular resistance offered by decreased alveolar pressure.

The effect of PEEP is not that of directly lowering the vascular resistance in nonventilated areas, but of increasing alveolar pressure and vascular resistance in areas of normally ventilated lung. The consequence is "relatively" lower vascular resistance in nonventilated areas, and increased shunt fraction through this zone.