RESTRICTIVE LUNG DISORDERS

Restrictive lung disorders can also significantly alter lung volumes and capacities in a characteristic manner (see table 1 and fig. 1).

Table 1 Manifestations of Restrictive Lung Disorders

Decreased tidal volume (V_T)
Decreased residual volume (RV)
Decreased functional residual capacity (FRC)
Normal residual volume/total lung capacity ratio (RV/TLC)
Decreased total lung capacity (TLC)
Decreased vital capacity (VC)
Decreased inspiratory capacity (IC)
Decreased expiratory reserve volume (ERV)

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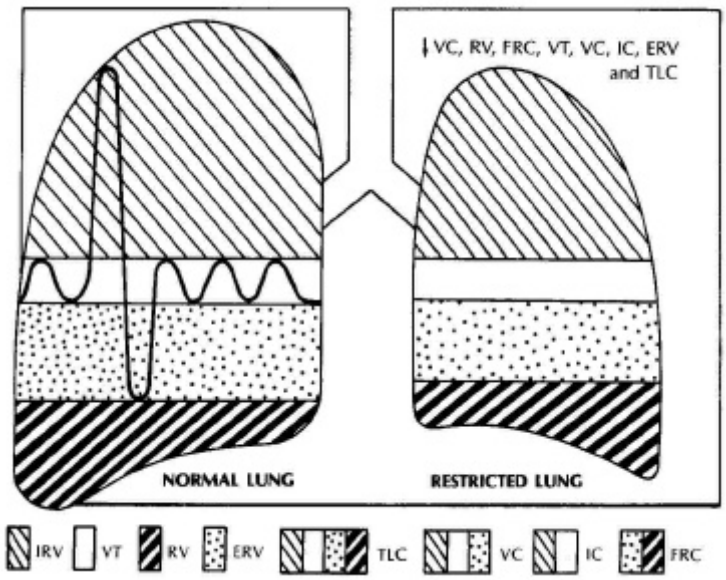

Figure 1 lung volumes and capacities typical of restrictive lung disorders

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Restrictive disorders can manifest certain changes in the anatomic structures in the alveoli and surrounding lung parenchyma.

- lung compression (e.g. secondary to kyphoscoliosis or pleural effusion)
- atelectasis (e.g. secondary to pneumothorax or flail chest)
- consolidation (e.g. pneumonia)
- calcification (e.g. tuberculosis or asbestosis)

- fibrosis (e.g. pneumoconiosis or chronic interstitial lung disease)
- bronchogenic tumor (e.g. squamous cell carcinoma)
- caseous tubercles (e.g. tuberculosis)
- cavitations (e.g. tuberculosis or lung abscess)

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VENTILATORY STRATEGIES

Ventilatory strategy in disorders of low lung compliance are aimed at recruiting collapsed lung, maintaining FRC, and improving oxygenation, while trying to decrease the risk of barotrauma.

PRESSURE CONTROL

Pressure control has been shown to increase oxygenation, improve gas exchange, reduce peak inspiratory pressures, and increase the mean airway pressure. Oxygenation often improves with the use of this mode, therefore it is used frequently with lung diseases manifesting with hypoxemia and reduced lung compliance.

When a patient on volume control begins to develop plateau pressures approaching 35 cm H_20 (the distending pressure above which volutrauma is thought to occur), the clinician should consider changing the patient to pressure control mode. Because pressure control limits the pressure in the ventilator circuit to a preset level, it helps to control the amount of alveolar hyperinflation. However, pressure control can be dangerous in that tidal volumes will vary with changes in patient's compliance and resistance. By increasing mean airway pressure, pressure control mode may cause adverse hemodynamic effects.

PEEP

PEEP has become an invaluable tool to increase FRC, improve oxygenation, and recruit collapsed lung in patients with ARDS or other lung disorders of low compliance. There are four mechanisms by which PEEP is expected to improve pulmonary function and gas exchange:

1. **Peep increases FRC**. In alveoli and small airways that are already distended with air, Peep causes further distention. The amount of the distention is based on the compliance of the lung. The application of peep also prevents small airways and alveoli from collapsing on expiration, and therefore increases FRC at end-expiration.

2. **PEEP recruits collapsed alveoli**. Studies have found that some alveoli that have collapsed in ARDS patients are recruitable with the application of PEEP above 10 cm H_20. This can dramatically improve the compliance of the lung.

3. **Redistribution of extravascular lung water**. Some studies have shown that in animal models, lungs flooded with water can be recruited and oxygenation improved by the application of PEEP, without decreasing the total lung water content.

4. **Improved ventilation/perfusion matching**. In acute lung injury, the application of PEEP can decrease shunt, while decreasing the blood flow to areas of high deadspace. Overall, PEEP can improve the V/Q mismatch in patients with acute lung injury or ARDS.

When ventilating a patient with ARDS, it is helpful to look at a pressure volume curve (see fig. 2).

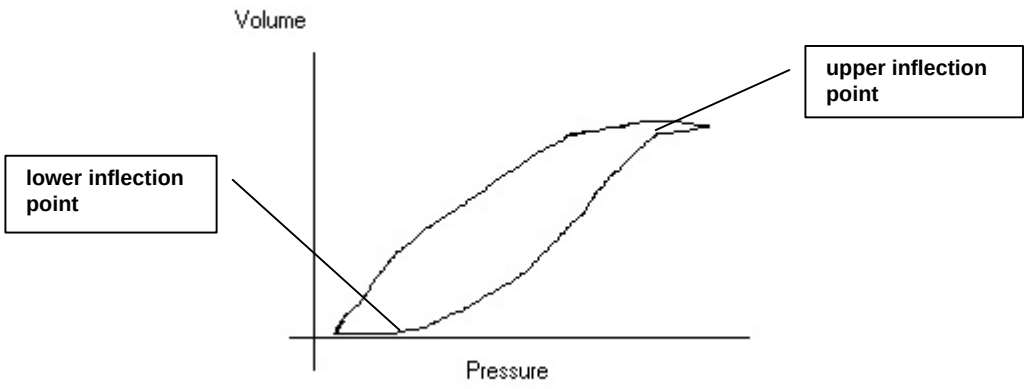

Figure 2 pressure-volume curve at inadequate PEEP

When the lung has low compliance, alveolar consolidation, and alveolar collapse as it does with ARDS, you can see at the bottom of the curve that it requires a large change in pressure to begin opening up the alveoli and causing a volume change at the beginning of inspiration. The point at which the lung begins to open is called the *inflection point*. At the end of the inspiration, there is an upper inflection point, past which an increase in pressure does not result in a further increase in volume. This represents the point at which the lung is being overinflated, and alveolar damage can occur. When the patient exhales, the lung recollapses, refloods, and the cycle begins again. When the lungs collapse and re-expand with each breath, it creates *shear stresses*, which can further perpetuate lung injury.

In studies done by Gattinoni et al in 1987, it was demonstrated by looking at the lungs of ARDS patients in CT scan that ARDS is a heterozygous disease, and parts of the lung are healthy, while other parts are non-recruitable. This leaves some of the lung available for recruitment by PEEP. By applying PEEP to expand the collapsed alveoli, the pressure-volume curve will shift, moving the curve above its lower inflection point and increasing pulmonary compliance **(see fig. 3).**

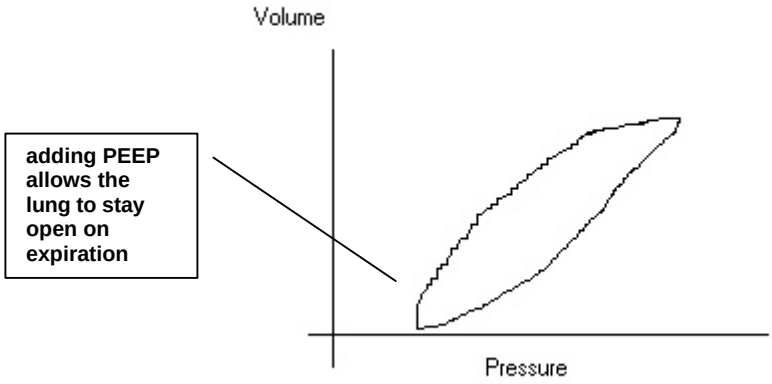

Figure 3 loss of inflection points

Ventilating a patient above the inflection point and preventing the lungs from collapsing and re-expanding with each breath has been demonstrated in several studies to protect the lung in ARDS from further injury.

The technique of using high PEEP to ventilate the non-compliant lung needs to be done with very close monitoring of ventilatory waveforms by an experienced technician.

INVERSE RATIO VENTILATION

Another method to improve oxygenation in patients with ARDS is to increase mean airway pressure. Mean airway pressure is the average pressure throughout the respiratory cycle (inspiration and expiration). Increasing the mean airway pressures without overdistending the alveoli can be done using two different methods. Each describes a method used to limit peak inspiratory and alveolar pressures while increasing the mean airway pressure and improving oxygenation.

The first is called pressure-controlled inverse ratio ventilation (PCIRV). In this method, pressure control is the chosen mode used for ventilation. This means that pressure is held constant throughout the breath, the flow waveform is variable and decreasing, and the inspiratory time is limited by time. The decelerating flow waveform may in itself recruit lung and improve oxygenation. An inverse ratio can be accomplished by prolonging the set inspiratory time for longer than the expiratory time.

Volume-controlled inverse ratio ventilation (VCIRV) accomplishes the same end by using slow inspiratory flowrates, an inspiratory pause, or a descending flow waveform with long inspiratory times.

The advantages and disadvantages in PCIRV vs. VCIRV are compared in table 2.

Table 1 VCIRV vs. PCIRV

	VCIRV	*PCIRV*
Advantages	Available on all ventilators	Peak distending pressures precisely controlled
	Guaranteed minute ventilation	Some patients may tolerate without deep sedation
	Precise control of flow pattern	Larger experience published in the literature
	Familiar mode to most clinicians	
	Peak inspiratory flow lower than PCIRV	
Disadvantage	Peak alveolar pressures can vary; pressures must be monitored carefully	Tidal volume varies with changing respiratory mechanics; minute ventilation must be monitored carefully
	Deep sedation usually necessary to prevent dysynchronous breathing	Not available on all ventilators
		Not a familiar mode to many clinicians
		? Greater shear forces

Hazards of inverse ratio ventilation include all of those that are inherent to mechanical ventilation, with an emphasis on the development of autopeep. Inverse ratio ventilation can risk the development of autopeep; as the ratio of inspiration to expiration becomes smaller, air trapping can result when the patient has less time to exhale.

PRONE POSITION

Turning a patient with ARDS into the prone position can dramatically increase their oxygenation. Recent studies have shown that 50-75% of patients will have some improvement in oxygenation, and the degree of improvement is usually enough to allow a reduction in PEEP or FiO_2.

Prone position is thought to have an effect on oxygenation due to improvement in ventilation in the dorsal lung regions, while perfusion is only marginally altered. This could be explained due to the weight and size of the heart, the extent to which the abdominal pressure affects the dependent lung units, the relative relationship between the front and back chest wall compliance, and enhanced drainage of airway fluid when patients are turned prone.

Hazards of prone position include difficulty in nursing care, possible time delay if the patient needs cardiopulmonary resuscitation, pressure necrosis from poorly padded and supported bony regions, and increased risk of accidental extubation.