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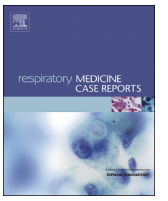

Case Report

Unexpected pulmonary mechanics during positive pressure mechanical ventilation in fibrotic lung disease with concomitant flail chest

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ABSTRACT

Understanding of pulmonary mechanics is essential to understanding mechanical ventilation. Typically, clinicians are mindful of peak and plateau pressures displayed on the ventilator and lung compliance, which is decreased in lung disease such as idiopathic pulmonary fibrosis (IPF). Decreased lung compliance leads to elevated peak and plateau pressures. We present a patient with IPF undergoing mechanical ventilation after cardiac arrest. Despite low lung compliance, he had normal peak and plateau pressures due to the presence of flail chest and increased chest wall compliance. This case highlights the role chest wall compliance plays in total respiratory system compliance and pulmonary mechanics.

Abbreviations

CAD	Coronary artery disease
C_{CW}	Chest wall compliance
C_L	Lung compliance
CPR	cardiopulmonary resuscitation
C_{RS}	Respiratory system compliance
CT	Computed tomography
IPF	Idiopathic pulmonary fibrosis
HFNC	High flow nasal cannula
UIP	Usual interstitial pneumonia
VC	Volume control
ROSC	Return of spontaneous circulation
FiO2	Fraction of inspired oxygen
Vt	Tidal volume
PEEP	Positive end-expiratory pressure
P_{plat}	Plateau pressure

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P_{peak}	Peak pressure

1. Introduction

Understanding of pulmonary mechanics is essential in understanding mechanical ventilation, particularly in those with underlying lung disease. In addition to ventilator settings, clinicians are typically mindful of peak pressure (P_{peak}), plateau pressure (P_{plat}), and lung compliance (C_L). In severe lung disease, such as idiopathic pulmonary fibrosis (IPF), C_L is typically reduced [1]. As compliance is inversely related to pressure, decrease in C_L typically results in elevated P_{plat} and P_{peak} [2]. However, this relationship does not take into account the role of chest wall compliance (C_{CW}) in total respiratory system compliance (C_{RS}) and pulmonary mechanics. We present a case of a patient with severe IPF undergoing mechanical ventilation after cardiac arrest who had normal peak and plateau pressures in the setting of flail chest.

2. Case description

A man in his 70's with idiopathic pulmonary fibrosis (IPF), pulmonary hypertension, and coronary artery disease (CAD) presented with worsening dyspnea and acute on chronic hypoxemic respiratory failure. His chronic home oxygen requirement was 6 L/min, but upon admission he required high-flow nasal cannula (HFNC) 30LPM with 100% FiO2. Aside from worsening dyspnea, he had no other localizing symptoms. His worsening hypoxemia was deemed to be due to his end-stage fibrotic lung disease. A computed tomography (CT) scan of his chest compared to a previous CT showed stable, severe usual interstitial pneumonia (UIP) pattern consistent with his IPF (Fig. 1). While hospitalized, he experienced asystole cardiac arrest in the setting of worsening hypoxemia. He underwent cardiopulmonary resuscitation (CPR) with return of spontaneous circulation (ROSC) at which point he was intubated and placed on positive pressure mechanical ventilation. The ventilator was set to volume control (VC)+ with a respiratory rate of 20 breaths/minute, tidal volume (Vt) of 480 mL, inspiratory time of 0.8 seconds, fraction of inspired oxygen (FiO_2) 100%, and positive end-expiratory pressure (PEEP) of 7.5 cm H_2O. The ventilator recorded a delivered Vt of 960 mL. Despite his severe pulmonary fibrosis, this high Vt only generated a peak pressure (P_{peak}) of 18 cm H_2O as captured by the ventilator (Fig. 2), while plateau pressure was 16 cm H_2O.

He was noted to have a sternal flail chest breathing pattern, with paradoxical movement of the chest wall inwards with inspiration (Video 1). Inward sternum displacement is seen, indicating that the patient was triggering the breath and causing a decrease in intrapleural pressure. Ventilator adjustments were trialed, with all showing normal peak pressure on the ventilator (Fig. 3). His low pressure readings were likely due to his sternal flail chest and increased C_{CW} counteracting his severe pulmonary fibrosis and decreased C_L. Unfortunately, his clinical status worsened. Before we could attempt chest binding or potentially consult thoracic surgery for repair of sternal fracture in order to restore normal compliance, the patient's family elected to transition to comfort-directed care.

Supplementary video related to this article can be found at https://doi.org/10.1016/j.rmcr.2022.101802

3. Discussion

3.1. Imaging discussion

The patient had known history of IPF, with severe fibrotic changes seen on his CT chest. A repeat CT chest on presentation did not show any acute changes, such as new groundglass opacities or consolidations to suggest a new infectious, inflammatory, or pulmonary process to explain worsening hypoxemia. The extent of fibrosis on his CT chest led us to believe his lung compliance was greatly reduced.

3.2. Clinical discussion

We reasoned that his low pressure readings were due to his sternal flail chest counteracting his severe pulmonary fibrosis. Given reduced chest wall resistance and likely increased chest wall compliance, he would continue to have low pressure readings regardless of what tidal volume or PEEP the ventilator was set to give.

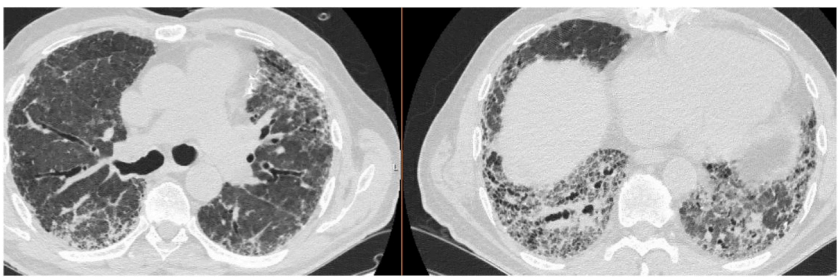

Fig. 1. Patient CT Chest.
CT chest showing usual interstitial pneumonia (UIP) pattern with subpleural reticular changes, traction bronchiectasis, and basilar honeycombing.

Fig. 2. Initial Ventilator Settings and Peak Pressure.
The ventilator is set to volume control (VC) + with a tidal volume of 480 mL. The patient is receiving 960 mL as seen at the top of the ventilator screen. Despite receiving this high tidal volume, peak pressure is normal at 18 cm H_2O.

In lung fibrosis, such as in IPF, the normal, compliant lung extracellular matrix composed of elastin is replaced by a fibrillar collagen-rich matrix. Affected alveolar areas create the UIP pattern, which is characterized by alternating areas of lesions and normal lung, fibroblastic foci interspersed in fibrotic areas, and honeycomb lesions in which airway walls are composed of fibrotic tissue. Alterations in surfactant composition or metabolism may also play a role in IPF pathogenesis, with impaired function seen in IPF [1].

Lung compliance (C_L) is defined as the change in lung volume divided by the change in transpulmonary pressure and describes the ability of the lung to expand. Normal C_L is approximately 200 mL/cm H_2O [2]. During full mechanical ventilatory support, C_L is a component of respiratory system compliance (C_{RS}), and plateau pressure (P_{plat}) is inversely related to C_{RS}. P_{plat}, as measured on a mechanical ventilator, approximates alveolar pressure and is a large component of P_{peak}, with the difference between the two attributable to resistance and flow in the airways and the mechanical ventilator circuit [2]. In IPF, the pathophysiological changes seen in the lung extracellular matrix and in pulmonary surfactant drives a reduction in C_L that is seen early in the disease process and worsens with disease progression [1]. Such decrease in C_L would subsequently decrease C_{RS} and result in increased P_{plat} and P_{peak}. A study of lung mechanics in patients with end-stage IPF receiving mechanical ventilation demonstrated such a relationship between C_{RS} and airway pressures [3].

Acceptable C_{RS} during mechanical ventilation is 50–100 mL/cm H_2O [2]. Both C_L and chest wall compliance (C_{CW}) function to compose total C_{RS}. Independently, C_L and C_{CW} have higher compliance than the lung-chest wall system, or C_{RS}. C_L opposes the outward pull of C_{CW} to create the net C_{RS}. The C_{RS} also factors in the opposing force of the chest wall muscles and diaphragm. These muscles provide the necessary pressure difference for air movement [4]. The opposing nature of C_L and C_{CW} in determining C_{RS} is quantified by the following equation: $1/C_{RS} = 1/C_W + 1/C_L$ [2]. If C_{RS} is constant, an increase in C_{CW} will result in subsequent decrease in C_L, and vice versa. If C_L is constant, an increase in C_{CW} results in increased C_{RS} and a decrease in C_{CW} results in decreased C_{RS}.

As discussed above, C_{RS} and P_{plat} have an inverse relationship, with P_{plat} serving as an approximation of alveolar pressure and a component of P_{peak}. This relationship is quantified by the following equation: $C_{RS} = Vt/(P_{plat} - PEEP)$ [2]. At a constant tidal volume, a change C_{RS} corresponds with an inverse change in pressure. It follows that changes in C_{CW} would have a similar relationship with an effect on pulmonary pressures.

Chest wall expansion occurs through two mechanisms. At low lung volumes, the elastic components of the chest wall promote passive outward recoil. At high lung volumes, the inspiratory muscles of the chest wall contract, promoting chest wall expansion and outward abdominal displacement. These changes during inspiration accommodate increases in lung volume [5]. In normal, non-pathologic states, the elastic components of the chest wall stretch easily, giving a relatively high C_{CW}. Normal C_{CW} is approximately 200 mL/cm H_2O, similar to that of C_L [2,5].

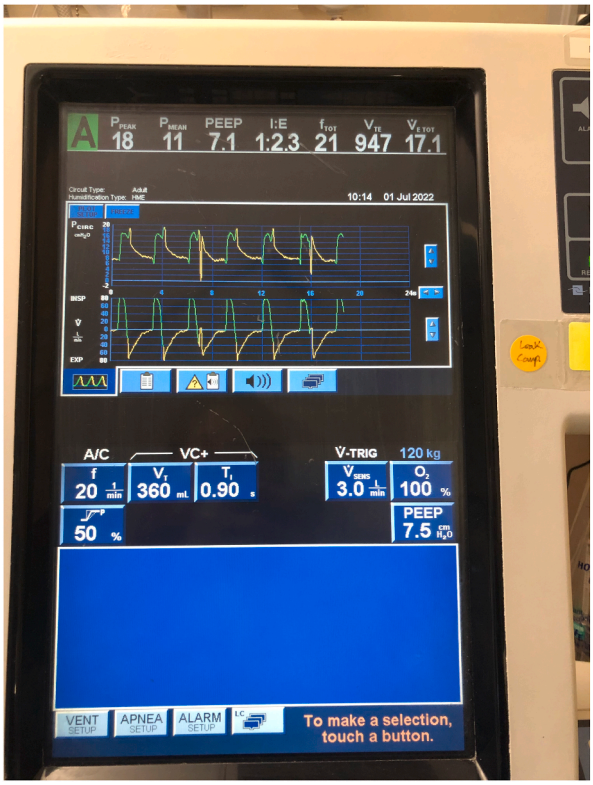

Fig. 3. Adjusted Ventilator Settings and Effect on Peak Pressure.
The ventilator was adjusted to a lower tidal volume. However, the patient still received a significantly higher tidal volume. Peak pressure remained low at 18 cm H_2O.

There are numerous conditions that affect chest wall compliance. Several diseases, such as obesity and kyphoscoliosis, cause chest wall stiffening and decrease in its compliance [5]. As a result, C_{RS} would decrease as well, requiring higher levels of intrathoracic pressure to allow for sufficient lung expansion and concomitant chest wall expansion. This can lead to hyperinflation, increased ventilatory workload on the respiratory muscles, and increased risk of barotrauma [6]. Flail chest is unique in that C_{CW} is actually decreased due to impaired integrity and loss of resistance of the chest wall [2].

Flail chest occurs when chest wall injuries cause the associated portion of the chest wall to move independently of the rest of the chest wall. In flail chest, the continuity of the chest wall is disrupted, leading to paradoxical motion of the flail segment of the chest wall; the flail segment will move inwards during inspiration while the rest of the chest wall moves outward [7]. Sternal fractures (anterior) and rib fractures (lateral) can both result in flail chest (Fig. 4).

Flail chest typically occurs in the setting of blunt trauma; in this case, it occurred in the setting of CPR. Chest compliance increases significantly and chest stiffness decreases over the course of CPR, with changes seen as quickly as 5 min into CPR. Increased C_{Cw} in this setting is at least in part due to the development of thoracic fractures, both rib and sternal, as well as due to alternating compression and decompression [8,9].

4. Conclusion

- The fibrotic changes and changes in surfactant production seen in IPF drastically decrease lung compliance in lung fibrosis; given the inverse relationship between compliance and pressure, pulmonary pressures (P_{plat} and P_{peak}) typically would be elevated.
- Chest wall compliance and lung compliance oppose each other to produce a net respiratory system compliance. In the setting of constant lung compliance, increase or decrease in chest wall compliance will have a respective increase or decrease in respiratory system compliance and the inverse effect on peak and plateau pressures.
- Flail chest results in the disruption of the chest wall continuity and in turn leads to increased chest wall compliance. This results in increased respiratory system compliance and decreased pulmonary pressures, given the inverse relationship between compliance and pressure.

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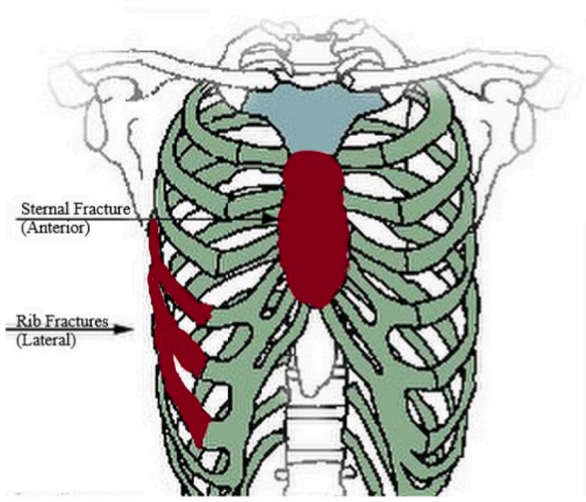

Fig. 4. Patterns of Flail Chest.
Both sternal fractures and multiple rib fractures can lead to the flail chest breathing pattern. Sternal fractures lead to an anterior flail chest breathing pattern, while rib fractures can lead to a lateral flail chest breathing pattern.

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Declaration of competing interest

None.

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