Predicting the Haemodynamic Response to Prone Positioning: A Novel and Simultaneous Analysis of the Guyton and Rahn Diagrams

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Graphical models of physiology are heuristically appealing as they facilitate qualitative conclusions at the bedside of the critically ill. Historically, the Rahn diagram has portrayed the physiology of the lungs, chest wall and respiratory system, while the Guyton diagram has illustrated cardiovascular physiology. As contemporary methods of haemodynamic monitoring, and their predecessors, are inexorably influenced by the interaction between the respiratory and cardiovascular systems, a qualitative graphical model for mechanical heart-lung interaction holds clinical utility. This short report will present an illustrative clinical case, briefly review the physiological underpinnings of the Rahn and Guyton Diagrams and then introduce a novel combination thereof. It is expected that this new diagram will clarify the case at hand, as well as facilitate the transfer of cardio-respiratory theory to clinical practice.

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Introduction

When Arthur Guyton combined the venous return and cardiac function curves onto a single graph, he created a compelling qualitative construct for physiologists and clinicians alike [1, 2]. His representation has aided our understanding of cardiovascular physiology for decades but largely neglects the intimate relationship between the cardiovascular system and the thoracic pump. Whereas Guyton correctly predicted that the cardiac function and venous return curves would be altered by intra-thoracic pressure (ITP) [3], his diagram is typically presented independent of the effects of changing ITP and lung volume.

Appreciation for the mechanical relationship between the heart and lungs has been especially prominent since the introduction of the flow-directed pulmonary artery catheter (PAC) in the early 1970s. Following widespread adoption of the PAC, it became apparent that its measured variables are significantly affected by mechanical heart-lung interaction and that the physiological linkage between the respiratory and cardiovascular systems can undermine both acquisition and interpretation of haemodynamic data [4, 5]. While attempts were made to correct these deficiencies [6, 7], the use of the PAC has waned significantly to the point where it was recently eulogized [8].

Newer, non-invasive methods of haemodynamic monitoring have largely taken the place of the PAC in modern intensive care units (ICUs) [9]. Simultaneously, functional haemodynamic monitoring (FHM) is gaining traction amongst intensivists [10]. FHM assays the functional state of the cardiovascular system by measuring a haemodynamic response (e.g. SVC calibre change) to a defined stressor (e.g. a change in ITP) [11]. Nevertheless, these contemporary means of haemodynamic assessment still obey fundamental principles of cardio-respiratory physiology. Thus, comprehension of the immediate interdependence of the heart and lungs within the confines of the thorax continues to be of paramount importance.

Illustrative Case

A 70 year old woman with a body mass index (BMI) of 39, severe chronic bronchitis and profound aspiration pneumonia complicated by acute respiratory distress syndrome (ARDS), is becoming progressively more hypoxaemic. Her partial

pressure of oxygen to fraction of inspired oxygen (P/F) ratio is less than 100 mmHg. She is heavily sedated and pharmacologically paralysed. Her plateau pressure (Pplat) is 37 cm H_20 and her oesophageal pressure (Poes) is 17 cm H_2O. Her mean arterial pressure (MAP) is 60 mmHg and her urine output has decreased during the last hour. Transoesophageal echocardiography (TOE) is carried out to help predict fluid responsiveness. In the supine position her superior vena cava (SVC) is noted to be engorged and unvarying with mechanical inspiration. A decision is made to place her in the prone position. Following this, her Pplat remains 37 cm H_2O, her Poes falls to 15 cm H_2O and P/F ratio rises to 150 mmHg. Importantly, her MAP increases slightly and her SVC now collapses with mechanical inspiration. Noting the change in SVC calibre, 500 ml of balanced crystalloid is infused; the patient's MAP increases further and urine output normalizes.

Clinical Overview

Turning a paralysed patient into the prone position may change both the pulmonary and chest wall compliances. The combined compliance of the lungs and the chest wall, the respiratory system compliance (Crs), determines the end-inspiratory alveolar pressure, commonly assessed on the ventilator as the plateau pressure. Assuming that the plateau pressure approximates end-inspiratory alveolar pressure (PAlv) and that the oesophageal pressure estimates intra-thoracic (or pleural) pressure, the pressure across the alveolus, called the trans-pulmonary pressure (Ptp), can be assessed at the bedside as the difference between these i.e. Pplat – Poes.

Consequently, application of the prone position in this patient resulted in an apparent fall in intra-thoracic (pleural) pressure (decreased Poes) with a contemporaneous rise in the trans-pulmonary pressure as the difference between Pplat and Poes increased.

Ptp (supine) = Pplat – Poes = [37 – 17] = 20 cm H_2O

Ptp (prone) = Pplat – Poes = [37 – 15] = 22 cm H_2O

However, it is important to note that the oesophageal pressure in the supine position tends to over-estimate the intra-thoracic (pleural) pressure due to mediastinal compression of the oesophagus. In healthy individuals, the compression-induced rise in oesophageal pressure may be as much as 40% [12]. It is also likely that in the critically ill ARDS patient the dense, oedematous lung and cardiomegaly from volume infusion may compress and increase the oesophageal pressure by an even greater degree [13]. Lastly, it has been known for decades that the oesophageal pressure in the upright, lateral and prone positions are all similar in value, reinforcing the impact of the mediastinum on oesophageal pressure in the supine position [14]. It is therefore reasonable to correct the oesophageal pressure measured in the supine position to more accurately reflect intra-thoracic pressure. A reduction in the measured supine Poes of approximately 40%, based on the data described above, will give a new 'corrected' supine oesophageal pressure in this patient of 10 cm H_2O i.e. an intra-thoracic (pleural) pressure (Ppl) of 10 cm H_2O. Using this corrected intra-thoracic (pleural) pressure we can insert the 'new' value for supine trans-pulmonary pressure (Ptp) as follows:

Ptp (supine) = Pplat – Poes = [37 – 10] = 27 cm H_2O

Ptp (prone) = Pplat – Poes = [37 – 15] = 22 cm H_2O

Taking the aforementioned corrections into consideration, on assuming the prone position the respective haemodynamic impacts upon the right ventricle (RV) are to:

1. diminish its venous return – as the pressure within the thorax rises relative to the body (i.e. the intra-thoracic pressure Ppl increased from 10 cm H_2O to 15 cm H_2O),

2. augment RV forward flow – as the fall in trans-pulmonary pressure (from 27 cm H_2O to 22 cm H_2O) reduces RV afterload.

In aggregate, therefore, the cardiac output is slightly increased and the RV becomes more preload responsive.

The Physiology and Novel Qualitative Diagram

The Rahn Diagram

A qualitative analysis of respiratory mechanics can be gleaned from the Rahn diagram, which simultaneously depicts the compliances of the passive chest wall, lungs and respiratory system (see Figure 1) [15].

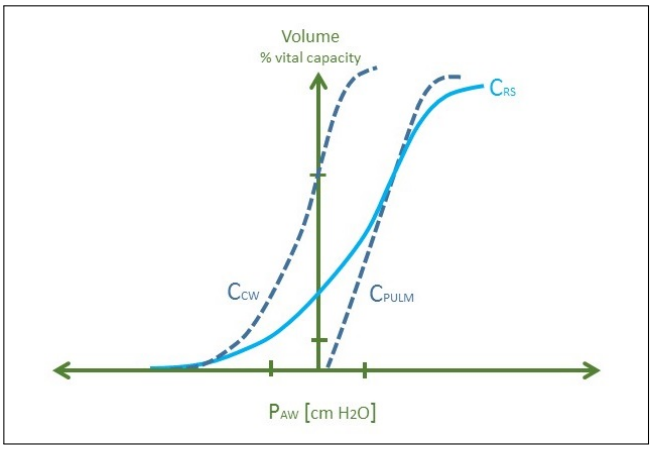

Figure 1. The Rahn diagram. The x-axis is airway pressure and the y-axis is the lung volume. The pulmonary and chest wall compliance curves (Cpulm and Ccw respectively) are depicted in dashed navy lines. The Crs curve in sky blue represents the summative compliance of the respiratory system.

Importantly, in the patient fully passive with the ventilator, the intra-thoracic pressure (or pleural pressure) follows the chest wall compliance curve as this pressure is the surface pressure generated between the lungs and the effortless chest wall [16]. Clinically, this pressure may be estimated by an oesophageal balloon (i.e. the Poes) [17]. Considering the patient while supine (see Figure 2), a given ventilator-delivered volume on the y-axis may be parsed into the plateau pressure (from the respiratory system compliance curve), and the pleural pressure (from the obese chest wall compliance curve) [18]; the lateral distance between the plateau pressure (Pplat) and the pleural pressure (Ppl) qualitatively approximates the trans-pulmonary pressure (Ptp) (dashed red line).

When a patient is moved from the supine position to the prone position, the following changes are expected on the Rahn diagram (see Figure 3).

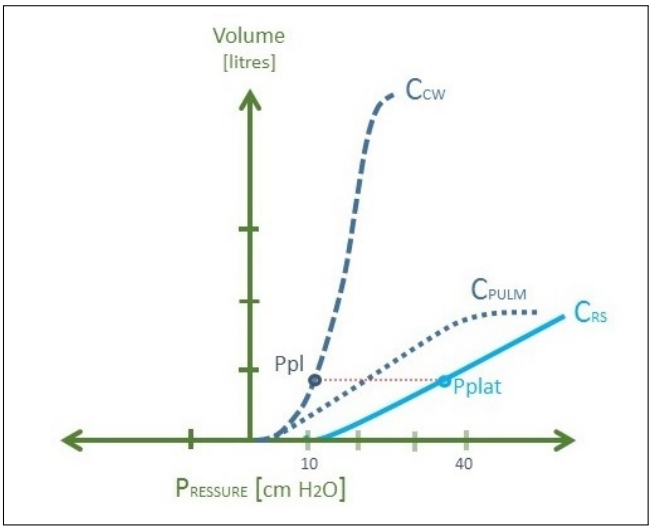

Figure 2. The patient in the supine position. The patient's obesity has caused shift of the chest wall compliance curve to the right. ARDS decreases pulmonary compliance reducing the slope of the Cpulm curve. The combined effect is a reduced Crs. For a given ventilator volume, the plateau pressure Pplat can be estimated from the Crs curve and the pleural pressure can be estimated from the Ccw curve, allowing the trans-pulmonary pressure (red dotted line) to be calculated (27 cm H_2O).

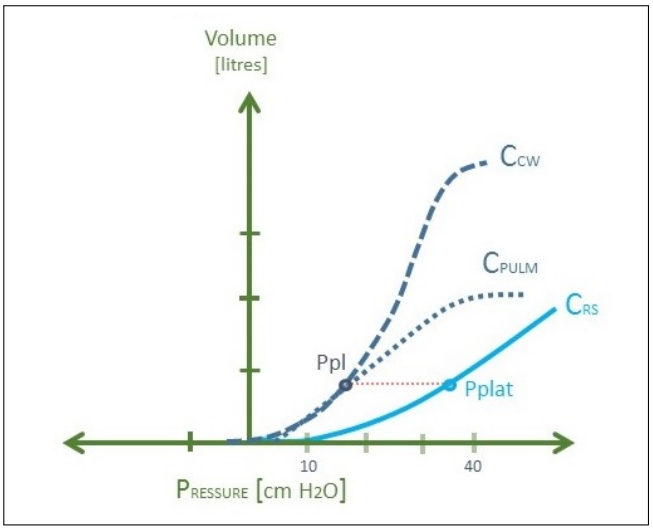

Figure 3. The patient in prone position. Chest wall compliance has fallen further resulting in reduced slope of the Ccw curve. Due to lung recruitment, the slope of the Cpulm has risen. Overall the Crs curve has not changed significantly. For the same ventilator volume, the Pplat (37 cm H_2O) remains the same but the pleural pressure Ppl has risen to 15 cm H_2O. The trans-pulmonary pressure (dashed red line) has therefore fallen and is now 22 cm H_2O.

Firstly, dorsal lung spaces are recruited and apparent pulmonary compliance increases (i.e. an increased slope of the Cpulm curve). Secondly, the chest wall is stiffened on moving to the prone position so its slope falls. Since pulmonary compliance rises and chest wall compliance falls, the combined respiratory system compliance does not vary (compare Figures 2 & 3). However, trans-pulmonary pressure falls because the pleural pressure rises relative to the plateau pressure. As elaborated below, the haemodynamic consequence of an increase in pleural pressure relative to plateau pressure is a reduction in both RV preload and afterload.

The Guyton Diagram

The venous return curve models blood flow into the thorax. The x-intercept of the venous return curve depicts the mean systemic filling pressure (Pms) [19] which is the equilibrium pressure the circulatory system assumes when blood flow ceases. The mean systemic filling pressure is, essentially, determined by venous blood volume (i.e. 'volume status') and venous tone; it is the upstream pressure for blood flow towards the thorax. When the venous return curve is superimposed on cardiac function curve, the Guyton diagram is formed.

Importantly, the relationship between the RV cardiac function curve and the SVC venous return curve illustrates the concepts of SVC collapse and fluid responsiveness. An inspiratory augmentation of pleural pressure shifts the cardiac function curve rightwards with respect to the venous return curve, because the pressure within the thorax rises relative to the extra-thoracic venous pressure. When the venous return curve intersects the flat portion of the RV cardiac function curve, that is, when a patient is not fluid responsive, the lateral distance between right atrial pressure (Pra, an estimation of the SVC pressure) and the intra-thoracic pressure (the x-intercept of the cardiac function curve) shrinks minimally. The absence of SVC collapse indicates that the patient is on the flat portion of her SVC transmural pressure–volume compliance curve [20]; a marker that the patient is not fluid responsive (see Figure 4).

Conversely, when the venous return curve intersects the ascending portion of the RV cardiac function curve, the patient is fluid responsive and an inspiratory augmentation of intra-thoracic pressure causes the distending pressure of the SVC to

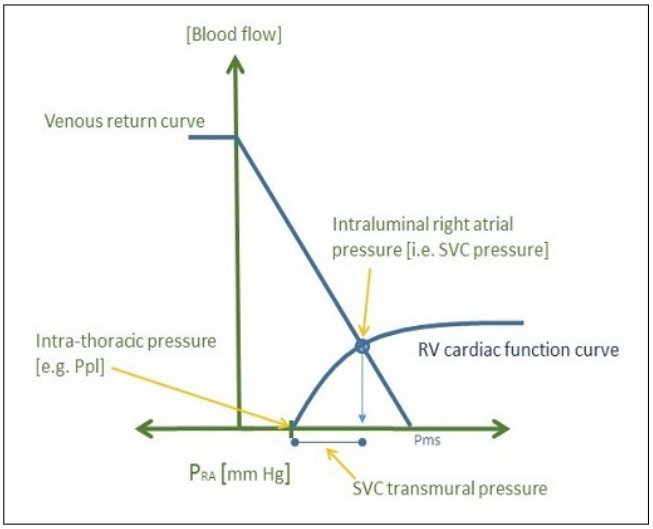

Figure 4. The Guyton diagram for a patient who is not fluid-responsive. The venous return curve intersects the plateau of the cardiac function curve. Cardiac output is on the y-axis and right atrial pressure (or SVC pressure) is on the x-axis. The intra-thoracic pressure (or pleural pressure), is the x-intercept of the cardiac function curve and shifts rightwards in response to a passive mechanical breath. The SVC transmural pressure is indicated at end-inspiration. The SVC transmural pressure is the pressure within the SVC less the intra-thoracic pressure (x-intercept of the cardiac function curve). This graphic is analogous to the patient in the supine position, that is, a mechanical breath results in a relatively small increase in Poes, but a large increase in Ptp and RV afterload. Pms is mean systemic pressure and the x-intercept of the venous return curve.

fall greatly. This collapse is an echocardiographic marker of fluid responsiveness [21] (see Figure 5).

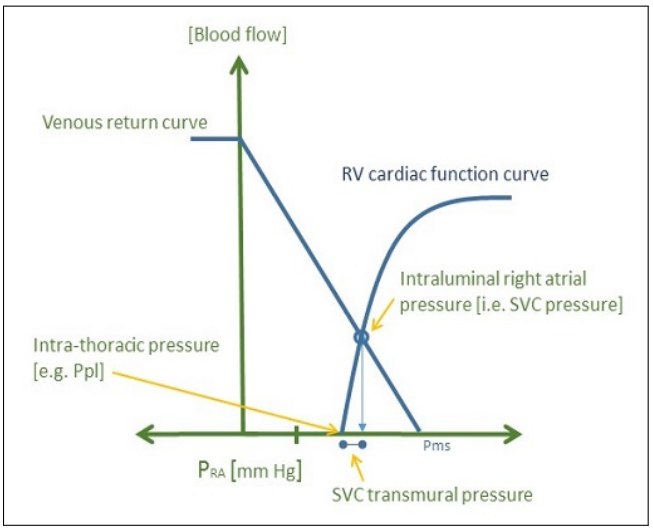

Figure 5. The Guyton diagram for a fluid-responsive patient. The venous return curve intersects the ascending portion of the cardiac function curve, such that provision of fluids will raise cardiac output. The transmural SVC pressure is much smaller, meaning that its inspiratory fall will lead to collapse. This graphic is analogous to the patient in the prone position, that is, a mechanical breath results in a relatively large increase in Poes, but a decrease in Ptp and, therefore, RV afterload. Again, this graphic represents end-inspiration.

Simultaneous Analysis

How does the Guyton analysis relate to the Rahn analysis and how does this inform the aforementioned clinical scenario? The Rahn diagram, described above, may be altered such that ventilator volume is moved to the z-axis (effectively directed 'into the page'); pressure remains on the x-axis. The superimposed Guyton diagram may share pressure on the x-axis while blood flow (cardiac output or venous return) remains on the y-axis. This composite diagram allows for simultaneous analysis of the ventilator-applied volume, Ppl, Pplat, Ptp, cardiac output and SVC transmural pressure.

Figure 6 represents the patient while supine, at end-inspiration. The primary insult is the excessive stress and strain placed upon the 'baby lung' [22] (see also Figure 2) such that Pplat increases much more than Ppl; Ptp therefore rises. Because RV afterload is partly determined by the distending pressure across the alveolus [23], a large Ptp retards RV ejection and the slope of the RV function curve falls.

Additionally, the rightward shift of the cardiac function curve relative to the venous return curve follows the rise in pleural pressure [3]. While supine, there is a comparatively small inspiratory augmentation of Ppl as compared to the stiffened chest wall when prone. In totality, when the patient is supine, mechanical inspiration drives high RV afterload with minimal reduction in RV preload such that the SVC is non-collapsible, the venous return curve continues to intersect the plateau of the RV function curve and the patient is fluid intolerant.

Figure 7, by contrast, depicts the changes that occur in the prone position. Firstly, the chest wall compliance falls, while the apparent pulmonary compliance improves [24]. Accordingly, the Pplat – derived from the respiratory system compliance curve – changes very little to the extent that the fall in the chest wall compliance is counterbalanced by the increase in lung recruitment. In contrast to the supine position, the Ppl rises notably relative to the Pplat and so the Ptp shrinks. A fall in the Ptp diminishes RV afterload and therefore steepens the slope of the RV cardiac function curve [25].

Additionally, the fall in chest wall compliance augments Ppl which prominently shifts the RV cardiac function curve relative to the SVC venous return curve. This magnification of end-inspiratory pressure within the thorax – relative to the extra-thoracic veins – diminishes venous return and potentially impairs stroke volume and hence cardiac output. However, this is coupled with a fall in RV afterload, and this fall may be sufficient to offset the impact of reduced venous return, and for stroke volume and cardiac output (i.e. the y-axis) to rise. Notably, the intersection of the cardiac function and venous return curves at their sloped portions shrinks the SVC transmural pressure. Therefore, on TOE, there is inspiratory collapse and provision of fluid will raise cardiac output further. The impact of these changes in summarised in Figure 8.

Pathophysiology of the Case

How does the prone position alter respiratory mechanics in this patient?

Assuming the prone position caused the patient's chest wall compliance to fall (i.e. her chest wall became 'stiffer'). This increased the intra-thoracic (pleural) pressure present for any given ventilator-delivered inspiratory volume. However, if lung is recruited in the prone position then lung compliance will rise (i.e. the lungs will become less stiff). Because the plateau pressure on the ventilator is the summation of the lung and chest wall in series, the plateau pressure may not change if the increase in lung compliance offsets the fall in chest wall compliance. If the plateau pressure stays the same, but the intra-thoracic pressure rises in the prone position then the net effect is a fall in trans-pulmonary pressure.

Caveats

Importantly, this analysis neglects (for diagrammatic simplicity) changes in the venous return curve in response to both prone position and cyclical mechanical ventilation [26]. In the prone position, pressurization of the abdomen may increase the mean systemic pressure (Pms) [27]. This is the pressure head for venous return to the RV and is the x-intercept of the venous return curve. Additionally, the patient's underlying volume status may alter resistance to venous return. Increased intra-thoracic pressure favours collapse of the great veins on entering the thorax, that is, vascular waterfall physiology [28]. As demonstrated in the IVC, hypervolaemia retards great-vein collapse while the converse is true when the abdomen's venous beds are relatively under-filled [29].

Conclusion

In conclusion, the sedated and paralysed patient placed into the prone position will have changes in chest wall and pulmonary compliance. These changes favour increased intra-thoracic pressure and diminished trans-pulmonary pressure. Consequent reduction in both RV preload and afterload renders the RV fluid-responsive and maintains the cardiac output respectively. Finally, the aforementioned principles are illustrated by a novel combination of two classic, graphical analyses, emphasising the mechanical linkage of the cardiovascular and respiratory systems for the clinical physiologist.

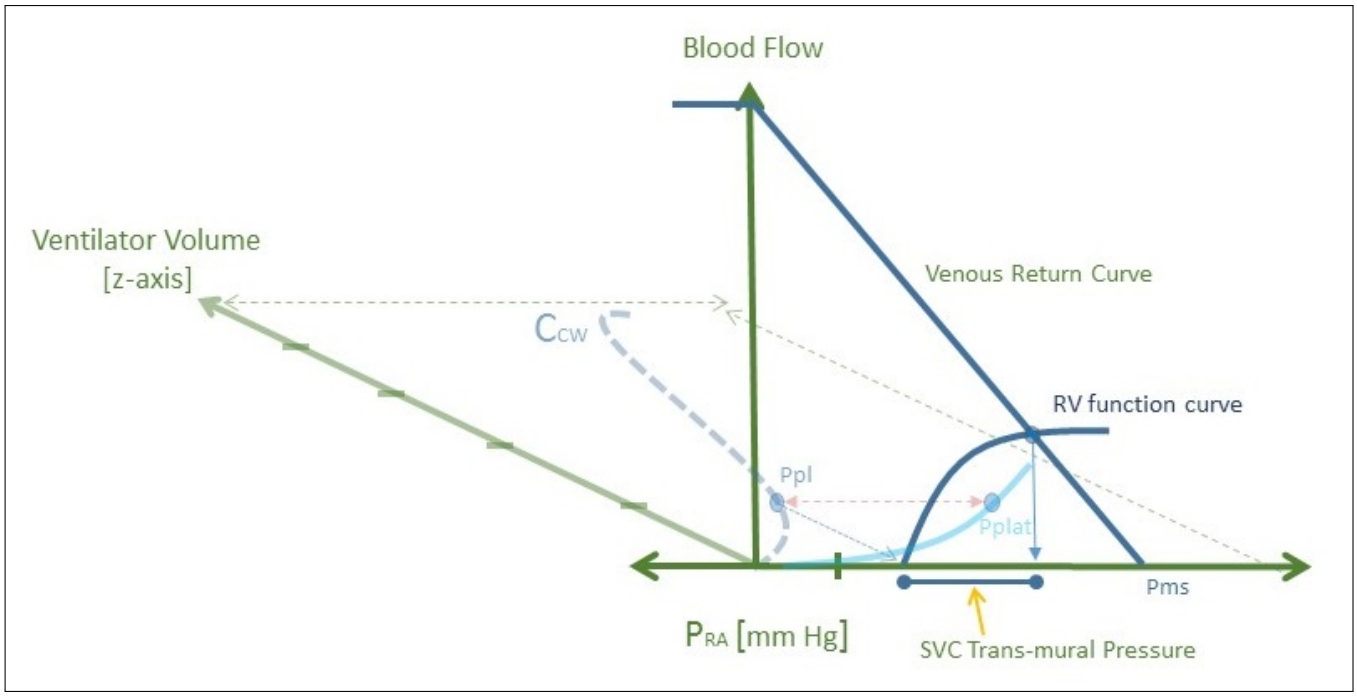

Figure 6. The simultaneous Rahn and Guyton analysis of the supine patient. This graphic represents the system at end-inspiration. The Rahn diagram in the z-axis matches Figure 2. Cpulm is omitted for simplicity. Pplat, Ppl and Ptp (dashed red) are depicted. The RV function curve x-intercept shifts in-step with the Ppl along the Ccw and parallels Figure 4. Because there is a large Ptp (RV afterload) without much fall in preload, the SVC trans-mural pressure remains high and the RV is fluid intolerant.

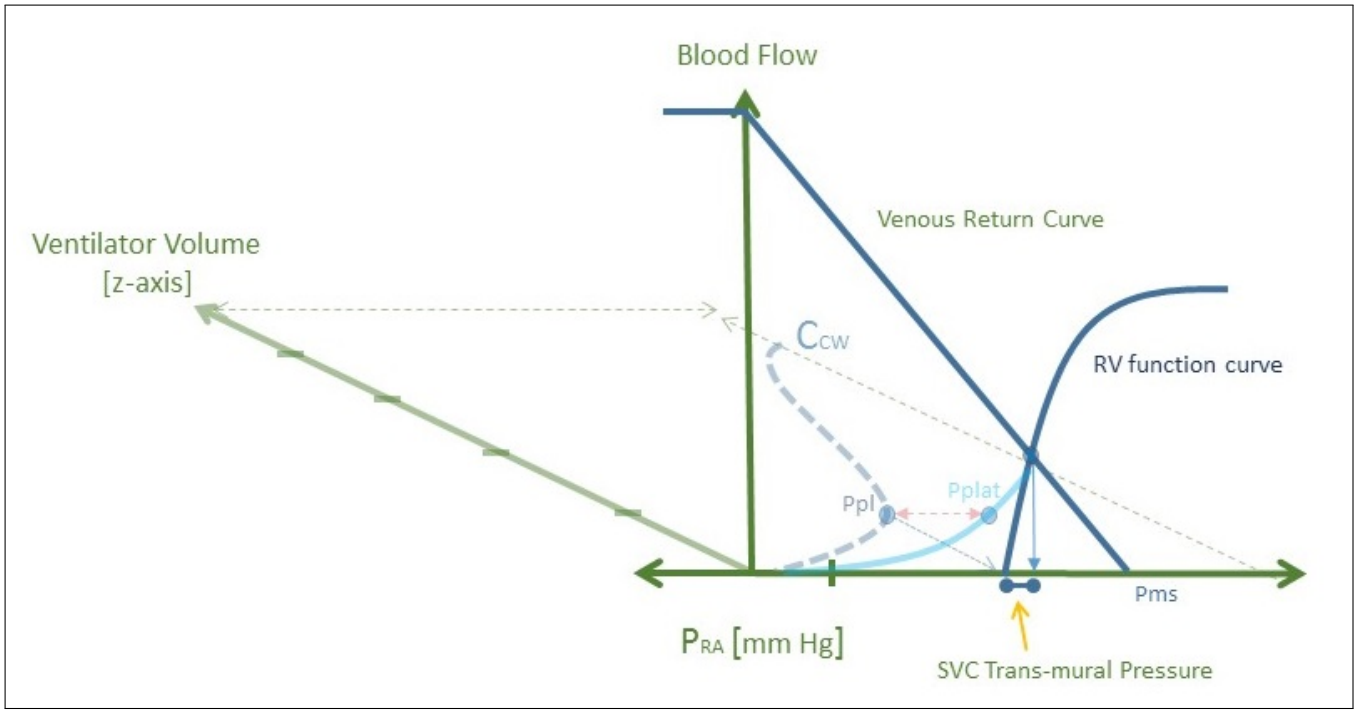

Figure 7. The simultaneous Rahn and Guyton Analysis of the prone patient. This graphic represents the system at end-inspiration. The Rahn diagram in the z-axis matches Figure 3. Cpulm (omitted for simplicity) rises, while Ccw falls, such that the overall respiratory compliance (Crs) curve remains unchanged. At the same ventilator volume, the Ppl increases to a greater degree and the Ptp (dashed red line) is reduced. The reduction in Ptp raises the slope of the RV cardiac function curve. Simultaneously, the RV function curve x-intercept rises in-step with the Poes such that preload is significantly diminished; the Guyton diagram parallels Figure 5. Because both RV afterload and preload are reduced, cardiac output, on the y-axis, is essentially unchanged or may even rise. The SVC transmural pressure falls and the patient is now fluid responsive.

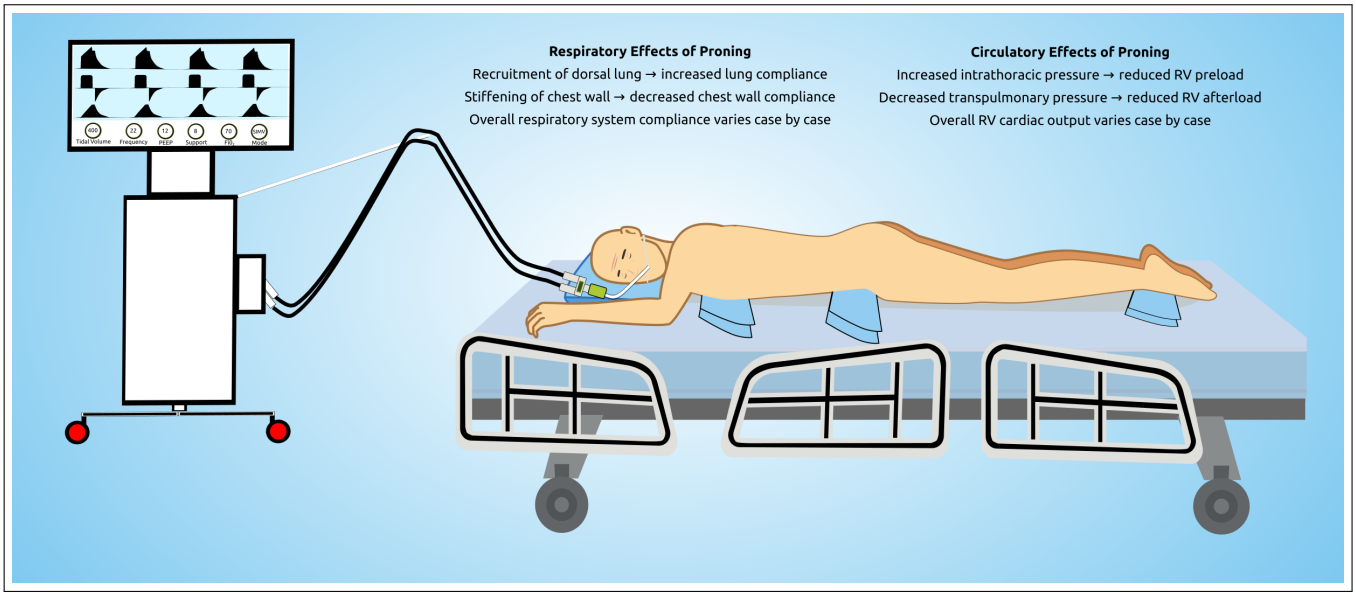

Figure 8. Summary of the respiratory and circulatory effects of the prone position.

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