Abdominal Circulatory Interactions

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KEYWORDS

• Mechanical ventilation • Ascites • Prone positioning • Vascular waterfall
• Abdominal zone conditions

KEY POINTS

- Ventilation whether spontaneous or mechanical affects abdominal pressure (Pab) by inducing diaphragmatic descent.
- Changes in Pab affect circulatory physiology through effects on venous return, preload, and LV and RV performance.
- Transmission of increased abdominal pressure to the thorax and cardiac chambers can make interpretation of static pressure in the cardiac chambers difficult.
- Changes in abdominal pressure may play a large role in common clinical conditions, such as weaning-induced cardiac dysfunction, postparacentesis circulatory dysfunction, prone positioning during ARDS, and laparoscopic surgery.
- Intensivists should have a fundamental understanding of the effects of abdominal pressure on circulatory and respiratory physiology.

The abdominal compartment is separated from the thoracic compartment by the diaphragm. Under normal circumstances, a large portion of the venous return crosses the splanchnic and nonsplanchnic abdominal regions before entering the thorax and the right side of the heart. Moreover, mechanical ventilation especially with positive end-expiratory pressure (PEEP) may affect abdominal venous return independent of its interactions at the thoracic level. Furthermore, changes in pressure in the intra-abdominal compartment may have important implications for organ function within the thorax, particularly if there is a sustained rise in intra-abdominal pressure as in abdominal compartment syndrome. It is therefore important to understand the consequences of abdominal pressure (Pab) changes on respiratory and circulatory

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physiology. This article elucidates important abdominal-respiratory-circulatory interactions and their clinical effects. The effect of intra-abdominal hypertension on systemic physiology is not a focus of this review. For a detailed discussion of this topic please see Sarani, Maluso, Olson: Abdominal Compartment Hypertension and Abdominal Compartment Syndrome, in this issue.

RELATIONSHIP BETWEEN RIGHT ATRIAL PRESSURE, ABDOMINAL PRESSURE, AND VENOUS RETURN

Traditionally, changes in intrathoracic pressure have been the sole focus of analyses of cyclic respiratory changes in venous return. It is generally assumed that spontaneous inspiration (negative intrathoracic pressure) enhances superior and inferior vena cava (IVC) blood flows secondary to decreases in right atrial pressure (Pra) induced by a fall in the intrathoracic pressure.[1] However, during inspiration, descent of the diaphragm also causes changes in intra-abdominal pressure,[2] which may affect venous return in complex ways. The abdominal vascular compartment has a large capacitance and is directly upstream to the intrathoracic compartment. IVC venous return accounts for two-thirds of the systemic venous return and insight into factors influencing changes in IVC venous return is crucial.

Previous work documented conflicting results of the effect of increasing Pab on venous return. Many studies found that IVC venous return increased with rising Pab.[3–5] However, other investigators reported a decrease in venous return that was dependent on the magnitude of stress and conditions of the circulatory system.[6,7]

In 1964, West and colleagues[8] proposed the pulmonary vascular zone theory to explain the relationships between pulmonary blood flow, vascular pressures, and alveolar pressures. To reconcile differences in observations of the influence of Pab on venous return Takata and colleagues[9] (analogous to West zones) put forth the concept of presence of similar vascular zones in the abdomen. In this elegant model, they hypothesized that a vascular waterfall[10] would develop within the IVC at the level of the diaphragm and that the occurrence of such a waterfall was dependent on pressure within the abdomen (Pab), pressure within the abdominal IVC (Pivc), and the critical transmural closing pressure of the IVC at the waterfall (Pc). In earlier work Lloyd[4] had demonstrated that flow in the abdominal IVC was in the forward direction unless Pivc was 5 cm H_2O or more below Pab and concluded that rather than behaving as a pure Starling resistor, the IVC had a tethering open capacity of Pc. Takata's model is illustrated in **Fig. 1**, wherein there is an upstream extra-abdominal venous compartment (Vu) and a downstream abdominal compartment (Vb), which is surrounded by Pab. Both empty into the thoracic IVC. Using this model, Takata and colleagues described two resting abdominal zone conditions: zone 2 and zone 3. In zone 3, IVC pressure at the level of diaphragm (Pivc) exceeds the sum of Pab and critical closing Pc. In this case, the effective back pressure to IVC flow is Pivc. In zone 2 conditions the sum of Pc and Pab is greater than Pivc and the effective backpressure to IVC flow is Pab + Pc. Changing Pab depending on initial resting state of the abdomen results in one of three scenarios: (1) zone 3 conditions are maintained with increase of Pab, (2) zone 2 conditions are maintained with increase of Pab, and (3) the abdomen transitions to zone 2 state from zone 3 after application of an increase in Pab. In the first scenario (zone 3 to zone 3), backpressure to IVC flow would not change but blood would be discharged from the abdominal compartment because of increased Pab, whereas blood volume in the extra-abdominal compartment would remain the same (extra-abdominal compartment, which is not surrounded by Pab, effectively sees Pivc as the back pressure), leading to a decrease in the total IVC volume. In the second