Progress in Pediatric Cardiology 39 (2015) 151–156

Contents lists available at ScienceDirect

Progress in Pediatric Cardiology

journal homepage: www.elsevier.com/locate/ppedcard

Review

Cardiopulmonary interactions in adults and children with congenital heart disease

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ARTICLE INFO

Available online 21 October 2015

Keywords:
Cardiopulmonary interactions
Heart-lung interactions
Ventricular interdependence
Pediatric cardiac surgery
Congenital heart disease
Cardiac intensive care

ABSTRACT

This review aims to clarify pathophysiologic mechanisms underlying interactions between the pulmonary and cardiovascular systems. Therapeutic interventions primarily targeting respiratory function during mechanical ventilation of critically ill cardiac patients may have an inevitable secondary effect on the heart and vice versa. A thorough understanding of these complex interactions is a prerequisite for clinical care and may lead to novel therapeutic strategies and improved outcomes.

1. Introduction

It was first appreciated in 1873 by Kussmaul that the heart and lungs are not just anatomically connected but physiologically interrelated. He first described the "pulsus paradoxus"—elimination of the radial pulse during inspiration in patients with constrictive tuberculous pericarditis. We now understand that the drop in systolic arterial blood pressure of more than 10 mmHg during inspiration is not a paradox, but rather an exacerbation of a normal phenomenon. Since then, our understanding of the complex interactions between the respiratory and cardiovascular systems in both health and disease are constantly evolving. Such an understanding is crucial when providing clinical care in critically ill patients, especially those requiring mechanical ventilation. Even in patients without preexisting cardiac disease, mechanical ventilation can result in hemodynamic compromise [1,2], and it becomes paramount in patients with cardiac disease and impaired cardiorespiratory reserve.

1.1. Cardiopulmonary interactions during normal breathing

1.1.1. Basic concepts

From a clinical hemodynamic standpoint, the main determinants of downstream flow through an elastic tube such as a blood vessel are: the inflow pressure, the outflow pressure (downstream pressure) and the transmural (distending) wall pressure. When transmural pressure is positive, flow is dependent upon the gradient between inflow and outflow pressure (downstream pressure gradient). If transmural pressure becomes negative, the vessel will collapse and flow will cease. This explains the normal variation in systemic venous return which occurs with respiration. Changes in intrathoracic pressure will not affect flow in vessels residing entirely within the thoracic cage, because the change in pressure affects the entire circuit, whereas changes in intrathoracic pressure alters flow in vessels crossing the diaphragm because respiration changes transmural pressure of the intrathoracic vessels only (Fig. 1).

1.1.2. Effect on right ventricular preload

According to Guyton, the pressure gradient between right atrium and mean systemic filling pressure determines right ventricular preload, because venous resistance is negligible. Mean systemic filling pressure is defined as the hypothetical pressure of the vascular compartment when all pressures have equilibrated and there is no flow [3,4]. Guyton found mean systemic filling pressure to be 7–12 mmHg in normal dogs [5]. Since the normal right atrial pressure is 2–3 mmHg, the pressure gradient driving venous return is typically only 5–10 mmHg so a change in atrial pressure of only 1–2 mmHg can have significant effect upon venous return [4]. In clinical scenarios, systemic filling pressure is primarily determined by vascular capacitance and circulatory volume [4]. Volume expansion and/or constriction of capacitance vessels increases mean systemic filling pressure to increase venous return to the right atrium.

Normal tidal breathing produces negative intrathoracic pressure which increases the downstream venous gradient between the inferior vena cava and right atrium. This is achieved through two mechanisms: first, negative intrathoracic pressure distends the compliant right atrial wall by increasing the transmural pressure across the right atrium (positive pressure inside the atrial wall minus the negative pressure outside the atrial wall), thus reducing right atrial pressure. Second, the pressure

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http://dx.doi.org/10.1016/j.ppedcard.2015.10.017
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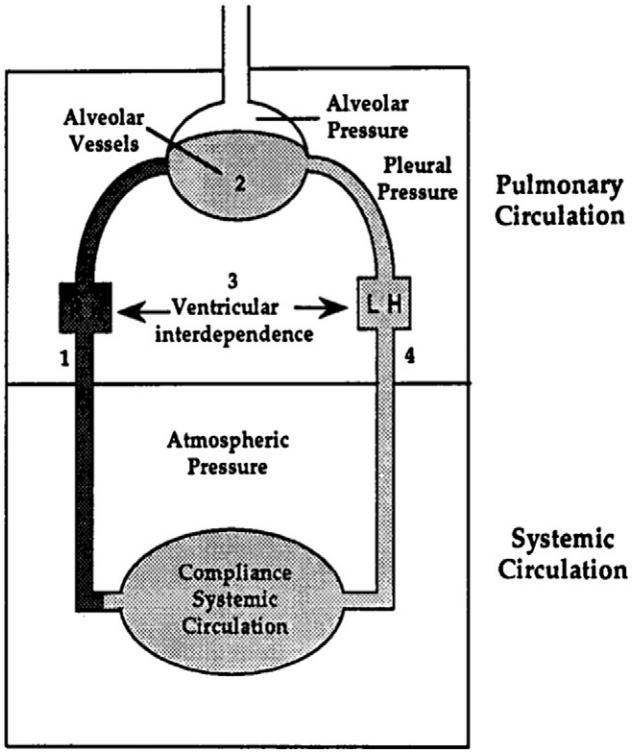

Fig. 1. Schematic representation of the vascular circuit in relation to different pressure compartments (intrathoracic versus extrathoracic).

in the extrathoracic (abdominal) part of the inferior vena cava rises as a result of the downward diaphragmatic excursion which causes an increase in intraabdominal pressure and mean systemic filling pressure. The net effect is an increase in flow from inferior vena cava to right atrium, which increases right ventricular preload. The relationship of venous return to right atrial pressure is demonstrated in Fig. 2. It is noted that maximum venous return occurs when right atrial pressure is zero which gives the highest downstream venous gradient. As evident by the graph, further reduction in right atrial pressure (as in airway obstruction or severe asthma) will not result in further augmentation of venous return.

In an experiment awarded the Nobel Prize in 1948, Cournand et al. demonstrated a 10–15% reduction in cardiac output when mean airway pressure was increased 10–15% through positive pressure ventilation in healthy volunteers [6]. Although this relationship is multifactorial, under physiologic conditions without an intracardiac shunt, cardiac output is determined by systemic venous return (since the two ventricles pump in series). Based on the Frank Starling relationship, an increase in preload improves cardiac output [7], so it makes physiological sense to examine the interposition of the venous return curve and Frank Starling curve. As demonstrated by Guyton, in 1973, cardiac output can be estimated by the intersection of these two curves (Fig. 3) [8].

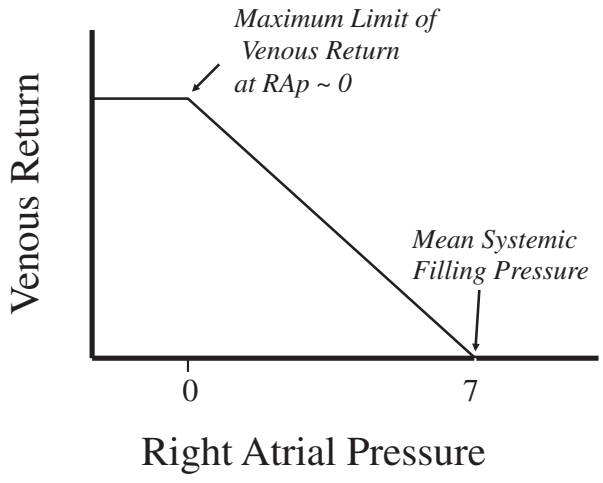

Fig. 2. Schematic representation of venous return curve from inferior vena cava in relation to right atrial pressure. Further reduction of right atrial pressure after the upper deflection point does not result to increased venous return.

1.1.3. Effect on left ventricular preload and afterload

Negative intrathoracic pressure during normal tidal breathing affects the left ventricle differently than the right ventricle. Left ventricular preload depends on the downstream gradient between pulmonary artery and pulmonary veins. Since the entire circuit is intrathoracic, changes in intrathoracic pressure do not alter the downstream pressure gradient. There is, however, a direct relationship of the left ventricular preload to right ventricular output and pulmonary vascular resistance. Since right ventricular output increases with inspiration, venous return to the left ventricle will also rise, augmenting left ventricular preload. The relationship of pulmonary vascular resistance to airway and intrathoracic pressure is complex and will be discussed later. In general, normal tidal breathing does not affect pulmonary vascular resistance significantly, so the net effect of normal breathing on left ventricular preload is positive. However, negative intrathoracic pressure increases the transmural gradient across the left ventricular wall and the intrathoracic part of aorta (mechanism as discussed above) which increases left ventricular afterload. When left ventricular function is normal, this afterload increase does not produce measurable changes in cardiac output, but it becomes quite important when left ventricular function is diminished.

2. Cardiopulmonary interactions during positive pressure ventilation

2.1. Effects on right ventricle

2.1.1. Effect on right ventricular preload

Intrathoracic pressure increases during a positive pressure breath, thereby causing a reduction of the right atrial transmural wall pressure and an increase in right atrial pressure. The rise in atrial pressure decreases the downstream pressure gradient (mean systemic filling pressure- right atrial pressure) that limits venous return so right ventricular preload is diminished.

The diaphragmatic excursion during a positive pressure breath also increases intraabdominal pressure, which further decreases venous return. Although a full description of the hydrodynamics governing this relationship is beyond the scope of this review, the crucial concept is the gradient between intraabdominal pressure and intraluminal inferior

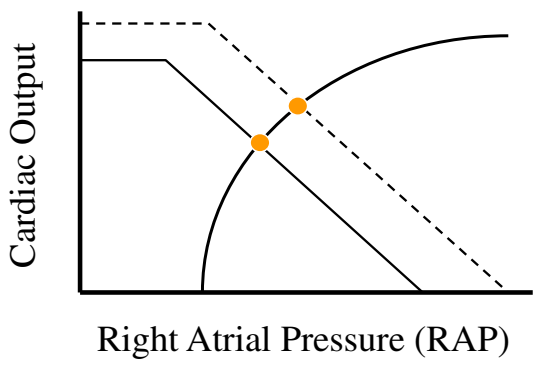

Fig. 3. Schematic representation of coupling between starling curve and venous return curves. Increased venous return results in increased cardiac output.