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Understanding venous return

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Cardiac output can increase fivefold to adapt to changing metabolic needs. Since normal contraction empties the heart of nearly all its blood, simply increasing the force or rate of contraction cannot explain the dramatic increase in cardiac output. Instead, the circulation mobilizes a large volume of blood from the compliant veins. The venous system stores two-thirds of the blood volume and serve as an adjustable reservoir [1, 2]. The Starling law explains that cardiac output is intrinsically coupled to the rate of blood return to the heart [1]. To increase cardiac output, the circulation increases venous return—the rate of blood flowing from the systemic veins into the right atrium. This review will discuss the mechanisms that regulate venous return.

A pressure gradient drives blood from the veins into the heart. The normal right atrial pressure at rest is 0 mmHg [1, 3–5]. Contraction empties the heart and maintains this normal (low) right atrial pressure. The mean systemic filling pressure (P_{sf}) is the physiologists' term for the upstream pressure that drives blood into the heart. This P_{sf} is the average non-pulsatile force exerted by the blood in the vasculature and is normally 7 mmHg [1]. The magnitude of the P_{sf}–right atrial pressure gradient is proportional to the force driving venous return to the heart [1, 4]. Mean systemic filling pressure is not a function of cardiac pumping. Rather, the volume of blood in the circulation and the vascular compliance determine P_{sf}. The veins have muscles in their walls and rich autonomic innervation. Blood volume expansion and constriction of the veins by sympathetic tone increase P_{sf}. Exercising limb muscles also constrict the veins. Because of valves, venoconstriction tends to increase the venous return gradient [1, 6].

The stressed blood volume is the amount of blood under vascular tension that contributes to P_{sf} and participates in venous return. Normally the majority of the blood volume is unstressed and does not contribute to P_{sf} or venous return. Constriction of the veins can mobilize the unstressed blood volume to augment stressed volume and venous return [7, 8]. Figure 1 shows the importance of stressed blood volume for venous return. Note that the pressure in the arterial system has little direct effect on P_{sf} and the gradient for venous return. For example, increasing systemic arterial pressure with a pure arterial constrictor (angiotensin) raises systemic blood pressure without increasing venous return and cardiac output [9]. However, balanced vasoconstrictors such as norepinephrine may be able to increase the gradient for venous return and cardiac output [6, 9–11].

Normally, a pressure gradient of just 7 mmHg sustains cardiac output. As shown in Fig. 1, increasing the gradient between P_{sf} and the right atrium increases the force driving venous return. High right atrial pressure is a backpressure that impedes venous return. Right atrial pressure is a determinant of cardiac output only insofar as it is kept low to allow cardiac filling. The circulation must keep the P_{sf} higher than right atrial pressure to maintain cardiac preload. Right atrial pressure rises as a result of filling—it is not a cause of filling. Equalization of right atrial

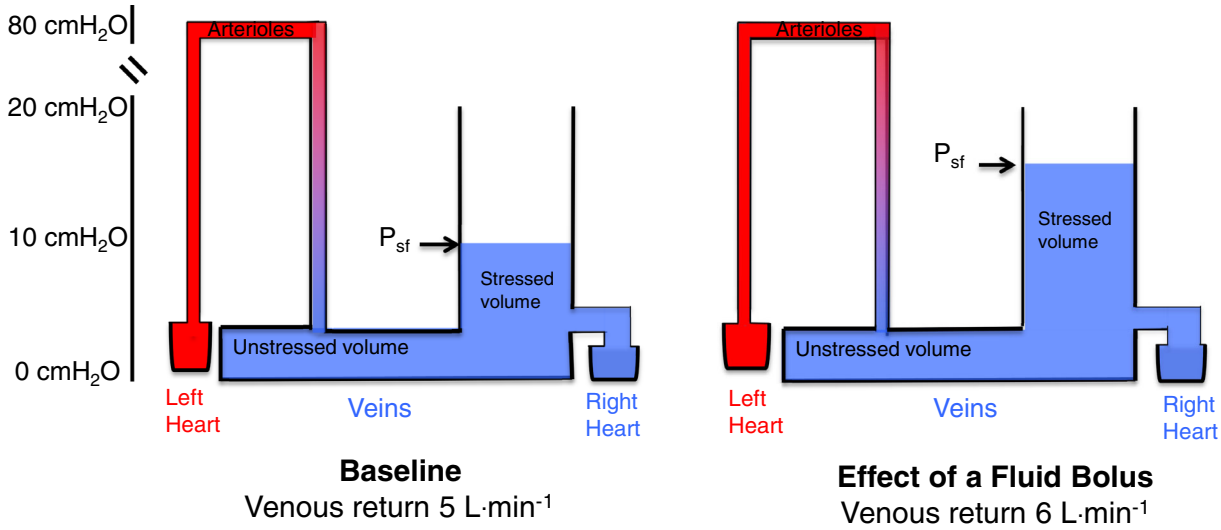

Fig. 1 Schematic description of an increase in venous return after a fluid bolus. Both panels show the systemic circulation in diastole and end-exhalation. For clarity, the pulmonary circulation is not shown. The *y*-axis represents the pressure of blood in different compartments. 0 cmH_2O represents atmospheric pressure. The arterial pressure is 80 cmH_2O (60 mmHg) in both panels. The *left panel* shows the baseline conditions with the mean systemic filling pressure (P_{sf}) of 10 cmH_2O (approximately 7 mmHg). The *right panel* shows an increase in the stressed blood volume and P_{sf} after fluid resuscitation. This generates a larger pressure gradient for venous return between P_{sf} and the right heart. The values depicted are theoretical. The actual response to fluid would also depend on cardiac performance, vascular resistance, and circulatory reflexes

pressure and the mean systemic filling pressure stops venous return and cardiac output [4]. Equalization can occur if P_{sf} falls as a result of volume depletion and venodilation, or if there is a pathological elevation of right atrial pressure [4, 5].

Spontaneous inspiration lowers right atrial pressure by reducing intra-thoracic pressure. Inspiration, therefore, increases the gradient for venous return and can augment cardiac output. Extremely forceful inspiratory effort can decrease intra-thoracic pressure enough to collapse the great veins entering the thorax. The collapse imposes a limit on the inspiratory increase of the venous return gradient [4, 5].

Right atrial pressure, not its transmural pressure, is the back-pressure that opposes venous return [2]. Positive pressure ventilation, cardiac tamponade, and ventricular afterload increase right atrial pressure and can decrease the gradient for venous return. We can envision this as raising the right heart reservoir in Fig. 1, while leaving the height of P_{sf} unchanged. A proportionate increase in P_{sf} can compensate for high right atrial pressure and sustain venous return [1].

Vascular resistance opposes venous return. Vasodilation increases venous return at any magnitude of pressure gradient. This phenomenon couples cardiac output to overall metabolic needs. Autoregulating tissues dilate their arterioles to recruit blood flow. Widespread arteriolar vasodilation reduces the resistance to venous return and raises cardiac output. Reduction in arteriolar resistance also explains much of the increase in cardiac output during sepsis, hypoxia, and anemia [5].

During exercise, sympathetic stimulation constricts the veins to raise P_{sf} and contracting skeletal muscles reduce the resistance to venous return by dilating their arterioles. These factors can dramatically increase venous return [5]. A similar situation occurs after massive transfusion. The expanded blood volume raises P_{sf} and also dilates the vasculature to reduce resistance to return [5].

The healthy heart can accommodate large increases in venous return. The heart has a number of mechanisms to adapt to increased myocardial stretch and tightly couple cardiac output to venous return. First, the stretch optimizes actin–myosin cross-bridges, which increases the force of contraction (the Frank–Starling mechanism). Stretching the right atrium also increases the automaticity of the sino-atrial nodal tissue to increase heart rate. Finally, myocardial stretch elicits a sympathetic reflex to augment myocardial contractility and heart rate [1].

There is a limit to the heart's capacity to accommodate increased venous return. The limit of accommodation is lower when the heart is abnormal, or there are arrhythmias or excessive ventricular afterload. If the circulation increases venous return into to a failing heart, blood wells up in the heart and raises diastolic pressure [1]. The rise in diastolic pressure decreases the rate of venous return and limits cardiac output. Interventions that improve cardiac contractility or relaxation or decrease ventricular afterload can increase the heart's ability to accommodate increased venous return.

Fluid therapy is an essential aspect of critical care [12]. The venous return model explains the physiologic basis of

fluid therapy and the management of hypoperfusion. Interventions such as fluids or vasopressors will recruit cardiac output only if they increase the gradient for venous return and the heart can accommodate the increased return [13].

Conflicts of interest The authors declare they have no financial conflict of interest.

Ethical standard There were no human or animal subjects used for this study and there is no requirement for institutional review approval.

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