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Importance of Venous Return, Venous Resistance, and Mean Circulatory Pressure in the Physiology and Management of Shock*

Michael A. Bressack, M.D.; † and Thomas A. Raffin, M.D., F.C.C.P.‡

Physiologic Background

A reasonable, unifying definition of shock at the cellular level is inadequate oxygen consumption by the cell for its metabolic needs. The negative consequence is the accumulation of an oxygen debt with eventual cellular dysfunction and death.

Inadequate oxygen consumption (shock) can result from the following: (1) inadequate oxygen content of the blood; (2) inadequate circulation of the blood (cardiac output); (3) failure to deliver the circulated blood to the cell (microperfusion); and (4) failure of the cell to utilize the delivered oxygen (cellular dysfunction).

In our discussion, we shall focus on inadequate circulation (cardiac output) as the limiting factor in oxygen consumption (shock). Traditionally, cardiac function has been viewed as the sole determinant of cardiac output. This function has been divided into preload, inotropy, afterload, and heart rate; cardiac output can be maximized by increasing preload and inotropy, decreasing afterload, and optimizing heart rate.^{1.2}

Guyton et al¹ emphasized that the regulation of cardiac output is determined by the interaction between the vasculature and the heart. In steady state, the heart cannot eject more blood than it receives from the vasculature and the vasculature cannot return more blood than it receives from the heart. Cardiac output must equal venous return.

The physiology of these separate systems can best be understood graphically. A family of Starling curves shows the regulation of cardiac output by the heart (Fig 1); maximal cardiac output requires adequate preload (indirectly measured as atrial pressure) and inotropy

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and minimal afterload (principally measured as vascular resistance).



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FIGURE 1. Cardiac function curves demonstrating the effects of preload (atrial pressure), inotropy, and afterload on cardiac outout.¹



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FIGURE 2. Venous return curve demonstrating the pressure-flow relationship of the venous system. The pressure-axis intercept equals mean circulatory pressure (Pmc). The inverse of the slope equals resistance to venous return (Rv). The plateau in the curve is due to the great veins functioning as Starling resistors.¹ Although our graphs refer to atrial pressure in general, it should be noted that right atrial pressure has a much greater effect than left atrial pressure on venous return.

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^{*}From the Departments of Pediatrics and Medicine, Stanford University, Stanford, CA.

^{*}Clinical Associate Professor of Pediatrics.

[‡]Associate Professor of Medicine.

Reprint requests: Dr. Raffin, Division of Respiratory Medicine, C-356, Stanford University Medical Center, Stanford 94305



FIGURE 3. Schematic view of the circulation as a single circuit with blood flowing from the pump (heart), through the artery/arteriole (vascular resistance vessels), into the reservoir (small vein/venule), and returning to the heart through large veins (resistance to venous return).

Guyton et al¹ developed a graphic representation of venous return which shows the regulation of cardiac output by the vasculature (Fig 2). This utilizes the concepts of mean circulatory pressure (Pmc) and resistance to venous return (Rv). If, as a reasonable approximation, we approach the circulation as a single circuit (Fig 3), the arteries/arterioles have dominant control over vascular resistance (arterial resistance is seven to ten times venous resistance³) and distribution of blood flow. The veins function as the principle intravascular reservoir (small veins/venules) (venous compliance is 20 to 60 times arterial compliance³) and the site of resistance to venous return (large veins³);



FIGURE 4. Schematic view of the venous side of the circulation with blood from the venous reservoir flowing into the atrium through large veins with a significant resistance (Rv). The rate of flow is determined by the upstream driving pressure in the reservoir (mean circulatory pressure [Pmc]), the downstream pressure (atrial pressure [Pa]), and the resistance to venous return (Rv). Mean circulatory pressure is determined by the total volume (V) and the elasticity (unstressed volume, Vo, and compliance, (C) of the reservoir.

this conceptual model is an oversimplification, since all types of veins have components of capacitance and resistance. Moreover, the systemic vasculature dominates the physiology of venous return because its capacitance is seven to ten times that of the pulmonary vasculature.³

If we focus on the venous side of the circuit (Fig 4), the venous return (equivalent to cardiac output in a closed system) equals

$$\frac{Pmc - Pa}{Rv}$$

where Pmc is mean circulatory pressure, Pa is atrial pressure, and Rv is resistance to venous return (primarily venous resistance since the intravascular reservoir is in the small veins/venules). The Pmc is the intravascular pressure measured when the heart has been stopped and the blood in the circulation has been redistributed so that pressures everywhere in the circulation are exactly equal.¹ During active circulation, Pmc is the pressure within the intravascular reservoir and is the upstream driving pressure for venous return; atrial pressure (Pa) is the downstream pressure (Fig 4). The Pmc is a function of the blood volume and the elastic properties of the circulation; thus,

$$Pmc = \frac{V - Vo}{C}$$

where V is the total intravascular (reservoir) blood volume, Vo the unstressed vascular volume (the volume contained within the vascular reservoir when the mean circulatory pressure is atmospheric), and C the vascular (reservoir) compliance (Fig 4); Vo and C characterize vascular elasticity.

Graphically (Fig 2), the venous return curve inter-



FIGURE 5. Effects of changes in mean circulatory pressure (Pmc) on the venous return curve. $^{1.4}\,$

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FIGURE 6. Effects of changes in resistance to venous return (Rv) on the venous return curve.¹⁴

cepts the pressure axis at a Pa which is equal to mean circulatory pressure (Pmc) and the inverse of the slope of the venous return curve is resistance to venous return (Rv). Although our graphs refer to atrial pressure (Pa), right atrial pressure has a much greater effect on venous return than left atrial pressure because the systemic compliance is so much larger than pulmonary compliance.³ The plateau in the venous return curve is due to the fact that the great veins entering the chest function as Starling resistors, collapsing as the right atrial pressure (Pa) becomes subatmospheric. A family of venous return curves can be constructed showing the effects of isolated changes in mean circulatory pressure or resistance to venous return. Venous return can be impaired by a decrease in mean circulatory pressure (Pmc) (Fig 5) or an increase in resistance to venous return (Rv) (Fig 6). The Pmc decreases due to the loss of intravascular blood volume (V) or a decrease in vascular elasticity (increased Vo, C). Decreased vascular elasticity is caused by dilatation/relaxation (an active process due to circulating vasoactive mediators or autonomic changes, or a passive relaxation due to increased flow) of the reservoir vessels (small veins/ venules).

The veins primarily determining the resistance to venous return include central large veins such as the vena cavae, which are passively affected, and peripheral large- and medium-sized veins which can be passively affected or responsive to circulatory vasoactive mediators and autonomic stimulation. Increased resistance to venous return can occur due to active vasoconstriction of the peripheral large- and mediumsized veins, passive narrowing of the veins (extrinsic pressure due to increased intraabdominal/pleural pressure or passive elastic recoil due to low flow), or to hyperviscosity (polycythemia).

The resistance to venous return (Rv) can be greatly

affected by the distribution of blood flow (flow distribution is determined by artery/arteriolar function). The systemic vasculature can be divided into vascular beds with short time constants (striated muscle, kidney) and long time constants (hepatosplanchnic); flow through vascular beds with short time constants will decrease resistance and therefore, improve venous return while flow through vessels with long time constants will increase resistance and therefore interfere with venous return.⁴

Guyton et al¹ realized that since venous return must equal cardiac output in a closed system, the same graphic parameters could be used for both the cardiac function and venous return curves. The superimposed curves demonstrate the interaction of the two systems in determining cardiac output (Fig 7). The intersection point of the curve (point A) will determine the cardiac output and atrial pressure based on the momentary pumping ability of the heart (inotropy, afterload, heart rate) and the characteristics of the circulation (V, C, Vo, Rv). Both the heart and the circulation may be abnormal. However, if cardiac function is the limiting factor in blood flow (decreased inotropy or increased afterload), there will be a low cardiac output associated with an increased atrial pressure (Fig 8, point B). On the other hand, if venous return is the principle limiting factor (decreased mean circulatory pressure or increased resistance to venous return), there will be a low cardiac output associated with a decreased atrial pressure (Fig 9, point B, C).

CLINICAL RELEVANCE

Weil and Shubin³ classified clinical shock as hypovolemic, cardiogenic, distributive, and obstructive. In cardiogenic and obstructive (pulmonary embolism, cardiac tamponade) shock, the primary abnormality is cardiac function; cardiac output is decreased in associa-



FIGURE 7. Superimposition of the cardiac function and venous return curves. The point of intersection (point A) determines the cardiac output and atrial pressure.¹

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FIGURE 8. Effects of decreased cardiac function with no change in venous return. Cardiac output decreases and atrial pressure increases (point A to B).¹⁴

tion with an increased atrial pressure (Fig 8, Point A to B) (the exact shape of the cardiac function curve will depend on the specific disorder). Although there can be primary or secondary abnormalities in the venous return curve, cardiac function curve abnormalities limit the cardiac output.

In hypovolemic shock, the principle abnormality is the loss of intravascular volume (V); although secondary changes in vascular and cardiac function may develop, venous return is primarily decreased due to a low mean circulatory pressure $(Pmc)^6$ (Fig 9, point A to C). Cardiac output is depressed in association with a decreased atrial pressure.

The mechanisms involved in distributive shock are much more complex and less understood; moreover, this form of shock is heterogeneous, including septic,



FIGURE 9. Effects of decreased mean circulatory pressure (Pmc) (point A to C) or increased resistance to venous return (Rv) (point A to B) with no change in cardiac function. Cardiac output and atrial pressure decrease.¹⁴

anaphylactic, and neurogenic shock. Focusing on only septic shock, we clinically find patients with a high, normal, or low vascular resistance and cardiac output; many of these findings are probably related to the age of the patient, the specific bacteria involved, and the associated vasoactive mediators.7 Although arterial tone determines total vascular resistance, perfusion pressure, and distribution of blood flow,3 venous tone with its effects on Pmc and Rv determines venous return, and therefore, cardiac output early in septic shock (before cardiac dysfunction). The study of the physiology of septic shock has dealt primarily with the arteries and not the veins. If the sepsis involves active venoconstriction, venous resistance (peripheral large veins) and elasticity (small veins/venules) will both be increased. The effect on venous return (cardiac output) will depend on which determinant (Pmc or Rv) dominates; increased Rv would decrease venous return while increased Pmc would increase venous return. Studies in dogs (E coli and endotoxin⁷) and piglets (group B streptococci⁸) have shown a decrease in venous return (cardiac output) because of the dominant effect of an increased resistance to venous return (Fig 10, point A to B). The Rv is further increased by passive elastic recoil of the veins (caused by a low cardiac output) and by the blockage of small veins/ venules (microvascular agglutination and/or clotting). If the venoconstriction is associated with significant arterioconstriction (elevated afterload), the cardiac output could be further harmed by a worsening of the cardiac function curve. Severe arterioconstriction could also elevate Rv by increasing the capacitance of proximal arteries.

If active venodilatation is part of the sepsis,⁹ resistance to venous return and elasticity will be decreased.



FIGURE 10. Sepsis with venoconstriction causing an increased resistance to venous return (Rv) and mean circulatory pressure (Pmc); cardiac function is unchanged. Cardiac output (venous return) and atrial pressure are decreased because of the dominant effect of Rv (point A to B). With fluid therapy cardiac output and atrial pressure are normalized by further increasing Pmc (point A).

The overall effect on venous return (cardiac output) will depend on the balance between a low Rv and Pmc; a dominant decrease in Rv (Fig 11, point A to C) would increase venous return (the increased cardiac output could further decrease Rv by passive relaxation of the veins), while a dominant decrease in Pmc (Fig 11, point A to B) would decrease venous return. If the venodilatation is associated with significant arteriodilatation (decreased afterload), the cardiac output could also be benefited by an improved cardiac function curve. When venous return (cardiac output) is normal or increased in septic shock, the patient can still deteriorate due to abnormalities in metabolic needs, microperfusion and/or cellular oxygen utilization.^{7.10}

Late in the course of both hypovolemic and septic shock, as the heart becomes injured because of an increasing oxygen debt within the cardiac cells, cardiac dysfunction dominates over abnormalities of venous return;¹¹ abnormalities in the cardiac function curve rather than the venous return curve limit cardiac output. However, there are also important late abnormalities in the venous return curve. Due to changes in microvascular hydrostatic pressure and cellular permeability, intravascular fluid moves into the interstitial and cellular spaces, decreasing Pmc because of the low intravascular volume (V).^{2,6,7,12} In addition, more than two decades ago, Lillelhei et al¹³ found evidence for increased resistance to venous return late in hypovolemic and septic shock.

THERAPEUTIC IMPORTANCE

The ultimate purpose of understanding the interaction between venous return and cardiac function in shock is to be able to approach therapy in a rational manner. Since cardiogenic and obstructive shock are principally due to cardiac function abnormalities, we must focus our therapy on optimizing preload, inotropy, afterload, and heart rate. On the other hand, when approaching hypovolemic and septic shock, we must focus our therapy on abnormalities in the venous return curves; there is no initial abnormality in cardiac function.

For hypovolemic shock, a logical approach is to replace the intravascular volume loss. Physiologically, this correction of a decreased intravascular volume (V) should normalize the mean circulatory pressure (Pmc), and therefore, cardiac output (Fig 9, point A). The controversy over whether to use crystalloid or colloid fluid therapy is of secondary importance as long as enough fluid is retained intravascularly. Difficulties arise when intravenous fluid is not retained because of increased vascular permeability, an increase in the microvascular hydrostatic pressure due to a rise in venous resistance, or fluid shifts into the cell.^{2,6,12}

Catecholamines, such as dopamine or epinephrine, can increase venous return in hypovolemic shock by



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FIGURE 11. Sepsis with venodilatation causing a decreased resistance to venous return (Rv) and mean circulatory pressure (Pmc); cardiac function is unchanged. Cardiac output (venous return) and atrial pressure may be decreased if Pmc dominates (point A to B) or increased if Rv dominates (point A to C). With fluid therapy cardiac output and atrial pressure are increased by normalizing Pmc (point D).

increasing venous elasticity and therefore mean circulatory pressure (Pmc).^{1,14,15} This can occur at a dosage that does not significantly affect cardiac function or total systemic vascular resistance. In fact, an increase in venous elasticity secondary to passive elastic recoil and endogenous catecholamine secretion (autonomic and circulatory) can stabilize a patient with hypovolemic shock by mobilizing blood from the body's venous reservoir.^{6,15}

Another treatment modality sometimes used in hypovolemic shock (trauma) is the pneumatic antishock garment. Its benefit is thought to be by compression of the systemic venules/small veins (increased elasticity), and therefore, elevation of the upstream driving pressure for venous return (Pmc). However, when the garment is applied to the abdomen, its inflation can distort and narrow large retroperitoneal and abdominal veins, thus increasing Rv. Holcroft et al^{6,16} have shown that in hypovolemic baboons, these two determinants of venous return counteract each other.

Recently, researchers have found that small amounts (4 ml/kg) of hypertonic sodium chloride (2400 mosm/L) may be beneficial in hemorrhagic shock by increasing mean circulatory pressure (Pmc) without changing the blood volume;¹⁷ presumably, vascular elasticity is increased (Vo, C are decreased). For this response to occur, hypertonic sodium chloride must be infused through the pulmonary circulation and the cervical vagus nerve must be intact, suggesting a peripheral

reflex of pulmonary origin.

In addition to specific antibiotic therapy, treatment for septic shock should depend on the specific abnormal determinants of venous return. When there is venoconstriction with an increase in resistance to venous return, the resulting low venous return (cardiac output) (Fig 10, point B) can be treated by elevation of Pmc with enough intravenous fluid therapy to overcome the increased Rv (increase the upstream driving pressure for venous return) (Fig 10, point A) or by normalization of the increased resistance to venous return. The increased resistance might be improved pharmacologically by venodilatation with agents such as nitroglycerin, calcium channel blockers, or beta-2 catecholamines. The ultimate effects of vasoactive agents on venous return are confusing and unresolved because of possible actions on venous elasticity (Vo, C), resistance to venous return (Rv), and the distribution of blood flow through vascular beds with long or short time constants.^{15,18,19} As already stated, small veins/ venules tend to be the site of the intravascular reservoir, while large veins are the primary site of resistance to venous return (Fig 3). Ideally, a therapeutic agent would improve venous return by decreasing venous resistance without decreasing venous elasticity and therefore Pmc. Unfortunately, researchers have so far not been able to find vasoactive agents that will clearly separate venous resistance and capacitance functions;^{15,18,19} this may partially be due to the fact that all types of vasoactive-responsive veins have components of capacitance and resistance. Vasoactive agents may also affect venous return by altering arteriolar tone; increased perfusion of vascular beds with short time constants will improve venous return while perfusion of vascular beds with long time constants will decrease venous return. Future research in these physiologic areas will be quite important for selecting vasoactive therapeutic agents. Finally, if the venoconstriction is associated with significant arterioconstriction (increased total vascular resistance), treatment of the elevated afterload may be important if cardiac function is abnormal.

When septic shock involves venodilatation, and therefore, increased capacitance, maintaining an adequate mean circulatory pressure (Pmc) with intravenous fluid therapy is crucial to maintaining a normal or elevated cardiac output (Fig 11, point D); the decreased Rv will help venous return. As with hypovolemic shock, catecholamines such as dopamine may benefit this form of septic shock by increasing venous elasticity and therefore Pmc.⁹ However, as already mentioned, vasoactive agents can have unpredictable effects on venous return because of differential effects on venous elasticity, resistance to venous return, and distribution of blood flow.^{15,18,19}

Vasoactive mediators are probably a major cause of



FIGURE 12. Effects of positive end-expiratory pressure (PEEP) on venous return and cardiac function curves. The venous return curve shows an increase in mean circulatory pressure (Pmc) and resistance to venous return (Rv). The cardiac function curve is shifted to the right and depressed. PEEP results in a decrease in cardiac output and an increase in atrial pressure (point A to B).²⁰

the distributive changes (venoconstriction or venodilatation) in septic shock.^{7.9} Discovering and reversing these mediators may reverse the abnormalities in venous return in a more specific manner. Therapy for venous occlusion (caused by microvascular agglutination and/or clotting) may be necessary to prevent abnormalities of Rv and Pmc.

Late in the course of all clinical types of shock, as cardiac decompensation becomes the dominant problem, therapy must focus on heart inotropy, afterload, and heart rate (cardiac function curve). However, as vascular and cellular permeability increase, a loss of intravascular fluid (V) into the interstitial and cellular space must be replaced with enough intravenous fluid to maintain an adequate mean circulatory pressure (Pmc) for venous return.^{2,6,7,12}

Finally, we must mention the important effects positive pressure ventilation has on venous return and cardiac function because of its frequent usage in critically ill patients in shock; this becomes relevant as the pleural and abdominal pressures increase at high levels of positive end-expiratory pressure. The venous return curve shows an increase in Pmc and Rv (Fig 12);²⁰ this is due to compression of intrathoracic and intraabdominal venous structures. The cardiac function curve is adversely affected by the increased afterload on the right ventricle and a decrease in cardiac compliance.²⁰ Even without any adverse cardiac effects, the cardiac function curve will be shifted to the right on the basis of the positive pleural pressure. Thus, the impairment in cardiac output during positive end-expiratory pressure (Fig 12, point A to B) can be explained by the relationship between the venous return and cardiac function curves.

CONCLUSION

We have discussed the physiology of venous return and its importance in the understanding and treatment of shock. Cardiac output is regulated by the interaction between the vasculature and the heart. When approaching a patient in shock, we must determine whether cardiac output is being limited by the vasculature (venous return) or cardiac function; therapy will be quite different for each. Moreover, the physiology of shock will probably change over time. Therapy must be chosen based on an understanding of the changing relationship between venous return and cardiac function. In order to understand and improve our therapeutic options, there is a great need for research into the differential effects of vasoactive agents on venous resistance, capacitance, and flow distribution. Adequate treatment for cardiac output does not assure survival. Abnormalities in microperfusion and/or cellular oxygen utilization can still lead to cellular dysfunction and death.

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