Reframing Shock

Bridging the Gap Between Theory and Practice

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KEYWORDS

• Shock • Hypoperfusion • Pressure interfaces

KEY POINTS

- Currently prevalent approaches to shock in the emergency department have two major limitations: reliance hypotension as a defining criterion, and confining evaluation and management within a four-category framework.
- Conventional approaches to shock have two major limitations: reliance on hypotension as a primary diagnostic criteria, and confining the clinical approach to shock within the four-category framework of hypovolemic, distributive, cardiogenic, and obstructive.
- Given the critical importance of early recognition of shock and timely initiation of appropriate management, equipping frontline providers with a more nuanced approach to shock may justify the additional cognitive investment required, particularly patients who continue to deteriorate despite initial interventions.
- This paper proposes a conceptual framework for approaching shock that attempts to balance physiological sophistication with bedside utility: Shock recognition: Approaching shock as a continuum progressing from physiologic stress to tissue hypoperfusion, rather than as a binary state defined by the presence or absence of hypotension. Shock differential diagnosis and management: Framing shock evaluation and treatment around identification and correction of failures in forward flow, rather than classification into one of four discrete categories with an associated set of proscribed interventions. Forward flow must be understood to be distinct from forward pressure, and may be conceptualized as the net sum of competing forward, backward, and external pressures.
- This framework is not meant to replace traditional approaches to shock, but instead aims to expand upon them, enabling a smoother cognitive shift toward a more nuanced mental model of shock physiology as patient complexity increases.

INTRODUCTION

Despite the depth of our scientific understanding of the complexity of circulatory physiology, the clinical approach to shock at the bedside remains anchored in 2 significant

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Abbreviations	
CVP	central venous pressure
LV	left ventricle
LV CO	left ventricular cardiac output
LVEDP	left ventricular end diastolic pressure
PVR	pulmonary vascular resistance
RAP	right atrial pressure
RV	right ventricle
RV CO	right ventricular cardiac output
SVR	systemic vascular resistance

oversimplifications. The first is the use of blood pressure as the defining criterion for diagnosing shock. The second is the classification of shock into 4 discrete categories: hypovolemic, distributive, cardiogenic, or obstructive.[1] The persistence of these reduction underscores the disconnect between our theoretic understanding of shock and the practical realities of clinical decision-making. This gap underscores the need for a reframed mental model of shock that is both physiologically sophisticated and readily operationalized at the bedside.

The mental model of shock described here incorporates two core components:

1. *Shock recognition*: Approaching shock as a continuum progressing from physiologic stress to overt tissue hypoperfusion, rather than as a binary state defined by the presence or absence of hypotension.
2. *Shock differential diagnosis and management*: Framing shock evaluation and treatment around identification and correction of disturbances in forward flow, rather than classification into one of 4 discrete categories with an associated set of proscribed interventions. Forward *flow* must be understood to be distinct from forward *pressure* and may be conceptualized as the net sum of competing forward, backward, and external pressures.

This mental model is not intended to function in opposition to current approaches to shock, but rather seeks to build upon them in order to facilitate a seamless cognitive transition to a more nuanced bedside approach to circulatory failure. Such an approach is particularly valuable where traditional models may prove insufficient, such as in patients with multiple comorbidities, ambiguous shock presentations, or when a clinical course deviates from expected trajectories. At the same time, it is important to recognize that acutely ill patients are not all equally complex. Emergency physicians must triage not only patient flow but also their cognitive and temporal resources, and many patients can be successfully managed using established approaches. Incorporating more sophisticated models of shock physiology into emergency care, however, does not necessarily require sacrificing clinical efficiency. The goal of bit is to provide a framework that facilitates the selective investment of additional time and mental resources where the use of higher-order approaches to shock is most likely to alter a patient's course.

CLINICAL RELEVANCE OF THE OVERSIMPLIFICATION OF SHOCK

Developing a more nuanced understanding of shock physiology is not merely an interesting theoretic exercise. Four recurrent pitfalls illustrate how reliance on reductive frameworks of shock physiology may adversely affect patient care:

- *Failure to identify shock*. Shock may be easily overlooked in patients who are not hypotensive, particularly in those with robust compensatory mechanisms. For

example, a young otherwise healthy patient presenting with abdominal pain due to a ruptured ectopic pregnancy may present with preserved blood pressure despite profound circulatory insufficiency, delaying recognition and early aggressive treatment. Hypotension may also be less prominent in presentations of certain shock phenotypes. For example, a patient who has circulatory failure due to depressed left ventricular function in the context of significantly elevated afterload may not exhibit significant hypotension even once they have progressed into later stages of shock.

- *Misdiagnosis of shock etiology*. Clinicians are often most comfortable diagnosing shock states defined by reduced forward pressures, such as hypovolemia requiring fluid resuscitation or vasodilation requiring vasopressors. In contrast, shock states that predominantly involve elevated back pressures are frequently underrecognized. For example, shock in a patient presenting with hypotension, hyperlactatemia, and abdominal distension may be easily misattributed to abdominal sepsis, whereas the true underlying etiology is acute decompensated right ventricular failure and congestive hepatopathy in the context of pulmonary hypertension.
- *Constrained shock management repertoire*. This narrow focus on forward pressure frequently extends to therapeutic decision-making. For example, while clinicians tend to be very comfortable administering fluids and vasopressors in a patient with shock due to Gram-negative bacteremia, they are often reluctant to administer diuretics and vasodilators in a hypotensive patient with acute decompensated left ventricular failure.
- *Difficulty with recognition and management of mixed shock states*. Many patients exhibit overlapping shock phenotypes, yet diagnostic and therapeutic frameworks tend to force classification—and therefore management strategy—into a single category. A common example is septic shock complicated by sepsis-induced cardiomyopathy, in which a blended physiology requires both vasopressor and inotropic support as well as a very careful approach to management of intravascular volume status.

DISENTANGLING SHOCK FROM HYPOTENSION

Reframing our approach to shock must begin with a clear conceptual delineation between *hypotension* and *hypoperfusion*. The extent to which the medical community equates hypotension with shock is underscored by "occult shock" being described as a distinct diagnostic entity in which a patient exhibits clear signs of tissue hypoperfusion despite being normotensive. While the characterization of shock based solely on the presence of hypotension is operationally convenient for integration into health care systems (eg, ICD-10 diagnostic classifications or vasopressor titration targeting a specific MAP), it drastically oversimplifies a complex physiologic state.

Shock is not a binary state defined by the presence or absence of hypotension but rather a dynamic interplay between physiologic stress and physiologic reserve. Compensated shock may be conceptualized as a state where physiologic reserve is adequate to counter the imposed physiologic stress. As this balance shifts—due either to escalating stress or declining reserve—the patient transitions into decompensated shock, marked by progressive tissue hypoperfusion. Viewed through this lens, shock may be best understood as a continuum triggered by one or more inciting events that induce a state of physiologic stress, which then evolves to tissue hypoperfusion and eventually end-organ dysfunction as compensatory mechanisms begin to fail (**Fig. 1**).

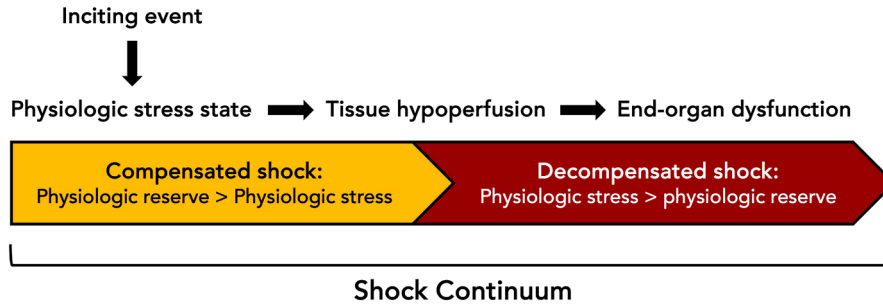

Fig. 1. Shock continuum.

It is essential to recognize that compensated shock represents a physiologic stress state wherein sympathetic drive often maintains normotension—or even hypertension—which can mask underlying hemodynamic instability. A classic example occurs in young, previously healthy trauma patients who, despite substantial blood loss, are hypertensive on initial presentation due to a robust catecholamine surge, and then rapidly deteriorate after administration of analgesics or sedatives that blunt their sympathetic drive and unmask the true severity of circulatory compromise. It is equally important to recognize that progression to decompensated shock is not necessarily associated with the development of hypotension, and that a patient may have severe tissue hypoperfusion and evolving end-organ dysfunction while maintaining a normal mean arterial pressure (MAP).

Shock Recognition

Reliance on hypotension as a defining feature of shock can delay recognition and initiation of appropriate management. However, if clinicians are to move beyond equating shock with hypotension, a key question emerges: *How, then, should shock be recognized?* Unfortunately, there is no single diagnostic test, laboratory value, or clinical score capable of definitively identifying shock. Shock may manifest in enormously different ways depending on factors such as the patient's age, comorbid conditions, physiologic reserve capacity, and underlying shock etiology. What shock looks like in an otherwise healthy 18 years old with massive hemorrhage is likely to be very different than what it looks like in an 85 years old with end-stage renal disease on beta blockers presenting with acute decompensated heart failure.

While blood pressure inarguably remains a critical physiologic parameter, it must be understood as one of multiple variables that inform the recognition and management of shock, rather than its defining feature. A pragmatic, multivariate bedside approach to the recognition of shock emphasizing clinically accessible parameters that can be readily evaluated by providers across diverse care settings is outlined in **Table 1** and is organized into three domains: physical examination, noninvasive hemodynamic monitoring, and laboratory evaluation. No single variable should be interpreted in isolation as definitive evidence for or against the presence of shock; instead, these data points must be integrated to form a clinical gestalt regarding the likelihood of shock in an individual patient. A number of these variables may reflect a patient's current position along the shock continuum, and whether the dominant physiologic process at the time of assessment is adaptive sympathetic activation or overt tissue hypoperfusion. For example, at the point along the continuum when a physiologic stress response predominates, the patient might be agitated and diaphoretic, and then become obtunded and mottled as tissue hypoperfusion evolves.

Table 1
Clinical data points suggestive of shock

Physical Exam	Laboratory Tests	Noninvasive Monitoring
• Mental status (agitation, confusion, obtundation) • Increased respiratory accessory muscle use • Skin examination (diaphoresis, mottling) • Capillary refill time >3 s • Oliguria	• Elevated lactate • Metabolic acidosis (HCO_3 and/or base excess) • Leukocytosis or leukopenia • Hyper- or hypoglycemia • Evidence of end-organ dysfunction (Cr, liver function tests [LFTs], troponin) • Elevated veno-arterial P_{CO_2} gap	• Hypotension • Tachycardia • Tachypnea • Hypothermia • Poor SpO_2 waveform • Low plethysmographic peripheral perfusion index

Shock Differential Diagnosis: A Physiology-First Approach

Once shock is recognized, the next critical task is identifying its underlying etiology. Timely and accurate differentiation among the various causes of shock is essential to initiate appropriate management, but it is critical not to conflate the *presence* of shock with the *etiology* of shock. One of the most common—and potentially most harmful—errors made by clinicians is to reflexively give intravenous fluids to a hypotensive patient with an elevated lactate. While an elevated lactate may suggest that a patient *is* in shock, it in no way reflects *why* the patient might be in shock.

Basing shock differential diagnosis on the classification of patients into one of four discrete categories—hypovolemic, distributive, cardiogenic, or obstructive—offers a fast, accessible heuristic that is a useful starting point for evaluation of shock in many patients; however, this approach risks oversimplifying a complex, dynamic physiologic state by emphasizing diagnostic labeling over a mechanistic approach to circulatory failure. The limitations of a categorical approach to shock are not simply academic. Classifying a patient into a specific shock subtype presumes that most clinical presentations may be cleanly categorized into a single pathophysiologic entity. It also implies that the precipitating cause of shock remains the dominant driver of ongoing shock physiology. Most importantly, assigning a patient to a given shock category then prescribes a limited set of stereotyped interventions: fluid resuscitation for hypovolemia, vasopressors for distributive shock, inotropes for cardiogenic shock, and decompression for obstructive shock. If a patient fails to respond to these interventions, clinicians often find themselves stranded in "now what?" moments for which they may lack a robust mental model to guide further diagnostic reasoning or management decisions.

Point-of-care ultrasound has become a central tool in shock differential diagnosis in the acutely ill patient.[2] While protocolized approaches such as the rapid ultrasound in shock exam provide an efficient and structured approach to obtaining key data points regarding shock etiology, if these data points are simply funneled back into the traditional four-category classification of shock, the same limitations and conceptual constraints remain. More importantly, interpreting ultrasound findings outside of the context of a thorough and nuanced understanding of shock physiology can be misleading and has the potential to inadvertently cause harm by bolstering clinician confidence in potentially erroneous conclusions. For example, a hyperdynamic left ventricle (LV) is often interpreted to reflect hypovolemia. While this is one possible

explanation for this finding, it may also be seen in the context of vasoplegia or acute valvular regurgitation—both of which require management strategies distinct from volume administration. With the growing use of point-of-care ultrasound in resuscitation of critically ill patients, a "physiology-first, ultrasound-second" approach is becoming increasingly essential: ultrasound should be used as a tool to test and refine physiologic hypotheses rather than as a standalone diagnostic endpoint. For a discussion of incorporating ultrasound in the assessment of shock based on a sophisticated yet bedside-applicable shock mental model of hemodynamic interfaces, see Rola and colleagues.[3]

PRESSURE VECTOR MENTAL MODEL OF SHOCK PHYSIOLOGY

Equating shock with hypotension is not only clinically problematic but also physiologically inaccurate. Two central issues underlie this disconnect: (1) the complex relationship between the microcirculation and macrocirculation and (2) the critical distinction between pressure and flow in macrocirculatory physiology.

Microcirculation Versus Microcirculation: Hemodynamic Coherence

There is increasing recognition of the potential for disassociation between systemic hemodynamics (eg, blood pressure, cardiac output) and the state of the microcirculation. This disassociation is clinically relevant because microcirculatory dysfunction seems to be associated with adverse patient outcomes in a manner that is independent of macrocirculatory variables,[4] and improvements in systemic hemodynamics are not always associated with parallel improvements at the microcirculatory level. Key mechanisms of microcirculatory dysfunction that may result in this type of disassociation include heterogeneous microcirculatory flow, reduced capillary density induced by hemodilution and anemia, microcirculatory flow reduction caused by arteriolar vasoconstriction or tamponade, and interstitial edema.

Although emerging technologies are beginning to offer novel approaches to directly assessing tissue perfusion,[5] many of these tools remain limited in availability, particularly in emergency settings. There are, however, multiple bedside surrogates of microcirculatory dysfunction that are easily accessible to emergency clinicians and are included in **Table 1**: mottling, capillary refill time, serum lactate concentration, the veno-arterial P_{CO_2} gap,[6] abnormalities in pulse oximetry waveforms, and the plethysmographic peripheral perfusion index.[7] Attention to these parameters, and an appreciation of their significance as markers of microcirculatory dysfunction, may help clinicians avoid overreliance on blood pressure in the recognition and management of shock. This article focuses primarily on macrocirculatory physiology, for a comprehensive review of mechanisms of microcirculatory shock, see Ince.[4]

Macrocirculatory Physiology: Pressure Versus Flow

While arterial blood pressure is the most readily measured and commonly referenced parameter in the assessment of systemic hemodynamics, it is in fact forward flow that serves as the principle determinant of tissue perfusion at the macrocirculatory level. In clinical practice, pressure and flow are often treated as equivalent; however, they represent very distinct physiologic concepts. Pressure refers to the force exerted per unit area, while flow refers to the volume of fluid moving through a vessel over time, and it is flow—*not* pressure—that is the critical determinant of tissue perfusion. The relationship between flow (Q), pressure gradient (P), and resistance (R) is a foundational concept of cardiovascular physiology and expressed as the equation $Q = \Delta P/$

R.[8] The clinically relevant upshot of this equation is that blood pressure—although correlated with flow under many conditions—does not necessarily reflect the adequacy of circulatory function. While upstream pressure is a critical driver of flow, absolute upstream pressure does not guarantee adequate flow, and therefore, blood pressure cannot be fully relied upon as a surrogate of tissue perfusion. To determine whether a given upstream pressure results in sufficient forward flow, it must be taken in the context of the pressure gradient (ie, the upstream-to-downstream pressure differential) and the resistance.

Three Pressure Vector Sum Shock Framework

While equation $Q = \Delta P/R$ is a foundational principle of circulatory physiology, translating this principle into actionable decisions in real time remains a challenge in clinical practice, especially under the time pressure of the initial resuscitation phase in a critically ill patient. The variables of pressure gradients and resistance are often understood in the form of isolated abstract concepts rather than as dynamic and interdependent components of a unified system governing tissue perfusion at the bedside. Furthermore, the flow equation does not encompass clinically important causes of compromised forward flow such as tension pneumothorax or cardiac tamponade.

Tissue perfusion requires adequate forward flow across capillaries. Forward flow is dictate by the vector sum of forward, backward and external pressures (Fig. 2). It is proposed that employing a vector sum heuristic simplifying the flow equation into components of forward pressure and back pressure—and then incorporating the concept of external pressure to account for pathologic states such as tension pneumothorax and cardiac tamponade—may offer a conceptual framework to facilitate bedside integration of complex hemodynamic principles by presenting them in a form that is more intuitive and adaptable to diverse clinical scenarios.

This framework requires a shift away from a narrow focus on forward pressures—blood pressure, left ventricular systolic function, and intravascular volume status—as the central organizing principles of shock evaluation and management. While these variables are unquestionably important, their primacy in shock assessment reflects a reductionist view that exclusively focuses on forward pressure as the principle determinant of perfusion. Viewing circulatory physiology instead through the lens of a vector sum framework enables clinicians to pose questions such as whether inadequate perfusion may be stemming from excessive back or external pressures rather than simply insufficient forward pressure, or—as is often the case—some combination thereof.

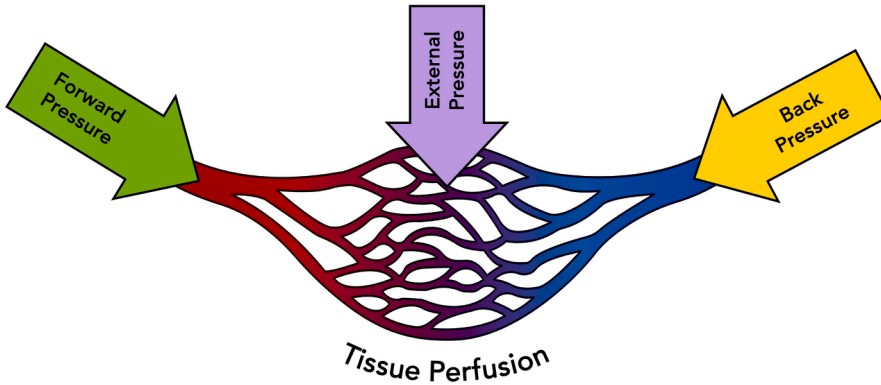

Fig. 2. Forward flow as the net vector sum forward, back, and external pressures.

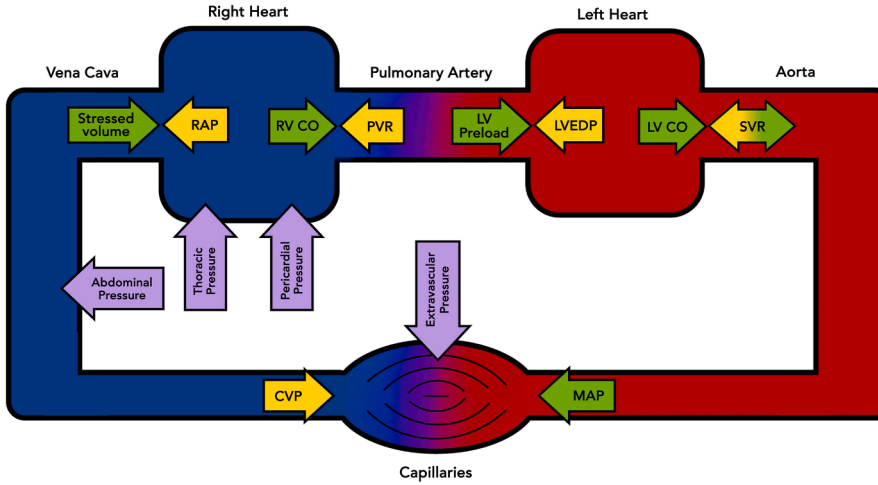

Fig. 3. Mapping of forward, back, and external pressures onto a simplified model of the circulatory system. CVP, central venous pressure; LV CO, left ventricular cardiac output; LVEDP, left ventricular end diastolic pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure; RV CO, right ventricular cardiac output; SVR, systemic vascular resistance.

MAPPING FORWARD AND BACK PRESSURES AS HEMODYNAMIC INTERFACES

Fig. 3 maps this vector sum model onto a simplified representation of the circulatory system in order to create a framework for translating clinical data into testable hypotheses about the various factors contributing to hypoperfusion in an individual patient. Rather than considering macrohemodynamic variables in isolation, forward, backward, and external pressures are organized around pressure interfaces, with the net vector sum at each interface determining the resultant forward flow. Significant impairment at any one of these interfaces can precipitate a shock state. However, shock may also arise from more modest impairments at multiple interfaces, and refractory shock may reflect a failure to recognize that more than one interface is compromised, or that an intervention to improve forward flow at one interface has inadvertently worsened forward flow at another. This highlights the importance of understanding these interfaces not only as discrete elements but also as components of an integrated system. While the core physiology underlying each interface is well established in medical education, clinicians at the bedside often struggle to apply this knowledge holistically. This may stem, in part, from the various components of circulatory physiology being taught as isolated concepts without a unifying framework to synthesize and operationalize these concepts in the context of clinical decision-making.

In this section, 5 key forward pressure–back pressure interfaces are described. The following section will explore the effect of various external pressures on these interfaces. Rather than prioritizing technical precision, this model attempts to frame physiologic concepts to capture the core meaning of each interface while intentionally favoring terminology that is most intuitive and familiar to the bedside clinician.

1. MAP and central venous pressure (CVP)
2. Left ventricular cardiac output (LV CO) and systemic vascular resistance (SVR)
3. Left ventricular preload (LV preload) and left ventricular end diastolic pressure (LVEDP)
4. Right ventricular cardiac output (RV CO) and pulmonary vascular resistance (PVR)

5. Stressed volume versus right atrial pressure (RAP)

The physiology of each of these key pressure interfaces will now be explored in more detail, beginning with the familiar concept of MAP.

Mean Arterial Pressure–Central Venous Pressure Interface

Forward pressure: MAP
Back pressure: CVP

Key physiology

- This interface represents the macrohemodynamic final common pathway across the capillaries that governs tissue perfusion. While clinician's have a theoretic knowledge of the equation perfusion pressure = MAP–CVP, it is often assumed that the magnitude of CVP (on the order of 5 mm Hg) is sufficiently negligible relative to that of MAP (on the order of 5 mm Hg) to be functionally ignored in practice. This perception, however, overlooks the fact that the most significant pressure drop in the circulatory system happens *before* the level of the capillaries (4a).[9] As a result, the relevant forward pressure driving the pressure gradient at the level of the precapillary arteriole is considerably lower—around 25 mm Hg—making a back pressure of 5 mm Hg far more competitive in determining the pressure gradient across the capillaries.

Clinical relevance

- Once it is understood that the driving pressure across systemic capillaries is approximately 20 mm Hg under normal physiologic conditions, it becomes readily apparent how even a modestly elevated CVP can exert a disproportionately large opposing force, particularly in the context of a reduced MAP. The various forward and back pressure vectors that ultimately determine the CVP will be discussed below, working backwards from the variables that determine MAP, the LV CO, and SVR

Left Ventricular Cardiac Output–Systemic Vascular Resistance Interface

Forward pressure: LV CO.
Forward and Back pressure: SVR.

Key physiology

- A fundamental principle of circulatory physiology is that volume is not usable until it is pressurized. SVR acts to pressurize the volume that is output by the LV, converting liters per minute into forward pressure. In conditions of vasodilation (eg, infection, inflammation), SVR can fall dramatically, and forward pressure decreases due to failure to adequately pressurize the stroke volume ejected by the LV.
- At the same time, the SVR also represents the primary component of left ventricular afterload, that is, the back pressure opposing LV CO. The LV is generally well adapted to handle afterload and is able to maintain a relatively stable stroke volume even when confronted with significantly elevated SVR (**Fig. 4**B).[10] In patients with systolic dysfunction, however, stroke volume may become highly sensitive to even modest increases in SVR.
- The ability of the LV to generate forward flow is dependent on adequate preload, as described by the Frank–Starling relationship. As ventricular filling increases, stroke volume rises accordingly—up to a point—beyond which further increases yield diminishing returns. In patients with impaired systolic function, this slope of the Frank–Starling curve is reduced, reflecting a blunted stroke volume response to changes in preload (**Fig. 4**C).[11]

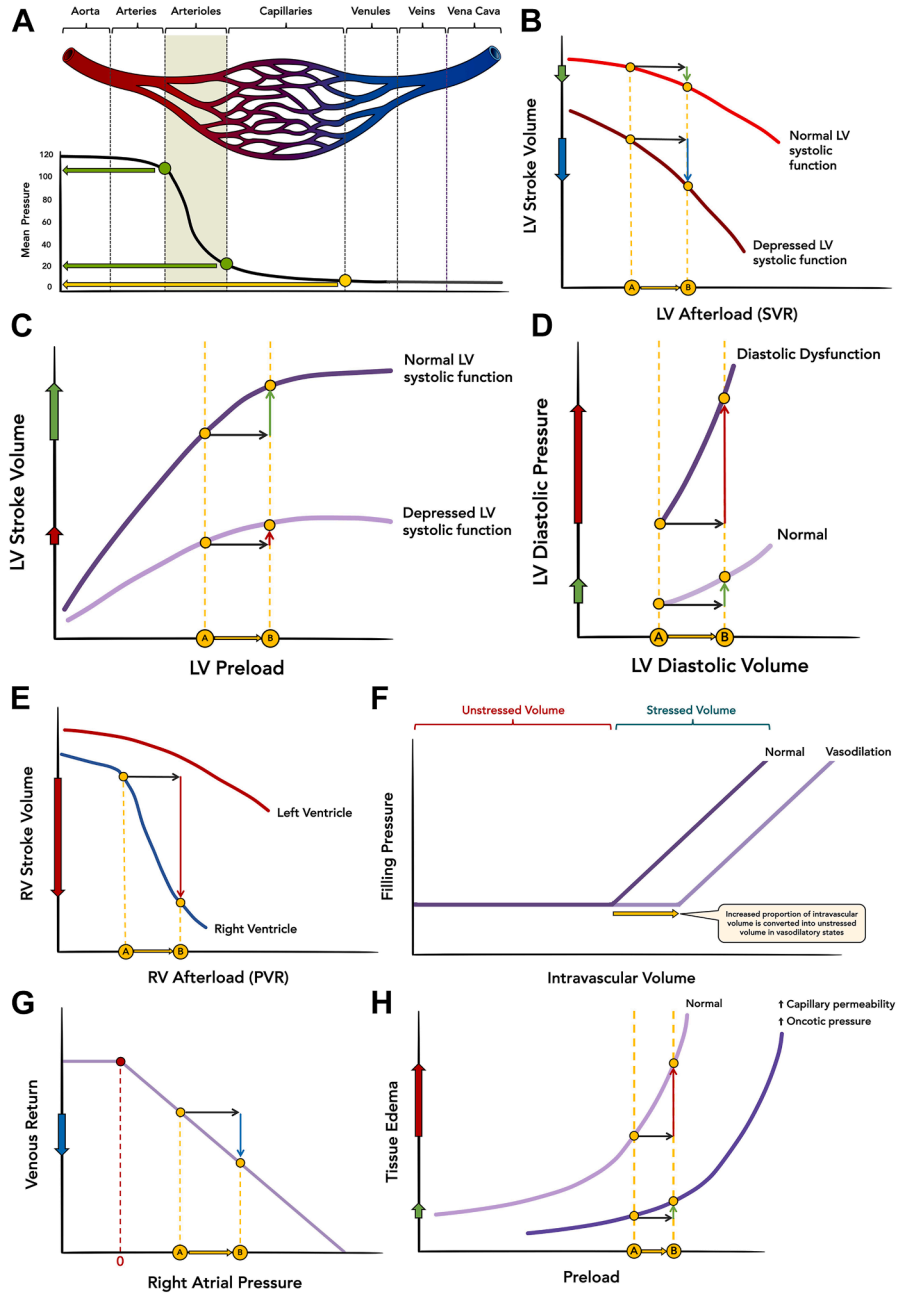

Fig. 4. (*A–H*) Physiology of pressure interfaces. LV, left ventricle; PVR, pulmonary vascular resistance; RV, right ventricle; SVR, systemic vascular resistance. ([*A*] Boryczko, K., Dzwinel, W. & A.Yuen, D. Dynamical clustering of red blood cells in capillary vessels. J Mol Model 9, 16–33 (2003). https://doi.org/10.1007/s00894-002-0105-x.[9–16])

Clinical relevance

- The hyperdynamic-appearing LV function often observed in low-SVR states is often not indicative of increased inotropy nor decreased intravascular volume but rather simply decreased back pressure at the LV CO–SVR interface. Conversely, patients with reduced systolic function may benefit from a reduction in SVR to optimize forward flow at this interface.
- Patients with systolic dysfunction are more likely to reside on the flatter portion of the Frank–Starling curve, meaning they are less likely to be able to augment stroke volume in response to increased preload. In such cases, reduced LV CO is not due to inadequate volume delivery to the LV, but to insufficient contractile force to convert preload into effective forward pressure. At the same time, it is critical not to conflate the slope of the Starling curve with a patient's position along its *x*-axis. Even in the setting of reduced systolic function, a patient may still reside on the ascending limb of the curve and thus benefit from fluid administration (eg, a patient with chronic systolic heart failure presenting in septic shock). Conversely, patients with normal LV contractile function may be positioned on the flat portion of the curve and therefore not fluid responsive.

Left Ventricle Preload–Left Ventricular End Diastolic Pressure Interface

Forward pressure: LV preload.
Back pressure: LVEDP.

Key physiology

- LV preload represents the volume of blood delivered to the LV at end-diastole and may be thought of as the forward pressure that drives filling of the LV in preparation for the next systolic contraction. Preload is often simplistically referenced in clinical practice as though it were a straightforward concept that is essentially equivalent to "volume status". In reality, preload is one of the most complex elements of macrohemodynamic physiology. While intravascular volume status is indeed one component that dictates preload, systemic vascular tone and right-sided cardiac output are also crucial determinants of LV preload, as will be discussed in the sections below.
- LVEDP acts as the back pressure to LV diastolic filling. In a normal LV, LVEDP does not generally act as a significant competing back pressure because LV compliance allows the chamber to accommodate increases in diastolic volume with relatively minimal increases in pressure. However, in the setting of a stiff or small LV due to diastolic dysfunction and/or LV hypertrophy, the pressure–volume curve becomes markedly steep (**Fig. 4**D),[12] and LVEDP begins to represent a significant back pressure to LV filling.

Clinical relevance

- Consequent to a steep pressure–volume curve patients with poor LV compliance tend to be highly sensitive to rapid volume shifts. On the one hand, small increases in volume may result in disproportionately large increases in back pressure that may be transmitted to the lungs and precipitate a rapid onset of pulmonary edema. On the other hand, removal of relatively small amounts of volume can lead to abrupt drops in blood pressure, and these patients may require additional volume administration to maintain filling pressures sufficient to overcome the back pressure posed by elevated LVEDP.

Right Ventricular Cardiac Output–Pulmonary Vascular Resistance

Forward pressure: RV CO.
Back pressure: PVR.

Key physiology

- A failure of forward flow at this interface effectively divorces systemic venous return and left heart filling because the LV cannot generate cardiac output if the right heart is not delivering any preload to it. In the presence of poor RV CO and/or elevated RV afterload, volume cannot be transferred to the left heart irrespective of the intravascular volume status.
- In contrast to the LV, the RV is exquisitely sensitive to afterload, with a dramatic drop in RV stroke volume in response to even relatively small increases in pulmonary pressures (**Fig. 4**E).[13] As a result, back pressure becomes a primary determinant of forward flow at this interface.

Clinical relevance

- RV failure is more often secondary to increased afterload (eg, pulmonary arterial hypertension, pulmonary embolism, lung disease) rather than intrinsic impairment of RV contractile function (eg, RV myocardial infarction). Elevated RV afterload also frequently occurs in the setting of increased back pressures transmitted from the left side of the heart (eg, left heart failure, mitral regurgitation).
- The frequently cited assertion that the RV is "preload dependent" and that RV failure should consequently be treated with volume expansion is only physiologically coherent under conditions of normal back pressure at the RV CO–PVR interface. Administration of volume under conditions of increased back pressures at the RV CO–PVR interface not only fails to improve forward flow but also can in fact precipitate clinical deterioration: the RV, unable to effectively move volume forward against high afterload becomes distended, leading to RV dilation with consequent worsening of contractile function and ultimately impaired LV filling due to decreased RV CO as well as interventricular septal shift increasing LVEDP as a back pressure to LV preload. In this context, effective management instead requires interventions aimed at reducing RV afterload such as pulmonary vasodilators to lower PVR and/or volume offloading to reduce elevated back pressures transmitted from the left side of the heart.

Stressed Volume–Right Atrial Pressure Interface

Forward pressure: stressed volume.
Back pressure: RAP.

Key physiology

- As discussed above, volume in the circulatory system is only usable if it is pressurized. As such, venous return to the right side of the heart depends not only merely on the absolute volume within the vascular compartment but also specifically on the portion of that volume that is pressurized (ie, stressed volume). Unpressurized intravascular volume (ie, unstressed volume) does not contribute meaningfully to forward pressure at this interface; until the venous system is filled to the point of creating wall tension to pressurize this volume, adding further volume to the system will not translate into an increase in pressure (**Fig. 4**F).[14]

- The RAP serves as the back pressure opposing venous return to the right side of the heart. Increases in RAP—whether due to volume overload, RV dysfunction, or elevated intrathoracic pressure—diminish the pressure gradient between the peripheral and the central circulation, thereby reducing venous return (Fig. 4G).[15] In this context, modest reductions in RAP may enhance venous return by increasing the pressure gradient between the peripheral venous system and the right atrium. This relationship, however, is nonlinear: as RAP falls toward zero, the collapsible nature of the venous system becomes a limiting factor, leading to a plateau in venous return despite further decreases in RAP.

Clinical relevance

- The clinical relevance of stressed volume becomes particularly apparent in patients with vasodilation or outright vasoplegia (eg, sepsis, systemic inflammation). Large volumes of fluid may be administered with minimal improvement in forward pressure; this is not due to absolute hypovolemia but rather the compromised ability to pressurize that volume. In this case, vasopressor support is required in order to improve vascular tone and pressurize the currently existent intravascular volume, converting it from unstressed to stressed volume.
- The key clinical consideration in understanding the back pressure at this interface is that elevated RAP should not necessarily be interpreted as a surrogate of intravascular volume status, but rather as a potential back pressure to venous return. Therapies that lower RAP, such as diuresis or PRV reduction in right heart failure, can actually improve LV preload by augmenting the volume delivered to the LV by the right side of the heart. On the other hand, patients with elevated LVEDP (ie, diastolic dysfunction, hypertrophic cardiomyopathy) often require higher RAP to maintain forward pressures that are able to effectively compete with back pressures resulting from the elevated LVEDP.

The convergence of these pressure interfaces now brings us full circle back to CVP, which acts as the back pressure at the macrocirculatory final common pathway for tissue perfusion, the MAP–CVP interface.

We now move on to explore the role of the third pressure in the vector sum model: external pressure.

INFLUENCE OF EXTERNAL PRESSURES ON HEMODYNAMIC INTERFACES

In addition to forward and back pressures, external pressures also exert a critical influence on forward flow in the circulatory system. This manifests most dramatically in presentations such as tension pneumothorax, cardiac tamponade, or abdominal compartment syndrome, where elevated external pressures become the dominant force driving hemodynamics. In these settings, the external pressure completely overwhelms the variables that normally dictate forward flow at a given pressure interface, often resulting in circulatory collapse. While the most striking consequences of elevated external pressures are these hyperacute—but relatively rare—presentations, external pressures also manifest in more subtle but nevertheless clinically significant ways.

Clinically important external pressures are mapped onto the hemodynamic framework in Fig. 3 to understand their interaction with the forward–back pressures interfaces discussed in the section above. These include:

1. Intrathoracic pressure
2. Pericardial pressure
3. Intra-abdominal pressure
4. Extravascular tissue pressure

Intrathoracic Pressure

Key physiology

- This external pressure acts as a compressive force at the stressed volume–RAP interface, reducing the venous return to the right side of the heart with a resultant decrease in the forward pressure that the RV CO is able to generate to fill the LV.

Clinical relevance

- The most dramatic clinical presentation of this physiology occurs in the context of a tension pneumothorax. It can also emerge in more subtle ways, such as the hemodynamic effects of positive pressure ventilation. While the extreme degree of external pressure elevation associated with a tension pneumothorax invariably results in circulatory compromise and represents a primary cause of shock, more modest increases in intrathoracic pressure—such as those induced by elevated positive end-expiratory pressure (PEEP)—can produce variable hemodynamic effects depending on the underlying etiology of shock. In states where cardiac output is dependent on enhanced venous return (eg, hypovolemia or vasodilation), the application of PEEP may exacerbate hypotension by further reducing venous return. Conversely, in patients with left ventricular dysfunction, a reduction in venous return may in fact lead to improved hemodynamics by decreasing pulmonary congestion and left ventricular afterload.

Pericardial Pressure

Key physiology

- The physiology of elevated pericardial pressures is functionally identical to that of elevated intrathoracic pressures, acting as a compressive force at the stressed volume–RAP interface.

Clinical relevance

- This physiology results in precipitous hemodynamic deterioration in the setting of overt pericardial tamponade. However, it may also present more insidiously in patients with chronic or small pericardial effusions, or in those with restrictive cardiomyopathy. In such cases, elevated pericardial pressures may not be the primary driver of shock but can serve as a contributing factor to impaired net forward flow when superimposed upon other hemodynamic derangements such as hypovolemia or vasodilation.

Intra-abdominal Pressure

Key physiology

- Elevated intra-abdominal pressures impair venous return to the right heart by acting as an external pressure that exerts a compressive force at the stressed volume–RAP interface. In addition, elevated intra-abdominal pressures can cause mesenteric ischemia by compromising forward flow at the MAP–CVP interface in the intestinal tissues.

Clinical relevance

- Similar to pericardial tamponade and tension pneumothorax, abdominal compartment syndrome represents a rapidly progressive form of shock that can only be addressed by emergent mechanical decompression. However, more modest elevations in intra-abdominal pressures—as seen in conditions such as ascites or bowel obstruction—may not independently precipitate a shock state but can be a variable contributing to decreased forward flow, particularly when superimposed on vasodilation and/or hypovolemia.

Extravascular Tissue Pressure

Key physiology

- Extravascular tissue pressure acts as an external pressure that compromises forward flow at the MAP–CVP interface by direct compression at the level of the capillaries.
- The relationship between preload and tissue edema is nonlinear and characterized by a curve with a significant inflection point (Fig. 4H).[16] Before the inflection point, increasing preload leads to relatively minimal increases in extravascular fluid; beyond the inflection point small increases in preload cause a rapid rise in extravascular fluid. It is important to note that major factors such as increased capillary permeability or decreased oncotic pressure that shift this curve to the left (ie, significant tissue edema develops at a lower preload) are independent of LV function.

Clinical relevance

- While the most overt example of extravascular tissue pressure impairing perfusion is compartment syndrome—which results in isolated regional hypoperfusion—more subtle elevations in tissue hydrostatic pressure (eg, total body fluid overload or inflammatory states with capillary leak) can also significantly compromise forward flow at the MAP–CVP interface. Although tissue hydrostatic pressures are typically low in absolute terms, their significance as an external pressure becomes evident when one considers that the forward-to-back pressure gradient across this interface is on the magnitude of 20 mm Hg under normal physiologic conditions (Fig. 4A).[9] As such, even relatively modest increases in external tissue pressure have the potential to reduce forward flow at this pressure interface.
- The effect of external pressure at this interface becomes magnified in the presence of increased back pressure at the MAP–CVP interface, and elevated extravascular tissue pressure causes a "second hit" in patients with both intravascular and extravascular volume overload (eg, acute decompensated heart failure), where both tissue hydrostatic pressure and CVP are increased.

APPLICATION OF PRESSURE VECTOR MODEL OF SHOCK

Approaching hemodynamics through the lens of pressure vectors attempts to provide a framework for shock physiology that balances sophistication with clinical utility and provides a more robust toolset for assessment and management of complex shock patients. This framework is not meant to replace the four conventional diagnostic categories but instead aims to expand upon them, enabling a smoother cognitive shift toward a more nuanced mental model of shock physiology as patient complexity increases.

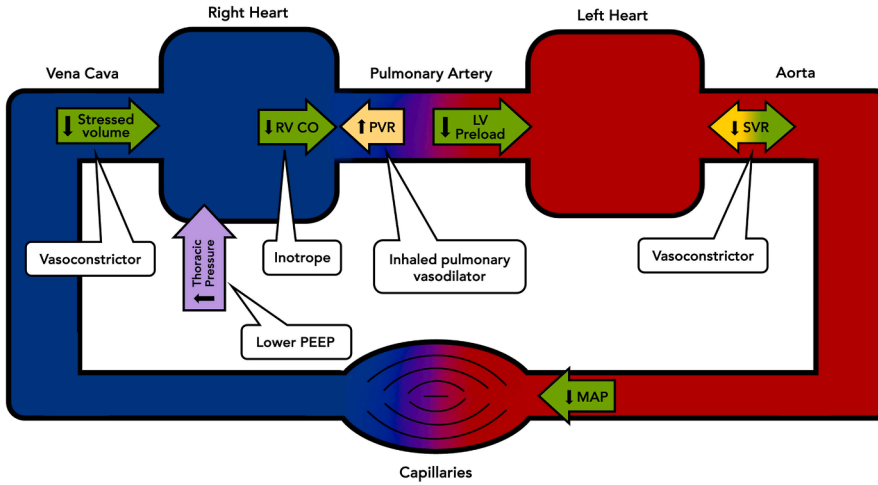

Fig. 5. Pressure interface map labeled with associated suggested management interventions using the example of a patient presenting with septic shock, subsequently develops a massive pulmonary embolus, and is intubated and initiated on high PEEP for hypoxemia.

Traditional shock classifications can be readily mapped onto this vector-sum framework and doing so can facilitate parsing of management-relevant distinctions within a given category. For instance, tension pneumothorax, pericardial tamponade, and massive pulmonary embolism all fall under obstructive shock in the conventional schema. Using the pressure-vector map, it is apparent that tension pneumothorax and pericardial tamponade have functionally similar pathophysiology: excessive external pressure at the stressed volume–RAP interface. Massive pulmonary embolus, however, represents a fundamentally different pathophysiology: increased back pressure at RV CO–PVR interface.

Ultimately, the real value of this framework arises from its ability to map complex shock states in a way that makes apparent more nuanced—and at times seemingly counterintuitive—management interventions. An example of this is outlined in Fig. 5 using the case of a cancer patient who initially presents with sepsis, subsequently develops a massive pulmonary embolus, and is then intubated and initiated on high PEEP for hypoxemia.

SUMMARY

This article proposes a conceptual framework for approaching shock that attempts to balance physiologic sophistication with bedside utility. The first component is based on shock recognition in a continuum progressing from physiologic stress to overt tissue hypoperfusion. The second is framing shock differential diagnosis and management around identification and correction of disturbances in forward flow, conceptualized as the net sum of competing forward, backward, and external pressures.

While this mental model introduces greater complexity compared to currently prevalent approaches to shock in the emergency department, the potential clinical yield of incorporating a more nuanced understanding of shock into clinical practice may justify the additional cognitive investment required. In shock patients—particularly those who continue to deteriorate despite initial interventions—decisions made within the first hours of care can profoundly influence the entire clinical trajectory. These early

choices may determine not only whether a patient survives hospitalization but also whether they experience a brief intensive care unit stay or a prolonged critical illness, and whether they are ultimately discharged home with full functional recovery or to a long-term care facility with significant residual morbidity. When considering the substantial time clinicians devote to engaging with medical literature, assimilating new guidelines and algorithms, and adopting emerging technologies, it is reasonable to advocate for a parallel investment in cultivating improved physiologic reasoning abilities.

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