Running from a Bear: How We Teach Vasopressors, Adrenoreceptors, and Shock

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ABSTRACT

Vasopressors are widely used in the management of shock among critically ill patients. The physiology of vasopressors and adrenoreceptors and their effects on end organs therefore represent important, high-yield topics for learners in the critical care environment. In this report, we describe our approach to teaching this core concept using the stereotypical human physiologic response when running from a bear, in the context of the relevant supporting literature. We use escaping from a threatening predator as a lens to describe the end-organ effects of activating adrenoreceptors together with the effects of endogenous and exogenous catecholamines and vasopressors. After reviewing this foundational physiology, we transition to the clinical environment, reviewing the pathophysiology of various shock states. We then consolidate our teaching by integrating the physiology of adrenoreceptors with the pathophysiology of shock to understand the appropriateness of each therapy to various shock phenotypes. We emphasize to learners the importance of generating a hypothesis about a patient's physiology, testing that hypothesis with an intervention, and then revising the hypothesis as needed, a critical component in the management of critically ill patients.

Vasopressors are fundamental to the management of shock in the intensive care unit (ICU), forming a critical topic for learners. The sympathomimetic effects of catecholamines and exogenous vasopressors typically enhance vascular tone and cardiac output (CO) and are intended to sustain critical end-organ function in states of hypoperfusion in various forms of shock. In addition to their excitatory properties on vascular smooth muscle and the myocardium, vasopressors have other effects on end organs that are important to be aware of.

Our goal is to describe a memorable and engaging blueprint for our approach to

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ATS Scholar Vol 4, Iss 2, pp 216–229, 2023

DOI: 10.34197/ats-scholar.2021-0132HT

teaching this high-yield topic in critical care medicine: vasopressors, their receptors, their physiologic effects on end organs, and their use in various shock states.

WHO ARE THE LEARNERS?

Our approach may be appropriate for learners across the medical education spectrum but was originally developed for medical students, residents from various medical and surgical subspecialties, pharmacy students and residents, critical care fellows, and other learners in the ICU. We encourage including learners from multiple disciplines to enhance and enrich discussion and facilitate interprofessional education. With this intended audience in mind, we have incorporated principles of adult learning theory, such as drawing on life experiences to contextualize learning, highlighting the practical relevance of the teaching, and encouraging self-directed learning (1).

WHAT IS THE SETTING?

Our approach is most naturally suited for the clinical setting of the ICU given the prevalence of shock among critically ill patients. Beyond this, we find our approach to be highly adaptable to various teaching formats. As outlined further below, we conceptualize our method to be composed of several discrete modules that may be "mixed and matched" (i.e., included or omitted) to fit the appropriate time constraints of the teaching setting and the needs of learners. The full sequence may be used for small-group teaching sessions with or without a whiteboard (i.e., a "chalk talk") or informal brief "talks" in the workroom. A truncated version could be adapted for bedside teaching. This framework could even be applied to the appropriate material within a formal lecture-based physiology course.

WHAT IS THE CONTENT?

For the purposes of this teaching method, we focus on α-1, β-1, and β-2 adrenoreceptors given the centrality of these receptors in understanding vasopressors. We briefly discuss hormone replacement therapy with vasopressin and cortisol and the inotropic agents dopamine and dobutamine. However, for the purposes of this approach, we do not discuss α-2 receptors (and their negative feedback role in downregulating sympathetic activity), β-3 receptors (and their role in lipolysis and thermogenesis in adipose tissue), angiotensin II, or other inotropes (i.e., levosimendan and milrinone), as these topics tend to distract from the core physiologic principles being conveyed and may be best handled in separate teaching sessions.

WHAT IS THE APPROACH?

Our teaching approach is centered on evolutionary biology. We explain that humans did not evolve to lie in hospital beds with continuous intravenous infusions. Rather, human physiology adapted to respond to survival in the wilderness, hunting for food and escaping ravenous predators, with success necessary to propagate genetic material. For our teaching method, we use the imagined scenario and accompanying physiologic response of running from a fast-approaching bear as a lens through which to illustrate the physiologic effects of sympathetic receptors, endogenous catecholamines, and exogenous vasopressors.

Our method is structured with five modules *1*) setting the scene, *2*) an overview of adrenoreceptors and their physiologic effects on end organs,

3) a review of vasopressors, *4*) a discussion of inotropes and hormone replacement therapy, and *5*) an overview of the pathophysiology of shock phenotypes, applying receptor physiology to the clinical setting.

We consider modules 1, 2, and 3 to represent the core of our teaching approach (uninterrupted, approximately 8–10 min). Module 4 (5 min) expands on the topic and adds complexity and nuance. Module 5 (5 min) challenges learners to apply their knowledge to the clinical setting. The entire teaching approach (20–30 min) progresses from module 1 through module 5. However, these five modules can be organized, divided, or combined in various sequences to suit the intended teaching format, time constraints, and learner level. For example, for a series of brief sessions, one could organize as follows: session 1, modules 1 and 2; session 2, modules 3 and/or 4; and session 3, module 5.

Learning Objectives

After attending this teaching session, learners should be able to *1*) describe the key end-organ effects of α-1, β-1, and β-2 adrenoreceptors; *2*) explain the adrenoreceptor profiles of epinephrine, phenylephrine, and norepinephrine and summarize the mechanisms of vasopressin, dopamine, and dobutamine; *3*) outline the physiology of major shock states; and *4*) apply vasopressor adrenoreceptor physiology toward understanding their clinical use in the management of shock.

MODULE 1: SETTING THE SCENE

We begin by prompting learners to imagine a camper in the wilderness. A bear appears in the distance, pauses, growls deeply, and begins to charge directly at our protagonist. We now pause to discuss and dissect the ensuing sympathetic response in our camper. The sympathetic nervous system is activated, generating a physiologic response (fight or flight) intended to facilitate a speedy and efficient escape, mediated primarily by epinephrine (adrenaline), the chief endogenous catecholamine. With increased epinephrine concentrations, a host of receptors are rapidly excited, all with the dedicated purpose of escaping the bear by activating useful physiologic mechanisms and deactivating those that, in the face of the imminent threat, are not beneficial to our camper's one goal: survival. This framework of "turning on" what is needed and "turning off" what is not is a key principle for conceptualizing vasopressors and their physiologic effects.

MODULE 2: OVERVIEW OF ADRENORECEPTOR PHYSIOLOGY

At this point in our approach, as we begin to discuss the effects of key adrenoreceptors, we suggest incorporating a visual aid (such as a whiteboard or chalkboard), if available, to depict an individual running from a bear, as demonstrated in Figure 1. This schematic template can then be filled in, highlighting the key effects of each adrenoreceptor as they are discussed in turn. Artistic ability is not required; in fact, this juncture represents an opportunity to infuse humor with cartoonish illustrations, keeping the learners engaged. For each figure, we have included a version that can be easily reproduced on a whiteboard paired with a high-quality version with an associated quick response code (allowing easy reference on the fly).

α-1

With the bear charging, our camper must first assess the threat. Pupils dilate to survey the landscape for other predators

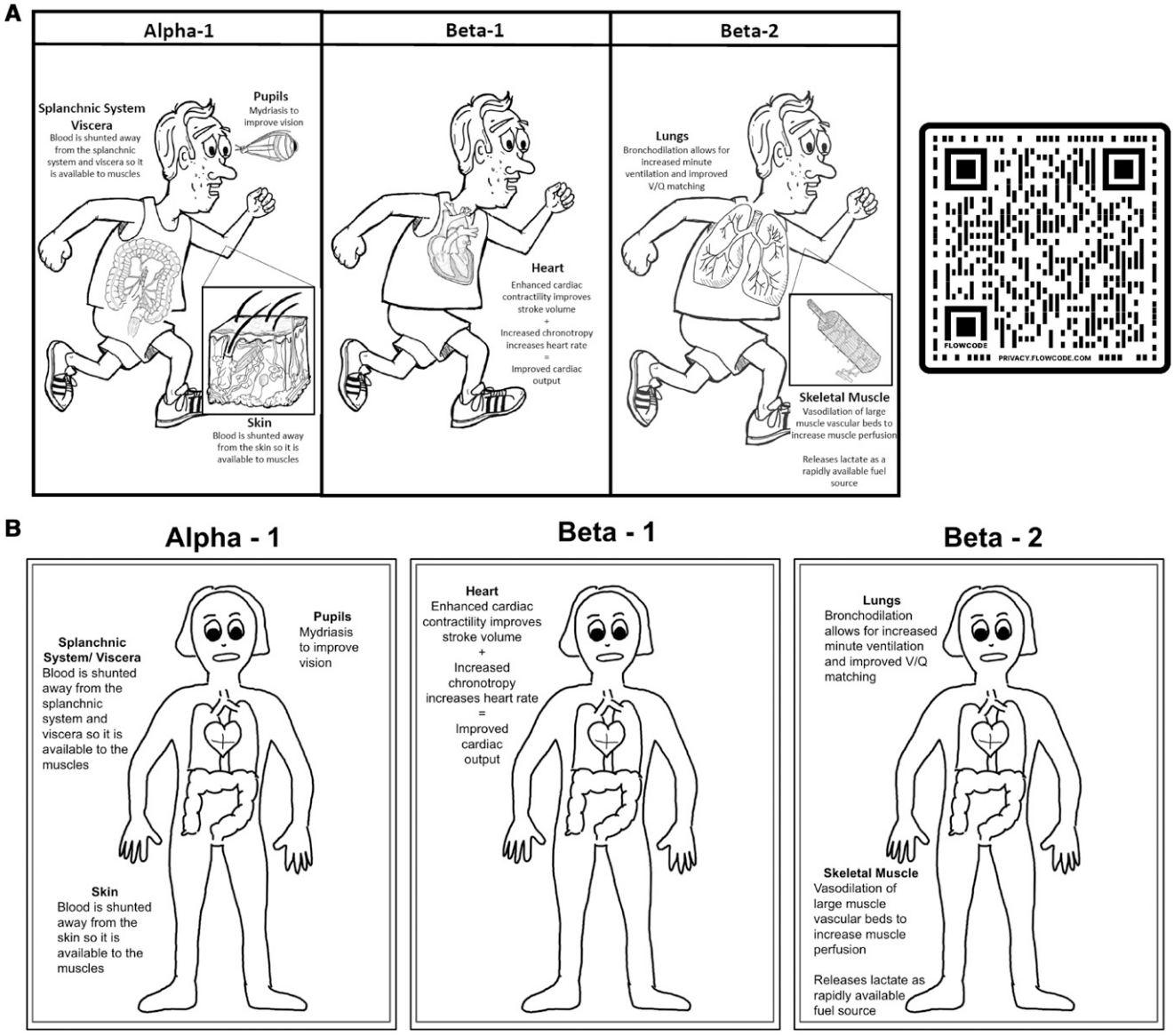


Figure 1. (*A*) End-organ effects of key adrenoreceptors. Agonism of key adrenoreceptors, including α-1, β-1, and β-2, leads to important effects on end organs that mediate the sympathetic response. As illustrated in the left panel, α-1 receptors dilate the pupils (enhancing vision) and mediate vasoconstriction of the splanchnic system and skin (to assist in shunting blood away from nonessential organs during a fight-or-flight response). β-1 receptors, depicted in the center panel, augment cardiac contractility and chronotropy, increasing stroke volume and heart rate, together resulting in enhanced cardiac output. β-2 receptors, shown in the right panel, lead to relaxation of smooth muscle. This results in bronchodilation (improving $\dot{V}/\dot{Q}$ matching and increased minute ventilation) as well as vasodilation of the vasculature in the smooth muscle, therefore achieving preferential shunting of blood to the muscles from nonessential organs. β-2 adrenoreceptor activation also leads to the release of lactate as a rapidly available energy source for the muscles and brain. (*B*) End-organ effects of key adrenoreceptors: easily reproducible illustration for whiteboard teaching. $\dot{V}/\dot{Q}$ = ventilation/perfusion.

and identify potential escape routes. Mydriasis is mediated primarily via α-1 receptors (2). In addition, and perhaps more clinically relevant to critical care, α-1 facilitates the diversion and optimization of blood flow for the sympathetic response, shunting blood away from the viscera, skin, and splanchnic system so that it is available to the muscles (3–5). With the assistance of α-1 adrenoreceptors, our camper prepares to escape, pupils dilated and blood shunting away from viscera and skin to where it will be of more use.

β-1

With eyes wide and blood being redirected, our camper is getting ready to move and must augment CO to meet a quickly rising demand for oxygen delivery (Do_2) to the muscles. β-1 adrenergic receptors enhance cardiac contractility and chronotropy by facilitating intracellular Ca^{2+} influx (6). This combination of increased inotropy and chronotropy augments CO and therefore increases Do_2.

Teaching Pearl: "Remember the first time your code pager alarmed? Perhaps you felt your heart thumping." We use this relatable example of fight or flight to remind our learners of some foundational physiology, perhaps on a whiteboard:

$$CO = [\text{Heart rate}] \times [\text{Stroke volume}]$$

$$Do_2 = CO \times (\text{Hemoglobin} \times Sa_{O_2} \times 1.34 + [Pa_{O_2} \times 0.0031])$$

Pa_{O_2} = Pressure of arterial oxygen

Sa_{O_2} = arterial oxygen saturation

β-2

Hopefully by now sprinting along with darting eyes, increased vascular tone, and a racing heart, our camper must meet rising demands for ventilation and oxygenation using β-2 adrenoreceptors. Located on vascular and airway smooth muscle cells, β-2 receptors lead to vasodilation of smooth muscle and bronchodilation of the airways (6, 7). Bronchodilation allows increased minute ventilation and improves ventilation/perfusion matching. In addition, β-2 agonism assists α-1 in the preferential shunting of blood flow: α-1 activation induces vasoconstriction to direct the available blood supply away from the periphery and viscera, where it is not needed acutely, while β-2 activation vasodilates the large muscle vascular bed to increase muscle perfusion.

Teaching Pearl: While discussing lactate, we find this a good time for a brief aside to remind learners that not all lactatemia signifies hypoperfusion: "Remember that patient with asthma with an elevated lactate?" Many other factors can raise serum lactate concentrations, causing type B lactic acidosis, including β-2 receptor excitement from epinephrine and albuterol use in acute asthma exacerbations.

Furthermore, as metabolic demand continues to rise with our subject racing along, an immediate fuel source is needed for the brain (to plan the escape) and for the heart and muscles (to execute that plan). There is no time for gluconeogenesis or glycogenolysis. Importantly, β-2 activation in skeletal muscle releases lactate as a rapidly available fuel source (8, 9), not to be thought of simply as a by-product of metabolism (10–12).

Our subject now sprints across the forest with eyes wide, bronchi dilated, a racing heart vigorously contracting, clamped splanchnic vasculature, muscles filled with blood, and a fighting chance of escaping the bear.

MODULE 3: CATECHOLAMINES AND VASOPRESSORS

After reviewing the physiologic response to normal stress (if being chased by a bear is normal for you) and the role of the major adrenergic receptors, we pivot to discussing catecholamines and vasopressors. Using the prior discussion of receptor physiology (leaving our whiteboard diagram intact for learners' reference), we move to the ICU, where our story informs vasopressor mechanisms of action and physiologic responses.

Bridging the Gaps: We find our teaching methodology to hold many opportunities for learners to create important links from physiology to bedside clinical practice. We find these "Aha!" moments to be valuable, even if not strictly within the scope of our session, but they can be omitted if time is short. As such, we remind learners that while "blown pupils" are often observed in the setting of recent cardiac arrest, assumed to be the consequence of neurologic injury, we can see that the α-1 adrenoreceptor effects of mydriasis from epinephrine, norepinephrine, or phenylephrine may in fact be responsible, confounding neurologic prognostication.

Epinephrine

As the chief endogenous catecholamine (and therefore primary mediator of the fight-or-flight response), we begin our discussion of vasopressors with epinephrine, a potent agonist of α-1, β-1, and β-2 receptors. We find it important to highlight that epinephrine demonstrates dose-dependent receptor effects: at lower doses, potent β-1 effects predominate, but with increasing doses, β-2 and then α-1 receptors gain potency (13). Higher doses do not equate to greater intensity of the same effect but rather slightly different effects. These varied effects may have clinically significant consequences. For example, at increasing doses with more potent β-2 activity, prolonged vasodilation of the musculature may worsen hypotension, and increased lactate production—although not necessarily due to hypoperfusion of end organs—may worsen metabolic acidosis and confound the clinical picture as we try to determine the state of end-organ perfusion (8). Although trials have demonstrated similar outcomes between norepinephrine and epinephrine (14, 15), epinephrine is not the preferred first-line agent for septic shock given its less discriminating receptor activation, with potentially undesirable effects, including the splanchnic vasoconstriction of α-1 (4), the tachyarrhythmias of β-1, and the vasodilation and hyperlactatemia of β-2 (15, 16). However, as an adjunctive vasopressor, it clearly has a role in the treatment of several shock states, especially in the presence of symptomatic bradycardia or decreased cardiac contractility given the strong β-1 effects on chronotropy and inotropy, respectively.

Bridging the Gaps: We remind our learners that epinephrine is the preferred first-line therapy for anaphylactic shock and that its adrenoreceptor activation profile helps explain why: β-1 increases cardiac contractility and CO, α-1 promotes vasoconstriction, and β-2 bronchodilates, mitigates against airway mucosal edema, and prevents further release of histamine and other allergic mediators.

Phenylephrine

We proceed by discussing phenylephrine, explaining that its potent α-1 agonism with minimal β activity (6) causes significant peripheral (and splanchnic) vasoconstriction with little to no inotropic or chronotropic effects. The increase in afterload can activate a baroreceptor-mediated reflex bradycardia (17). After highlighting this physiology, we explain to our learners that phenylephrine may be the preferred vasopressor in the setting of shock, in which there is rapid atrial fibrillation (minimal β-1 effects and therefore no positive chronotropy, as well as some reflex bradycardia), whereas it may be a poor choice for cardiogenic shock (increased afterload with no positive

inotropy) or prolonged shock states (splanchnic vasoconstriction potentially leading to impaired gut integrity).

Norepinephrine

We next discuss norepinephrine, emphasizing that it is a potent α-1 agonist with some β-1 activity and, to a much lesser degree, β-2 activity. Therefore, infusions of norepinephrine lead to peripheral vasoconstriction and some positive inotropy, increasing systolic and diastolic blood pressure, peripheral vascular resistance, and left and right ventricular function (18–20). These effects are beneficial in the vasodilatory pathophysiology of septic shock and may also be in the low-flow state of cardiogenic shock. We take this opportunity to remind our learners that multiple seminal trials have concluded that norepinephrine is superior to dopamine and at least equivalent to epinephrine and vasopressin, establishing norepinephrine as the preferred first-line agent in septic and cardiogenic shock, as per the Surviving Sepsis Campaign guidelines (14–16, 21–25).

MODULE 4: INOTROPES AND HORMONE REPLACEMENT

Inotropes

Dopamine. Dopamine, a precursor to norepinephrine and epinephrine in the catecholamine synthetic pathway, demonstrates dose-dependent effects. At lower doses, it targets D1 (located in the cerebral, coronary, mesenteric, and renal vasculature) and D2 (in the vasculature and renal tissue) receptors, stimulating vasodilation and increased blood flow (26, 27). Although a renally protective dose of dopamine had been previously proposed (28, 29), several recent publications and guidelines have discouraged this use for critically ill patients (30). At intermediate doses, stimulation of D1 and D2 increases and dopamine gains weak affinity for β-1 and stimulates norepinephrine release. As doses increase, affinity for β-1 rises, followed by affinity for α-1, which continues to increase with escalating concentrations (6). With these increasing doses, positive inotropy and chronotropy are achieved and systemic vascular resistance (SVR) increases, explaining the clinical utility of dopamine in hemodynamically significant bradycardia and other forms of cardiogenic shock.

Dobutamine. Dobutamine strongly binds β-1 and, to a lesser degree, β-2 receptors in the heart and peripheral vasculature, respectively. With increases in contractility and heart rate, as well as vasodilation from β-2, dobutamine augments stroke volume and heart rate and therefore CO. It has been suggested that dobutamine also has weak α-1 activity by binding vascular smooth muscle, theoretically offsetting some of the vasodilatory effects of β-2 (31). However, it is largely accepted that dobutamine, despite its potent inotropic effects, may lead to vasodilation and hypotension under some physiologic conditions. Thus, dobutamine may be a prudent choice in cardiogenic shock (with impaired left ventricular pump function) while being less desirable in distributive shock.

Hormone Replacement Therapy

Vasopressin. We emphasize to our learners that vasopressin, or "antidiuretic hormone," is not a catecholamine but a stress hormone produced in the hypothalamus and stored in the posterior pituitary. Vasopressin produces vasoconstriction and releases adrenocorticotropic hormone (6, 17, 32). Although vasopressin does not significantly contribute to maintaining vascular tone

in nonshock states, in vasodilatory shock, hypersensitivity to vasopressin due to relative vasopressin deficiency induces vasoconstriction to mitigate hypotension (33–40).

Vasopressin use, although not associated with improvements in overall mortality, has been shown to decrease norepinephrine dose requirements and may be more effective in early shock states (22, 23), along the same lines as the catecholamine sparing effects of corticosteroids (41–43). Exogenous vasopressin administration has been increasingly adopted over time in patients with sufficiently severe septic shock (16), however, significant heterogeneity remains in its use between institutions (44).

To tie the above physiology back to our allegory, we explain to our learners that in a stress state, vasopressin release contributes to adrenocorticotropic hormone release (to aid in the stress response), peripheral vasoconstriction (to enhance redistribution of blood flow), and increased renal free water reabsorption to maintain vascular volume (and, perhaps, to prevent the urge to urinate while running from a bear). However, the posterior pituitary has limited storage capacity and did not evolve to sustain vasopressin stores for prolonged shock states. Therefore, a short burst of vasopressin release may help while running from a bear, but when stores run out after a prolonged episode of septic shock, hypotension may ensue, and hormone replacement therapy may be necessary.

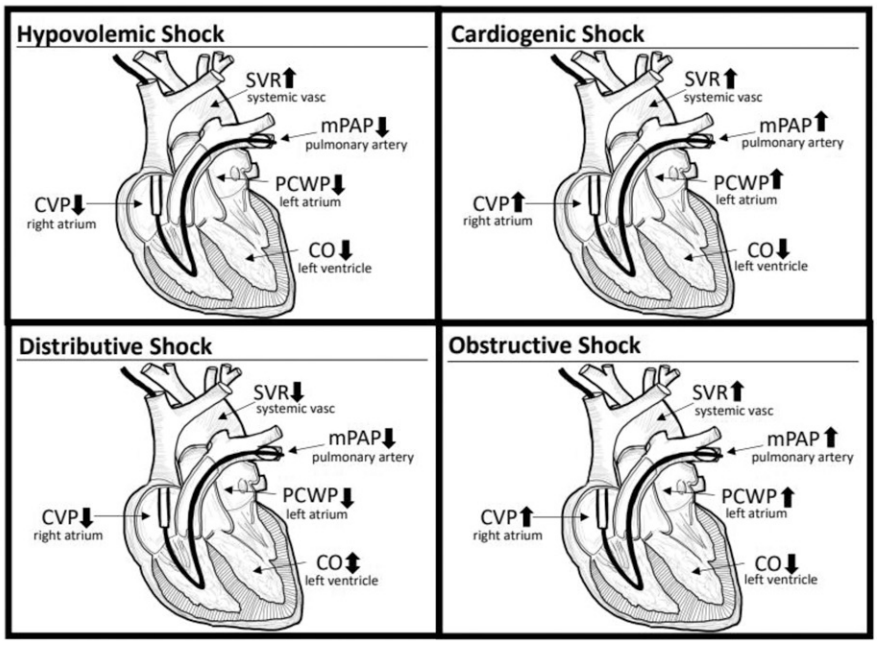
Cortisol. Cortisol assists in maintaining vascular tone, but as with vasopressin, endogenous stores may not be adequate to support the stress response, a phenomenon termed critical illness–related corticosteroid insufficiency, which poses a strong biological rationale for exogenous administration of corticosteroids in patients with septic shock. Despite inconsistent results on short- or long-term mortality, administration of corticosteroids has accelerated resolution of shock, decreased vasopressor requirements, increased ventilator-free days, and decreased ICU length of stay (41–43, 45). For adult patients with septic shock with escalating vasopressor requirements, we suggest to our learners that intravenous hydrocortisone 200 mg/d be considered (25).

MODULE 5: SHOCK STATES

After discussing adrenoreceptor pathophysiology and the effects of vasopressors, we find it useful to discuss the underlying pathophysiology of different shock states to contextualize the appropriateness of various treatment options on the basis of mechanistic principles. At this point, we suggest using a whiteboard to reproduce Figure 2 (either as an illustration [Figure 2A] or as a table [Figure 2B]), illustrating the expected hemodynamic changes in each shock state as they are explained below in turn.

Distributive (e.g., septic) shock is characterized by inappropriate systemic vasodilation. In septic shock specifically, excess proinflammatory mediators and cytokines enter the systemic circulation, resulting in vasoplegia and organ dysfunction (38, 46). We encourage our learners to visualize a recent patient with septic shock (or perhaps at this point, we take a walk to the bedside), as we explain that patients with distributive shock typically present with warm extremities (because of physiologically inappropriate peripheral vasodilation), high CO, widened pulse pressure (indicative of high

A



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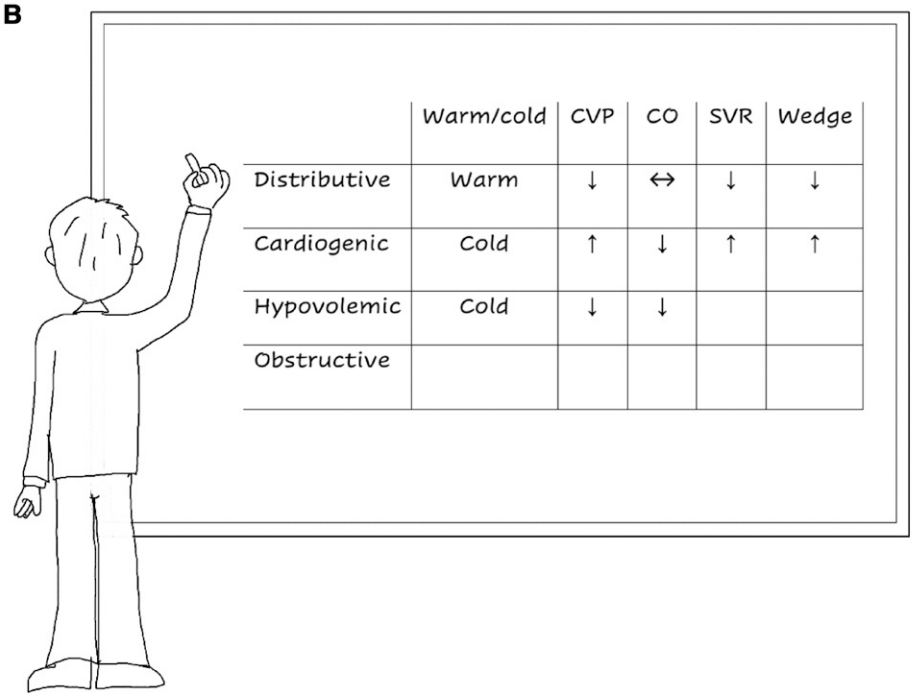

Figure 2. (*A*) Expected changes in hemodynamics during key shock states. Hypovolemic shock is mediated primarily by intravascular volume depletion. As a result, intracardiac pressures are decreased (i.e., central venous pressure [CVP], mean pulmonary arterial pressure [mPAP], and pulmonary capillary wedge pressure [PCWP]), as is cardiac output (CO), a result of decreased stroke volume. In contrast, systemic vascular resistance (SVR) increases to compensate before the delivery of adequate volume resuscitation. In cardiogenic shock, impaired CO leads to increases in the preceding circulatory pressure (i.e., PCWP, mPAP, and CVP) as well as a compensatory increase in SVR. The inappropriate systemic vasodilation of distributive shock leads to reduced SVR, CVP, and mPAP. CO can be variable and is often elevated with increases in heart rate and stroke volume. Finally, in obstructive shock, defined by an obstruction to forward flow, CO is diminished and preceding pressures are elevated (i.e., PCWP, mPAP, and CVP), as is SVR. (*B*) Expected changes in hemodynamics during key shock states: easily reproducible table for whiteboard teaching.

stroke volume and low SVR), low central venous pressure (CVP), low pulmonary capillary wedge pressure (PCWP), and low SVR. We highlight that preferred agents include those that increase vascular tone (norepinephrine, phenylephrine, epinephrine, and vasopressin), whereas those that vasodilate (dobutamine) may have deleterious effects in a typical vasodilatory shock patient, because of worsening vasodilation and therefore SVR.

In contrast, we challenge our learners to recall a recent patient with cardiogenic shock (or we take another walk to the bedside), explaining that cardiogenic shock is caused primarily by impaired CO, and patients will have cool extremities (because of poor CO and α-1 activation in the skin), narrow pulse pressure (low stroke volume and high SVR), high CVP, high PCWP, and high SVR. Preferred agents for cardiogenic shock include those with positive inotropy or that lower vascular tone (dobutamine, dopamine, norepinephrine, and epinephrine) whereas those that primarily increase vascular tone and therefore afterload (e.g., phenylephrine, vasopressin) will likely be problematic.

We briefly mention that hypovolemic shock (hemorrhagic or nonhemorrhagic) is caused by intravascular volume depletion, and patients will typically have narrow pulse pressure, low CO, low CVP, low PCWP, cool extremities, and high SVR (compensatory). Primary management is volume repletion (or blood if hemorrhagic), but agents that support vascular tone may be necessary.

Last, we explain that obstructive shock is defined by an obstruction of forward flow, with impaired CO (e.g., cardiac tamponade, pulmonary embolism, tension pneumothorax). CO, pulse pressure, and PCWP are low, CVP is elevated, extremities are cool, and SVR can be normal or elevated. Treatment primarily involves alleviation of the obstruction, although resuscitation may be needed in the interim.

Implications for the Management of Shock

We now emphasize to our learners that in the ICU, with abundant information available, medical decisions are based on hypotheses about underlying pathophysiology. Armed with a hypothesis based on initial observations, interventions are implemented to target and test the proposed physiology at play. The impact of the intervention is then evaluated in real time. If the effects of the intervention are concordant with the hypothesis, that reinforces the underlying assumptions; if they are discordant, the hypothesis is revised and new interventions attempted. We advise staying at the bedside whenever possible to witness the impact of a chosen intervention to best assess the strength of the hypothesis and plan the most appropriate next steps. This cycle is paramount to learning and caring for patients in the ICU.

For a patient with unspecified shock, effects of particular pharmacologic agents may provide insight into the underlying pathophysiology. For example, we ask our learners to imagine a patient who may have septic or cardiogenic shock or mixed shock. If the patient is treated with phenylephrine and worsens, a component of cardiogenic shock is more likely to be contributing, and a trial of dopamine or dobutamine might be prudent. Conversely, if a patient is given dobutamine and worsens, the vasodilatory component may be more prominent than previously considered, and treatment plans should be revised. By proposing a hypothesis, acting on the

suspected physiology, and reacting appropriately to the observed effects, intensivists both treat patients and elucidate the underlying pathophysiology, making further observations and adjusting accordingly.

WHY IS THIS THE APPROACH?

This approach was developed, revised, and honed over many years by one of the authors, an experienced educator and critical care physician, while teaching learners of various educational levels and backgrounds in the ICU. The teaching format was designed mindful of adult learning theory, including principles of goal-oriented and activity-oriented learning. Our approach is engaging and interactive, rooted in experiential learning and encouraging immediate, practical application of new knowledge. The allegory was chosen to emphasize the evolutionary basis of human physiology rooted in ensuring survival, which may not neatly translate to sustaining life amid a prolonged shock state. Furthermore, we find that this frame is widely understandable and relatable to most learners regardless of background and experience. If instructors so choose, alternative narratives may certainly be considered while applying the same approach and material. The content, structure, and flow have evolved on the basis of learner feedback and emerging data relevant to the topic material. Anecdotally, this teaching approach has received a consistently positive reception among learners and has subjectively been effective for retaining these complex but fundamental principles. Formal evaluation of the effectiveness and generalizability of this approach in other settings is encouraged.

WHAT CAN BE CHALLENGING?

Although we find this teaching approach to be engaging and enjoyable for learners, certain challenges can be anticipated. We have found that visual supplementation with diagrams on a whiteboard can be helpful, but limitations of space and resources in the ICU clinical setting may present challenges when choosing this teaching setting. We have provided examples of high-quality diagrams that may serve as a template for instructors or even be disseminated to learners (using a provided quick response code) in the absence of a physical whiteboard or artistic prowess. In addition, we have paired each high-quality image with a simplistic version that can be more easily reproduced on a whiteboard. Also, the inherently active, busy nature of the ICU may limit learners' attention spans and provide time constraints. Uninterrupted, the core module of this teaching approach may be offered in 8–10 minutes, and additional modules can be added as time permits and as appropriate to the learners. The entire teaching session, with all modules, typically takes 20–30 minutes (although it may take slightly longer with less practice). Therefore, if time is limited, we would encourage that this approach be divided into multiple sessions at the discretion of the instructor.

CONCLUSIONS

In this report, we describe our approach to teaching the physiology of adrenoreceptors, the pathophysiology of vasopressors, and their use in shock states, while providing a review of the relevant literature. We hope that this overview provides a model for educators and trainees in the critical care environment.

Author disclosures are available with the text of this article at www.atsjournals.org.

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