Utilizing Vasopressors: Critical Care Advances in the Emergency Department

José A. Rubero, MD, FACEP, FAAEM
Associate Program Director
University of Central Florida/HCA GME Consortium
Emergency Medicine Residency Program

Objectives

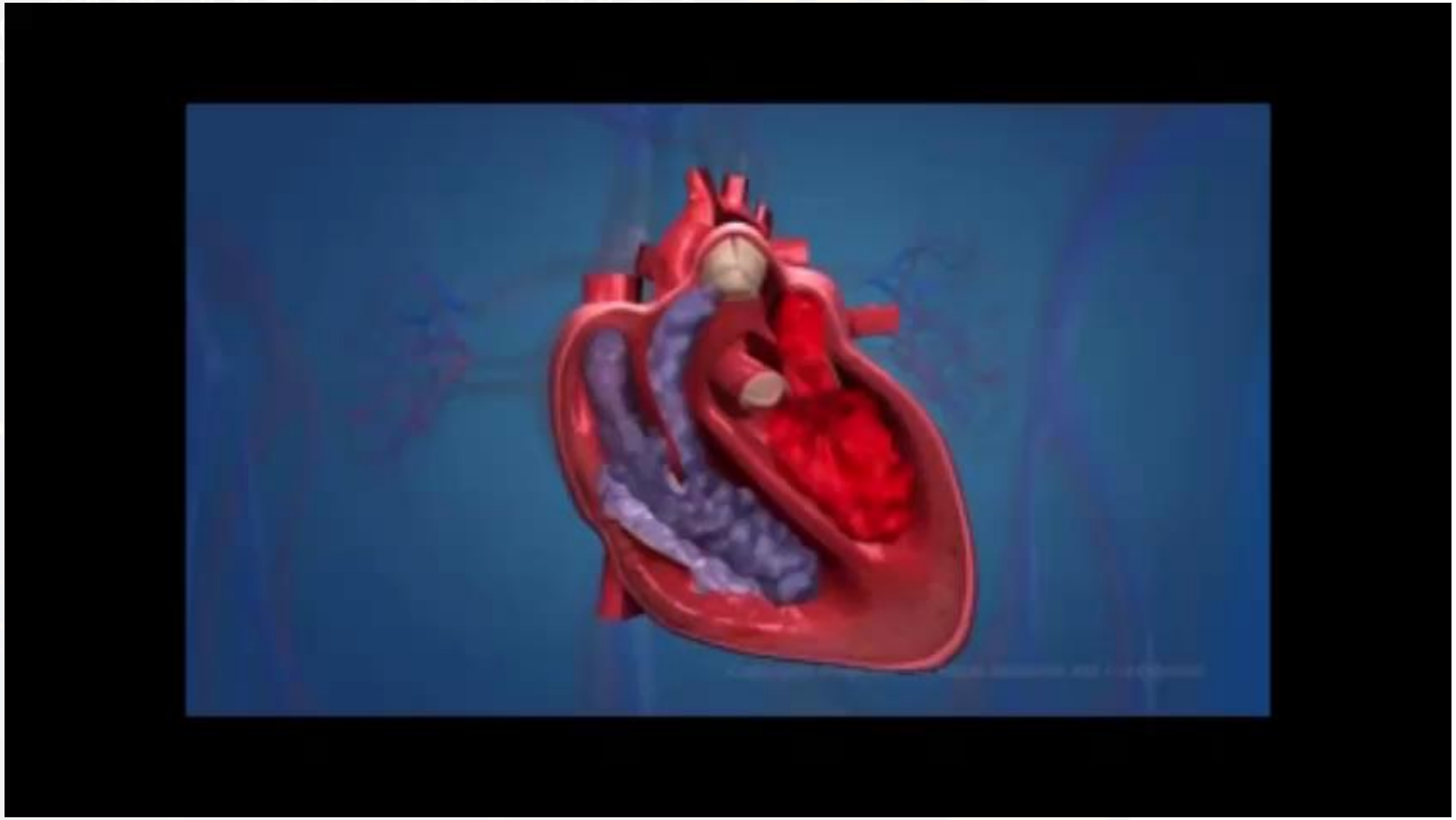
- Review and recognize the types of shock and their presentations
- Discuss and understand the mechanisms of the commonly used vasopressors
- Identify when to use vasopressors to improve perfusion and oxygenation in the Emergency Department

Disclosures

- I have no actual or potential conflict of interest in relation to this program/presentation.

Shock

- The PUMP
  - Oxygen delivery and utilization
    - Ventilation
    - Blood transfusions
    - Dobutamine

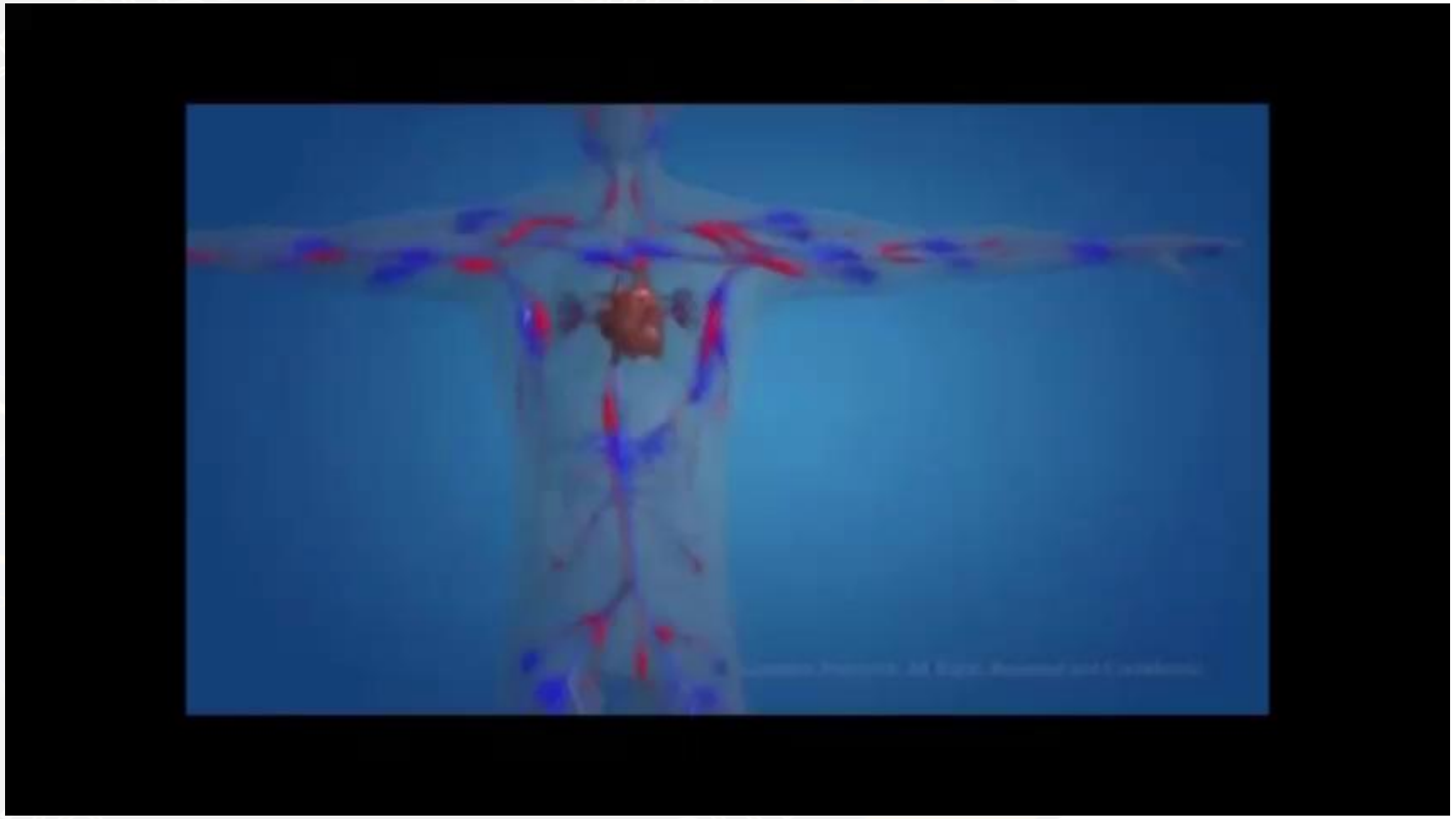


Shock

- The TANK
  - Volume status
    - IVF's
      - 30 ml/kg in sepsis

Shock

- The PIPES
  - Vascular resistance, MAP
    - Norepinephrine
    - Epinephrine
    - phenylephedrine



Shock

- Shock is a final common pathway associated with regularly encountered emergencies including myocardial infarction, microbial sepsis, pulmonary embolism, significant trauma, and anaphylaxis.
- Shock results in impaired tissue perfusion, cellular hypoxia, and metabolic derangements that cause cellular injury.
- Although this early injury is often reversible, persistent hypoperfusion leads to irreversible tissue damage, progressive organ dysfunction, and can progress to death.

Ellender, TJ, et al. Emerg Med Clin N Am 26 (2008) 759–786

Shock

- Cardiovascular collapse (shock) is a common life-threatening condition that requires prompt stabilization and correction
- Shock may be caused by
  - Primary decrease in CO (cardiogenic-obstructive shock)
  - Low circulating blood volume (hypovolemic shock)
  - Vasodilatation (distributive shock)

Ellender, TJ, et al. Emerg Med Clin N Am 26 (2008) 759–786

Shock

- Cardiogenic shock can be defined by
  - Intrinsic dysfunction
    - Myopathies, infarction, acute valvular dysfunction, and arrhythmias
  - Extrinsic dysfunction caused by obstructive disorders
    - Pulmonary embolism, constrictive pericarditis, pericardial tamponade, or tension pneumothorax
  - Most common cause of death among patients hospitalized for acute myocardial infarction.
  - Treating cardiogenic shock may require multiple agents, but despite appropriate inotropic and vasopressor support, mechanical assistance or even cardiac transplant may be required.

Ellender, TJ, et al. Emerg Med Clin N Am 26 (2008) 759–786

Shock

- Hypovolemic shock can be defined as
  - A relative or absolute decreased circulating blood volume, results in a decreased preload that alters stroke volume and leads to a decreased CO.
- Hypovolemic shock can be caused by
  - Hemorrhage from trauma, aneurysm rupture, or gastrointestinal bleeding
  - Basic fluid loss caused by diarrhea, burns, or ‘‘third spacing.’’

Ellender, TJ, et al. Emerg Med Clin N Am 26 (2008) 759–786

Shock

- Hypovolemic shock
  - Treated with volume resuscitation using isotonic crystalloid.
  - If hemorrhage was the cause of volume loss, red cell transfusion may be necessary to maintain adequate oxygen-carrying capacity in the blood.
  - If the blood pressure is dangerously low, it is reasonable to use vasopressors to increase blood pressure during volume resuscitation and methods to prevent ongoing volume loss are performed.
  - Vasopressors are no substitute for adequate fluid resuscitation and should be discontinued once blood pressure has normalized with sufficient volume resuscitation.

Ellender, TJ, et al. Emerg Med Clin N Am 26 (2008) 759–786

Shock

- Distributive or vasodilatory shock
  - Results from vascular changes that lead to a decrease in vasomotor tone (vasodilation) and a loss of peripheral vascular resistance. Characterized by a mismatch in perfusion and oxygen demand.
  - There are multiple subcauses of distributive shock
    - Septic shock
    - Anaphylaxis
    - Neurologic shock

Ellender, TJ, et al. Emerg Med Clin N Am 26 (2008) 759–786

Shock

- Septic shock
  - Most commonly seen in the ED.
  - Inflammatory mediators released by the body in response to an infection may have multiple deleterious effects — including inappropriate vasoconstriction and vasodilation, increased vascular permeability, and impaired cardiac contractility — that can lead to maldistribution of perfusion.
  - Volume resuscitation is the initial therapy in the resuscitation of patients with septic shock.
  - The inciting infection should be identified, with the early administration of antibiotics chosen according to expected pathogens.
  - Surgical removal of infected tissue may be necessary for localized infections. Inotropic and vasopressor support is often necessary.

Otero RM, et al. Chest 2006;130: 1579-95.

Shock

- Anaphylaxis
  - Form of distributive shock caused by an immediate-type hypersensitivity response to an allergen, provoking a severe, systemic inflammatory response.
  - This response leads to increased vascular permeability, with intravascular volume loss, decreased SVR, and impaired myocardial contractility.
  - Bronchospasm with increased resistance to airflow is common in anaphylaxis.
  - Epinephrine is the drug of choice in the treatment of anaphylactic shock due to its potent inotropic and vasopressor effects, as well as the ability to decrease bronchospasm.

Ellender, TJ, et al. Emerg Med Clin N Am 26 (2008) 759–786

Shock

- Neurogenic shock
  - Form of distributive shock, normally arises from injuries or damage to the cervical spinal cord.
  - A unique feature of neurogenic shock is that tachycardia in response to hypotension is uncommon.
  - Intravenous fluid is the first-line in therapy for neurogenic shock.
  - Vasopressor support may be required.
  - If bradycardia is present, dopamine or another vasopressor that will provide chronotropic (heart rate) stimulation as well as increased vascular resistance may be preferred.

Ellender, TJ, et al. Emerg Med Clin N Am 26 (2008) 759–786

Shock

- It is also important to note that vasodilatory shock is the final common pathway of prolonged and severe shock of any cause.
- Vasoconstriction in the peripheral circulation is the normal response to conditions in which the arterial pressure is too low for adequate tissue perfusion, such as acute hemorrhagic or cardiogenic shock.
- In other conditions, hypotension occurs as a result of failure of the vascular smooth muscle to constrict.
- Such so-called vasodilatory shock is characterized not only by hypotension due to peripheral vasodilatation but also by a poor response to therapy with vasopressor drugs.

Landry, DW, et al. N Engl J Med 345 (2001) 588-5956

Shock

TABLE 1. CAUSES OF VASODILATORY SHOCK.*

Sepsis
Inadequate tissue oxygenation
Nitrogen intoxication (hypoxic lactic acidosis)
Carbon monoxide intoxication
Prolonged and severe hypotension
Hemorrhagic shock
Cardiogenic shock
Cardiopulmonary bypass
Shock with probable vasodilatation
Metformin intoxication
Some mitochondrial diseases
Cyanide poisoning
Cardiac arrest with pulseless electrical activity

*Anaphylaxis, liver failure, and glucocorticoid deficiency are sometimes listed among the causes of vasodilatory shock, but the data are inconclusive.

Landry, DW, et al. N Engl J Med 345 (2001) 588-5956

Shock

- MAP is derived from the product of systemic vascular resistance (SVR)
- and CO.
- SVR is governed by blood viscosity, vessel length, and the inverse of vessel diameter.
- SVR and CO are important clinical concepts that distinguish the different forms of shock.

Ellender, TJ, et al. Emerg Med Clin N Am 26 (2008) 759–786

Shock

- Consequently, any basic approach to hypotension should begin with an assessment of the patient's volume status and CO.
  - Low CO states are clinically linked to a narrowed pulse pressure, a rising shock index, and a delayed capillary refill with cool peripheral extremities.
  - Widened pulse pressures with low diastolic pressures, bounding pulses, warm extremities, and normal capillary refill can be seen with increased CO states.

Ellender, TJ, et al. Emerg Med Clin N Am 26 (2008) 759–786

Shock Type	HR	SVR	CO
Hypovolemic	↑	↑	↓
Distributive	↑	↓	↑ early; ↓ late
Cardiogenic	↑	↑	↓
Obstructive	↑	↑	↓

Shock

- Conditions that cause high output and low resistance are classically linked to inflammatory states.
  - Septic shock
  - Severe pancreatitis
  - Anaphylaxis
  - Burns
  - Liver failure
- Conditions with suspected hypoperfusion and clinical evidence of low CO, an assessment of cardiac volumes and global intravascular volume must be reassessed.
  - Hemorrhage (trauma, GI bleed)
  - Volume loss (diarrhea, vomiting)
- Clinical features, such as elevated jugular venous pulses, peripheral edema, a cardiac gallop, or pulmonary rales, help to distinguish the hypotensive patient with low CO and high intravascular volumes.
  - These patients tend to be cold and clammy because of their increased SVR and usually have historical features and clinical signs (EKG changes) that help further differentiate the cardiac origins of shock.

Ellender, TJ, et al. Emerg Med Clin N Am 26 (2008) 759–786

Management of shock

- The management of shock first focuses on identifying the underlying cause and applying some combination of fluid resuscitation, vasoconstrictors, inotropic agents, and potentially vasodilators in a coordinated attempt to right physiologic irregularity, correct perfusion deficits, and maintain oxygen delivery.
- Clinically, this is achieved by improving blood pressure and CO through the optimization of preload, augmentation of SVR, and the increase of cardiac contractility.

Holmes CL. Curr Opin Crit Care 2005;11:413–7; Kellum JA, et al. Curr Opin Crit Care 2002;8:236–41.

Management of shock

- Vasopressor agents largely improve perfusion pressure and preserve regional distribution of CO through an increase in MAP above autoregulatory thresholds.
- Vasopressor agents may also improve cardiac preload and increase CO by decreasing venous compliance and augmenting venous return.
- Inotropes improve oxygen delivery and CO through an increase in rate and contractility.

Holmes CL. Curr Opin Crit Care 2005;11:413–7; Kellum JA, et al. Curr Opin Crit Care 2002;8:236–41.

Vasoactive drugs

- Vasoactive drug therapy is used to manipulate the relative distribution of blood flow and restore tissue perfusion.
- These agents are classically subdivided, based on their predominant pathway of activity, into two separate class types:
  - Vasopressors and inotropes.
- Vasopressors modulate vasoconstriction and thereby increase blood pressure
  - norepinephrine, vasopressin, metaraminol, vasopressin, methylene blue
- Inotropes increase cardiac performance and thereby improve cardiac output (CO).
  - milrinone, levosimendan

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Vasoactive drugs

- Vasopressor and inotropic agents function primarily through stimulation of adrenergic receptors or through the induction of intracellular processes that mimic sympathetic end points (increased cAMP).
- Many of the drugs in use have varied effects because of their mixed receptor activity.
- Most of these act directly or indirectly on the sympathetic nervous system with effects that vary according to the strength of sympathetic receptor stimulus and affinity.
  - Direct-acting drugs operate by stimulating the sympathetic nervous system receptor
  - Indirect-acting drugs cause the release of norepinephrine, which produces the effect.

Ellender, TJ, et al. Emerg Med Clin N Am 26 (2008) 759–786

Vasoactive drugs

- Inodilators are agents with inotropic effects that also cause vasodilation leading to decreased systemic and/or pulmonary vascular resistance (SVR, PVR)
  - milrinone, levosimendan
- Some agents don’t fit these categories easily!
  - dopamine
- No inotropic agents have been shown to have superiority over any others in good quality trials.

Vasoactive Medication Receptor Activity and Clinical Effects

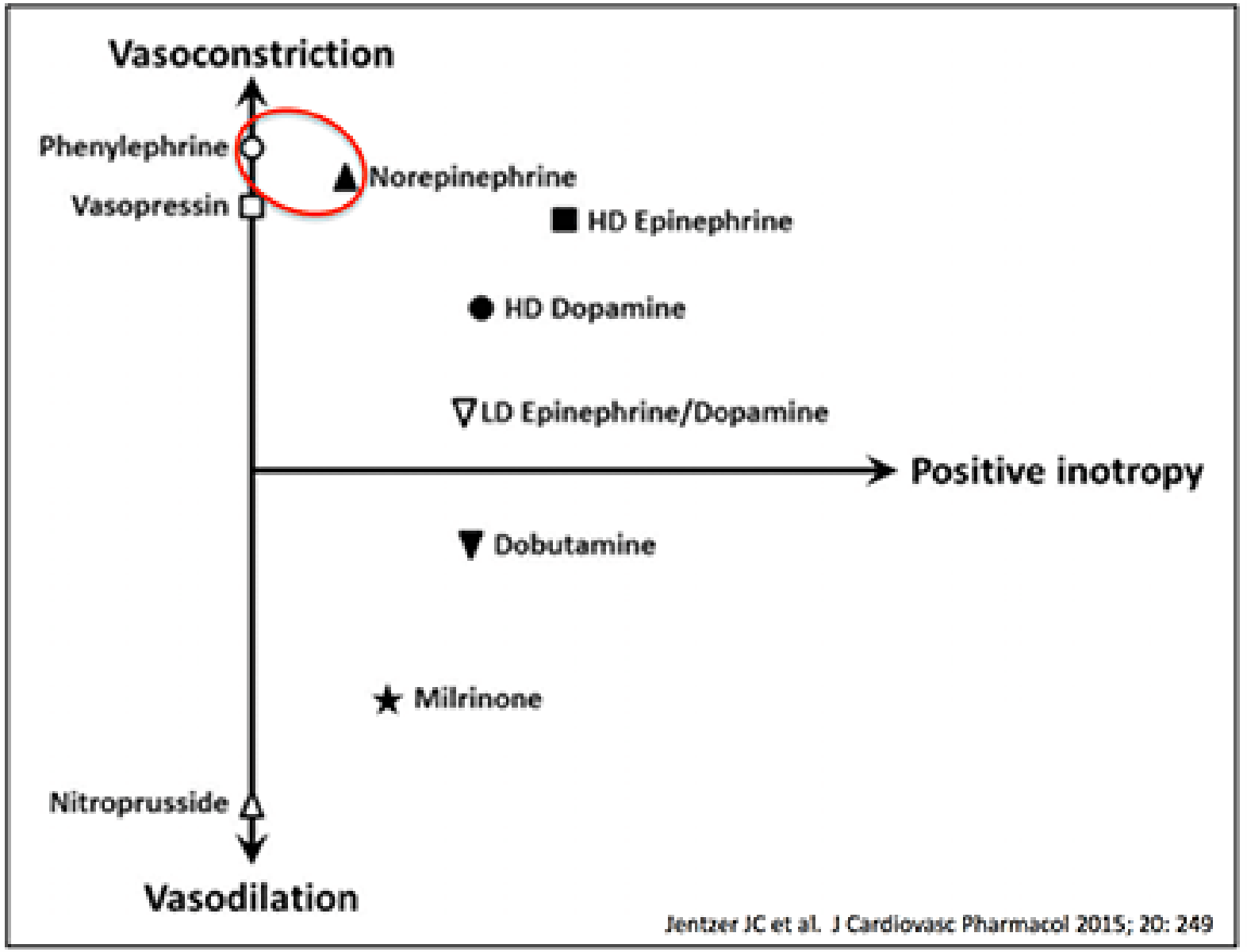
Drug	Alpha-1	Beta-1	Beta-2	Dopaminergic	Predominant Clinical Effects
(Neosynephrine) Phenylephrine	***	0	0	0	SVR ↑ ↑, CO ↔/↑
(Levophed) Norepinephrine	***	**	0	0	SVR ↑ ↑, CO ↔/↑
(Adrenalin) Epinephrine	***	***	**	0	CO ↑ ↑, SVR ↓ (low dose) SVR/↑ (higher dose)
(Intropin) Dopamine (mcg/kg/min)					
0.5 to 2	0	*	0	**	CO
5 to 10	*	**	0	**	CO ↑, SVR ↑
10 to 20	**	**	0	**	SVR ↑ ↑
Dobutamine	0/*	***	**	0	CO ↑, SVR ↓
Isoproterenol	0	***	***	0	CO ↑, SVR ↓

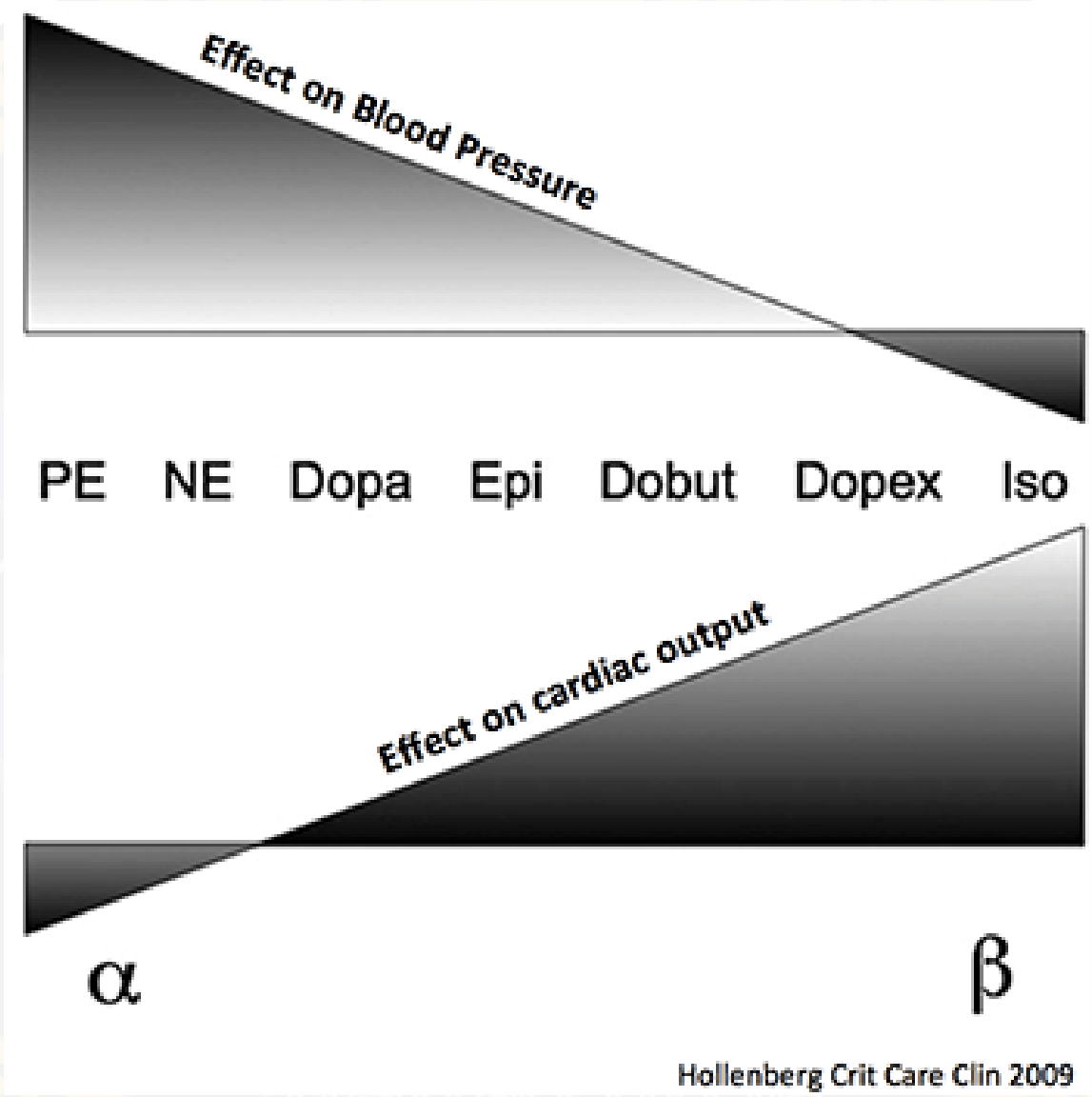
*** Very Strong Effect, ** Moderate effect, * Weak effect, 0 No effect.

Agent	Receptor Agonist Activity*				Initial Dose	Onset
	α	β1	β2	DA		
Phenylephrine	++++	–	–	–	10 mcg/min	2 minutes
Norepinephrine	++++	+++	–	–	2 mcg/min	1-2 minutes
Epinephrine	+++	++++	+++	–	1 mcg/min	1 minute
Dopamine	++	++++	++	++++	5 mcg/kg per min	5 minutes
Dobutamine	+	++++	++	–	1 mcg/kg per min	1-2 minutes
Isoproterenol	–	++++	++++	–	5 mcg/min	1-5 minutes
* Receptor activity may be dose dependent						

Receptor Physiology

Receptor		Location	Effect
Alpha-1 Adrenergic		Vascular wall	Vasoconstriction
		Heart	Increase duration of contraction without increased chronotropy
Beta Adrenergic	**Beta-1**	Heart	↑Inotropy and chronotropy
	Beta-2	Blood vessels	Vasodilation
Dopamine		Renal Splanchnic (mesenteric) Coronary Cerebral	Vasodilation
	Subtype		Vasoconstriction


Vasoconstriction
Phenylephrine
Norepinephrine
Vasopressin
HD Epinephrine
HD Dopamine
LD Epinephrine/Dopamine
Positive inotropy
Dobutamine
Milrinone
Nitroprusside
Vasodilation
Jentzer JC et al. J Cardiovasc Pharmacol 2015; 20: 249


Effect on Blood Pressure
PE NE Dopa Epi Dobut Dopex Iso
Effect on cardiac output
α
β
Hollenberg Crit Care Clin 2009

	Epinephrine (Adrenaline)	Norepinephrine (Noradrenaline)	Dopamine	Dobutamine
Class	endogenous catecholamine	endogenous catecholamine	endogenous catecholamine	synthetic catecholamine
Mechanism	beta > alpha	alpha > beta	DA>beta>alpha	beta 1 and 2
Effects	b1 -> +HR, +inotropy, +CO; alpha at higher doses preserves coronary and cerebral blood flow; b2 -> +vasodilation, +brochodilation	a1-> +SVR +beta at higher doses	dose (mcg/kg/h) 1-5 = DAR 1+2 5-10 = betaR > 10 = alphaR + aldosterone	Inotropy; + VO2 ; + myocardial work; mild + HR + coronary perfusion; mild – SVR; mild - PVR
Pharmacokinetic	onset in mins Met: COMT + MAO t/12 = mins	onset in mins Met: COMT + MAO t/12 = mins	onset in mins Met: COMT + MAO t/12 = mins	onset in mins methylation + conjugation t/12 = mins urine + bile -> inactive metabolites
Use	cardiac arrest low CO state cardiac surgery	septic shock vasodilation	nil (kids!)	low CO state part of EGDT cardiac surgery
Pros	$; titratable	$; titratable	$; titratable	titratable
Cons	lactic acidosis low K, low PO4	reflex bradycardia hypertension peripheral ischemia	arrhythmia pulmonary vasoconstriction N&V; immune dysregulation - TSH and PRL release	$$ myocardial ischemia tachydysrhythmias tachyphylaxis
Evidence	Annane 2007: vs dobtumine + norad = no difference in septic shock CAT: vs norad in septic shock = no difference	CAT: vs adrenaline in septic shock = no difference Annane 2007: with dobutamine vs adrenaline = no difference in septic shock	Bellomo 2000: no 'renal dose' dopamine De Backer 2010 and Patel 2010: vs norad = more tachydysrhythmias in septic shock	SURVIVE: no mortality benefit vs levosimendan Annane 2007: with norad vs adrenaline = no difference in septic shock

	Vasopressin	Levosimendan	Milrinone
Class	endogenous peptide	calcium sensitiser (inodilator)	bipyridine inodilator
Mechanism	V1R = vasoconstriction V2R = renal + endothelium V3R = pituitary OTR = oxytoxin type receptors	modulates troponin C activates vascular ATP-dependent K channels	cAMP PDE-3 inhibitor
Effects	antidiuresis + SVR + platelet aggregation + PVR - splanchnic flow		inotropy - SVR - PVR +CO
Pharmacokinetic	peptidases t1/2 = 10-20 min	slow onset (can give loading dose) liver + renal metabolism t1/2 = 1 hour but has active metabolites effects last up to 1/52	t1/2 = 2.3h renal no effect >8h
Use	septic shock (cardiac arrest}	low CO state	low CO state cardiac surgery support RV
Pros	fast onset/ offset (except renal effects) catecholamine resistance	OK if b-blockers catacholamine resistance faster decrease in BNP cf. dobutamine	pulmonary vasodilation OK if b-blockers catacholamine resistance little +HR
Cons	$$$$ pulmonary hypertension; splanchnic ischaemia; uterine contraction?; Thrombosis	$$$$$ tachycardia; low BP; headache; not if LVOTO not if liver/ renal disease	$$$ hypotension; may need norad little evidence
Evidence	VASST: no benefit vs norad in septic shock	SURVIVE: no mortality beneift vs dobutamine	faster weaning off bypass

	Phenylephrine
Class	endogenous non-catecholamine
Mechanism	alpha and some beta1
Effects	venoconstriction, thereby increasing venous return to the heart Increased preload may increase cardiac output Increased afterload and reflex bradycardia may reduce cardiac output
Pharmacokinetic	onset in mins Met: COMT + MAO t/12 = mins
Use	septic shock vasodilation
Pros	$; titratable
Cons	reflex bradycardia hypertension peripheral ischemia
Evidence	Morelli 2008a randomized 32 patients with hyperdynamic sepsis to receive phenylephrine vs. norepinephrine as a first-line vasopressor (with additional open-label dobutamine as needed). The only difference detected between the groups was that patients in the norepinephrine group had a slightly higher blood pressure. There were no differences in heart rate, cardiac output, systemic vascular resistance, lactate, gastric mucosal perfusion, renal function, or dobutamine requirement:

Figure 1. Algorithm For The Assessment and Treatment Of Hypotension

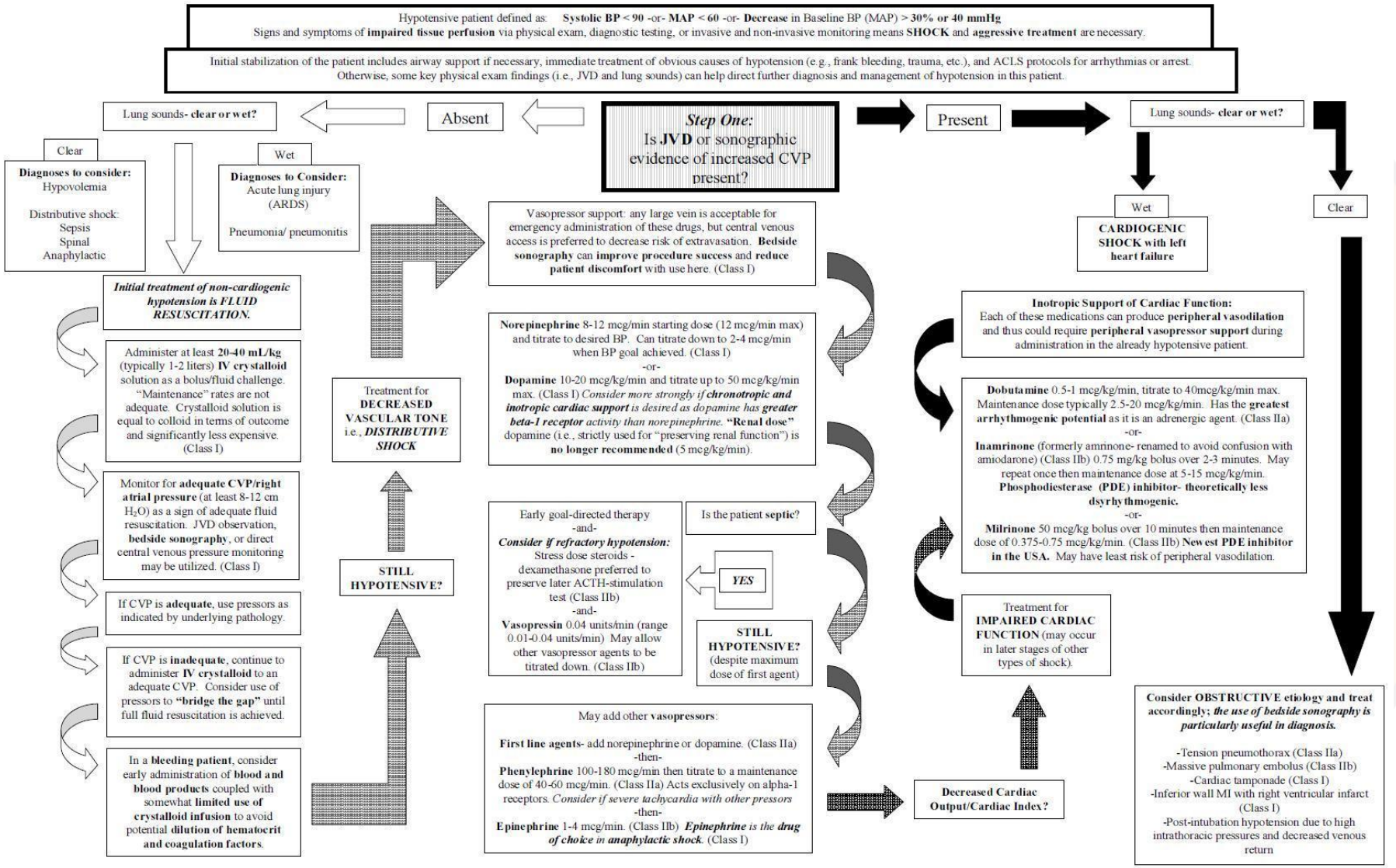
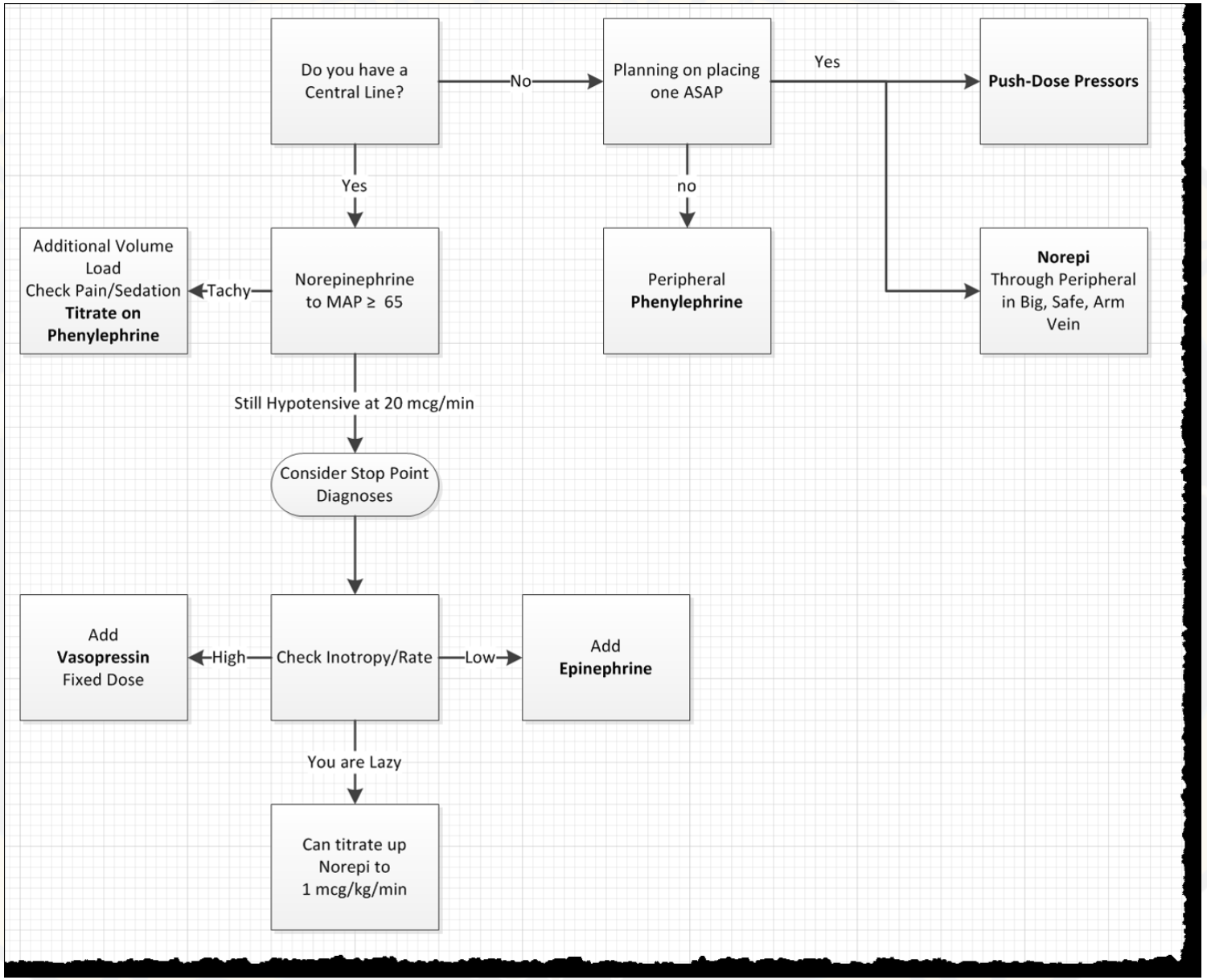


Table 5. Drugs By Adrenergic Receptor Type: Indications, Advantages, And Disadvantages

Pressor	Indications	Advantages	Disadvantages
Dopamine	• Dopamine is FDA indicated for all forms of shock and for treatment of decreased cardiac output • Poor cardiac function with poor perfusion • Post arrest hypotension/ myocardial stunning	• Effective at multiple receptors • Graded, dose-dependent receptor activity (not all or nothing) • Titrate to patient specific responses and hemodynamic monitoring	• "Dopaminergic" doses may improve urine output but do not improve renal function and generally are not helpful in addressing hypotension • May be arrhythmogenic at higher "alpha" doses • High doses may compromise urine output (consider using with dobutamine)
Norepinephrine	• Septic shock due to low SVR • Can be used in anaphylactic shock	Excellent at increasing systemic vascular resistance (SVR)	Increased risk of dysrhythmias and myocardial ischemia; increased oxygen consumption; may decrease intestinal perfusion and increase lactate levels
Phenylephrine	FDA indicated for use in hypotension	Good choice if tachycardia/arrhythmia limiting use	No effect on cardiac output
Dobutamine	• FDA indicated for decreased cardiac output and CHF • Best if used when there are signs/symptoms of shock without severe hypotension (< 90 mmHg)	• Inotropic agent: increases cardiac output • Good for congestive heart failure *without* hypotension	Can decrease SVR; may provoke hypotension. Potential solution: add dopamine or epinephrine to increase SVR **OR** consider switching to another class of inotropic agents, such as phosphodiesterase inhibitor (e.g., inamrinone and milrinone)
Epinephrine	• FDA indicated for use in anaphylactic shock • Intravenous form is FDA indicated for cardiac arrest	Does not require volume resuscitation prior to use (for the purely anaphylactic cause of shock)	Increased risk of dysrhythmias and myocardial ischemia
Vasopressin	Consider in septic shock refractory to volume expansion and first line catecholamines	May decrease amount of other vasopressors needed	• Not a first line agent • Delayed onset of action • Its use in septic shock and for cardiac arrest are off-label


Do you have a Central Line?
No
Planning on placing one ASAP
Yes
Push-Dose Pressors
Yes
Yes
no
Additional Volume Load
Check Pain/Sedation
Titrate on Phenylephrine
Tachy
Norepinephrine to MAP ≥ 65
Peripheral Phenylephrine
Norepi
Through Peripheral in Big, Safe, Arm Vein
Still Hypotensive at 20 mcg/min
Consider Stop Point Diagnoses
Add Vasopressin Fixed Dose
High
Check Inotropy/Rate
Low
Add Epinephrine
You are Lazy
Can titrate up Norepi to 1 mcg/kg/min

Clinical Application

		1st Line Agent	2nd Line Agent
Septic Shock		Norepinephrine (Levophed) Phenylephrine (Neosynephrine)	Vasopressin Epinephrine (Adrenalin)
Heart Failure		Dobutamine	Milrinone
Cardiogenic Shock		Norepinephrine (Levophed) Dobutamine	
Anaphylactic Shock		Epinephrine (Adrenalin)	Vasopressin
Neurogenic Shock		Dopamine	Phenylephrine (Neosynephrine)
Hypotension	Anesthesia-induced	Phenylephrine (Neosynephrine)	
	Following CABG	Epinephrine (Adrenalin)	

Case 1

- 72 year-old woman with DM type II, hypertension and Stage II CKD is transferred from a Skilled Nursing Facility for altered mental status. Her vitals upon arrival are as follows: Temp 101F, BP 70/45, Hr 140, RR 20, O2 Sat 95% RA. Pertinent lab findings: WBC 21, Cr 3.5, Lactic Acid 3.4, Positive UA.
- After adequate IVF resuscitation, pt continues to remain hypotensive BP 60-70s/30-40s and tachycardic Hr 130s. What is the most appropriate 1st line vasopressor/inotropic agent?

A. Epinephrine (Adrenalin)
B. Dobutamine
C. Norepinephrine (Levophed)
D. Dopamine

Pathophysiology

- Septic Shock: results in a ↓ SVR and a systemic inflammatory response syndrome with diffuse capillary leak
- Cardiac Output: typically elevated, but may be depressed in some cases

Treatment

- Vital Signs: inadequate endpoints in determining a response to resuscitation efforts in sepsis
- Lactate Measurements: serial will guide ongoing resuscitation efforts

Ann Emerg Med 2006; 48: 28 - 54

Treatment

- Epinephrine in Septic Shock:
  - Comparison Between Epinephrine and Norepinephrine: prospective, double blinded, randomized trial of 280 patients in shock compared epinephrine and norepinephrine for the ability to reach MABP goals
  - No Difference: in ability to reach MABP goals or 28-day or 90-day mortality between groups

Intensive Care Med 2008 34:2226-2234

Treatment

- Epinephrine in Septic Shock:
  - Comparison Between Epinephrine alone versus Norepinephrine and Dobutamine: prospective, multicenter, double blinded, randomized trial of 330 patients in septic shock compared epinephrine and norepinephrine for efficacy and safety
  - No Difference: in 28-day all cause mortality, no difference in time to hemodynamic success or time to vasopressor withdrawal

Lancet 2007 370:676-684

- No Difference: Currently EBM supports Norepinephrine over Dopamine; and equivalent to Epinephrine
- Assess Volume: Utilize Ultrasound, arterial wave form analysis or pulse pressure variation to determine intravascular volume
- Dobutamine Care: vasodilator properties of Dobutamine may reduce MABP

Case 2

- 64 year-old man with PMH significant for CAD s/p MI and PCI (2004; drug-eluting stents), ischemic cardiomyopathy (EF 20-25%) with AICD (2007), who presents to ED with 1 week history of progressively worsening shortness of breath, orthopnea and bilateral lower extremity edema, and chest pain after running out of all medications about 10 days ago.
- In ED, vitals: Temp 99F, BP 75/48, Hr 75, RR 25, O2 Sat 91% on RA. CXR reveals vascular congestion and bilateral pleural effusion. Bedside ultrasound reveals significantly diminished EF. EKG reveals new Q waves in leads v1-v5.
- What is the most appropriate 1st line vasopressor/inotropic agent?

A. Epinephrine (Adrenalin)
B. Dobutamine
C. Norepinephrine (Levophed)
D. Dopamine

Pathophysiology

- Primary Pump Failure
  - Decreased Contractility: acute coronary syndrome related ischemia
- Limited Cardiac Output
- Reduced Coronary Perfusion pressure with reduced MABP
- Increased Heart Rate corresponds to raised myocardial oxygen demand

Treatment

- First Line Therapy: Dobutamine with or without Norepinephrine
- Dopamine and Epinephrine: are 2nd and 3rd line agents
- Phosphodiesterase Inhibitors: have long half lives that limits their utility in acute settings (milrinone)
- Phenylephrine: offers pure alpha stimulation that can cause ↑afterload without improved contractility, resulting in reflex bradycardia

Surg Clin N Am 86 (2006) 1503 - 1521

Case 3

- 56 year-old obese man with PMH significant for COPD and OSA, who was initially admitted to the medicine floor for acute COPD exacerbation secondary to community-acquired pneumonia, was found to be in acute respiratory failure.
- Versed and Succinylcholine were given for emergent intubation. Vitals after intubation are as follows: Temp 99.8F, BP 74/48, Hr 74. What is the most appropriate 1st line vasopressor/inotropic agent?

A. Phenylephrine (Neosynephrine)
B. Dobutamine
C. Norepinephrine (Levophed)
D. Dopamine

Case 4

- A 19 y/o man has sustained a high c-spine injury at C-2 due to a trampoline accident. His neurological injury is complete at the C-2 / C-3 level and he is intubated.
- Vital Signs: Temp 97.8F, BP 78/50, HR 62, RR 18
- He has been given 4 L of NS and his BP has not responded.
- What are the options for vaso-active agents in the treatment of spinal shock?
    a. Milrinone
    b. Dobutamine
    c. Phenylephrine
    d. Dopamine

Pathophysiology

- Hypotension of Spinal Shock: due to the loss of sympathetic tone of the heart and vasculature.
- Resultant Bradycardia & ↓ SVR: may further exacerbate cord injury-the penumbra is at risk.

Treatment

- Maximizing MABP with fluids and Dopamine offers the best choice for improvement in neurological outcome without adverse events.

J Neurosurg 1997; 87: 239

Clinical Application

		1st Line Agent	2nd Line Agent
Septic Shock		Norepinephrine (Levophed) Phenylephrine (Neosynephrine)	Vasopressin Epinephrine (Adrenalin)
Heart Failure		Dobutamine	Milrinone
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	Following CABG	Epinephrine (Adrenalin)	