

The Internet Book of Critical Care

Vasopressors

February 20, 2020 by [Josh Farkas](#)



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[overview](#)

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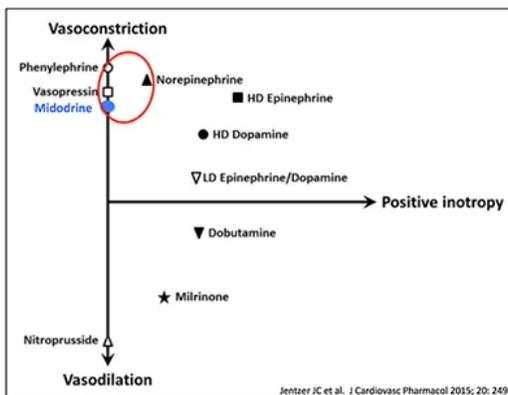
The table below categorizes vasoactive medications. This might seem like a lot, but grouping drugs together into classes can simplify things.

Rough properties of various vasopressors

Drug Typical dose range	Target	Effect on - Heart rate - Inotropy - Ectopy	Effect on systemic vascular resistance	Effect on cardiac output	Effect on blood pressure	Effect on pulmonary vascular resistance	Main uses	Safe for peripheral use?
Inodilators								
Dobutamine 2-20 mcg/kg/min	$\alpha\beta\beta\beta$	$\uparrow\uparrow\uparrow$	\downarrow	$\uparrow\uparrow\uparrow$	Variable	\downarrow	Cardiogenic shock	
Milrinone 0.375-0.75 mcg/kg/min	cAMP	$\uparrow\uparrow\uparrow$	$\downarrow\downarrow$	$\uparrow\uparrow\uparrow$	Variable	$\downarrow\downarrow$	Cardiogenic shock	
Isoproterenol 2-10 mcg/min	$\beta\beta\beta$	$\uparrow\uparrow\uparrow\uparrow\uparrow$	\downarrow	$\uparrow\uparrow\uparrow$	Variable		Bradycardia	Yes
Pure Vasopressors								
Vasopressin 0.01-0.06 U/min	V1 & V2	\downarrow	$\uparrow\uparrow\uparrow$	$\leftrightarrow/\downarrow$	$\uparrow\uparrow\uparrow$	\downarrow	Distributive shock, Pulmonary HTN	No.
Phenylephrine 40-180 mcg/min	$\alpha\alpha\alpha$	\downarrow	$\uparrow\uparrow\uparrow$	Variable	$\uparrow\uparrow\uparrow$	$\uparrow\uparrow$	Distributive shock	Yes
InoPressors								
Norepinephrine 0-40 mcg/min*	$\alpha\alpha\alpha\beta$	\uparrow	$\uparrow\uparrow\uparrow$	\leftrightarrow/\uparrow	$\uparrow\uparrow$	\leftrightarrow	Shock (most types)	Yes, for short period with monitoring
Epinephrine 0-20 mcg/min*	$\alpha\beta\beta\beta$	$\uparrow\uparrow\uparrow$	\uparrow	$\uparrow\uparrow\uparrow$	$\uparrow\uparrow$		Bradycardia, cardiogenic shock, sepsis, anaphylaxis	Yes
Dopamine, low 1-4 mcg/kg/min	Dopa-R	\leftrightarrow	\downarrow	\uparrow	\downarrow		Zombie apocalypse (absence of better agents).	Probably not
Dopamine, medium 4-10 mcg/kg/min	$\alpha\beta\beta\beta\delta$	\uparrow	Variable	$\uparrow\uparrow$	Variable			
Dopamine, high 10-20 mcg/kg/min	$\alpha\alpha\alpha\beta\delta$	$\uparrow\uparrow$	$\uparrow\uparrow$	\uparrow	$\uparrow\uparrow\uparrow$	\uparrow		

*Listed ranges are typically used doses in the United States, but there is no true "maximal" dose. Some countries may tend to use higher doses than others. At very high doses, pressors may lose some receptor specificity. The best dose is the dose required to keep the patient alive – in some cases very high norepinephrine or epinephrine doses may be needed.

-The Internet Book of Critical Care, by @PulmCrit



inodilators

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classic inodilators (milrinone, dobutamine)

- Mechanism
 - Dobutamine stimulates mostly beta-receptors, with very little stimulation of alpha-receptors.
 - Milrinone inhibits intracellular adenylyl cyclase, thereby increasing intracellular cyclic AMP levels.
- Physiologic effect
 - Primary effect is positive inotropy, with positive chronotropy as well.
 - Secondary effect is peripheral vasodilation.
 - Cardiac output is increased due to both inotropic effect and vasodilation.
 - Effect on blood pressure is variable, depending on how responsive the heart is to inotropy. If the heart responds strongly (with increased stroke volume and heart rate), it is possible for these drugs to increase blood pressure. However, if the heart is already working as hard as it can, then the vasodilator effect may be dominant, causing a drop in blood pressure. Overall, the effect on blood pressure is unpredictable.
- Clinical use

- (1) Low-output cardiogenic shock. Care is required for patients with hypotension, since there is a risk of exacerbating hypotension.
- (2) Septic shock with inadequate cardiac output (as an add-on agent). However, *epinephrine* could be a superior choice in that situation, if blood pressure is tenuous.
- How to titrate
 - Ideally, the inodilators should be titrated against cardiac output or a surrogate of cardiac output (e.g. urine output).
 - They shouldn't be titrated against blood pressure, because they have no predictable effect on blood pressure.
- Pro/con of different agents?
 - Milrinone causes a bit more vasodilation, so it might be better for cardiogenic shock. However, milrinone is renally eliminated, which can make it difficult to titrate in patients with renal failure.
 - Dobutamine is easier to titrate due to its short half-life, so it is often a preferred agent if the patient's response to inotropy isn't entirely predictable.
 - Prolonged infusion of dobutamine may cause desensitization of beta-receptors and reduced efficacy. This may be a problem, but it can also help wean the patient off dobutamine once the infusion has been running for a long time (it may be easier to wean off than would be expected).

isoproterenol

- Mechanism: Pure beta-agonist.
- Physiologic effect: Very powerful chronotrope, with positive inotropic effects as well.
- Clinical use: Bradycardia (will work in some patients who are refractory to epinephrine).
- How to titrate: typically, against heart rate.
- Pro/Con?
 - Isoproterenol is probably the most powerful chronotrope.
 - It doesn't cause vasoconstriction, so it's safe to give peripherally.
 - Its main drawback is pricing & availability: in the United States the price is astronomical and some hospitals don't have it. If you don't have it, epinephrine can usually be used as an alternative agent.

pure vasopressors

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vasopressin

- Mechanism: Stimulates V1 and V2 receptors, causing vasoconstriction and renal water retention.
- Physiologic effects:
 - It increases systemic vascular resistance (SVR).
 - It does cause venoconstriction, which may increase preload.
 - Its dominant effect on cardiac output is often to cause a reduction (but this may depend on the heart's ability to tolerate increased afterload).
- Clinical use:
 - Vasodilatory shock (particularly sepsis). Typically given in low doses (0-0.06 U/min), either as primary or secondary agent (27483065).
 - Front-line agent for hepato-renal syndrome (HRS) in countries lacking terlipressin (such as the United States).
 - Central diabetes insipidus (very low doses needed, e.g. 0.01 units/minute or less).
 - Variceal gastrointestinal hemorrhage (theoretically an attractive agent, but pragmatically it's impossible to titrate adequately).
- How to titrate: typically, against blood pressure.
- Pro/Con
 - Vasopressin may preferentially cause vasoconstriction of post-glomerular arterioles in the kidney, causing improvement in renal function.
 - It may cause some pulmonary vasodilation, which can be helpful in the context of pulmonary hypertension.
 - Vasopressin shouldn't generally be given peripherally (if it extravasates, there is no antidote).
 - Vasopressin can cause digital ischemia, especially when combined with norepinephrine – **must pay careful attention to perfusion of hands and feet; shut off vasopressin at first sign of ischemic digits.**

phenylephrine

- Mechanism: Pure alpha-agonist, causes arterial and venous vasoconstriction.
- Physiologic effect
 - Increased systemic vascular resistance (SVR).
 - Venos constriction increases the *preload*.
 - Effect on cardiac output depends on preload-responsiveness versus ability of the heart to handle increased afterload. For example, in a patient with systolic heart failure and volume overload, added preload won't help, whereas the heart may be unable to tolerate afterload – so the net effect is to reduce the cardiac output. Alternatively, for a patient who is preload-responsive with a stronger ejection fraction, phenylephrine could cause a net *increase* in cardiac output.
 - Available evidence in sepsis suggests that phenylephrine has a very similar physiologic effect compared to norepinephrine (reviewed [here \(https://emcrit.org/pulmcrit/phenylephrine-infusion/\)](https://emcrit.org/pulmcrit/phenylephrine-infusion/)). Both agents are predominantly alpha-agonists.
 - Phenylephrine can cause a mild reflex bradycardia due to elevation in blood pressure.
- Clinical use:
 - Vasodilatory shock.
 - Useful in patients with critical aortic stenosis (who have a *fixed* afterload imposed on the left ventricle by the stenotic valve).
 - Atrial fibrillation with fast ventricular response (increases blood pressure while causing reflex reduction in heart rate).
- How to titrate: typically, against blood pressure.
- Pro/Con
 - It has classically been feared that phenylephrine would drop the cardiac output. This seems to occur with phenylephrine *boluses*, but not with *infusions* (available evidence indicates that a phenylephrine infusion functions pretty similarly compared to a norepinephrine infusion).
 - It is safe to give peripherally.
 - Phenylephrine is about ten times less potent than norepinephrine (i.e. 10 mcg/min phenylephrine is roughly equivalent to 1 mcg/min norepinephrine). Phenylephrine is generally supplied as a fairly *dilute* solution, which can make this logistically problematic for patients requiring high-dose vasoconstriction. Thus, phenylephrine monotherapy is largely restricted to patients with mild to moderate vasodilatory shock due to logistic constraints.

inopressors

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norepinephrine

- Mechanism: Predominantly an alpha-agonist, with some beta-agonism as well.
- Physiology
 - Increases systemic vascular resistance (SVR), causes venos constriction (increasing preload), and has an inotropic/chronotropic effect.
 - Increases blood pressure and may increase urine output.
 - Tends to cause cardiac output to increase or remain stable (depending on how responsive the heart is to preload, afterload, and inotropy).
- Clinical use
 - Widely popular first-line agent for a variety of shock states (septic shock, cardiogenic shock with severe hypotension).
 - Good “broad spectrum” vasoactive agent when it's unclear precisely what is going on.
- How to titrate:
 - Typically, against blood pressure.
 - **There is no “maximal dose”** (<https://emcrit.org/pulmcrit/high-dose-vasopressor/>) of norepinephrine.
- Pro/Con
 - Strong track record in septic and cardiogenic shock.

epinephrine

- Mechanism: At lower doses the beta-agonist effects may predominate; with ongoing up-titration there are increasing alpha-agonist effects as well.
- Physiology
 - Causes chronotropy and inotropy, thereby increasing the cardiac output.
 - Increases systemic vascular resistance and also causes venos constriction (increasing preload).

- Stabilizes mast cells, blocking the pathophysiology of anaphylaxis.
- Beta-2 agonist stimulation causes bronchodilation, decreases potassium levels, and stimulates the generation of *aerobic lactate production* by the liver. This is often feared, but lactate may be used as a metabolic fuel by the heart, so this mechanism of action is **probably beneficial** (<https://emcrit.org/pulmcrit/understanding-lactate-in-sepsis-using-it-to-our-advantage/>) (in the absence of profound pre-existing metabolic acidosis).
- Clinical uses
 - (1) Bradycardia and bradycardic shock (given inotropic effects).
 - (2) Septic shock (shown in the 🐱 **CAT trial** (<https://www.ncbi.nlm.nih.gov/pubmed/18654759>) to be an adequate alternative to norepinephrine). It seems to work especially well in patients with inappropriately low heart rate and/or low cardiac output, who likely have a poor sympathetic response to sepsis (more on this [here](https://emcrit.org/pulmcrit/epinephrine-challenge-septic-shock/) (<https://emcrit.org/pulmcrit/epinephrine-challenge-septic-shock/>)).
 - (3) At low doses (below 5-10 mcg/kg/min), the predominant effect is as an inotrope, so it can be used for patients with low-output cardiogenic shock. Compared to dobutamine/milrinone, low-dose epinephrine has a touch of alpha-activity which will tend to prevent hypotension.
 - (4) Push-dose epinephrine is useful for patients crashing from a variety of causes (e.g. **bradycardic peri-arrest** (<https://emcrit.org/pulmcrit/epinephrine-atropine-bradycardia/>)). Epinephrine is generally a good choice for the nearly-dead patient.
 - (5) First-line agent for anaphylaxis. Note that epinephrine may be indicated for treatment for anaphylaxis even if the hemodynamics are stable (see **IBCC anaphylaxis chapter** (<https://emcrit.org/ibcc/anaphylaxis/>)).
- How to titrate: depends on clinical application.
- Pro/Con
 - Epinephrine is a powerful drug with established efficacy in sepsis, also useful in bradycardia and cardiogenic shock.
 - The main concern is that at high doses for long periods of time, it may promote a stress cardiomyopathy.
 - It causes lactate production which isn't dangerous (may be physiologically **beneficial** (<https://emcrit.org/pulmcrit/understanding-lactate-in-sepsis-using-it-to-our-advantage/>)). However, practitioners must be aware of this issue; otherwise they may senselessly chase lactate values.
 - Epinephrine causes a small decrease in potassium, which is generally not a problem. Effects on potassium may be useful in patients with hyperkalemia and bradycardia (**BRASH syndrome** (<https://emcrit.org/pulmcrit/brash-syndrome-bradycardia-renal-failure-av-blocker-shock-hyperkalemia/>)).

dopamine

- Mechanism/physiology
 - Dopamine hits a variety of receptors at different dose ranges ("dirty" drug).
 - It's often difficult to figure out what it is doing to your patient. For example, low-dose dopamine can actually cause *hypotension* (due to a predominant effect of vasodilation), which can make it difficult to wean off the dopamine.
- Reasons dopamine should be abandoned:
 1. Dopamine increases mortality in RCTs: Dopamine increased mortality compared to norepinephrine in the subgroup of patients with cardiogenic shock (**De Backer 2010** (<https://www.ncbi.nlm.nih.gov/pubmed/20200382>)). It also increased mortality compared to epinephrine among septic children (**Ventura 2015** (<https://www.ncbi.nlm.nih.gov/pubmed/26323041>)).
 2. It's often impossible to figure out what dopamine is doing (given the variety of different effects at different doses in different patients). This makes it impossible to titrate in any rational fashion (up-titration may cause dopamine to function via a different mechanism entirely).
 3. Dopamine has unique adverse endocrine effects.
 4. Dopamine may directly stimulate diuresis via action on dopamine-receptors, thereby falsely suggesting that renal perfusion is adequate.
 5. There is a relatively high risk of tissue necrosis if it extravasates.
 6. Better agents exist: there is nothing dopamine does that can't be achieved with the use of norepinephrine and/or epinephrine.
 7. Dopamine may cause greater malperfusion of the gut compared to norepinephrine.

peripheral pressors

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peripheral IV line

- Hemodynamic stabilization should never wait until central access is obtained. Thus, peripheral vasopressors should be started immediately if the blood pressure or perfusion is inadequate.
- Norepinephrine is safe for short periods of time through a large peripheral vein. Ongoing peripheral infusion also appears safe, but this should ideally be done within the context of a well-designed protocol involving frequent monitoring of the extremity and preparation for management of extravasation reaction (more on this [here \(https://emcrit.org/emcrit/peripheral-vasopressors-extravasation/\)](https://emcrit.org/emcrit/peripheral-vasopressors-extravasation/)). Ongoing infusion should be *avoided* in deep ultrasound-guided peripheral IVs, where it may be impossible to monitor the tissue surrounding the end of the IV cannula.
- Phenylephrine and epinephrine have *not* been reported to cause tissue necrosis. Peripheral infusion of these agents appears to be generally safe, although this should still ideally be done via a well-functioning cannula proximal to the wrist (more on this [here \(https://emcrit.org/pulmcrit/phenylephrine-epinephrine-central-access/\)](https://emcrit.org/pulmcrit/phenylephrine-epinephrine-central-access/)).
- Vasopressin should arguably be avoided for peripheral administration, because if it extravasates there is no vasodilatory agent which can counteract its action.

midline catheter

- These are catheters placed in the arm, similar to a PICC, but shorter (typically 10-20 cm in length, terminating before the shoulder).
- Clinician-placed midlines are evolving as an alternative to either ultrasound-guided peripheral IVs or central lines.
- This is a rapidly emerging topic. Overall, vasopressor administration via midline catheters appears to be safe.
- More on midline catheters: see [EMCrit RACC Midlines part 1 \(https://emcrit.org/emcrit/midlines-1/\)](https://emcrit.org/emcrit/midlines-1/) & [part 2 \(https://emcrit.org/emcrit/midlines-part-2/\)](https://emcrit.org/emcrit/midlines-part-2/).

midodrine

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basics

- Oral alpha-1 agonist, which acts as a pure vasopressor.
- Overall behavior is similar to that of phenylephrine (may be conceptualized as “oral phenylephrine”).

more common clinical indications in critical care:

- Septic shock
 - It can be used to accelerate liberation from vasopressor infusions.
 - Midodrine has the same mechanism of action as phenylephrine. Prior to initiation of oral midodrine, it may make sense to transition the patient to IV phenylephrine. If the patient responds favorably to low-dose phenylephrine, this suggests that they may be a good candidate to transition to oral midodrine.
- Cirrhosis & hepatorenal syndrome
 - Midodrine is a component of oral therapy for hepatorenal syndrome.
 - Some evidence suggests that *ongoing* midodrine therapy in patients with cirrhosis may support renal perfusion (given that these patients suffer from chronic vasodilation).

dose

- The usual starting dose is 10 mg PO q8hr. *Make sure the drug is dosed q8hr and not “three times daily with meals,” which is what the computer may default to.*
- Dose range is 5-40 mg q8hr (26953217).
- Midodrine is cleared by the kidney, so exercise caution in renal dysfunction.

contraindications/cautions

- Reflex bradycardia can occur.
- The major limitation is that midodrine should be weaned off after the patient is discharged from the ICU to the ward. Failure to wean off midodrine in a timely fashion could potentially lead to harm. One study showed that midodrine was commonly continued and administered alongside antihypertensives. Although this may be justifiable in patients with heart failure and reduced ejection fraction, in many cases it was likely an error (31107279, 31524705).

more on midodrine:

- PulmCrit blog [here \(https://emcrit.org/pulmcrit/midodrine-icu/\)](https://emcrit.org/pulmcrit/midodrine-icu/).
- Mayo Clinic experience with 1,119 patients treated with oral midodrine ([Rizvi 2018 \(https://www.ncbi.nlm.nih.gov/pubmed/29613861\)](https://www.ncbi.nlm.nih.gov/pubmed/29613861)).

methylene blue

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mechanisms of action

- (1) Methylene blue causes vasoconstriction by inhibiting nitric oxide synthase.
 - This is a potentially dangerous way to increase blood pressure, because it could potentially impair microvascular perfusion.
 - Historically, a nitric oxide synthesis inhibitor was shown to increase mortality in septic shock ([14707556 \(https://www.ncbi.nlm.nih.gov/pubmed/14707556\)](https://www.ncbi.nlm.nih.gov/pubmed/14707556)).
- (2) Methylene blue inhibits the conversion of guanine triphosphate to cGMP (an intracellular signaling molecule which increases vasodilation).
- (3) Methylene blue may be able to accept electrons from NADH and transfer them to cytochrome C in the mitochondria, thereby *bypassing* parts of the electron transport chain. This could restore mitochondrial function in some situations where parts of the electron transport chain are dysfunctional, for example metformin toxicity (28840449).

more common indications

- Refractory vasoplegic shock of any etiology.
 - *Especially* following cardiothoracic surgery.
 - Possibly also: septic shock, anaphylaxis.
- Metformin poisoning

dosing

- #1) Test dose of 2 mg/kg infused over 15 minutes.
 - If no response, then try another medication or treatment strategy.
 - If response seen, then consider initiating an infusion...
- #2) Infusion:
 - Dose range from 0.25 – 2 mg/kg/hour.
 - May be continued for up to 48-72 hours. Wean off when hemodynamics improve.

potential adverse effects / contraindications

1. Inhibition of cGMP may increase pulmonary vascular resistance, thereby impairing right ventricular function and impairing oxygenation. This may be more of a problem at higher doses.
2. High levels of methylene blue can interfere with pulse oximetry (a problem mostly when giving the bolus dose).
3. Methylene blue can act as an oxidizing agent at high doses (e.g. >7 mg/kg). This may cause methemoglobinemia. In patients with G6PD deficiency, this could also cause hemolytic anemia.
4. Methylene blue inhibits monoamine oxidase A (MAO), thereby increasing brain serotonin levels. This could cause serotonin syndrome in the presence of other serotonergic agents.
5. Methylene blue may inhibit CYP enzyme metabolism, leading to accumulation of some medications (e.g. digoxin, warfarin, fentanyl).
6. Methylene blue is contraindicated in pregnancy (due to a potential for placental vasoconstriction and fetal hypoxemia).

podcast

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<https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/apps.40518.14127333176902609.7be7b901-15fe-4c27-863c-7c0dbfc26c5c.5c278f58-912b-4af9-88f8-a65fff2da477.jpg>

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questions & discussion

To keep this page small and fast, questions & discussion about this post can be found on another page [here](https://emcrit.org/pulmcrit/pressors/).



<https://i0.wp.com/emcrit.org/wp-content/uploads/2016/11/pitfalls2.gif>

- Any use of dopamine (there are better agents).
- Failure to aggressively up- and down-titrate vasopressors to determine the physiologic effect of each on any specific patient. Medications may not behave as described in a textbook (even this book!). By continually adjusting infusion rates, it is often possible to get a sense of which agents are most effective. The goal is always to use the minimal total dose of vasopressors necessary to achieve hemodynamic targets, so if a drug doesn't seem to be having any effect then wean it off.
- Ongoing infusion of vasopressin despite evidence of malperfused digits.
- Excess use of vasopressors (especially the combination of norepinephrine and vasopressin), which can lead to a phenomenon of iatrogenic vasoconstrictive shock in patients with poor cardiac function (wherein excessive vasoconstriction causes a drop in cardiac output, *causing low cardiac output and shock*). Beware of this phenomenon. When encountered, consider the *addition* of epinephrine and down-titration of vasoconstrictors.
- Use of agents with beta-agonist activity in patients with atrial fibrillation and rapid ventricular response.
- Failure to up-titrate norepinephrine beyond arbitrary "upper limits" imposed by hospitals or local culture (there is actually no upper limit on the norepinephrine dose).
- Fear of using epinephrine due to concerns that it may increase the lactate (increased lactate levels due to epinephrine are probably beneficial in most cases).
- Delaying vasopressor initiation until central access is obtained (instead, peripheral vasopressor infusion should be used to immediately stabilize the patient).

Going further:

- Pressor overview
 - [EMCrit RACC 138](https://emcrit.org/emcrit/vasopressor-basics/)
- Pure pressors
 - [Alternative viewpoint on phenylephrine infusions](https://emcrit.org/pulmcrit/phenylephrine-infusion/)
 - [Oral vasopressors to accelerate liberation from ICU](https://emcrit.org/pulmcrit/midodrine-icu/)
- Epinephrine & lactate
 - [Epinephrine & significance of lactate](https://emcrit.org/pulmcrit/understanding-lactate-in-sepsis-using-it-to-our-advantage/)
- Titration & selection issues
 - [Why we fail at hemodynamics: cognitive errors](https://emcrit.org/pulmcrit/hemodynamics-swan-curse/)
 - [Epinephrine responsiveness & challenge](https://emcrit.org/pulmcrit/epinephrine-challenge-septic-shock/)
- Peripheral pressors & midlines
 - [Peripheral pressors](https://emcrit.org/emcrit/peripheral-vasopressors-extravasation/) (EMCrit RACC 107)
 - [Are peripheral vasopressors safe](https://rebelem.com/rebel-cast-ep73-are-peripheral-vasopressors-safe/) (Salim Rezaie, RebelCast)
 - [Phenylephrine & epinephrine can be infused peripherally](https://emcrit.org/pulmcrit/phenylephrine-epinephrine-central-access/)
 - Midlines: [EMCrit RACC part 1](https://emcrit.org/emcrit/midlines-1/) & [part 2](https://emcrit.org/emcrit/midlines-part-2/).
- Vasopressin
 - [Vasopressin... & renal microvascular hemodynamics](https://emcrit.org/pulmcrit/renal-microvascular-hemodynamics-in-sepsis-a-new-paradigm/)
 - [Vasopressin, vepinephrine, & VANISH](https://emcrit.org/pulmcrit/vanish-renoresuscitation-vasopressin-vepinephrine/)
 - [VANCS trial: vasopressin for post-CABG shock](https://emcrit.org/pulmcrit/vasopressin-vancs/)

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