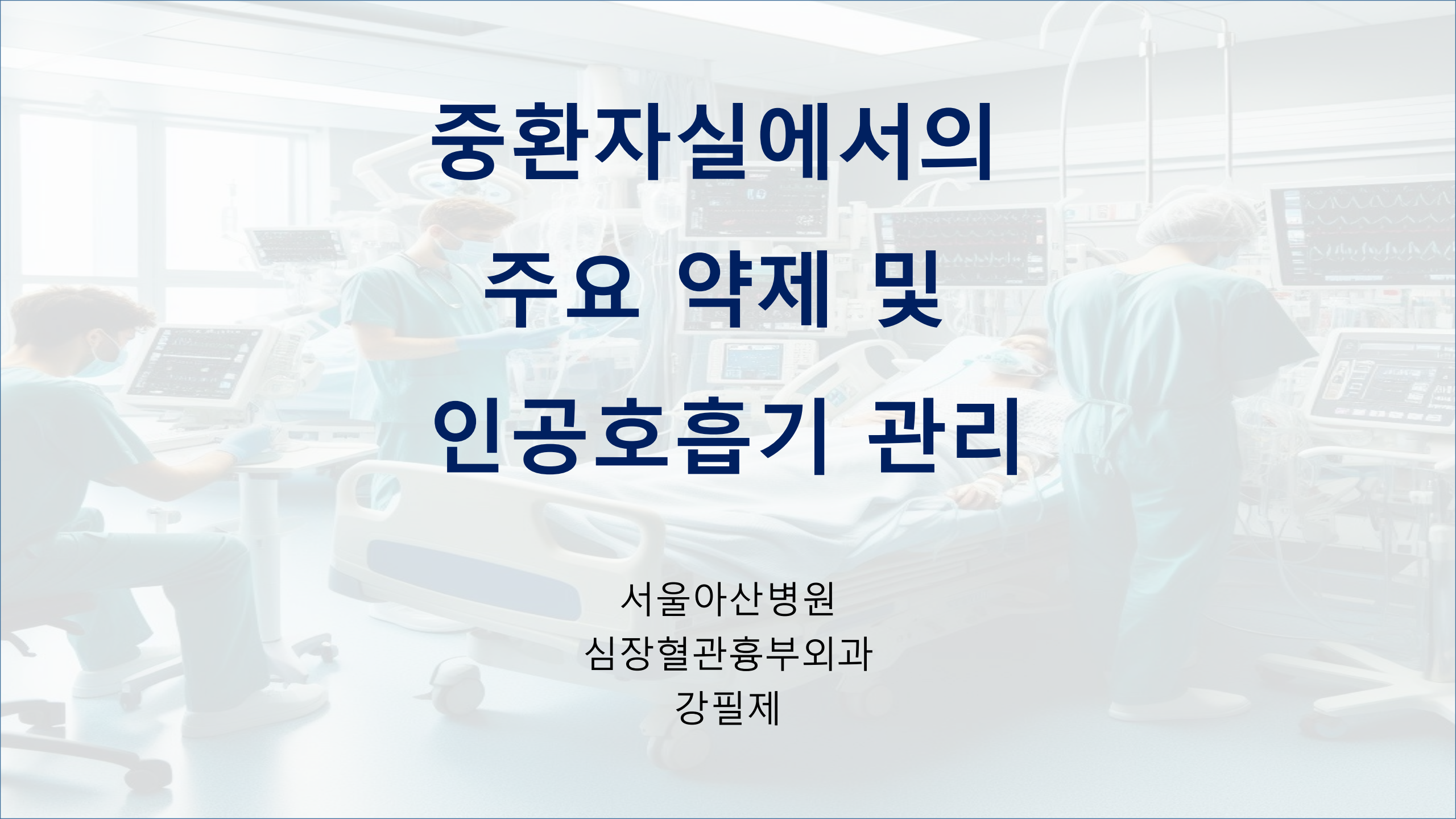

중환자실에서의 주요 약제 및 인공호흡기 관리

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강필제

Contents

- Inotropics & Vasopressors
- Sedatives-analgesia
- Mechanical Ventilation Management

Inotropics & Vasopressors

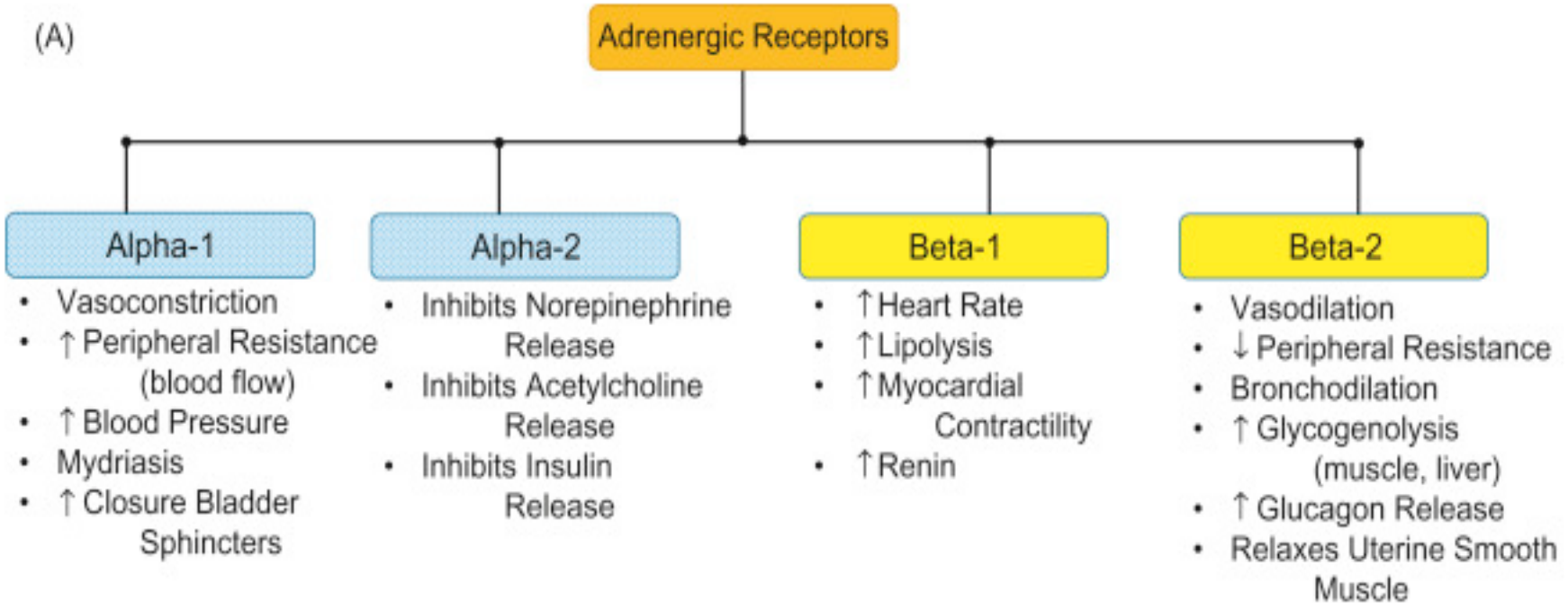
- Inotropic and vasopressor agents have increasingly become a **therapeutic cornerstone** for the management of several important cardiovascular syndromes.
- They are generally administered with the assumption that short- to medium-term clinical recovery will be facilitated **by enhancement of cardiac output (CO) or vascular tone** that has been severely compromised by often life-threatening clinical conditions.

Inotropics & Vasopressors

- Catecholamines mediate their cardiovascular actions predominantly through **α, β_1, β_2, and dopaminergic receptors**, the density and proportion of which modulate the physiological responses of inotropes and pressors in individual tissues.
- The relative binding affinities of individual inotropes and vasopressors to adrenergic receptors can be altered *by hypoxia or acidosis*, which mutes their clinical effect.

Inotropics & Vasopressors

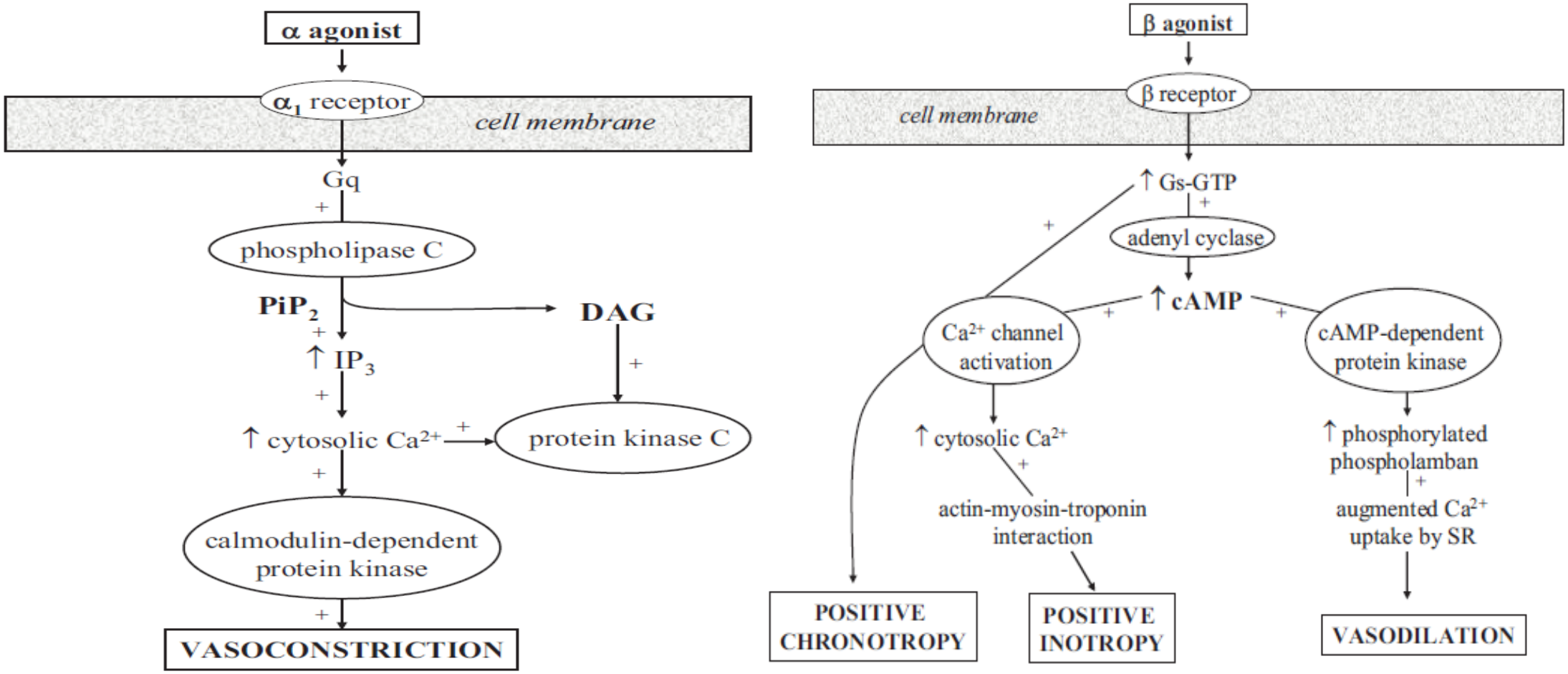
(A)



(B)

Alpha-1	Alpha-2	Beta-1	Beta-2
NE > E	E > NE	E = NE	E >> NE
NE = Norepinephrine; E = Epinephrine			

Inotropics & Vasopressors



Inotropics & Vasopressors

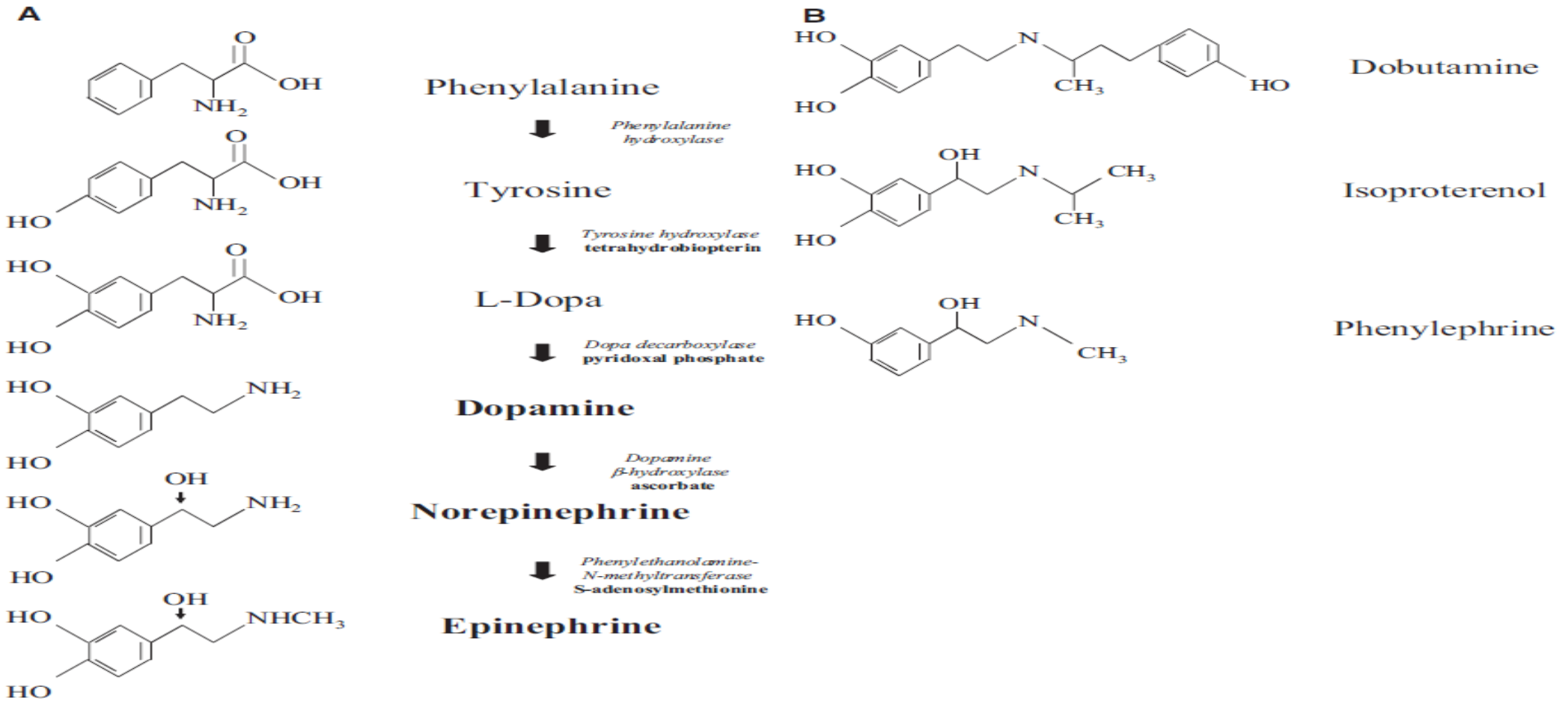
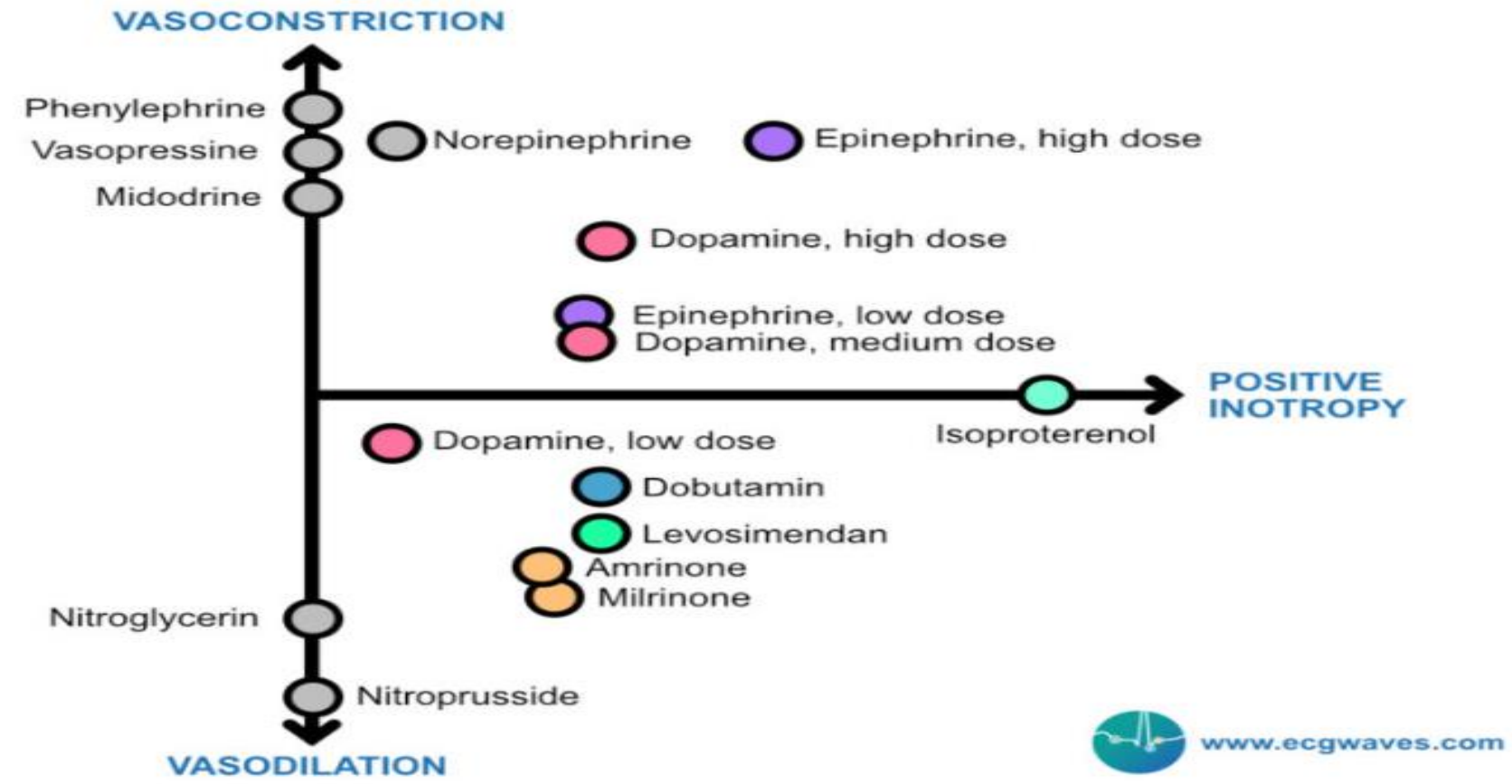


Figure 3. A, Endogenous catecholamine synthesis pathway. Left, chemical structures; Right, names of compounds with conversion enzymes (italics) and cofactors (bold). B, Chemical structures and names of common synthesized catecholamines.

Inotropics & Vasopressors



Inotropes and vasopressors.

Table 1. Drug manual for inotropes and vasopressors

Inotropics & Vasopressors

Dopamine (2 to 20 mcg/kg/minute)

- An alternative to norepinephrine in septic shock in **highly selected** patients (eg, with absolute or relative bradycardia and a low risk of tachyarrhythmias).
- More adverse effects (eg, **tachycardia**, arrhythmias particularly at doses ≥20 mcg/kg/minute) and less effective than norepinephrine for reversing hypotension in septic shock.
- Lower doses (eg, 1 to 3 mcg/kg/minute) **should not** be used for renal protective effect and can cause hypotension during weaning.

Inotropics & Vasopressors

Dobutamine (beta1 adrenergic, 2 to 20 mcg/kg/minute)

- **Initial agent of choice** in cardiogenic shock with low cardiac output and maintained blood pressure.
- Add-on to norepinephrine for cardiac output augmentation in septic shock with myocardial dysfunction (eg, in elevated left ventricular filling pressures and adequate MAP) or ongoing hypoperfusion despite adequate intravascular volume and use of vasopressor agents.
- **Increases cardiac contractility and rate**; may cause hypotension and tachyarrhythmias.

Inotropics & Vasopressors

Norepinephrine (alpha-1 adrenergic, 0.02 to 0.5 mcg/kg/minute)

- Initial vasopressor of **choice** in septic, cardiogenic, and hypovolemic shock.
- Wide range of doses utilized clinically.

Inotropics & Vasopressors

Epinephrine (alpha-1 adrenergic, 0.01 to 0.5mcg/kg/minute)

- Initial vasopressor of choice in **anaphylactic shock**.
- Typically an add-on agent to norepinephrine in septic shock when an additional agent is required to raise MAP to target and occasionally an alternative first-line agent if norepinephrine is contraindicated.
- Increases heart rate; may induce tachyarrhythmias and ischemia.
- For inotropy, doses in the higher end of the suggested range is needed.
- *Elevates lactate concentrations during initial administration*

(ie, may preclude use of lactate clearance goal); may decrease mesenteric perfusion.

Inotropics & Vasopressors

Phenylephrine (alpha-1 adrenergic, 0.25 to 5 mcg/kg/min)

- Pure alpha-adrenergic vasoconstrictor.
- May be considered when tachyarrhythmias preclude use of norepinephrine.
- Alternative vasopressor for patients with septic shock who:

(1) develop tachyarrhythmias on norepinephrine, epinephrine, or dopamine,

(2) have persistent shock despite use of two or more vasopressor/inotropic agents including vasopressin (salvage therapy), or

(3) high cardiac output with persistent hypotension.

- May decrease stroke volume and cardiac output in patients with cardiac dysfunction.
- May be given as **bolus** dose of 50 to 100 mcg to support blood pressure during rapid sequence intubation.

Inotropics & Vasopressors

Vasopressin (0.01 to 0.1 units/min)

- Add-on to norepinephrine to raise blood pressure to target MAP or decrease norepinephrine requirement.
  Not recommended as a replacement for a first-line vasopressor.
- Pure vasoconstrictor; may decrease stroke volume and cardiac output in myocardial dysfunction or precipitate ischemia in coronary artery disease.

Inotropics & Vasopressors

Milrinone (nonadrenergic, PDE3 inhibitor, 0.125 to 0.75 mcg/kg/min)

- Alternative for short-term cardiac output augmentation to maintain organ perfusion in cardiogenic shock refractory to other agents.
- Increases cardiac contractility and modestly increases heart rate at high doses; may cause peripheral vasodilation, hypotension, and/or ventricular arrhythmia.
- Renally cleared; dose adjustment in renal impairment needed.

Inotropics & Vasopressors

COMPLICATIONS

- Vasopressors and inotropic agents have the potential to cause a number of significant complications, including hypoperfusion, dysrhythmias, myocardial ischemia, local effects, and hyperglycemia.

In addition, a number of drug interactions exist.

  - Hypoperfusion — Excessive vasoconstriction in response to hypotension and vasopressors can produce inadequate perfusion of the extremities, mesenteric organs, or kidneys. Excessive vasoconstriction with inadequate perfusion, usually with an systemic vascular resistance (SVR) >1300 dynes x sec/cm5, commonly occurs in the setting of inadequate cardiac output or inadequate volume resuscitation.

Inotropics & Vasopressors

COMPLICATIONS

- The initial findings are dusky skin changes at the tips of the fingers and/or toes, which may progress to frank necrosis with autoamputation of the digits. Compromise of the renal vascular bed may produce renal insufficiency and oliguria, while patients with underlying peripheral artery disease may develop acute limb ischemia.
- Inadequate mesenteric perfusion increases the risk of gastritis, shock liver, intestinal ischemia, or translocation of gut flora with resultant bacteremia. Despite these concerns, maintenance of MAP with vasopressors appears more effective in maintaining renal and mesenteric blood flow than allowing the MAP to drop, and maintenance of MAP with vasopressors may be life-saving despite evidence of localized hypoperfusion.

Inotropics & Vasopressors

COMPLICATIONS

- Dysrhythmias — Many vasopressors and inotropes exert **powerful chronotropic effects** via stimulation of beta-1 adrenergic receptors. This increases the risk of sinus tachycardia (most common), atrial fibrillation(potentially with increased atrioventricular nodal [A-V] conduction and therefore an increased ventricular response), re-entrant atrioventricular node tachycardia, or ventricular tachyarrhythmias. **Adequate volume loading** may minimize the frequency or severity of dysrhythmias. Despite this, dysrhythmias often limit the dose and necessitate switching to another agent with less prominent beta-1 effects.

Inotropics & Vasopressors

COMPLICATIONS

- Myocardial ischemia — The chronotropic and inotropic effects of beta-adrenergic receptor stimulation can increase **myocardial oxygen consumption**. While there is usually coronary vasodilation in response to vasopressors, perfusion may still be inadequate to accommodate the increased myocardial oxygen demand. Daily electrocardiograms on patients treated with vasopressors or inotropes may screen for occult ischemia, and excessive tachycardia should be avoided because of impaired diastolic filling of the coronary arteries.
- Hyperglycemia — Hyperglycemia may occur due to **the inhibition of insulin secretion**. The magnitude of hyperglycemia generally is minor and is more pronounced with **norepinephrine and epinephrine** than dopamine. Monitoring of blood glucose while on vasopressors can prevent complications of untreated hyperglycemia.

Inotropics & Vasopressors

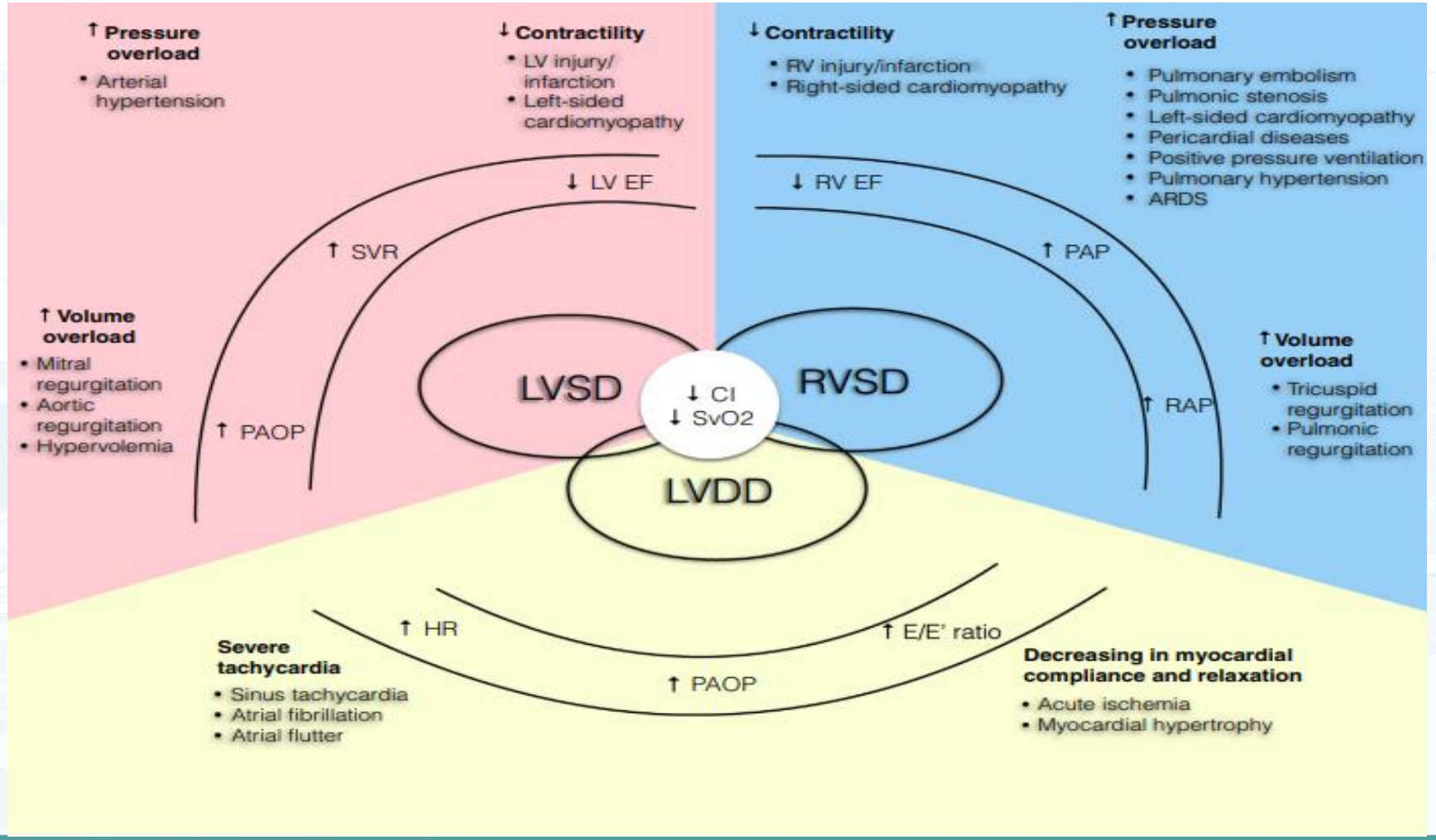
Table. Inotropic and Vasopressor Drug Names, Clinical Indication for Therapeutic Use, Standard Dose Range, Receptor Binding (Catecholamines), and Major Clinical Side Effects

Drug	Clinical Indication	Dose Range	Receptor Binding				Major Side Effects
			α1	β1	β2	DA	
Catecholamines							
Dopamine	Shock (cardiogenic, vasodilatory) HF Symptomatic bradycardia unresponsive to atropine or pacing	2.0 to 20 $\mu g \cdot kg^{-1} \cdot min^{-1}$ (max 50 $\mu g \cdot kg^{-1} \cdot min^{-1}$)	+++	++++	++	+++++	Severe hypertension (especially in patients taking nonselective β-blockers) Ventricular arrhythmias Cardiac ischemia Tissue ischemia/gangrene (high doses or due to tissue extravasation)
Dobutamine	Low CO (decompensated HF, cardiogenic shock, sepsis-induced myocardial dysfunction) Symptomatic bradycardia unresponsive to atropine or pacing	2.0 to 20 $\mu g \cdot kg^{-1} \cdot min^{-1}$ (max 40 $\mu g \cdot kg^{-1} \cdot min^{-1}$)	+	+++++	+++	N/A	Tachycardia Increased ventricular response rate in patients with atrial fibrillation Ventricular arrhythmias Cardiac ischemia Hypertension (especially nonselective β-blocker patients) Hypotension
Norepinephrine	Shock (vasodilatory, cardiogenic)	0.01 to 3 $\mu g \cdot kg^{-1} \cdot min^{-1}$	+++++	+++	++	N/A	Arrhythmias Bradycardia Peripheral (digital) ischemia Hypertension (especially nonselective β-blocker patients)
Epinephrine	Shock (cardiogenic, vasodilatory) Cardiac arrest Bronchospasm/anaphylaxis Symptomatic bradycardia or heart block unresponsive to atropine or pacing	Infusion: 0.01 to 0.10 $\mu g \cdot kg^{-1} \cdot min^{-1}$ Bolus: 1 mg IV every 3 to 5 min (max 0.2 mg/kg) IM: (1:1000): 0.1 to 0.5 mg (max 1 mg)	+++++	++++	+++	N/A	Ventricular arrhythmias Severe hypertension resulting in cerebrovascular hemorrhage Cardiac ischemia Sudden cardiac death
Isoproterenol	Bradyarrhythmias (especially torsade des pointes) Brugada syndrome	2 to 10 μg/min	0	+++++	+++++	N/A	Ventricular arrhythmias Cardiac ischemia Hypertension Hypotension
Phenylephrine	Hypotension (vagally mediated, medication-induced) Increase MAP with AS and hypotension Decrease LVOT gradient in HCM	Bolus: 0.1 to 0.5 mg IV every 10 to 15 min Infusion: 0.4 to 9.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$	+++++	0	0	N/A	Reflex bradycardia Hypertension (especially with nonselective β-blockers) Severe peripheral and visceral vasoconstriction Tissue necrosis with extravasation

Inotropics & Vasopressors

Drugs	HR	MAP	CO	PVR	Bronchodilation	RBF
Epinephrine	↑↑	↑	↑↑	↑/↓	↑↑	↓↓
Ephedrine	↑↑	↑↑	↑↑	↑	↑↑	↓↓
Norepinephrine	↓	↑↑↑	↓/↑	↑↑↑	0	↓↓↓
Dopamine	↑/↑↑	↑	↑↑↑	↑	0	↑↑↑
Dopexamine	↑/↑↑	↓/↑	↑↑	↑	0	↑
Isoproterenol	↑↑↑	↓	↑↑↑	↓↓	↑↑↑	↓/↑
Dobutamine	↑	↑	↑↑↑	↓	0	↑

Low-Cardiac-Output Syndrome After Cardiac Surgery

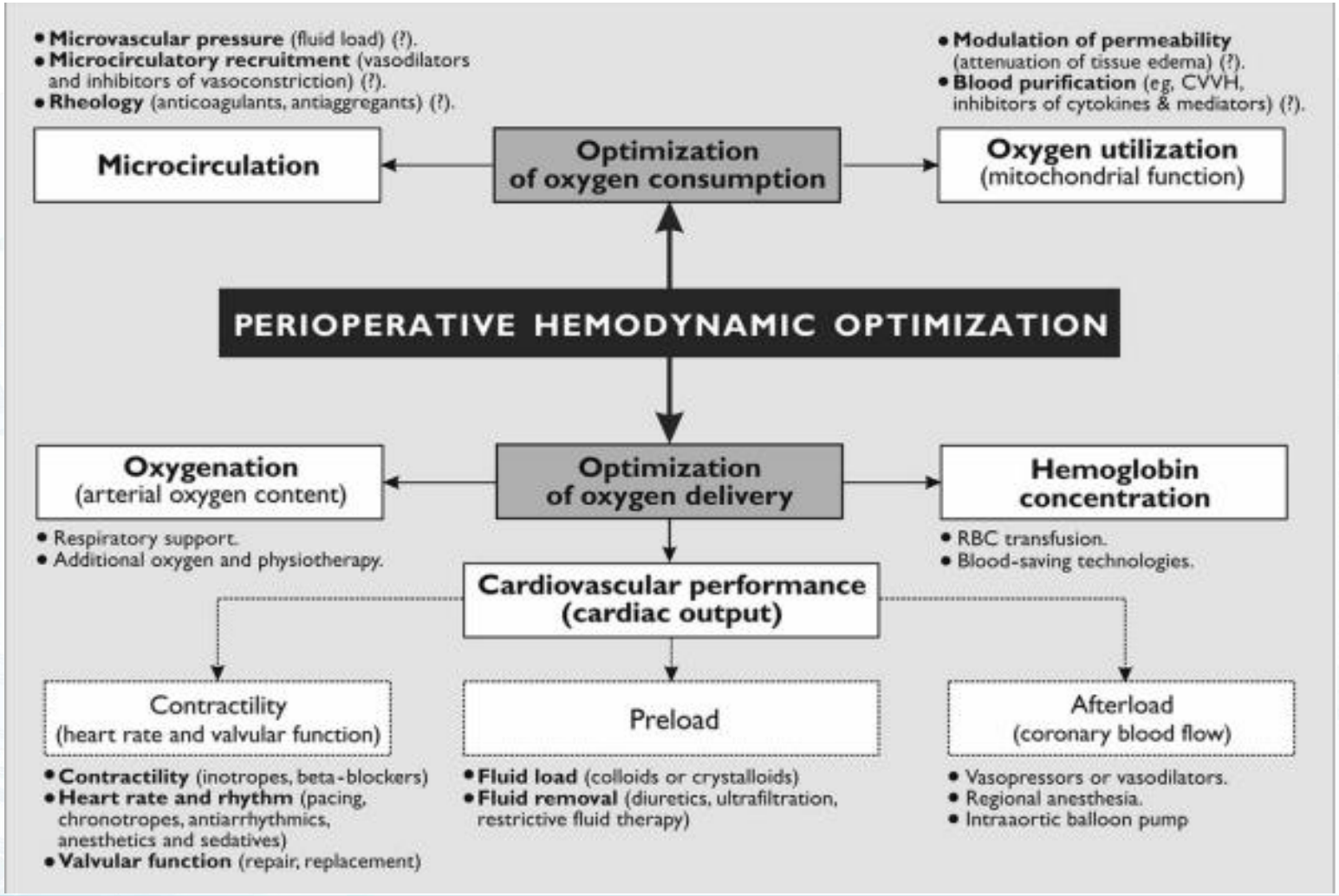


Low-Cardiac-Output Syndrome After Cardiac Surgery

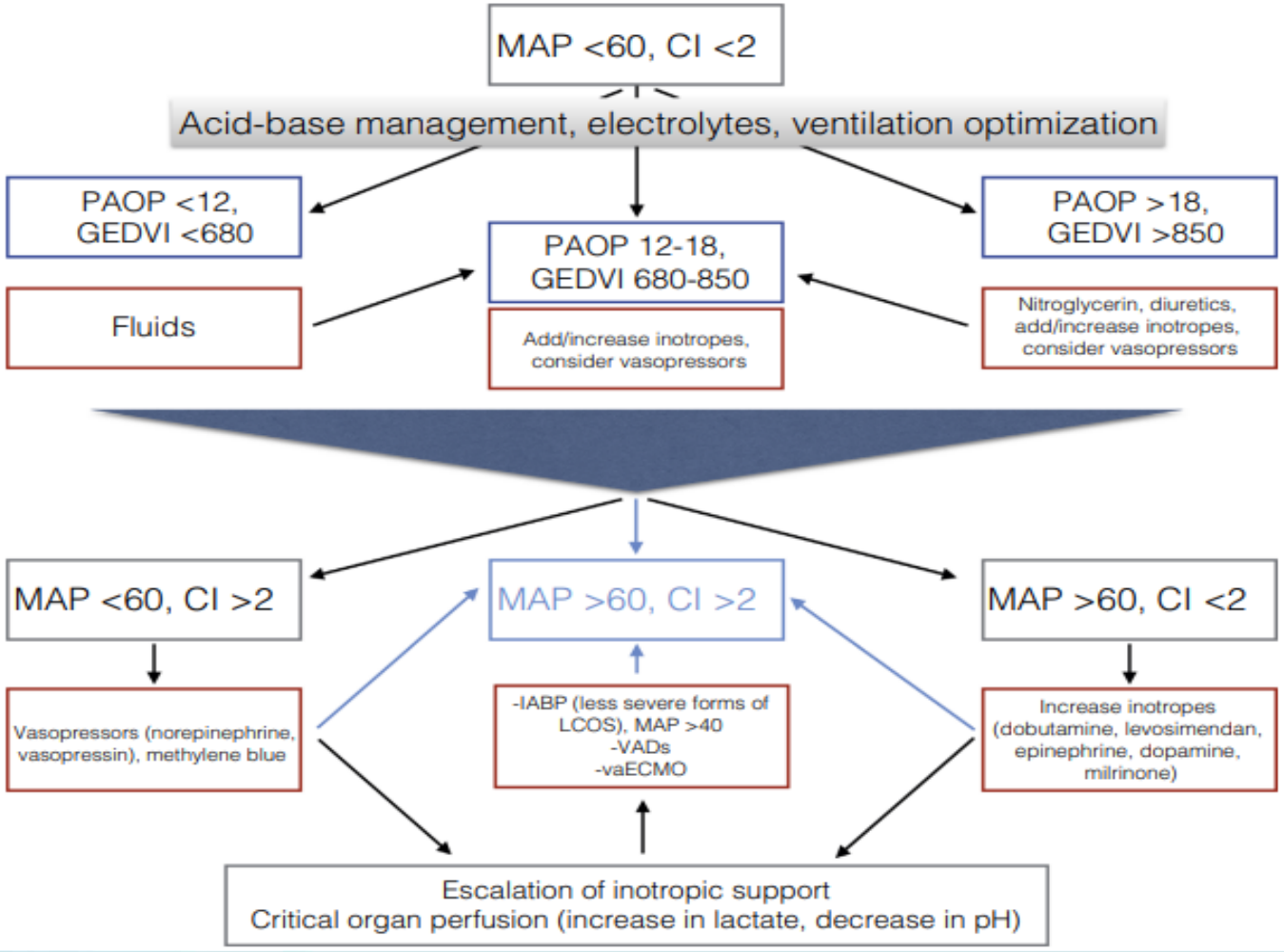
Advantages and Disadvantages of Different Hemodynamic Monitoring Devices in Cardiac Surgery

	Invasiveness	Reliability in Cardiac Surgery Patients	Ease of Use	Ability to Monitor CO in Real Time	Ability to Measure Variables Other Than CO
Ultrasound techniques*	+	++	+	+	+++
Pulmonary artery catheter	+++	+++	+	++†	++
Transpulmonary thermodilution	++	+++	++	+++	+++
Lithium dilution	++	+++	+	+++	++
Uncalibrated pulse contour analysis	+	+	++	+++	+
Applanation tonometry	0	+	+++	+++	+
Estimated continuous cardiac output	0	+/-	+++	+++	+
Bioreactance	0	+/-	++	+++	+

Low-Cardiac-Output Syndrome After Cardiac Surgery



Low-Cardiac-Output Syndrome After Cardiac Surgery



Sedative-analgesic medications

Fentanyl (1 to 3 mcg/kg/hour infusion)

- **Advantages**: Potent analgesic-sedative with immediate onset and less hypotension than other opioid analgesic choices due to relative lack of histamine release. Metabolized hepatically by CYP3A4 to inactive metabolites.
- **Disadvantages**: Highly lipophilic parent drug accumulates in adipose and other tissue with repeated or prolonged administration. Chest wall rigidity may occur with higher dosing.
- **Role**: A good choice for analgesia for most critically ill patients.

Sedative-analgesic medications

Remifentanil 0.5 to 15 mcg/kg/hour infusion

- **Advantages:** Ultra-short-acting. Cleared by nonspecific plasma esterases to inactive metabolites. Does not accumulate in renal or hepatic impairment. Prompt reversal of analgesia and sedation upon discontinuation.
- **Disadvantages:** Anticipate pain and discomfort upon abrupt cessation. Glycine excipient may accumulate in renal impairment.
- **Role:** An alternative to fentanyl for patients requiring frequent neurologic assessments or those with multiorgan failure.

Sedative-analgesic medications

Remifentanil

기계적 환기 중인 중환자의 진통 및 진정을 위해서 이 약을 초기에 단독투여할 수 있다.
이 약의 초기투여속도로 0.1~0.15μg/kg/분(6~9μg/kg/h)이 권장된다. 원하는 만큼의 진정 및 진통 효과에 도달할 때까지 0.025μg/kg/분(1.5μg/kg/시간)의 증가속도로 이 약의 주입속도를 조절해야 한다. 용량을 조정할 때에는 최소 5분의 간격을 두어야 한다. 진정 및 진통 정도를 주의깊게 모니터링하고 규칙적으로 재평가하여 이 약의 주입속도를 적절하게 조정해야 한다. 주입속도가 0.2μg/kg/분(12μg/kg/시간)에 도달했으나 원하는 만큼의 진정효과에 도달하지 않았을 때에는 아래 표에 따라 적절한 양의 진통제 투여를 시작해야 한다. 원하는 진정 효과에 도달할 때까지 진정제의 용량을 조절해야 한다. 추가적인 진통이 필요할 경우 0.025μg/kg/분(1.5μg/kg/시간) 증가속도로 이 약의 주입속도를 증가시킬 수 있다.
대조임상시험에서 최대 3일 동안 중환자에게 이 약을 시험하였다. 3일을 초과하여 환자에게 이 약을 시험한 바는 없으므로 더 장시간 동안 투여시의 안전성과 유효성에 대한 증거는 확보되지 않았다.
각 피험자에서 진통 및 전정 효과가 나타났던 정맥용 연속 투여시의 초기주입속도 및 유지속도를 다음 표에 요약하였다.

정맥용 연속투여 μg/kg/분(μg/kg/시간)	
초기유지속도	유지속도
0.1~0.15 (6~9)	0.006~0.74 (0.38~44.6)

중환자에서 이 약의 정맥용 단회 주입은 권장되지 않는다.
이 약은 병용투여 중인 다른 진정제의 투여량을 감소시킬 것이다. 필요할 경우, 진정제의 초기용량은 다음과 같다.

진정제	정맥용 단회 주입(mg/kg)	정맥용 연속투여 (μg/kg/시간)
프로포폴 미다졸람	~ 0.5 ~ 0.03	0.5 0.03

각 약물을 독립적으로 조절하기 위해서 진정제를 혼합물 형태로 투여해서는 안 되며, 별도의 정맥투여 세트(running IV set)를 이용하여 투여하도록 한다.

Sedative-analgesic medications

Acetaminophen Oral, rectal: 325 to 1000 mg every 4 to 6 hours, Maximum ≤4 g/day

- **Advantages**: Lacks dependence and tolerance of opioids.

  Lacks antiplatelet effect and gastrointestinal toxicity of NSAIDs.

- **Disadvantages**: Lacks significant anti-inflammatory effect. IV preparation requires administration over 15 minutes. Can cause hepatotoxicity in chronic or acute overdose. Avoid or use a lower daily dose in older adults and patients at risk for hepatotoxicity (eg, heavy alcohol use or malnourished). Interacts with warfarin (may prolong INR) and CYP-inducing drugs (elevated risk of hepatic inflammation).

- **Role**: **First choice** for treatment of mild to moderate acute pain and febrile conditions. Adjunctive analgesic that may reduce opioid requirements. When hepatic dysfunction is significant, consider avoiding or reducing dose (eg, ≤2 g/day total).

Sedative-analgesic medications

Ketorolac

- **Advantages**: Lacks dependence and tolerance of opioids. Effective anti-inflammatory.
- **Disadvantages**: Can cause or worsen renal insufficiency. Dose-related risk of gastropathy. Reversibly inhibits platelet functioning. May alter cardioprotective effect of aspirin.
- **Role**: Adjunctive analgesic that may reduce opioid requirements.
- Avoid in renal impairment, gastrointestinal bleeding, platelet dysfunction, ischemic heart disease, heart failure, reduced cardiac output, hypovolemic state, asthma, or cirrhosis. Contraindicated in treatment of perioperative pain in coronary artery bypass graft surgery. Patients should be well hydrated.

Sedative-analgesic medications

Ibuprofen Oral: 400 mg orally every 4 hours (maximum 2.4 g/day chronic), IV: 400 to 800 mg IV every 6 hours (maximum 3.2 g/day acute)

- **Advantages:** Lacks dependence and tolerance of opioids. Effective anti-inflammatory.
- **Disadvantages:** Can cause or worsen renal insufficiency. Dose-related risk of gastropathy. Reversibly inhibits platelet functioning. Can alter cardioprotective effect of aspirin.
- **Role:** Short-term treatment of moderate acute pain and febrile conditions. Adjunctive analgesic that may reduce opioid requirements.
- **Avoid** in renal impairment, gastrointestinal bleeding, platelet dysfunction, ischemic heart disease, heart failure, reduced cardiac output, hypovolemic state, asthma, or cirrhosis. Contraindicated in treatment of perioperative pain in coronary artery bypass graft surgery. Patients should be well hydrated.

Sedative-analgesic medications

Propofol 5 to 50 mcg/kg/minute

- **Advantages**: Potent sedative-hypnotic associated with an immediate onset and rapid awakening upon discontinuation when administered for short-term use. Metabolism is reportedly unaltered in hepatic or renal impairment and subject to few significant drug interactions. Infusion is readily titratable to desired depth of sedation, minimizing risk of oversedation. Propofol effectively decreases intracranial pressure, lowers cerebral metabolism, controls intractable seizures, and may reduce shivering in the rewarming phase of induced hypothermia following resuscitation from cardiac arrest.

Sedative-analgesic medications

Propofol 5 to 50 mcg/kg/minute

- **Disadvantages**: Adverse effects include hypotension, bradycardia, respiratory depression, decreased myocardial contractility, elevated triglycerides, peripheral injection site pain, and rarely, propofol infusion syndrome. Specific product presentations may include potential allergens (egg, soy, peanut, others). Consult product label information. No analgesic effects.
- **Role**: A good choice in conjunction with appropriate analgesia for short-term sedation of patients in whom rapid awakening is advantageous. Also a good choice to decrease elevated intracranial pressure or for short-term sedation in a general critical care population that is likely to be ready soon for ventilator weaning trials.

Sedative-analgesic medications

Ketamine 0.05 to 0.4 mg/kg/hour

- **Advantages:** A potent dissociative sedative-anesthetic with marked analgesia that **maintains cardiac output** and **MAP without inhibition of respiratory drive**. Does not inhibit protective reflexes. May reduce acute opioid tolerance.
- **Disadvantages:** Sympathetic stimulation (ie, increased HR and myocardial oxygen demand, elevated intracranial pressure and systemic blood pressure) may be intolerable depending upon clinical setting. Rarely, cardiorespiratory depression associated with rapid administration or higher doses. Adverse effects may include hallucinations, delirium upon withdrawal, tonic-clonic movements, dissociative experiences, unpleasant recall, hypersalivation, nausea, and vomiting. Complex metabolism includes CYP3A4, 2C9, 2B6, and non-CYP hepatic transformations and an active metabolite (norketamine), which may accumulate in renal and/or hepatic impairment or due to drug interactions.
- **Role:** An **alternate choice** for postsurgical pain management, severe agitation, or as an adjunctive analgesic in patients with severe refractory pain in clinical settings where increased myocardial oxygen demand and sympathetic tone are tolerable.

Sedative-analgesic medications

Midazolam 0.02 to 0.1 mg/kg/hour infusion

- **Advantages**: A potent amnestic and anxiolytic agent with an immediate onset of action and a short duration of effect when administered **short term** (<48 hours). It is the only IV benzodiazepine that is not delivered in propylene glycol.
- **Disadvantages**: Hepatically metabolized by CYP3A4 to active metabolites that may accumulate and cause prolonged sedation if delivered long term. Half-life may be prolonged in critically ill patients with hepatic or renal impairment. Risk of delirium. Also, it interacts with drugs used in the ICU (eg, some antiretrovirals, azole antifungals) that alter CYP metabolism such that excess sedation can occur with concomitant use of midazolam and drugs metabolized by CYP3A4.
- **Role**: A **good choice for short-term** anxiolysis and treatment of acute agitation. Dose adjustment and gradual titration are needed for patients with renal and/or hepatic impairment.

Sedative-analgesic medications

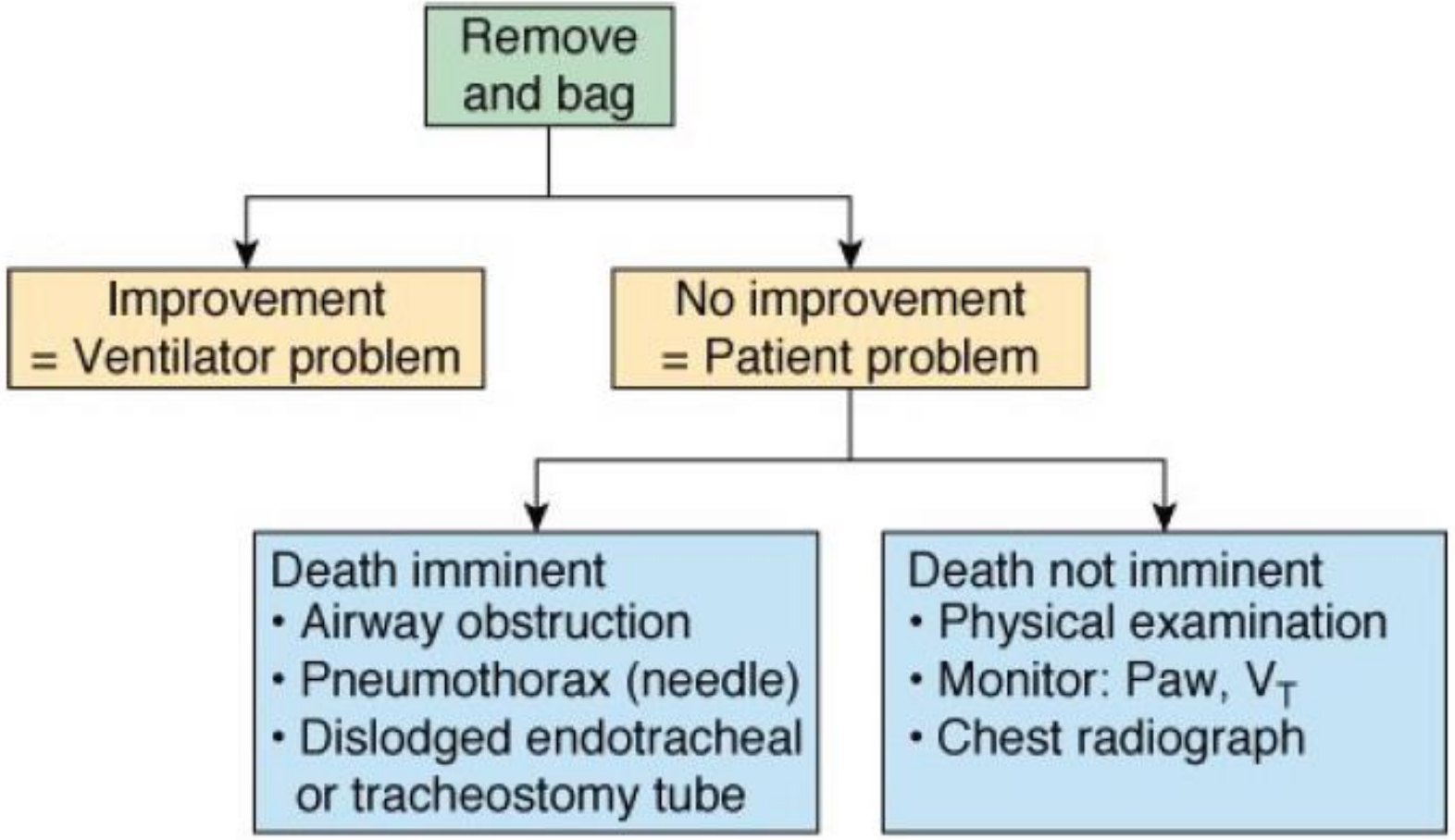
Quetiapine Oral: Initially 50 mg every 12 hours

- **Advantages:** Less risk of extrapyramidal symptoms and possibly less risk of QT prolongation than haloperidol.
- **Disadvantages:** Requires enteral route of administration and scheduled dosing due to slow onset of action and relatively gradual titration schedule. Adverse effects may include sedation or orthostatic hypotension, and risk of QT prolongation remains. Hepatically metabolized by CYP3A4 to active and inactive metabolites.
- **Role:** A potential choice as adjunct to as-needed IV haloperidol for treatment of agitation and/or delirium. In advanced hepatic impairment, initiate with reduced dose and titrate in lower increments.

Fights the Ventilator

- "Fighting the ventilator" is a phrase used to describe a ventilator-supported patient who **displays agitation and/or respiratory distress.**
- Such "fighting" is common at the time of intubation and initiation of mechanical ventilation, and is due largely to the anxiety that is to be expected under these circumstances.

Fights the Ventilator



Fights the Ventilator

- The major physical signs of respiratory distress include *tachypnea, diaphoresis*, nasal flaring, recruitment of the accessory muscles (scalenes and sternomastoids), recession of the suprasternal/supraclavicular/intercostal spaces, rib cage abdominal asynchrony and paradox, tracheal tug, *abnormal auscultatory findings, tachycardia, arrhythmias, and hypotension.*
- In addition to symptoms and signs of respiratory distress, the physician may utilize several cardiorespiratory variables that are usually monitored. Alterations in the pattern of breathing can be very helpful. A sudden increase in respiratory rate is an extremely sensitive sign of respiratory embarrassment, but further assessment is necessary to determine the precise cause of the disturbance.

Fights the Ventilator

- Patient related causes
  - Artificial airway problems
  - Movement of the endotracheal tube
  - Cuff herniation external
  - Compression of the endotracheal tube by the cuff
  - Cuff leak
  - Endotracheal tube kinking
  - Foreign body
  - Tracheoesophageal fistula
  - Innominate artery rupture
  - Malpositioning of the nasogastric tube
  - Buildup of secretions
  - Bronchospasm

Fights the Ventilator

- Patient related causes
  - Pneumothorax
  - Pulmonary edema
  - Pulmonary embolism
  - Acute hypoxemia
  - Blood in the endotracheal tube
  - Dynamic hyperinflation
  - Abnormal respiratory drive
  - Alteration in body posture
  - Drug induced
  - problems
  - Abdominal distension
  - Agitation

<Causes of Sudden Respiratory Distress in a Patient Receiving Mechanical Ventilation>

Fights the Ventilator

- Ventilator related causes
  - Ventilator malfunction
  - External ventilator circuit
  - Leaks or disconnects
  - Condensate
  - Inline nebulizers
  - Inadequate ventilator support
  - Patient–ventilator dyssynchrony

<Causes of Sudden Respiratory Distress in a Patient Receiving Mechanical Ventilation>

Fights the Ventilator

- Ventilator related problems
  - Airway malfunction
  - External circuit malfunction
  - Ventilator malfunction
  - Inappropriate ventilator settings

- Progression

  of an underlying disease process
  - Acute respiratory distress syndrome
  - Cardiogenic pulmonary edema
  - Pneumonia
  - Sepsis
  - Acute exacerbation of asthma or chronic obstructive pulmonary disease

<Causes of Worsening Oxygenation in the Ventilated Patient>

Fights the Ventilator

- Onset of a new problem
  - Pneumothorax
  - Atelectasis
  - Aspiration (gastric or oropharyngeal)
  - Ventilator associated
  - pneumonia
  - Sepsis
  - Pulmonary thromboembolism
  - Fluid overload
  - Bronchospasm
  - Retained secretions
  - Shock
  - Seizure

<Causes of Worsening Oxygenation in the Ventilated Patient>

Fights the Ventilator

- Effects of interventions and procedures
  - Endotracheal suctioning
  - Changes in body position
  - Chest physiotherapy
  - Bronchoscopy
  - Thoracentesis
  - Peritoneal dialysis
  - Hemodialysis
- Medications
  - Bronchodilators
  - Vasodilators
  - βblockers

<Causes of Worsening Oxygenation in the Ventilated Patient>

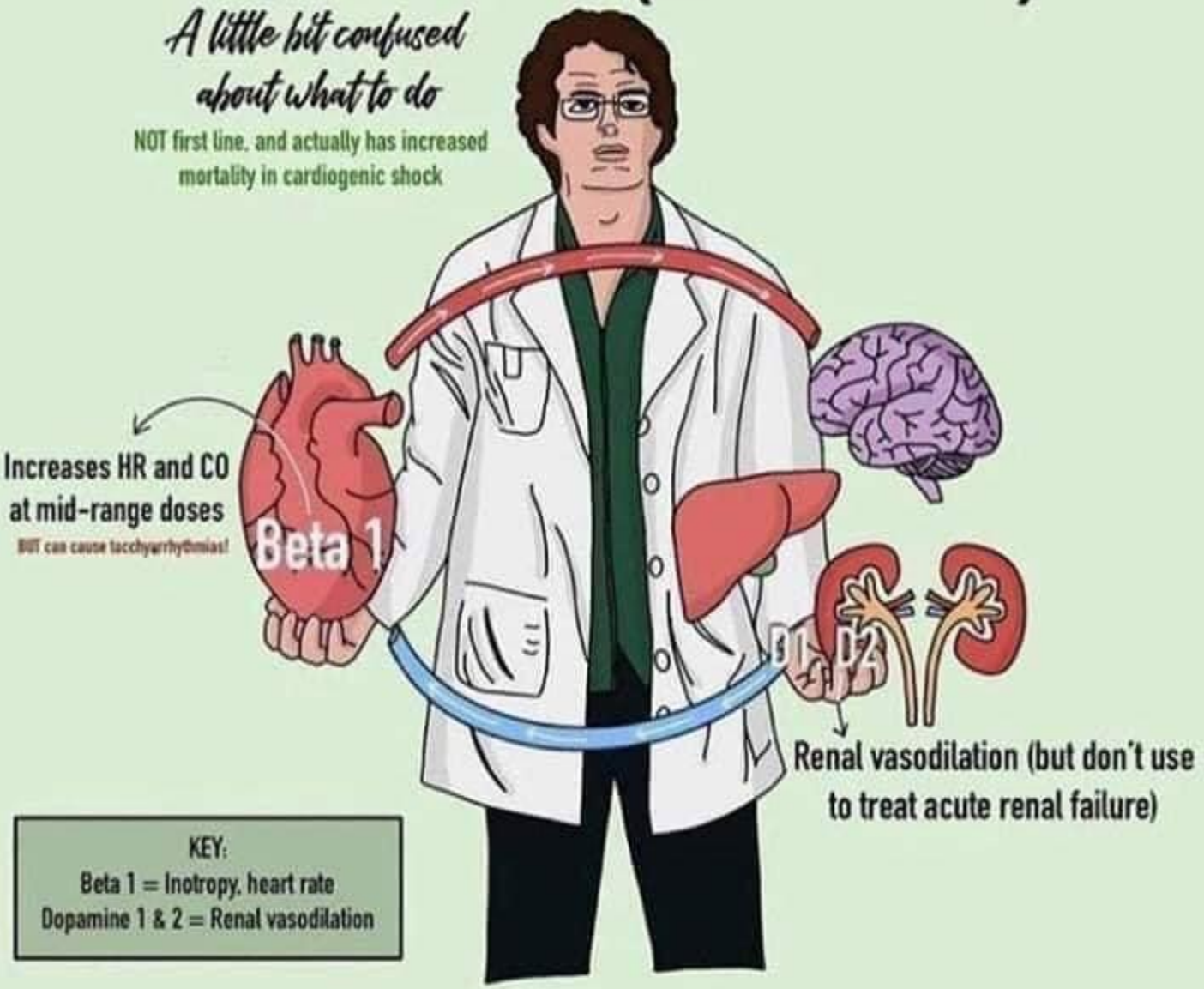
Fights the Ventilator

- Sudden distress in a ventilator supported patient is a medical emergency.
- The first rule is to ensure adequate ventilation.
- The patient should be disconnected from the ventilator and manually ventilated with 100% oxygen.
- While this is being performed, a systematic effort should be made to try to determine the cause of distress and correct it.
- If the distress is the result of poor coordination of a patient's respiratory efforts with the rhythm of the ventilator, this can usually be resolved by careful adjustment of the ventilator settings and the administration of analgesic or sedative agents, or both.

Dopamine (Low Dose)

A little bit confused about what to do

NOT first line, and actually has increased mortality in cardiogenic shock

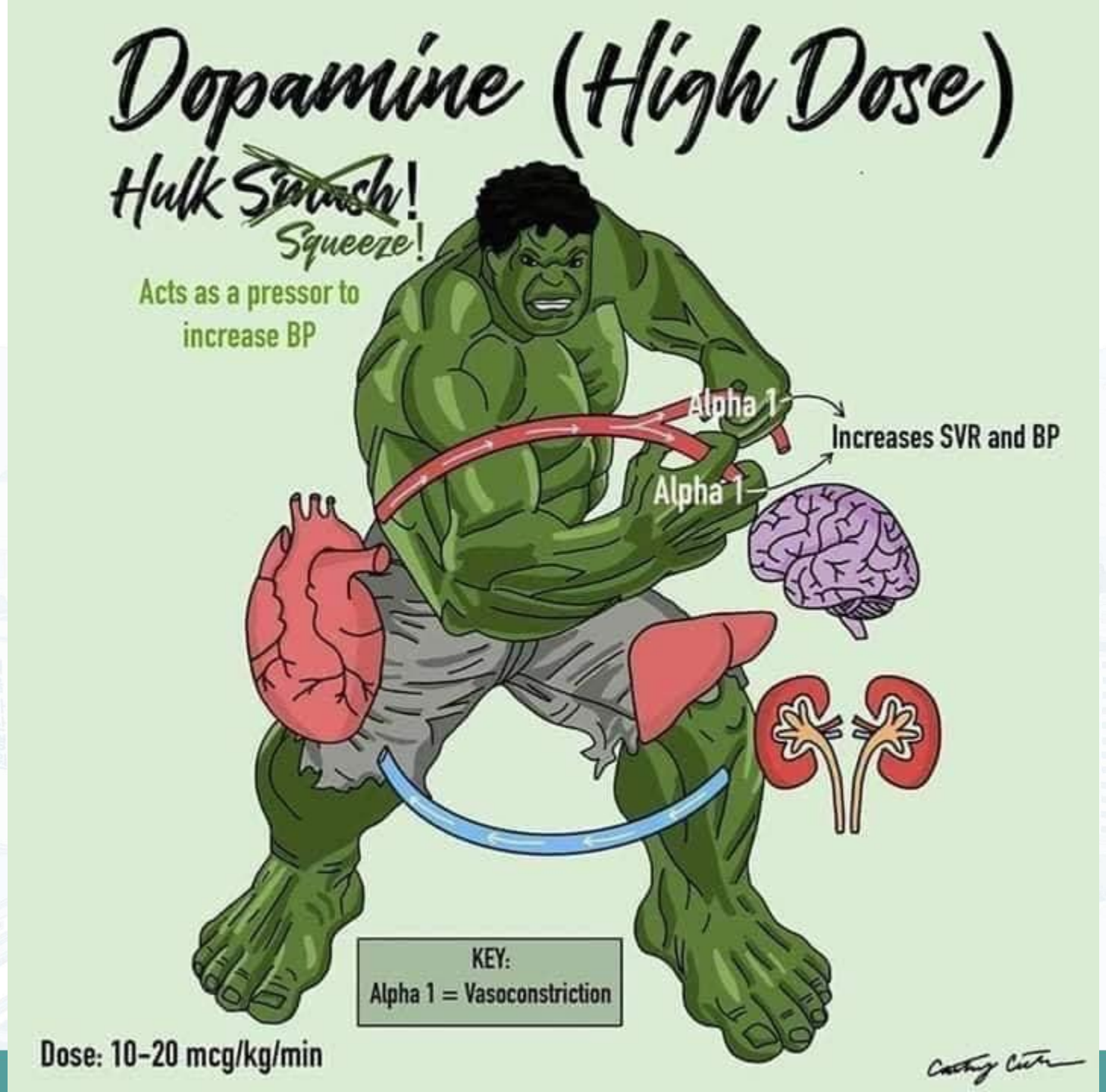


KEY:
Beta 1 = Inotropy, heart rate
Dopamine 1 & 2 = Renal vasodilation

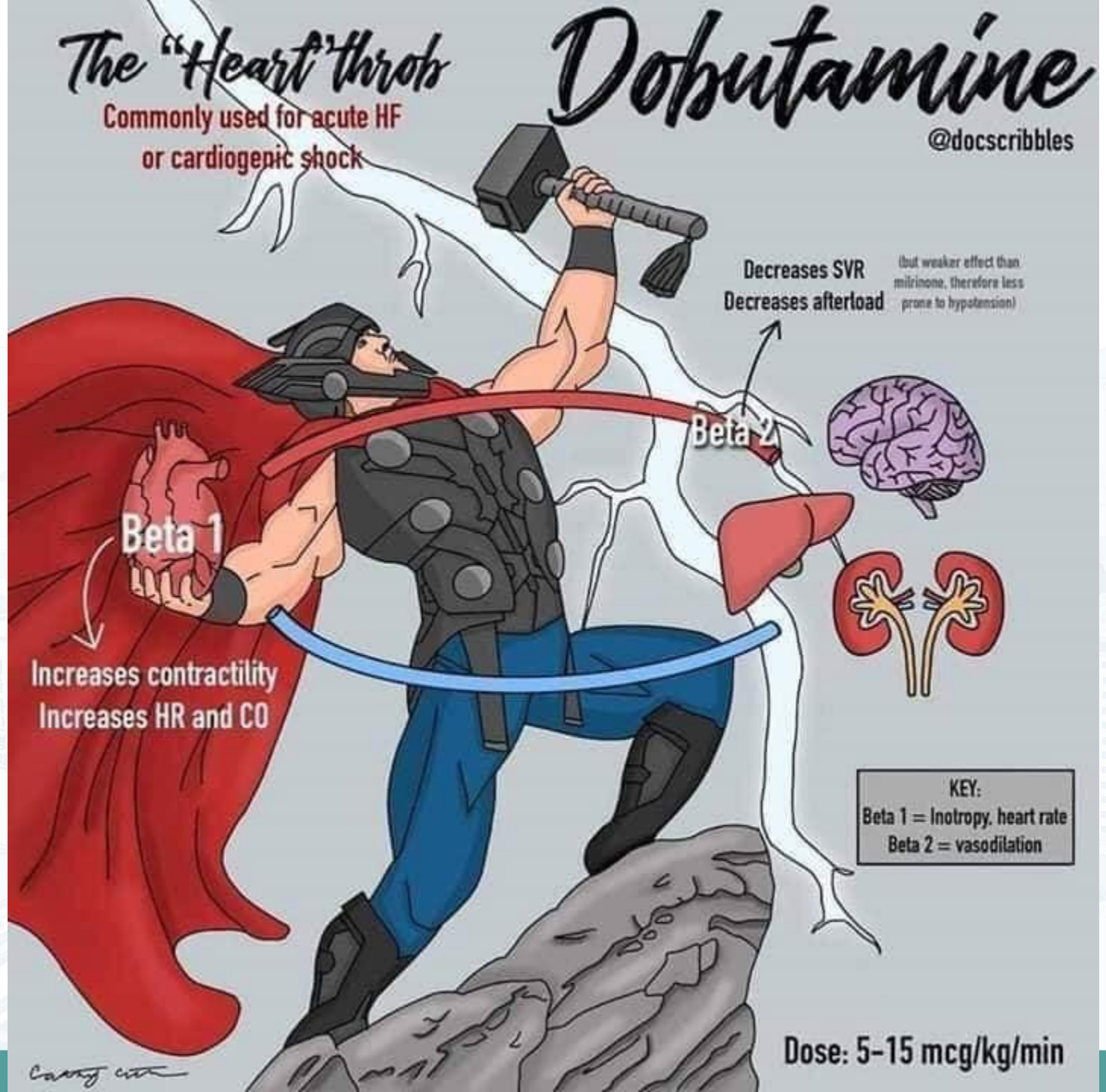
Dose: 0-5 mcg/kg/min

Cathy Cetin

Dopamine (High Dose)



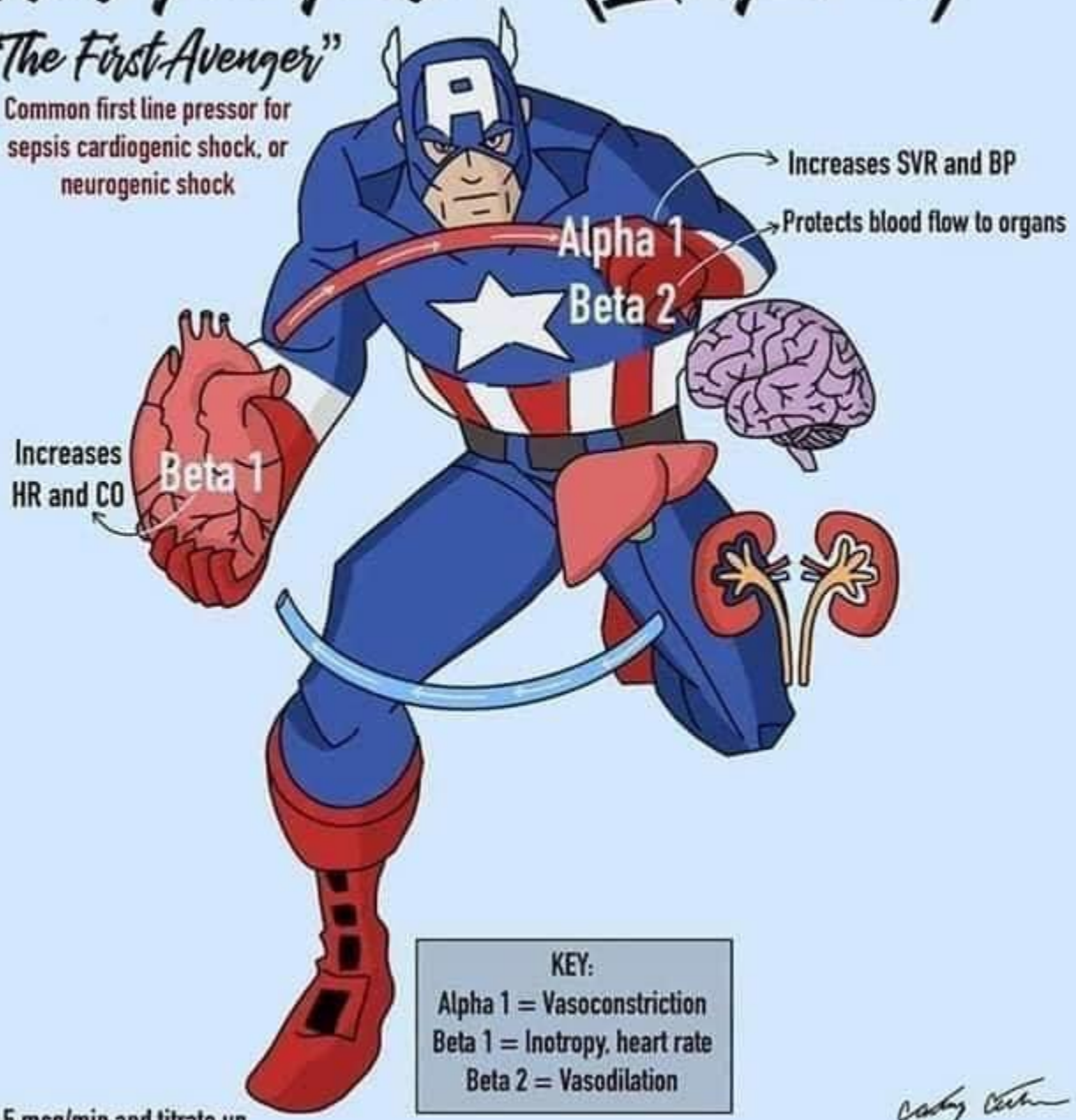
Dose: 10–20 mcg/kg/min


The "Heart"throb
Dobutamine
Commonly used for acute HF
or cardiogenic shock
@docscribbles
Decreases SVR
Decreases afterload
(but weaker effect than milrinone, therefore less prone to hypotension)
Beta 2
Beta 1
Increases contractility
Increases HR and CO
KEY:
Beta 1 = Inotropy, heart rate
Beta 2 = vasodilation
Dose: 5-15 mcg/kg/min

Norepinephrine (Levophed)

"The First Avenger"

Common first line pressor for sepsis cardiogenic shock, or neurogenic shock

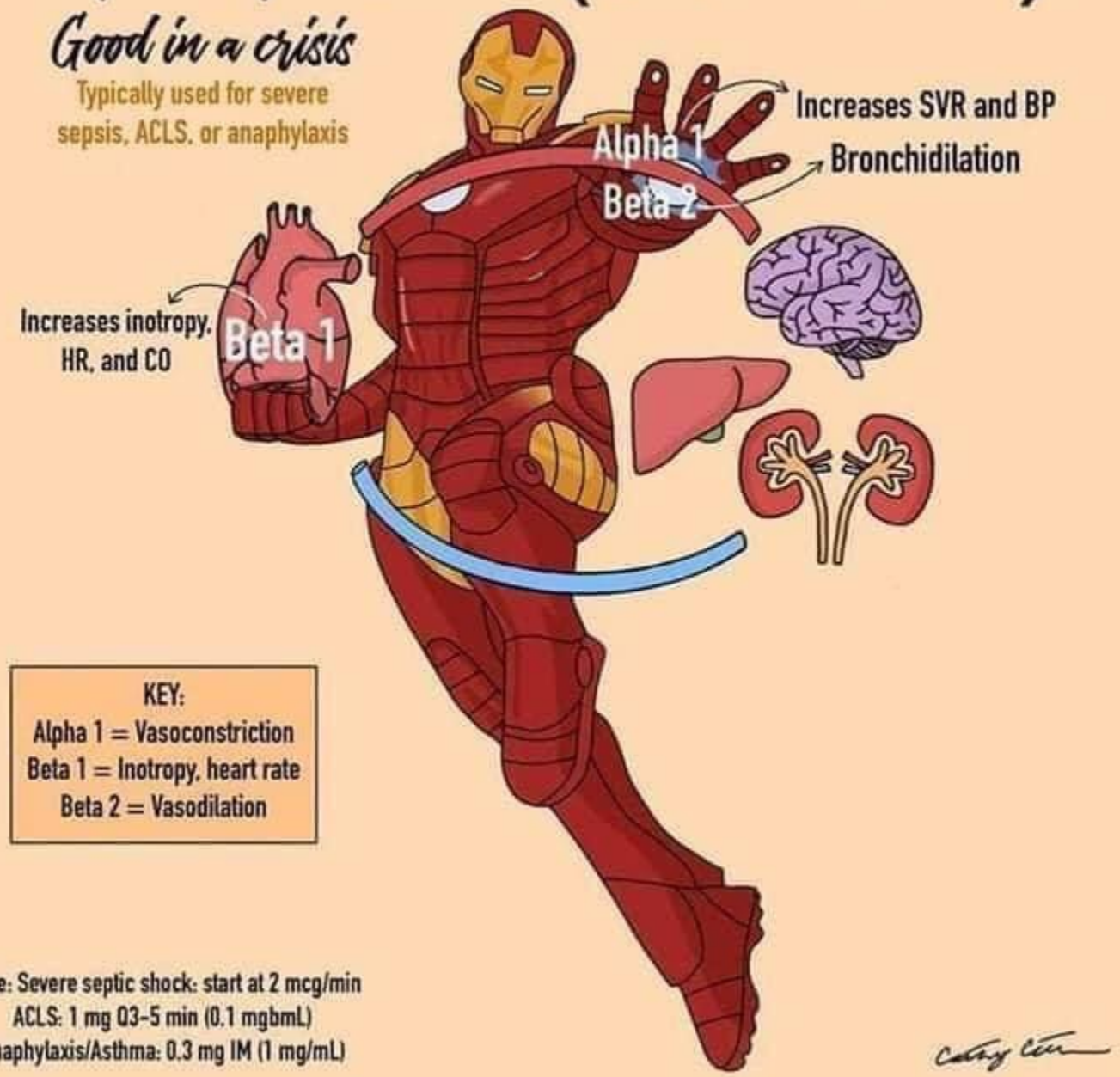


Dose: Start at 2-5 mcg/min and titrate up

Epinephrine (Adrenaline)

Good in a crisis

Typically used for severe sepsis, ACLS, or anaphylaxis



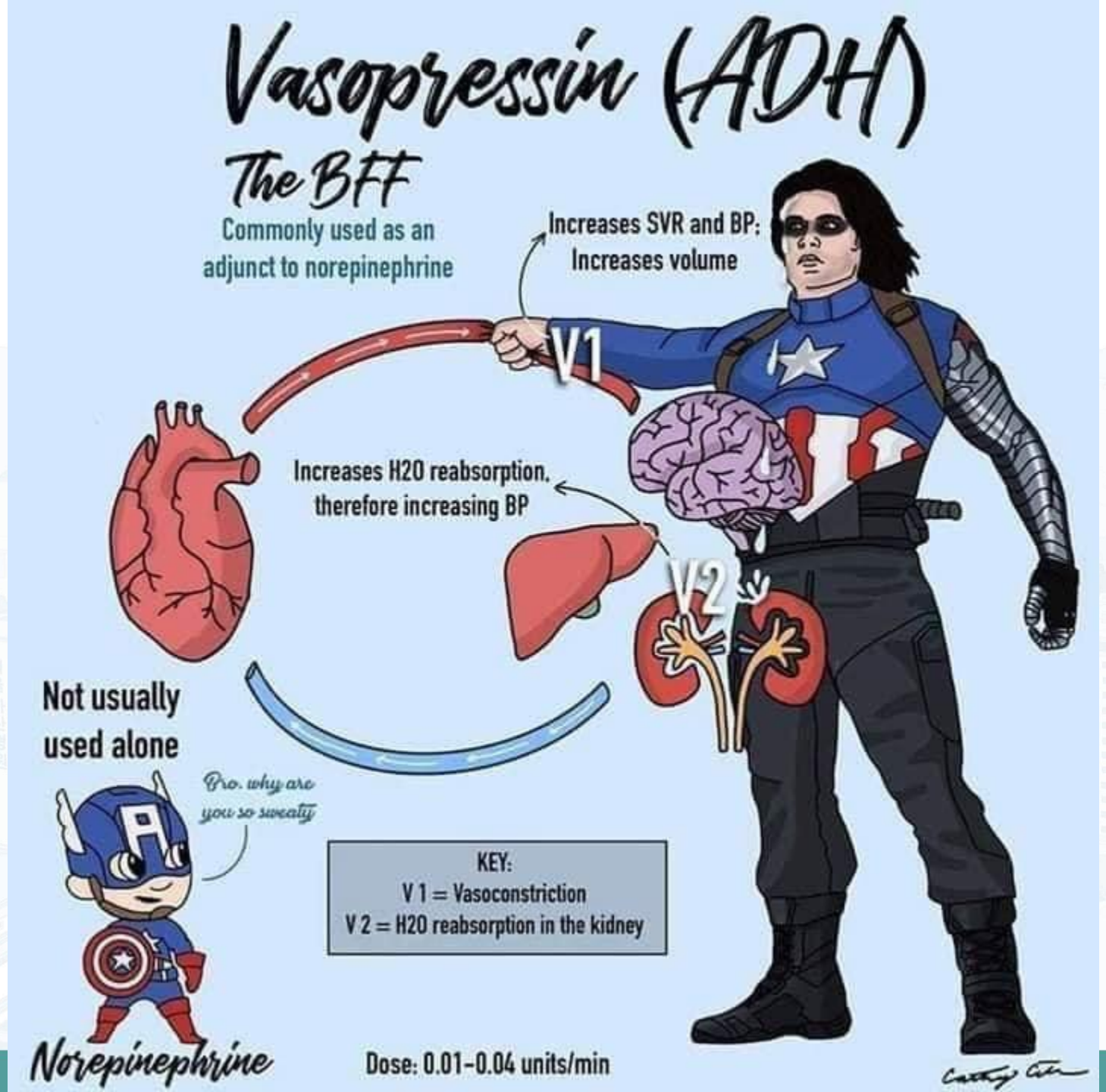
KEY:
Alpha 1 = Vasoconstriction
Beta 1 = Inotropy, heart rate
Beta 2 = Vasodilation

Dose: Severe septic shock: start at 2 mcg/min
ACLS: 1 mg Q3-5 min (0.1 mgbmL)
Anaphylaxis/Asthma: 0.3 mg IM (1 mg/mL)

Vasopressin (ADH)

The BFF

Commonly used as an adjunct to norepinephrine



Milrinone

Use with caution

Can cause hypotension and arrhythmias; Typically used for acute HF if there's BP room

KEY:

PDE3 in vessels: Vasoconstriction

Therefore blocking PDE3 leads to vasodilation.

PDE3 in heart: Breaks down cAMP. cAMP activates protein kinase A, which phosphoroyates calcium channels and allows for Ca2+ influx and increased contractility.

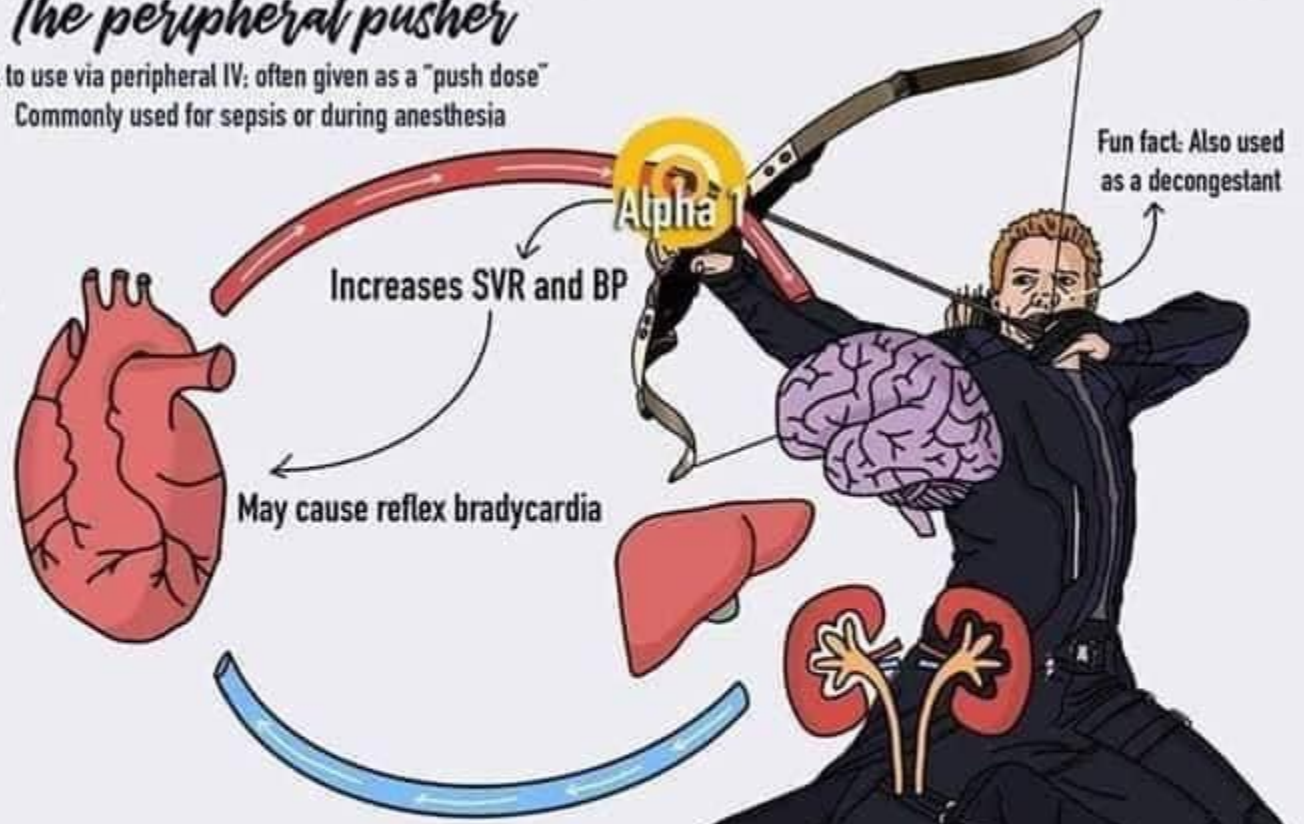
Therefore blocking PDE3 leads to increased inotropy.

Dose: 0.125-0.75 mcg/kg/min

Phenylephrine (NeoSynephrime)

The peripheral pusher

Ok to use via peripheral IV; often given as a "push dose"
Commonly used for sepsis or during anesthesia



Fun fact: Also used as a decongestant

KEY:
Alpha 1 = Vasoconstriction

Dose: 20–200 mcg/min

Conclusions

- It's always important to know the patient's condition.
- Proper combination drugs, not a lot of drugs

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Thank You for Listening