Shock

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Outline…

Introduction

Pathophysiology

Types of Shock

Resuscitation

Management principles

Introduction

A life-threatening condition of circulatory failure

It is a state of cellular tissue hypoxia caused by inadequate oxygen delivery that is unable to meet cellular metabolic needs and oxygen consumption requirements

The effects of shock are initially reversible, but can rapidly become irreversible, resulting in multiorgan failure (MOF) and death

When a patient presents with undifferentiated shock…

- It is important that the clinician **immediately initiate** therapy while rapidly identifying the etiology so that definitive therapy can be administered to reverse shock and prevent MOF and death

Blood Pressure is often used as an indirect estimator of tissue perfusion

It is crucial to recognize that a patient in shock can present hypertensive, normotensive, or hypotensive

Hypotension

- **In Adults:**
  - **systolic BP ≤ 90 mm Hg**
  - **mean arterial pressure ≤ 60 mm Hg**
  - **⇩ systolic BP > 40 mm Hg from the patient’s baseline pressure**

Oxygen delivery…

- is an interaction of Cardiac Output, Blood Volume, Systemic Vascular Resistance

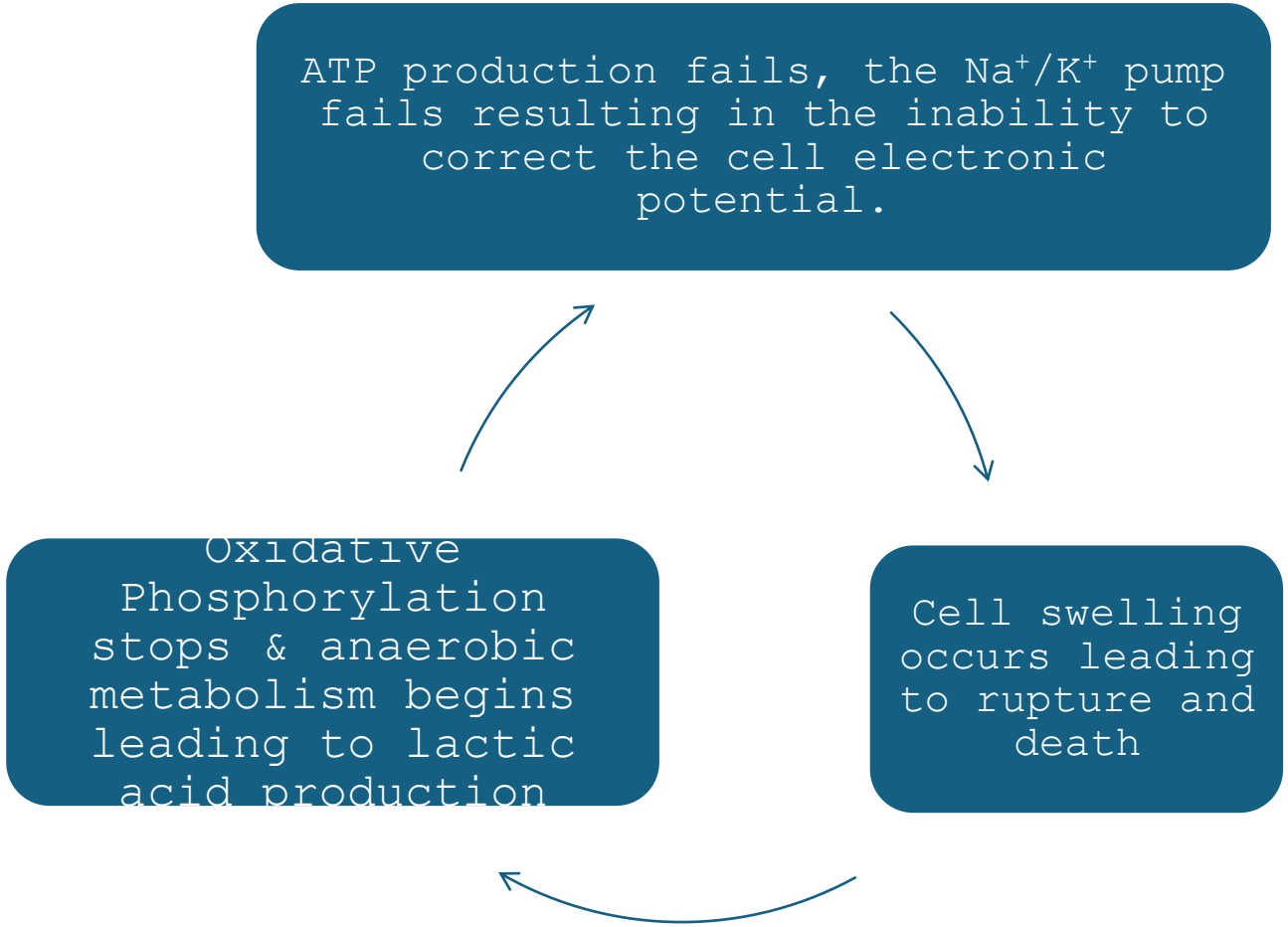
Pathophysiology

$ATP + H_2O \Rightarrow ADP + P_i + \underline{\mathbf{H^+}} + Energy$

Acidosis results from the accumulation of acid when during anaerobic metabolism the creation of ATP from ADP is slowed

H^+ shift extracellularly and a metabolic acidosis develops

Pathophysiology (2)



O_2 Delivery - volume of gaseous O_2 delivered to the LV/min

O_2 Consumption - volume of gaseous O_2 which is actually used by the tissue/min

O_2 Demand - volume of O_2 actually needed by the tissues to function in an aerobic manner

Demand > consumption = anaerobic metabolism

Mixed Venous Oxygenation

Used as a main marker of end organ perfusion and oxygen delivery

True mixed venous is drawn from the pulmonary artery (mixing of venous blood from upper and lower body)

Often sample will be drawn from central venous catheter (superior vena cava, R atrium)

Mixed Venous Oxygenation

Normal oxygen saturation of venous blood 68% – 77%

Low $S_{CV}O_2$

Tissues are extracting far more oxygen than usual, reflecting sub-optimal tissue perfusion (and oxygenation)

Following trends of $S_{CV}O_2$ to guide resuscitation (fluids, RBC, inotropes, vasopressors)

Physiologic Determinants

Global tissue perfusion is determined by:

- Cardiac output (CO)
  - CO = **Heart rate** (**HR**) times **Stroke Volume** (**SV**)
  - SV = function of **Preload, Afterload, Contractility**
- Systemic vascular resistance (SVR)
  - Variables: **Length,** Inverse of **Diameter, Viscosity**

Resuscitation Goals

CI = 4.5 L/min/m^2

Oxygen Delivery (DO_2I) = 600 mL/min/m^2

- < 400 is bad sign

Oxygen Consumption (VO_2I) = 170 mL/min/m^2

- If VO_2I < 100 suggest tissues are not getting enough oxygen

Maintain a mean arterial pressure of 60 (1/3 systolic + 2/3 diastolic)

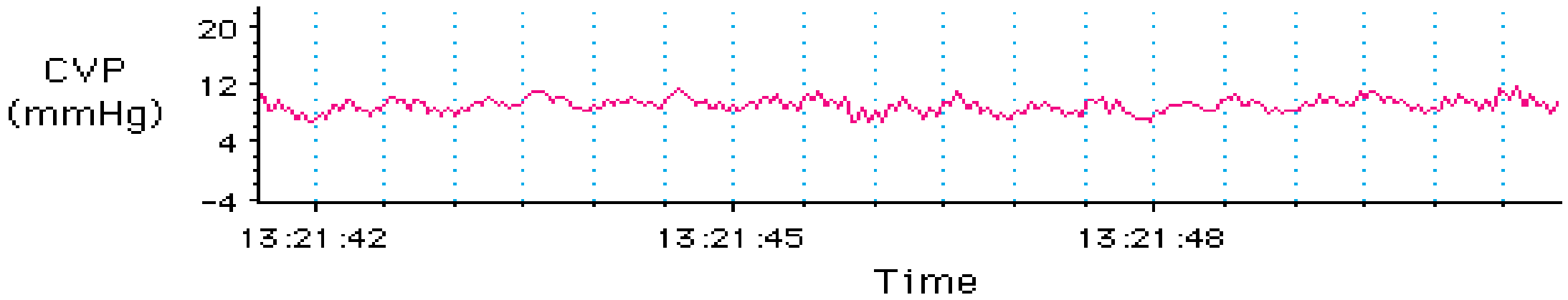
Keep O_2 sats >92%, intubate if neccesary

CVP

CVP of SVC at level of right atrium

pre-load “assessment”

normal 4 – 10 mm Hg



Pulmonary Artery Catheter

A 5 - lumen Swan Ganz catheter has either an **infusion port or a pacing port,** allowing insertion of a transvenous pacing wire; usually color coded white.

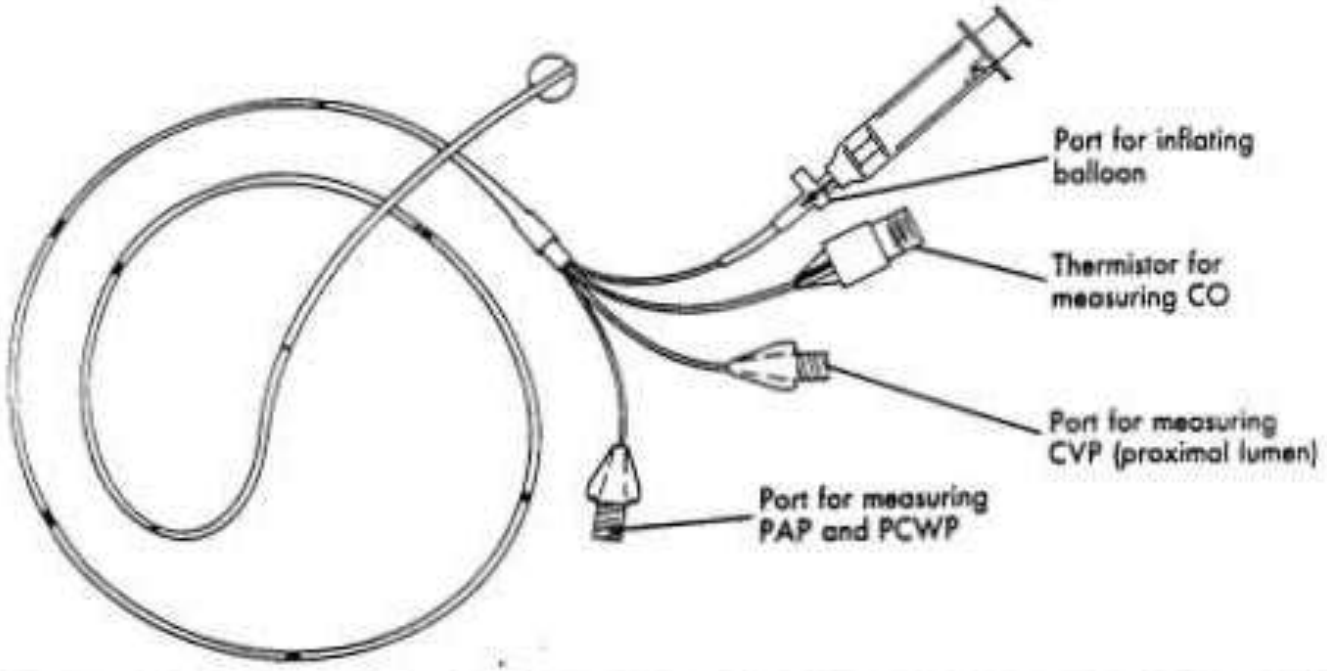
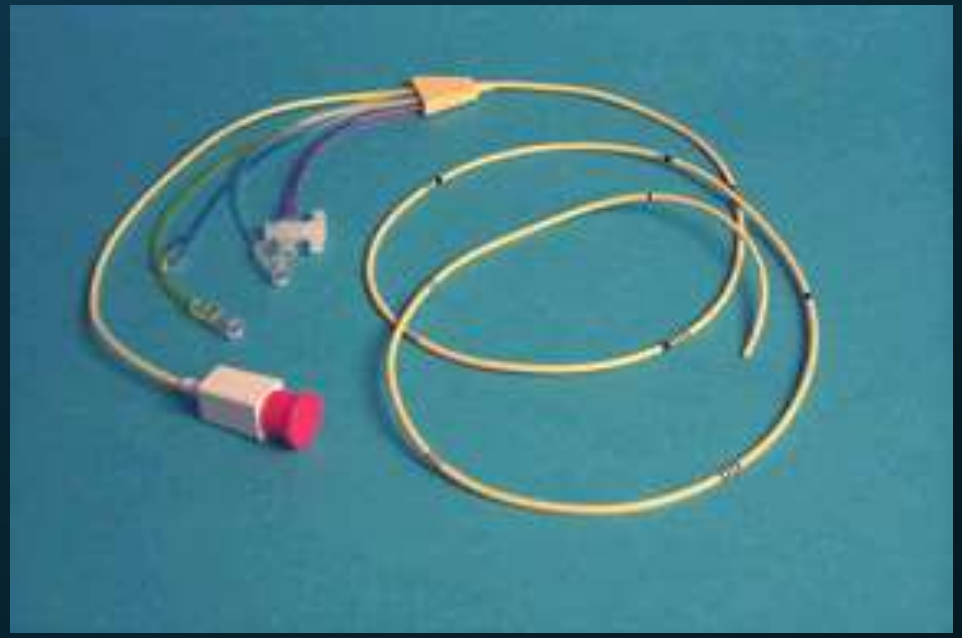
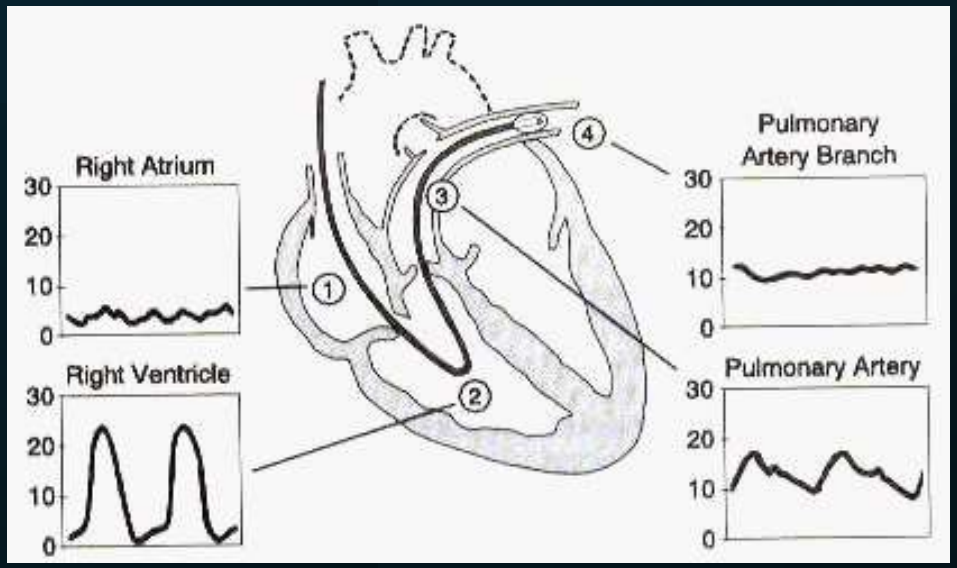


FIGURE 26-16 Four-lumen thermodilution pulmonary artery catheter for measuring cardiac output *(CO)*, central venous pressure *(CVP)*, pulmonary artery pressure *(PAP)*, and pulmonary capillary wedge pressure *(PCWP)*.




Right Atrium
30
20
10
0
Right Ventricle
30
20
10
0
1
2
3
4
Pulmonary Artery Branch
30
20
10
0
Pulmonary Artery
30
20
10
0

“Shock is a symptom of its cause.”

Types of Shock

- HYPOVOLEMIC
- CARDIOGENIC
- DISTRIBUTIVE
- OBSTRUCTIVE

Common Features of Shock

Hypotension (not an absolute requirement)

- SBP < 90mm Hg, not seen in "preshock"

Cool skin

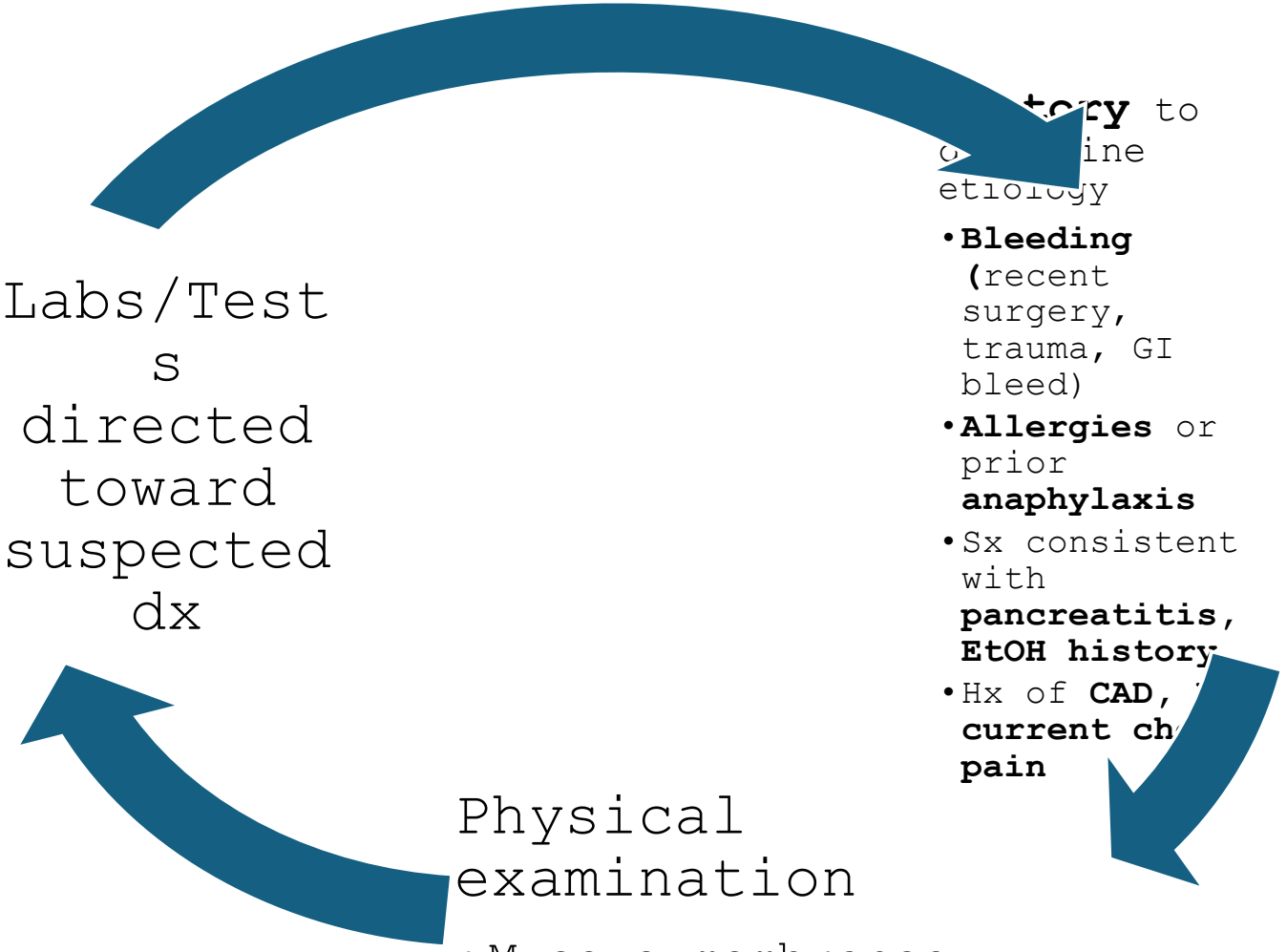
- Vasoconstrictive mechanisms to redirect blood from periphery to vital organs
- Exception is warm skin in early distributive shock

Oliguria (↓kidney perfusion)

Altered mental status (↓brain perfusion)

Metabolic acidosis

Work-up


tory to
ine
etiology
•**Bleeding** (recent surgery, trauma, GI bleed)
•**Allergies** or prior **anaphylaxis**
•Sx consistent with **pancreatitis, EtOH history**
•Hx of **CAD,**
current ch
pain
Physical examination
•Mucous membranes, JVD, lung sounds, cardiac exam, abdomen, rectal (blood), neuro exam,
Labs/Tests directed toward suspected dx

Hypovolemic Shock

Reduced circulating blood volume with secondary decreased cardiac output

- ***Causes***
  - hemorrhage
  - vomiting
  - diarrhea
  - dehydration
  - third-space loss
  - burns
- ***Signs***
  - ⇩ cardiac output
  - ⇩ PAOP
  - ⇧ SVR

Presentation of Hypovolemic Shock

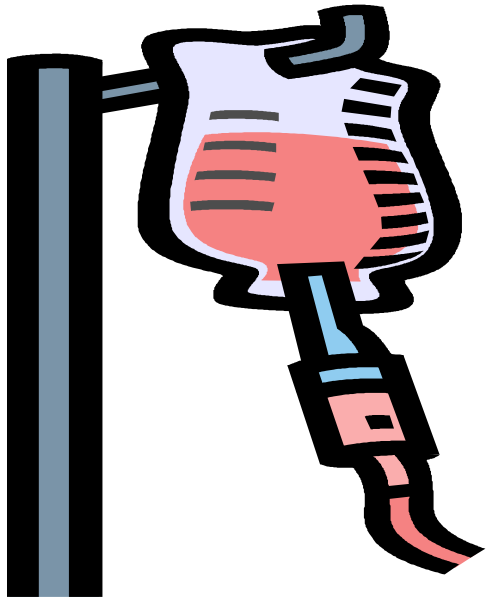
- Hypotensive
- flat neck veins
- clear lungs
- cool, cyanotic extremities
- evidence of bleeding?
  - Anticoagulant use
  - trauma, bruising
- oliguria

Classes of Hypovolemic Shock

	Class I	Class II	Class III	Class IV
Blood Loss	< 750	750-1500	1500-2000	> 2000
% Blood Vol.	< 15%	15 – 30%	30 – 40%	> 40%
Pulse	< 100	> 100	> 120	> 140
Blood Pressure	Normal	Normal	Decreased	Decreased
Pulse Pressure	Normal	Decreased	Decreased	Decreased
Resp. Rate	14 – 20	20 – 30	30 – 40	> 40
UOP	> 30	20 – 30	5 – 15	negligible
Mental Status	sl. Anxious	mildly anx	confused	lethargic
Fluid	crystalloid	crystalloid	blood	blood

SHOCK/HYPOVOLEMIA

- FLUIDS… FLUIDS… FLUIDS…
- BLOOD & PRODUCTS TRANSFUSION
- CORRECT
  - ACIDOSIS
  - COAGULOPATHY
  - HYPOTHERMIA



Initial Resuscitation

- CVP: 8- 12 mm Hg
- MAP ≥ 65 mm Hg
- UOP ≥ 0.5 cc/kg/hr
- Mixed venous Oxygen Sat ≥ 70%
- Consider:
  - Transfusion to Hb ≥ 10
  - Dobutamine up to 20 µg/kg/min

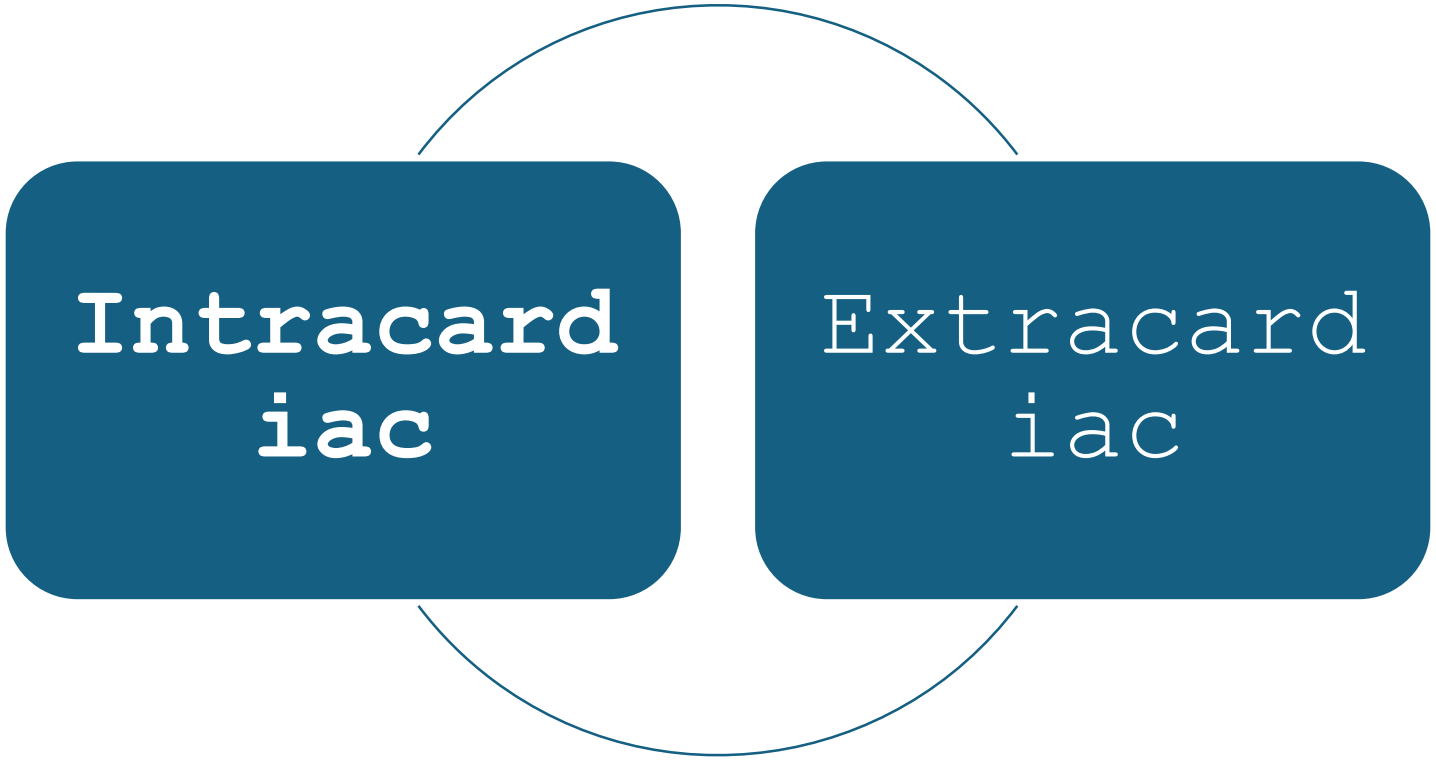
Treatment

Reverse hypovolemia vs. hemorrhage control

Crystalloid vs. Colloid

Pressors?

Cardiogenic Shock



Intracardiac

- Myocardial Injury or Obstruction to Flow
  - Arrythymias
  - valvular lesions
  - AMI
  - Severe CHF
  - VSD
  - Hypertrophic Cardiomyopathy

- ***Signs***
  - **⇩ cardiac output**
  - **⇧ PAOP**
  - **⇧ SVR**
  - **⇩ left ventricular stroke work (LVSW)**

Extracardiac (Obstructive)

- Pulmonary Embolism
- Cardiac Tamponade
- Tension Pneumothorax
- Presentation will be according to underlying disease process.

Treatment

Improve myocardial function, C.I. < 3.5 is a risk factor

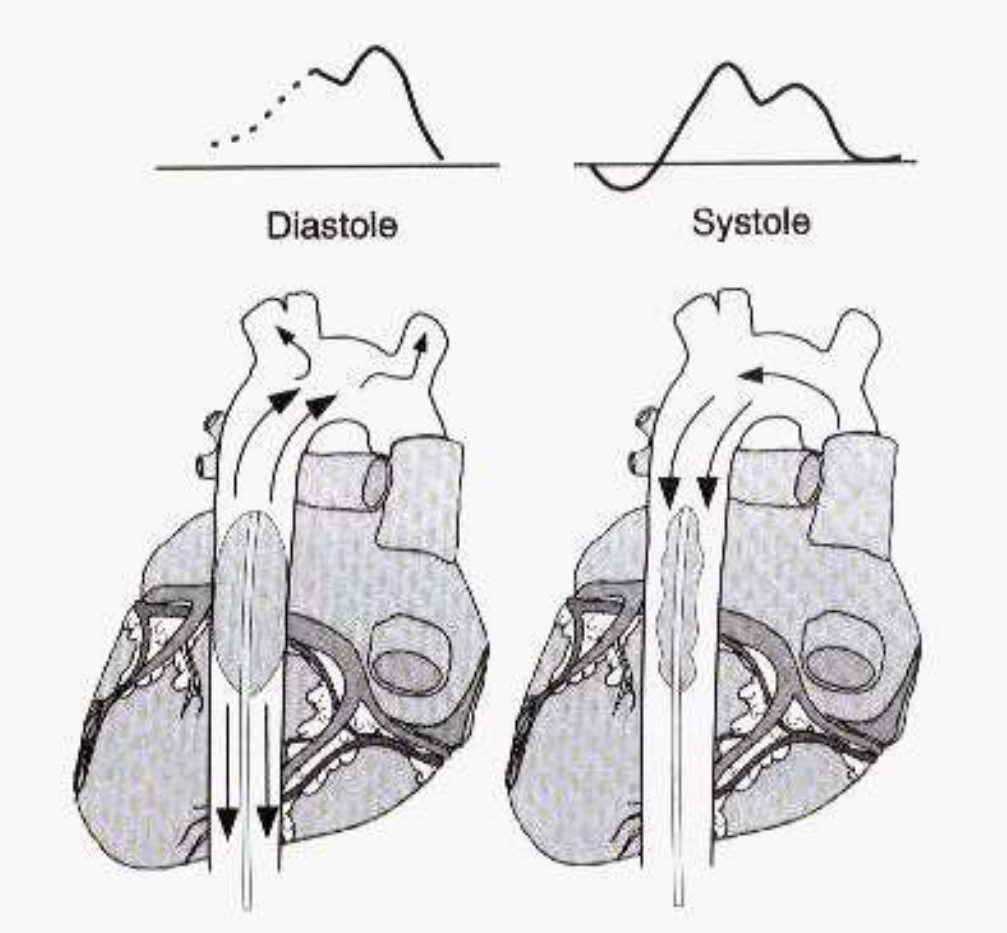
Catheterization if ongoing ischemia

Fluids first guided by CVP & PAOP, then cautious pressors

Aortic DIASTOLIC pressures drives coronary perfusion (DBP-PAOP = Coronary Perfusion Pressure)
GOAL - Coronary PP > 50 mm Hg

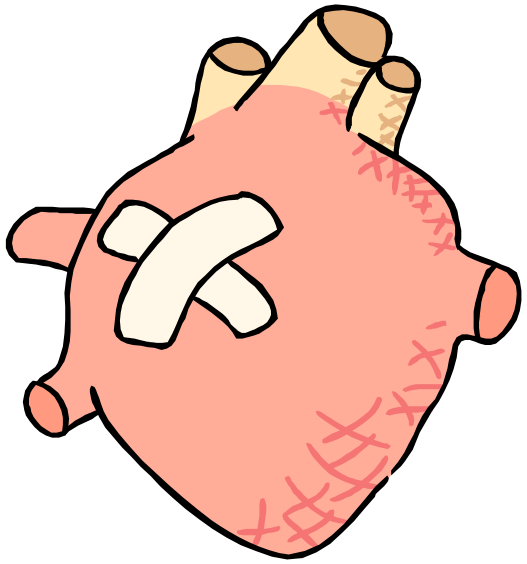
If inotropes and vasopressors fail, intra-aortic balloon pump

Intra-Aortic Balloon Pump



Cardiogenic Shock

- **Preload augmentation** - Consider Fluids
- **Contractility**
  - Dopamine
  - Dobutamine
  - Phosphodiesterase inhibitor
- **Afterload reduction**
  - Nitroglycerin
  - Dobutamine



Distributive Shock

Types

Sepsis
Anaphylactic
Acute adrenal insufficiency
Neurogenic

Signs

± cardiac output
± PAOP
⇩SVR

Old definition of sepsis..

Infection + 2 or more SIRS criteria

Box 1. SIRS (Systemic Inflammatory Response Syndrome)

Two or more of:

- Temperature >38°C or <36°C
- Heart rate >90/min
- Respiratory rate >20/min or $Paco_2$ <32 mm Hg (4.3 kPa)
- White blood cell count >12 000/mm^3 or <4000/mm^3 or >10% immature bands

Terms and Definitions

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection.

Organ dysfunction can be identified as an acute change in total SOFA score ≥2 points consequent to the infection.

Septic shock is a subset of sepsis in which underlying circulatory and cellular/metabolic abnormalities are profound enough to substantially increase mortality.

Patients with septic shock can be identified with a clinical construct of sepsis with persisting hypotension requiring vasopressors to maintain MAP ≥65 mm Hg and having a serum lactate level >2 mmol/L (18 mg/dL) despite adequate volume resuscitation. With these criteria, hospital mortality is in excess of 40%

qSOFA (Quick SOFA) Criteria

- Respiratory rate ≥22/min
- Altered mentation
- Systolic blood pressure ≤100 mm Hg

Table 1. Sequential [Sepsis-Related] Organ Failure Assessment Score[a]

System	Score 0	1	2	3	4
Respiration					
Pao_2/Fio_2, mm Hg (kPa)	≥400 (53.3)	<400 (53.3)	<300 (40)	<200 (26.7) with respiratory support	<100 (13.3) with respiratory support
Coagulation					
Platelets, $\times 10^3/\mu L$	≥150	<150	<100	<50	<20
Liver					
Bilirubin, mg/dL (μmol/L)	<1.2 (20)	1.2-1.9 (20-32)	2.0-5.9 (33-101)	6.0-11.9 (102-204)	>12.0 (204)
Cardiovascular	MAP ≥70 mm Hg	MAP <70 mm Hg	Dopamine <5 or dobutamine (any dose)[b]	Dopamine 5.1-15 or epinephrine ≤0.1 or norepinephrine ≤0.1[b]	Dopamine >15 or epinephrine >0.1 or norepinephrine >0.1[b]
Central nervous system					
Glasgow Coma Scale score[c]	15	13-14	10-12	6-9	<6
Renal					
Creatinine, mg/dL (μmol/L)	<1.2 (110)	1.2-1.9 (110-170)	2.0-3.4 (171-299)	3.5-4.9 (300-440)	>5.0 (440)
Urine output, mL/d				<500	<200

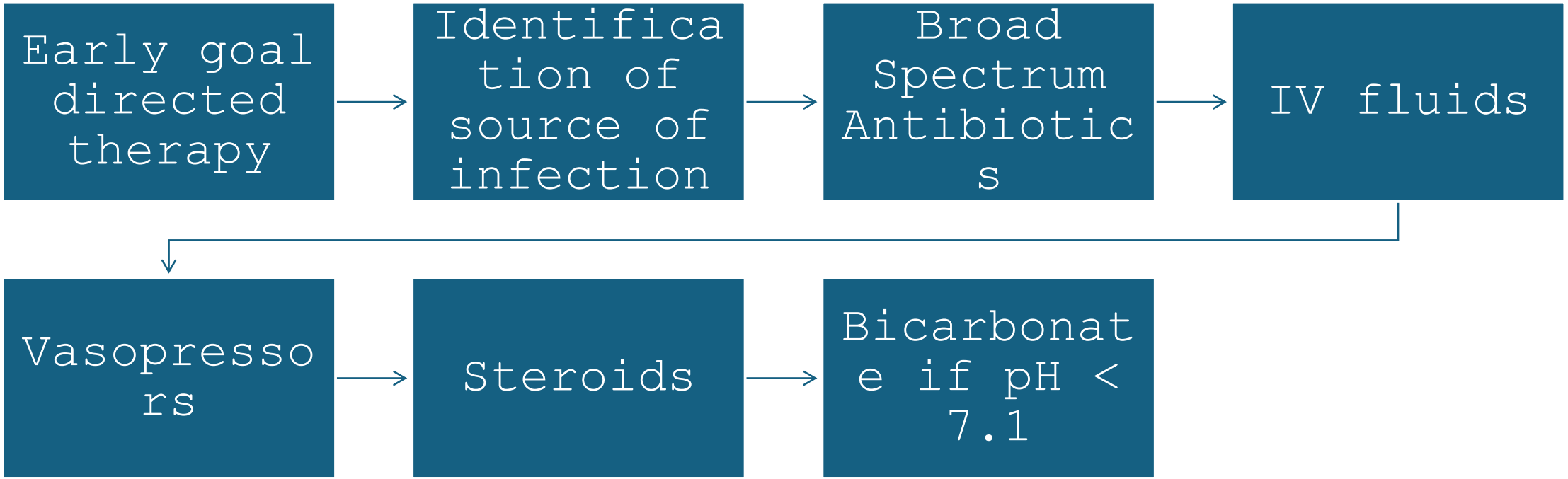
Abbreviations: Fio_2, fraction of inspired oxygen; MAP, mean arterial pressure; Pao_2, partial pressure of oxygen.

[a] Adapted from Vincent et al.[27]

[b] Catecholamine doses are given as μg/kg/min for at least 1 hour.

[c] Glasgow Coma Scale scores range from 3-15; higher score indicates better neurological function.

Management of Septic Shock



Steroid Use in Sepsis

Refractory shock 200-300 mg/day of hydrocortisone in divided doses for 7 days

ACTH test

Once septic shock resolves, taper dose

Add fludrocortisone 50 µg po q day

Sepsis…

- **Fluids**
- **Correct the cause**
  - Antibiotics
  - Debridement
- **Vasopressors**
  - Phenylephrine
  - Levophed

Adrenal Crisis
Distributive Shock

Causes

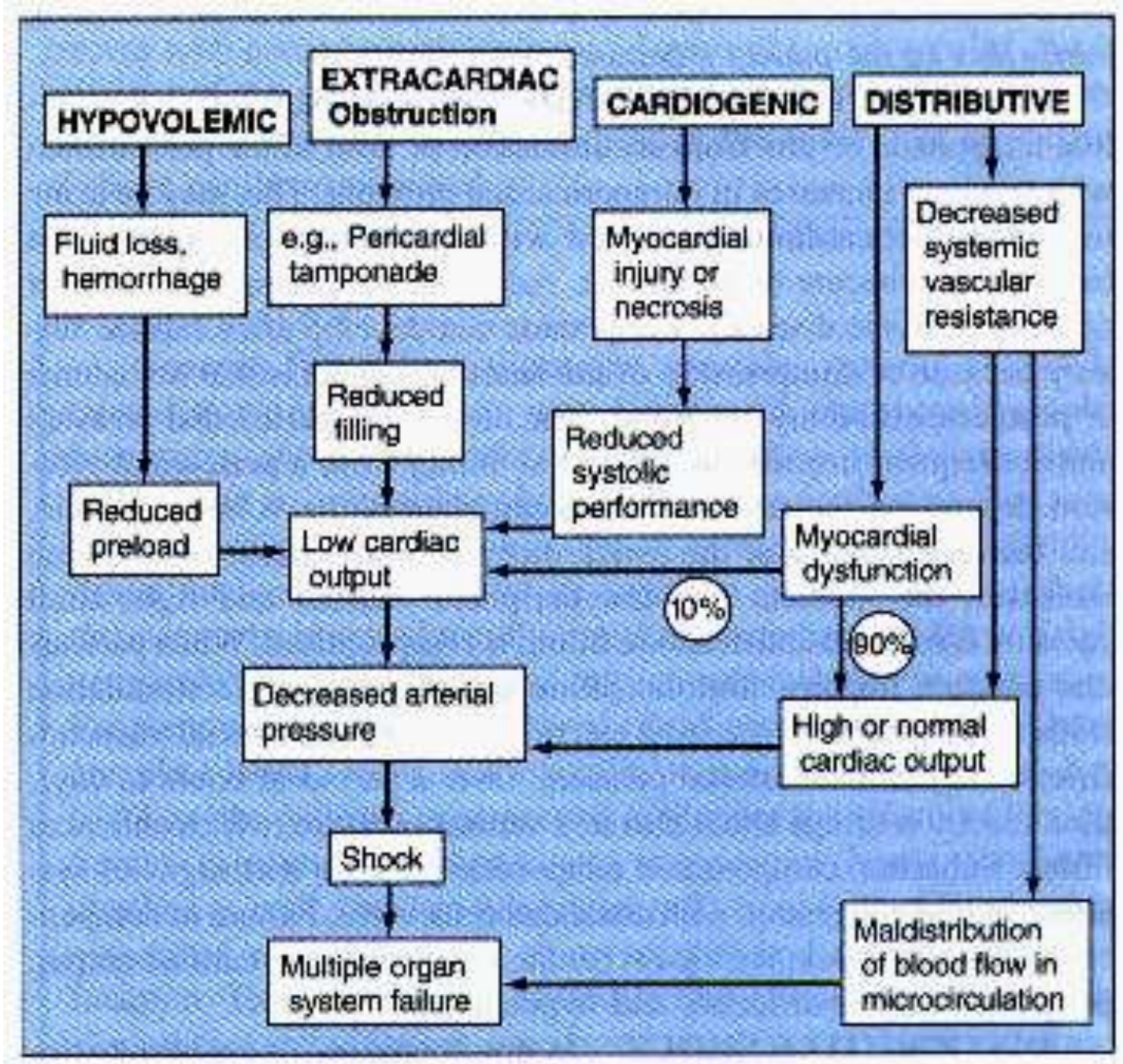
Autoimmune adrenalitis

Adrenal apoplexy = B hemorrhage or infarct

Steroids may be lifesaving in the patient who is unresponsive to fluids, inotropic, and vasopressor support.

Summary

Type	PAOP	C.O.	SVR
HYPOVOLEMIC	↓	↓	↑
CARDIOGENIC	↑	↓	↑
DISTRIBUTIVE	↓ or N **varies**	↓	
OBSTRUCTIVE	↑	↓	↑


HYPOVOLEMIC
EXTRACARDIAC Obstruction
CARDIOGENIC
DISTRIBUTIVE
Fluid loss, hemorrhage
e.g., Pericardial tamponade
Myocardial injury or necrosis
Decreased systemic vascular resistance
Reduced filling
Reduced systolic performance
Reduced preload
Low cardiac output
Myocardial dysfunction
10%
90%
Decreased arterial pressure
High or normal cardiac output
Shock
Multiple organ system failure
Maldistribution of blood flow in microcirculation

Vasopressors

- Assure adequate fluid volume
- Administer via CVL
- Do not use dopamine for renal protection
- Requires arterial line placement
- Vasopressin:
  - Refractory shock
  - Infusion rate 0.01 – 0.04 Units/min

Vasopressor Agents?

Augments contractility, after preload established, thus improving cardiac output.

Risk tachycardia and increased myocardial oxygen consumption if used too soon

increased C.I. improves global perfusion

Vasopressors & Inotropic Agents

- Dopamine
- Dobutamine
- Norepinephrine
- Epinephrine
- Amrinone

Dopamine

Low dose (0.5 - 2 µg/kg/min) = dopaminergic

Moderate dose (3-10 µg/kg/min) = β-effects

High dose (> 10 µg/kg/min) = α-effects

SIDE EFFECTS

tachycardia

> 20 µg/kg/min Δ to norepinephrine

Dobutamine

β-agonist

5 - 20 μg/kg/min

potent inotrope, variable chronotrope

caution in hypotension (inadequate volume) may precipitate tachycardia or worsen hypotension

Norepinephrine

Potent α-adrenergic vasopressor

Some β-adrenergic, inotropic, chronotropic

Dose 1 - 100 μg/min

Unproven effect with low-dose dopamine to protect renal and mesenteric flow.

Epinephrine

α- and β-adrenergic effects

potent inotrope and chronotrope

dose 1 - 10 μg/min

increases myocardial oxygen consumption particularly in coronary heart disease

Amrinone

Phosphodiesterase inhibitor, positive inotropic and vasodilatory effects

increased cardiac stroke output without an increase in cardiac stroke work

most often added with dobutamine as a second agent

load dose = 0.75 -1.5 mg/kg → 5 - 10 µg/kg/min drip

main side-effect - thrombocytopenia

Thank you