

Basics of Shock

Supplementary Educational Material

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Outline

0030 Objectives

- A. **0045 Shock**
 - a. When supply does not meet demand
 - i. The metabolic supply to tissues, and thereby organ systems, does not meet the demand
 - ii. **0115** It is not defined by a blood pressure
 - iii. **0152** Inadequate perfusion relative to metabolic demands-> tissue hypoxia (anaerobic metabolism) -> cell death
 - 1. **0217** Anaerobic metabolism: Less efficient energy production, lactate production
- B. **0250 Lactate Production**
 - a. **0302**- Glycolysis
 - b. **0310**- Pyruvate
 - i. Krebs cycle
 - ii. **0335** Lactate production in normal circumstances is due to inadequate oxygen supply
 - iii. **0410** Lactate production in shock is due to adrenergic stimulation of the B2 receptors by epinephrine leading to increased glycolysis
 - iv. **0510** Lactate production as energy source
 - 1. **0540** Normally always have low levels of lactate production that is cleared by the kidneys and the liver
 - c. **0545** Causes of Hyperlactemia
- C. **0638 Categories of Shock**
 - a. **0710** Cold Shock
 - i. Decreased / inadequate stroke volume:
 - 1. decreased preload, contractility
 - 2. increased afterload
 - ii. **0807**- narrow pulse pressure due to compensatory vasoconstriction and increase in systemic vascular resistance
 - b. **0742** Warm Shock
 - i. Decreased systemic vascular resistance
 - ii. Movement of blood flow from vital organs to non-vital organs (e.g skin and muscles)
 - iii. **0822**- wide pulse pressure due to the low systemic vascular resistance
- D. **0829 Stroke Volume**
 - a. Definition: the amount of blood pumped out of the heart with each heartbeat
 - i. **0844** Preload: the volume of blood present in the ventricle at the end of diastole
 - 1. Causes for decrease in preload – hypovolemia, hemorrhage or vasodilation
 - ii. **0905** Contractility: the strength of contraction
 - 1. Causes for decrease contractility- ischemia, toxins, myocarditis, congenital heart disease
 - iii. **0927** Afterload: resistance against the ventricular contraction
 - 1. Causes for increase afterload- hypertensive emergency, increased vasoconstriction
- E. **0947 Defining Blood Pressures**
 - a. **0954**- Systolic Blood pressure
 - i. Determined by the blood volume in the arteries + aortic compliance
 - ii. Systolic blood pressure ~ stroke volume
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 - i. As the ventricle is relaxing, tissue perfusion determined by systemic vascular resistance
 - ii. Diastolic blood pressure ~ systemic vascular resistance

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- iii. **1033** Chronically low diastolic blood pressure, therefore an increased pulse pressure, could be associated with aortic regurgitation
- F. **1150** Compensation for Shock
 - a. Decreased stroke volume->. Low cardiac output state and decreased tissue perfusion
 - i. **1223** 1- Increase heart rate
 - ii. **1234** 2- Increase systemic vascular resistance (if heart rate is inadequate)
 - b. **1310** Determinates of vascular tone
 - i. **1315** Adrenal glands: catecholamines that cause vasoconstriction
 - ii. **1328** Kidneys: (+) RAAS -> angiotensin II and aldosterone release
- G. **1424** Categories of Shock
 - a. **1448**- Cold Shock: decrease stroke volume (i.e. decreased SBP) + increased systemic vascular resistance (i.e. increased DBP)=> narrow pulse pressure
 - i. **1519** Cardiogenic shock: trouble with cardiac contractility
 - 1. Examples: ischemia, congenital heart disease, toxins
 - 2. Note: increasing the systemic vascular resistance will increase the afterload, thereby making it worse by further decreasing the stroke volume
 - ii. **1615** Hypovolemic shock: trouble with preload
 - 1. Examples: hemorrhage, gastrointestinal losses, **1708** venodilation (most of the blood volume is in the venous vasculature) leading to relative hypovolemia
 - iii. **1850** Obstructive shock (SICK): decreased pre-load due to "obstruction" of venous return
 - 1. Examples: pulmonary embolism, pneumothorax, tamponade
 - H. **2125** Warm Shock: Distributive shock (meaning the problem is the "distribution" of blood flow)= decrease stroke volume (i.e. decreased SBP) + decreased systemic vascular resistance (i.e. decreased DBP)=> widened pulse pressure
 - a. **2220** Venodilation: increase in venous capacitance leading to blood pooling in the venous system
 - b. **2234** Increased capillary permeability- leading to loss of plasma volume into the interstitial space (so has a component of hypovolemic shock)
 - c. **2240** Increased heart rate + increased contractility(compensation)
 - i. Sepsis, adrenal insufficiency, liver failure, anaphylaxis
 - ii. **2357** Sepsis- myocardial dysfunction due to cytokine release
 - d. **2602** Decreased/ normal heart rate
 - i. Neurogenic shock (unopposed vagal tone)

Learning objectives 0030



BE ABLE TO DEFINE
SHOCK



BE ABLE TO DISCUSS
WHY LACTATE MAY BE
ELEVATED IN SHOCK



LIST THE MAIN
CATEGORIES OF
SHOCK

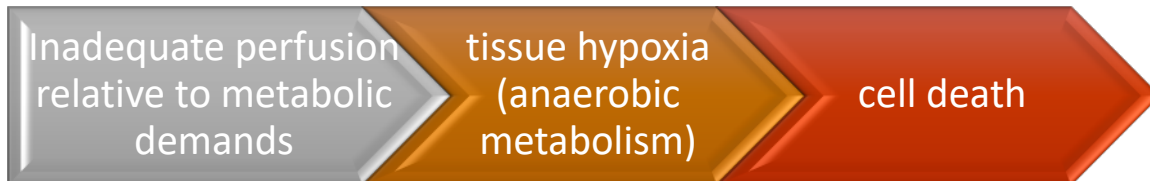
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Shock 0045

When supply does not meet demand

The metabolic supply to tissue, and thereby organ systems, does not meet the demand

It is **NOT** defined by a blood pressure



Anaerobic metabolism: Less efficient energy production, lactate production



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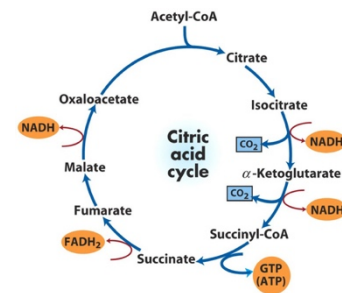
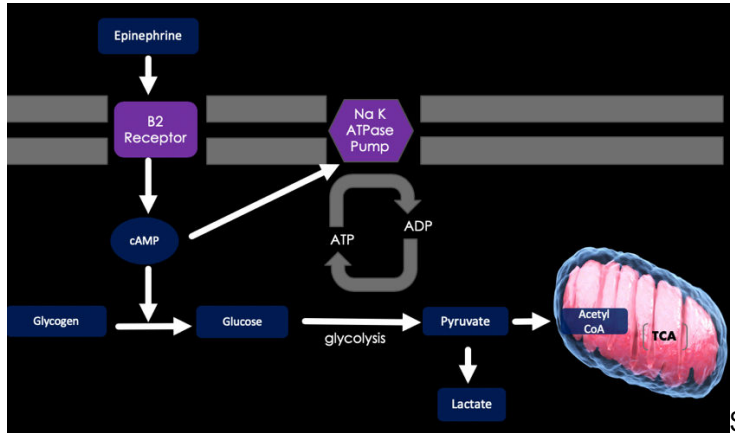
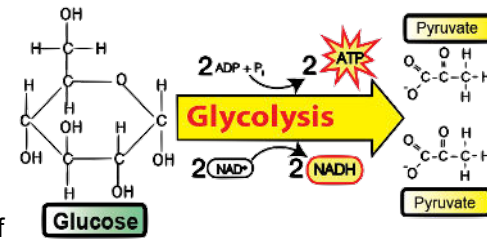
Lactate Production 0250

Glycolysis and Pyruvate

Krebs cycle

Lactate production in normal circumstances is due to inadequate oxygen supply

Lactate production in shock is due to adrenergic stimulation of the B2 receptors by epinephrine leading to increased glycolysis



Lactate production as energy source

J Cereb Blood Flow Metab. 2009 Jun;29(6):1121-9. doi: 10.1038/jcbfm.2009.35. Epub 2009 Apr 1.

Blood lactate is an important energy source for the human brain.

van Hall G¹, Strømstad M, Rasmussen P, Jans O, Zaar M, Gam C, Quistorff B, Secher NH, Nielsen HB.

Normally always have low levels of lactate production that is cleared by the kidneys and the liver

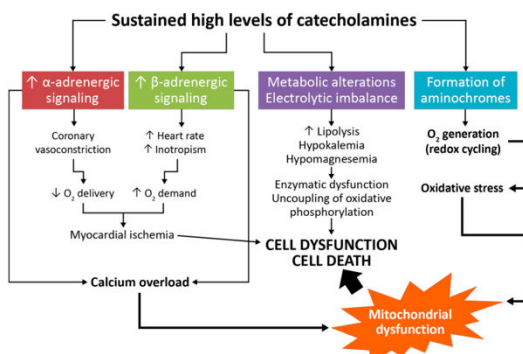


Figure 7: Deleterious effects of sustained catecholamine surge³²

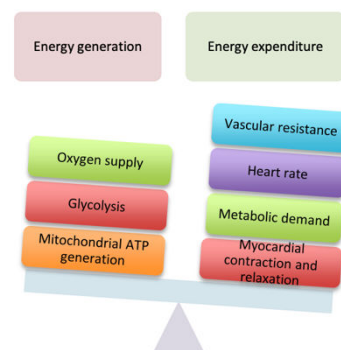


Figure 9: Imbalance between energy generation and expenditure⁴⁷

Causes of Hyperlactemia

- × Liver Disease
- × Accelerated glycolysis (increased metabolism): fever, adrenalin, hyperthyroidism, albuterol use
- × Mitochondrial dysfunction (inborn errors of metabolism)
- × Thiamine deficiency (malnutrition, chronic alcoholism)

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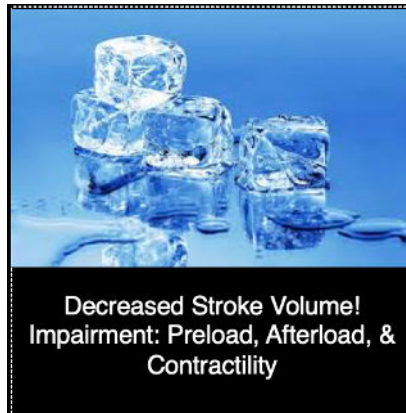
- × Anaerobic metabolism (ischemic gut)
- × Carbon monoxide & cyanide toxicity: alter oxidative phosphorylation
- × Metformin
- × Hyperventilation (alkalemia)
- × Sepsis (multifactorial)

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Categories of Shock 0638

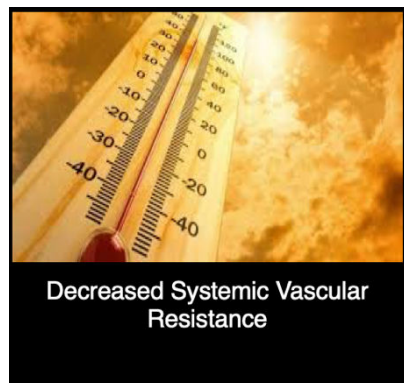
Cold shock 0710

- × Decreased / inadequate stroke volume
- × decreased preload, contractility
- × increased afterload
- × Narrow pulse pressure due to compensatory vasoconstriction and increase in systemic vascular resistance



Warm shock

- × Decreased systemic vascular resistance
- × Movement of blood flow from vital organs to non-vital organs (e.g. skin and muscles)
- × Wide pulse pressure due to the low systemic vascular resistance



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Stroke volume 0829

- × The amount of blood pumped out of the heart with each heartbeat

Preload:

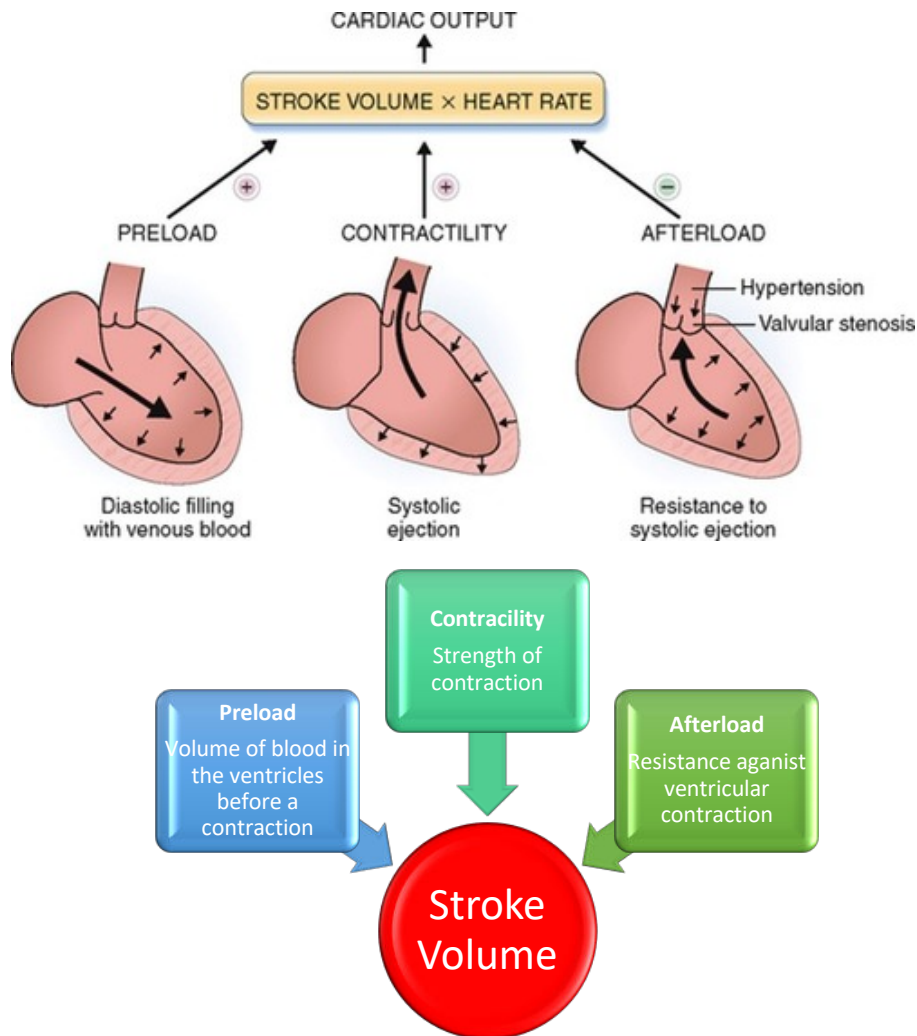
- × The volume of blood present in the ventricle at the end of diastole
- × Causes for decrease in preload – hypovolemia, hemorrhage or vasodilation

Contractility:

- × The strength of contraction
- × Causes for decrease contractility- ischemia, toxins, myocarditis, congenital heart disease

Afterload:

- × Resistance against the ventricular contraction
- × Causes for increase afterload- hypertensive emergency, increased vasocontraction



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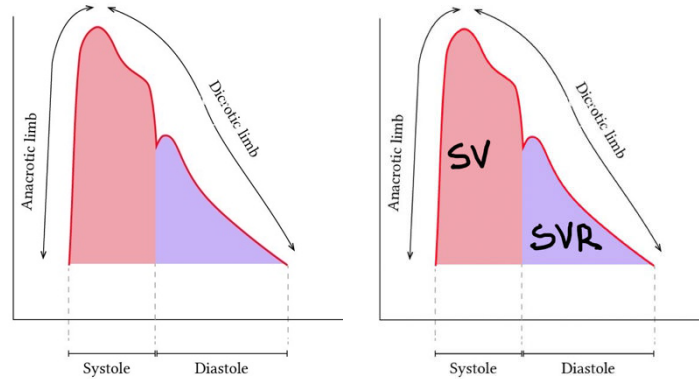
Defining blood pressures 0947

Systolic blood pressure

- × Determined by the blood volume in the arteries + aortic compliance
- × Systolic blood pressure ~ stroke volume

Diastolic blood pressure

- × As the ventricle is relaxing, tissue perfusion determined by systemic vascular resistance
- × Diastolic blood pressure ~ systemic vascular resistance



Chronically low diastolic blood pressure, therefore an increased pulse pressure, could be associated with aortic regurgitation

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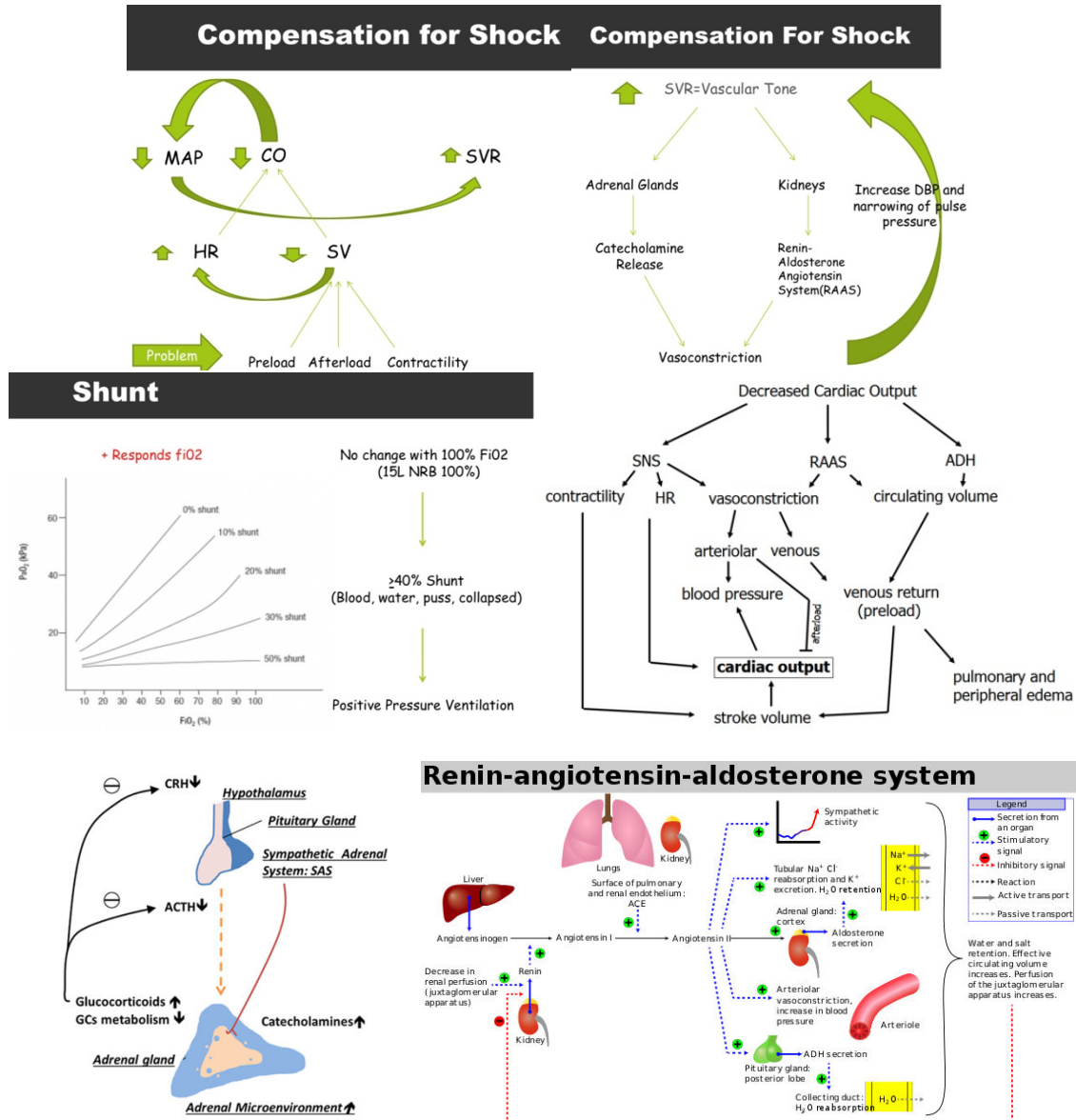
Compensation for shock 1150

Decreased stroke volume \rightarrow low cardiac output state & decreased tissue perfusion

1. Increase heart rate
2. Increase systemic vascular resistance (if heart rate is inadequate)

Determinates of vascular tone

- × Adrenal glands: catecholamines that cause vasoconstriction
- × Kidneys: (+) RAAS \rightarrow angiotensin II and aldosterone release



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Categories of Shock 1424

Cold shock:

Decrease stroke volume (i.e. decreased SBP) + increased systemic vascular resistance (i.e. increased DBP)=> narrow pulse pressure

Cardiogenic shock:

- × Trouble with cardiac contractility
- × Examples: ischemia, congenital heart disease, toxins
- × Note: increasing the systemic vascular resistance will increase the afterload, thereby making it worse by further decreasing the stroke volume

RV Failure

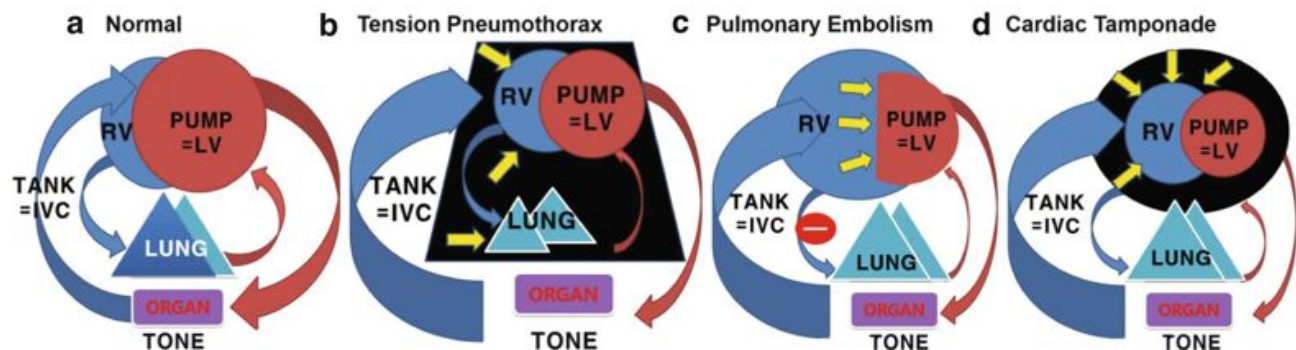
- × Signs: elevated JVD, pulmonary heave, loud P2, clear lung
- × Causes: Increased pulmonary vascular resistance (as seen in vascular occlusion (PE), PEEP or alveolar hypoxia)
- × Bedside echo can help differentiate (e.g. RV dilation in RV failure, LV with poor contractility in LV failure)

Hypovolemic shock:

- × Trouble with preload
- × Examples: hemorrhage, gastrointestinal losses, venodilation (most of the blood volume is in the venous vasculature) leading to relative hypovolemia

Obstructive shock (SICK):

- × Mechanism: Decreased preload/ increased afterload; normal contractility
- × Examples: Massive pulmonary embolism, cardiac tamponade, tension pneumothorax, HTN crisis, aortic dissection, restrictive/ constrictive pericarditis, very high positive end expiratory pressure (PEEP), abdominal compartment syndrome



Critical Care Fundamentals: Basics of Shock

Warm shock:

Distributive shock (meaning the problem is the “distribution” of blood flow) = decreased stroke volume (i.e. decreased SBP) << decreased systemic vascular resistance (i.e. decreased DBP) => widened pulse pressure

1. Venodilation – Increased venous capacitance leading to blood pooling in the venous system
 2. Increased capillary permeability - leading to loss of plasma volume into the interstitial space (so has a component of hypovolemic shock)
 3. Increased heart rate + increased contractility(compensation)
- × **Sepsis, adrenal insufficiency, liver failure, anaphylaxis**
 - Sepsis - myocardial dysfunction due to cytokine release

Decreased/ normal heart rate

- × Neurogenic shock (unopposed vagal tone)

