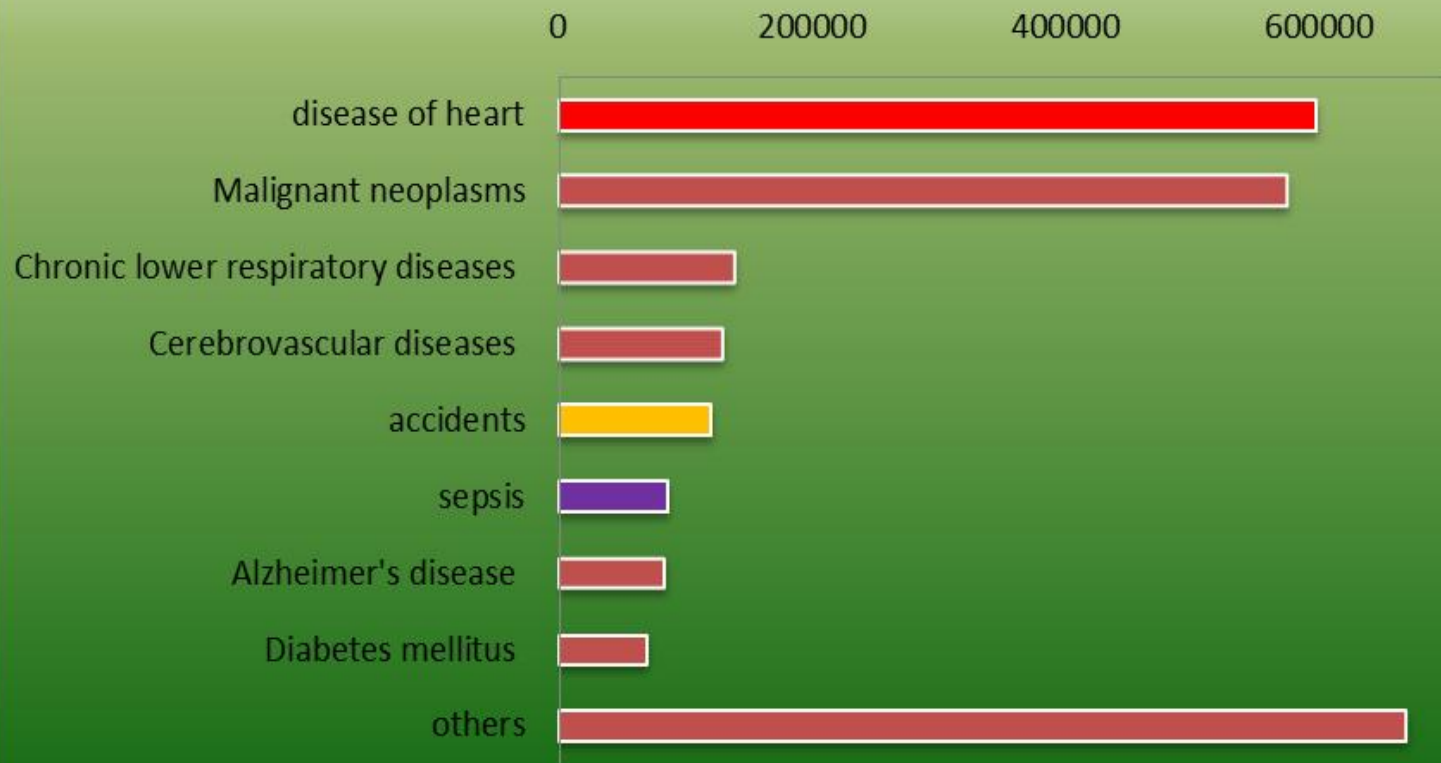


Shock

Shao Mian

Emergency Department, Zhongshan Hospital

Causes of death in US



What is shock

- THE BEGINNINGS OF UNDERSTANDING: THE LATE 19TH CENTURY
 - THE AGE OF REASON: 1890—1925
 - THE MODERN ERA: BLALOCK'S EPIPHANY
 - POSTMODERNISM: CELLULAR PHYSIOLOGY OF SHOCK 1945—1965
 - REACHING THE NEW MILLENNIUM: 1990--PRESENT
-

definition

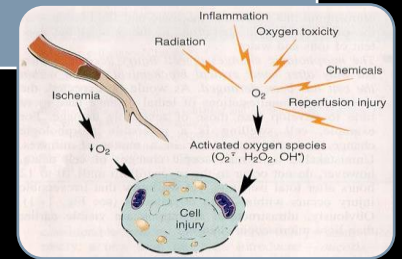
Trauma



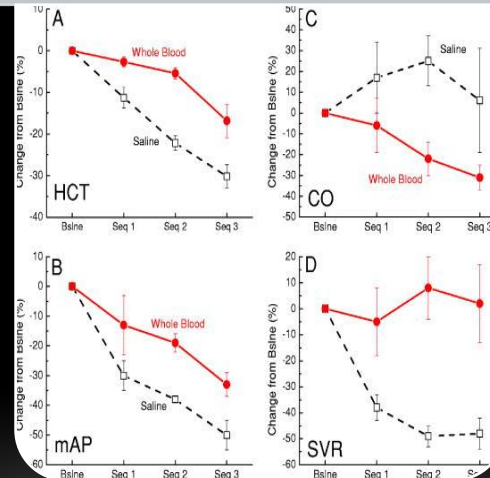
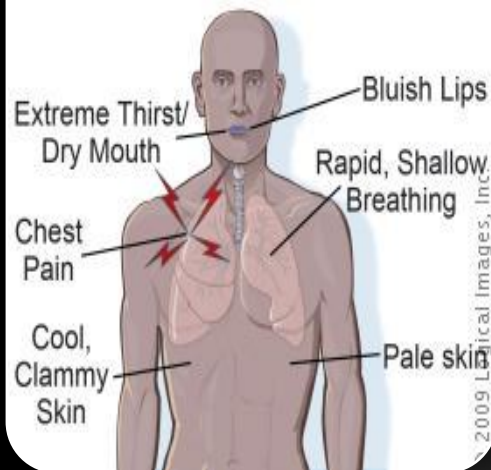
Bood pressure



Celluar injury

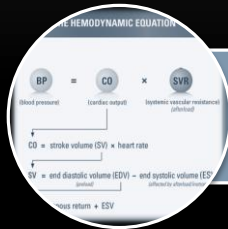


definition



The state in which profound and widespread reduction of effective tissue perfusion leads first to reversible, if prolonged, to irreversible cellular injury

Pathophysiology



Hemodynamic basis of shock

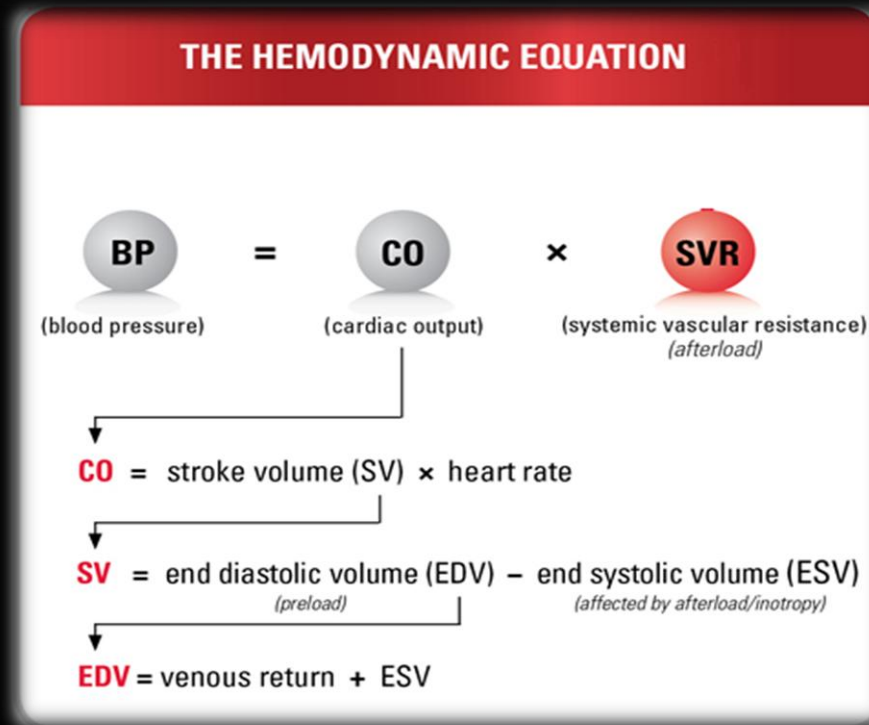


Compensatory mechanism

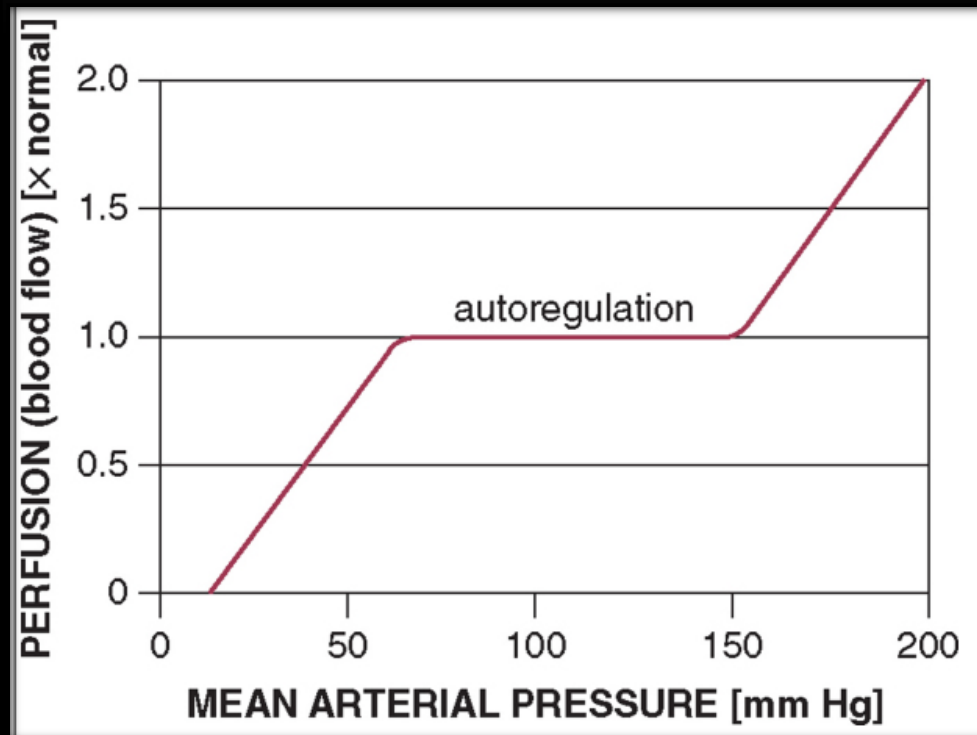


Organ dysfunction

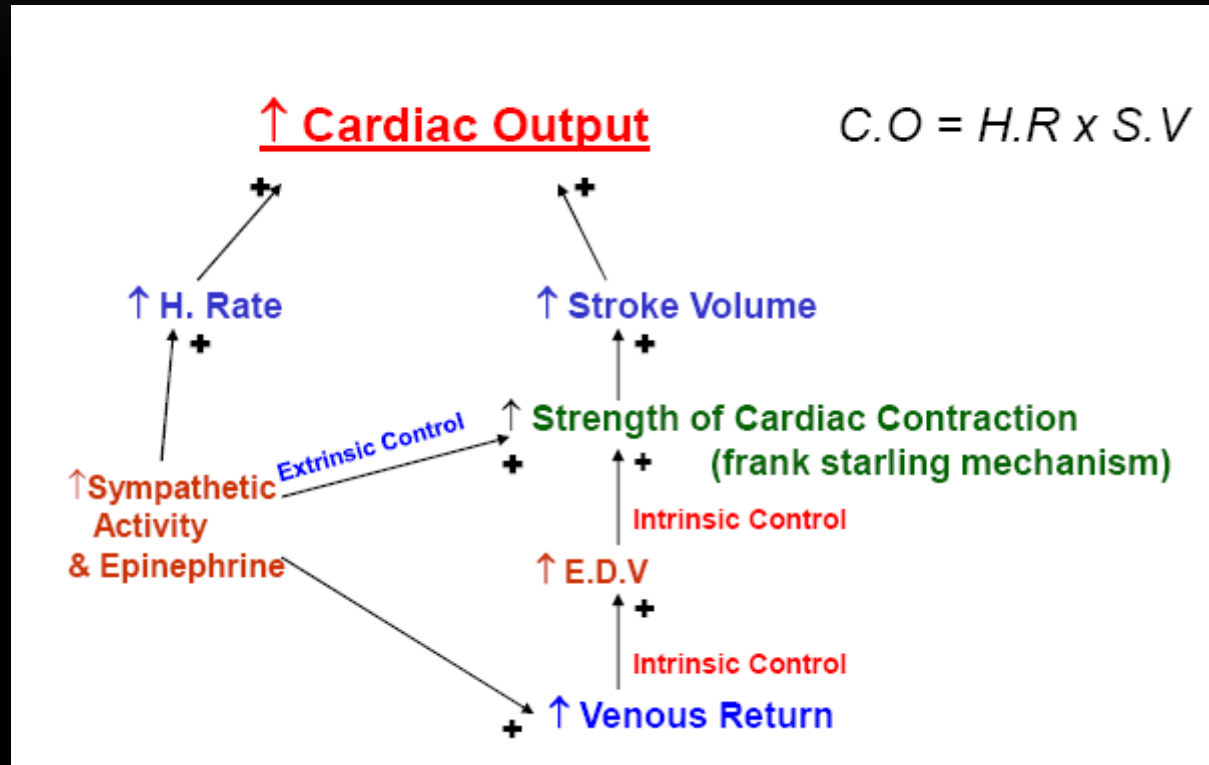
Hemodynamic Basis of Shock



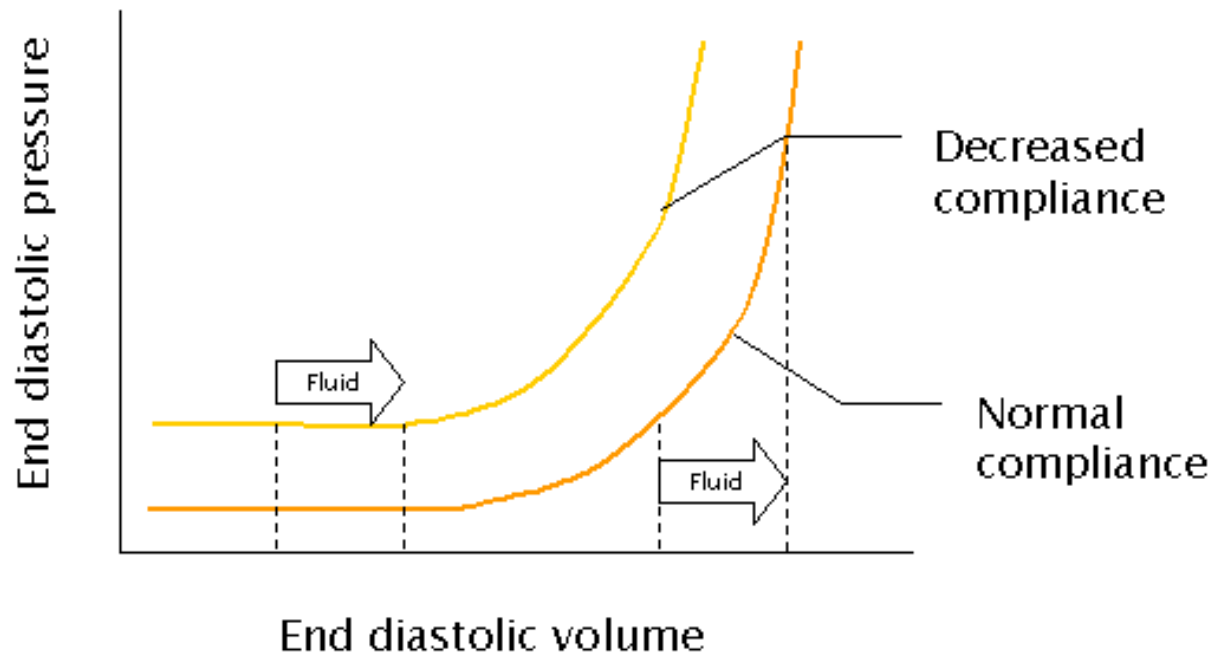
Blood flow autoregulation



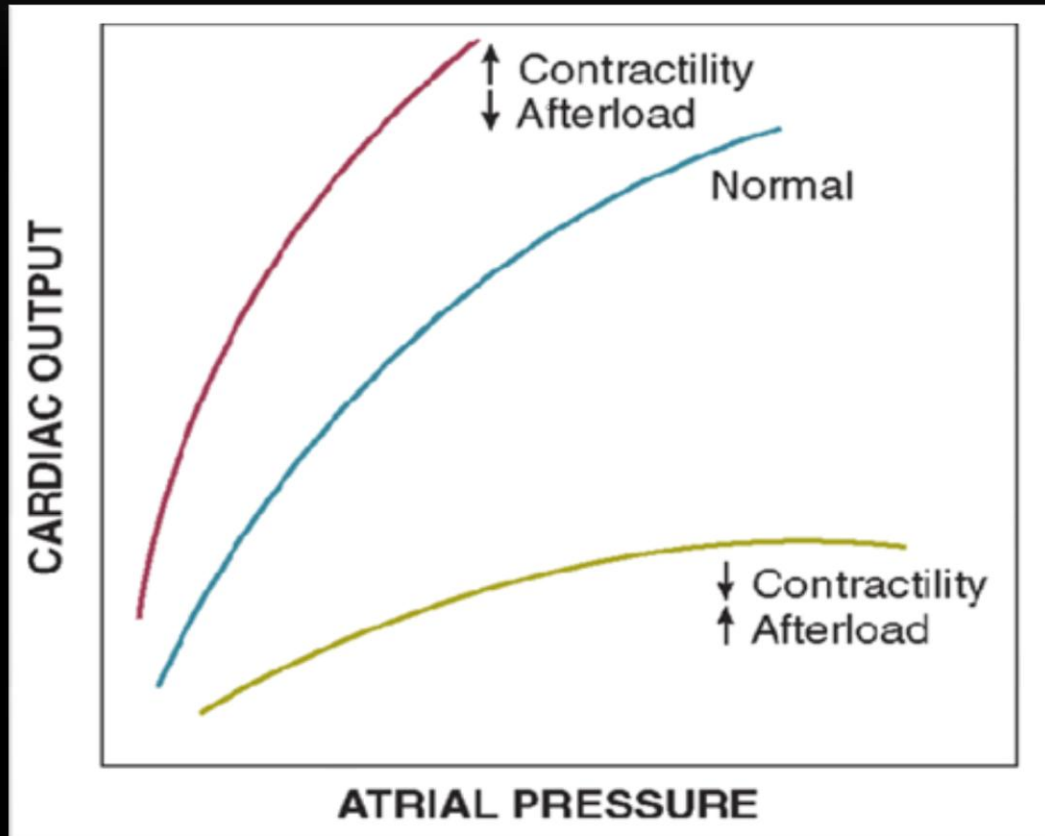
Cardiac output



Preload



Cardiac function curve



Venous Function

- A fundamental determinant of cardiac performance
- Maximal venous return is described by the equation:

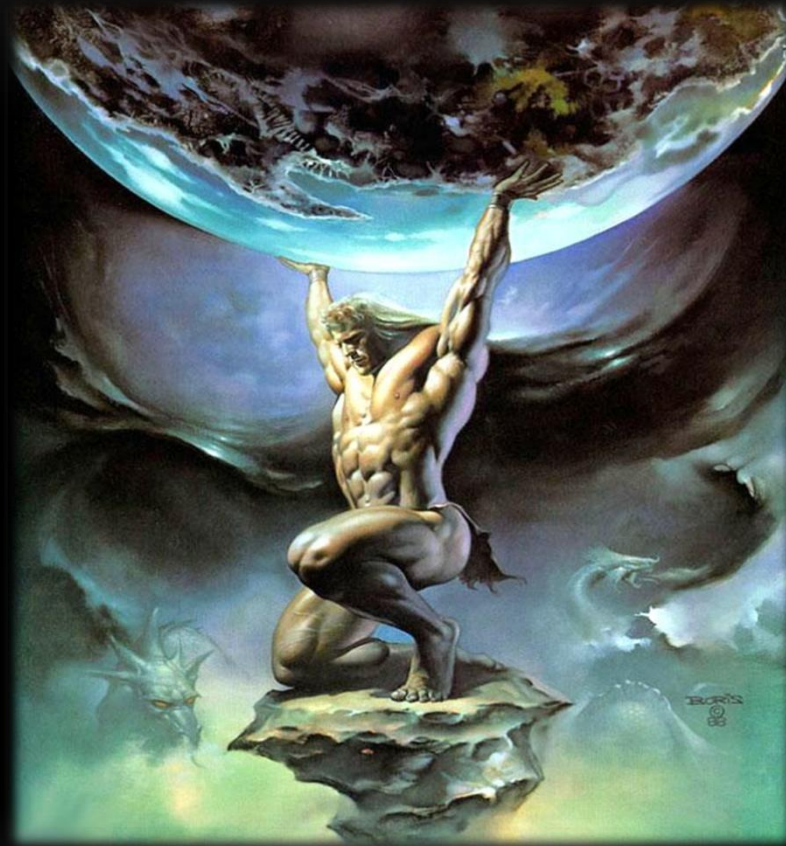
$$(P_{mc}-P_a)/R_v$$

- P_{mc} : the mean circulatory pressure
 - P_a : the right atrial pressure
 - R_v : the venous resistance
-

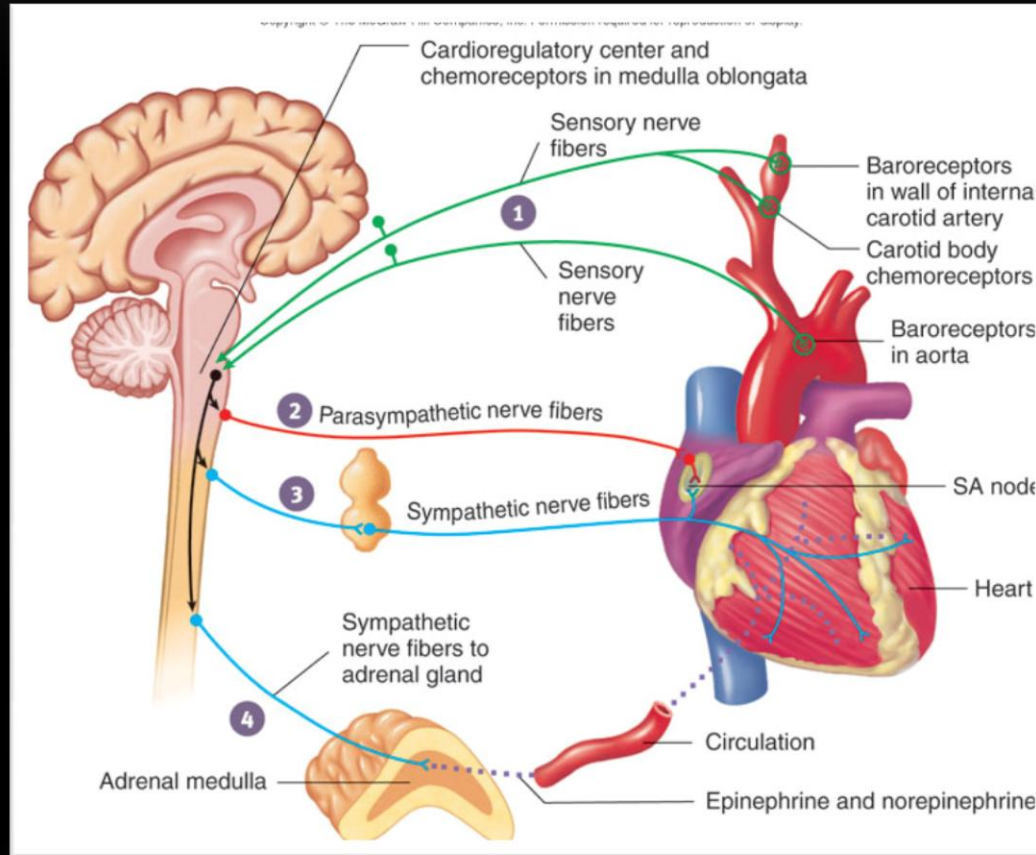
Microvascular Function

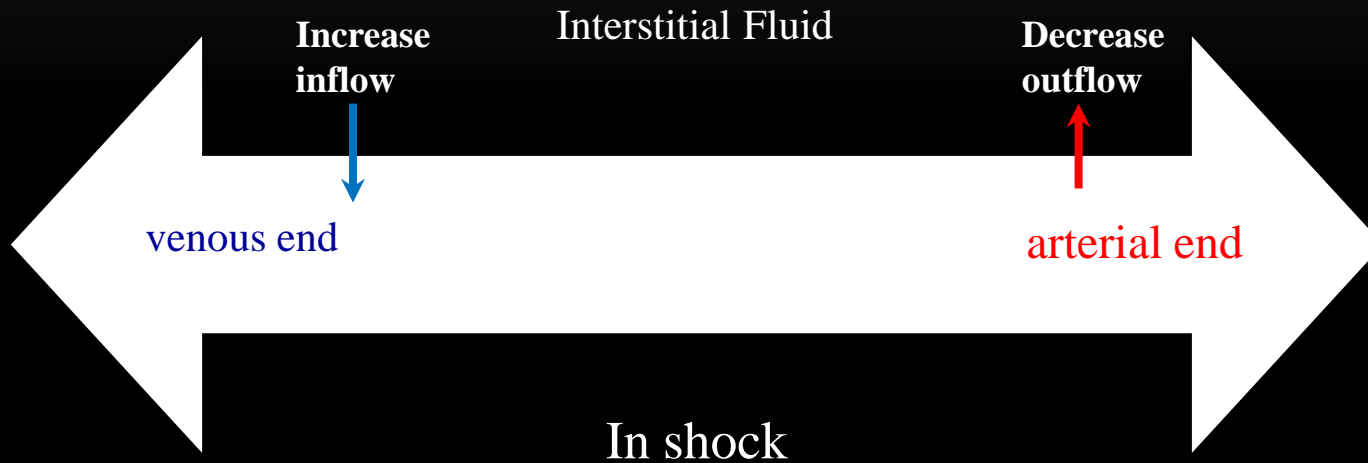
- Effective tissue perfusion requires intact microvascular function.
 - Match blood flow to areas of highest metabolic activity.
 - Intrinsic control of blood flow: endothelial stretch receptors, metabolic activity
 - Extrinsic control of blood flow: autonomic nervous system
-

Compensatory mechanism



Baroreceptors and chemoreceptors



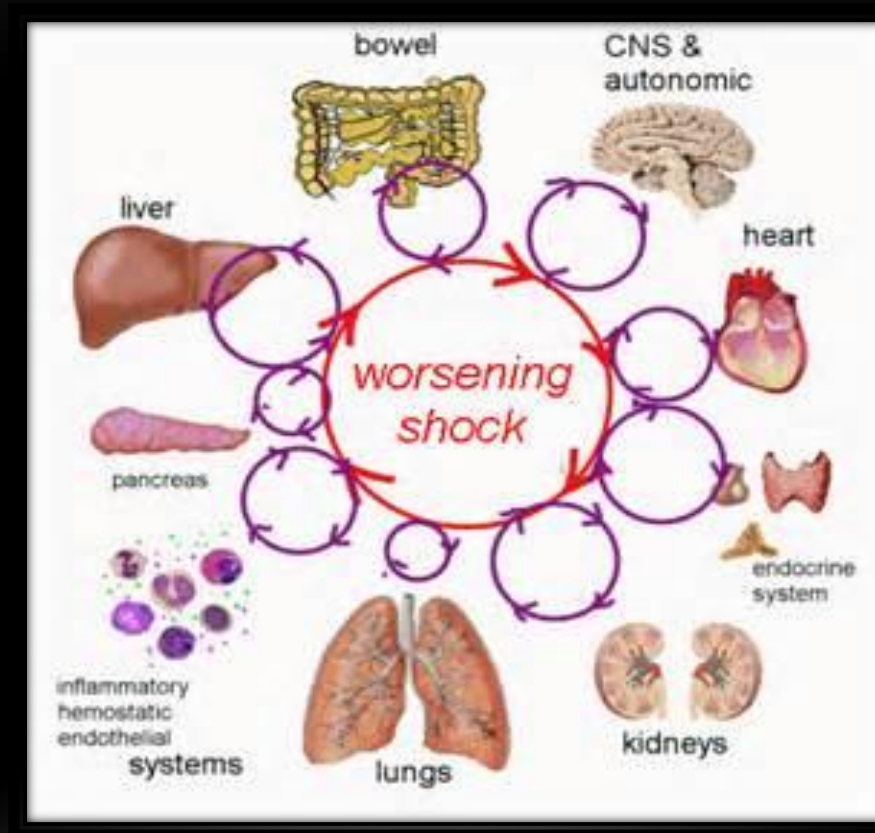


Fluid shift system

Renal compensatory mechanisms

- the juxtaglomerular apparatus : renin , aldosterone , angiotensin II
- the posterior pituitary : vasopressin
- Effects: water retention , vasoconstriction

Organ System Dysfunction



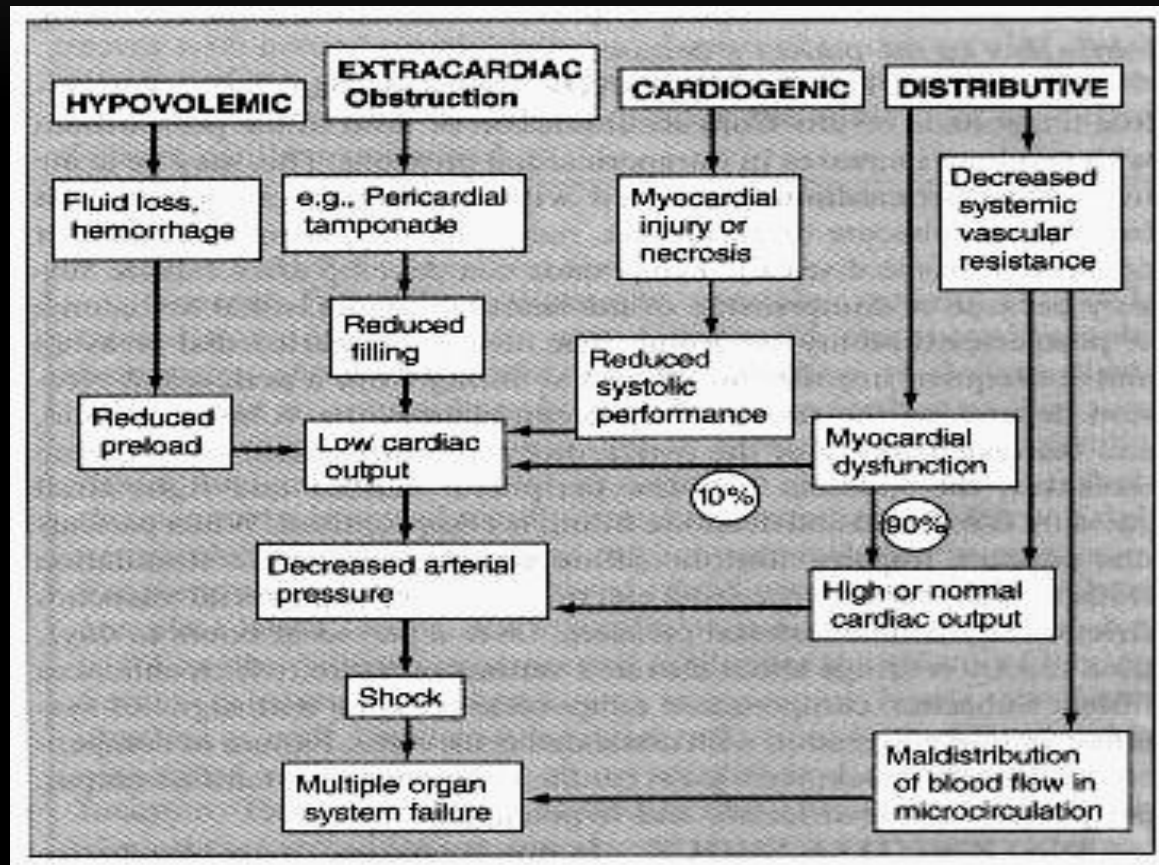
Central nervous system	Encephalopathy (ischemic or septic)
	Cortical necrosis
Heart	Tachycardia, bradycardia
	Supraventricular tachycardia
	Ventricular ectopy
	Myocardial ischemia
	Myocardial depression
Respiratory	Acute respiratory failure
	Adult respiratory distress syndrome
Kidney	Prerenal failure
	Acute tubular necrosis
Gastrointestinal	Ileus
	Erosive gastritis
	Pancreatitis
	Acalculous cholecystitis
	Colonic submucosal hemorrhage
	Transluminal translocation of bacteria/antigens

Liver	Ischemic hepatitis
	“Shock” liver
	Intrahepatic cholestasis
Hematologic	Disseminated intravascular coagulation
	Dilutional thrombocytopenia
Metabolic	Hyperglycemia
	Glycogenolysis
	Gluconeogenesis
	Hypoglycemia (late)
	Hypertriglyceridemia
Immune system	Gut barrier function depression
	Cellular immune depression
	Humoral immune depression

- Initial signs of end organ dysfunction
 - Tachycardia
 - Tachypnea
 - Metabolic acidosis
 - Oliguria
 - Cool and clammy skin
-

- End Organ Dysfunction
 - Progressive irreversible dysfunction
 - Oliguria or anuria
 - Progressive acidosis and decreased CO
 - Agitation, obtundation, and coma
 - Patient death
-

Classification of Shock



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19 December 2011 Last updated at 03:20 GMT



Obituary: Kim Jong-il



The BBC's James Robbins reports on the life of North Korea's Kim Jong-il

Kim Jong-il was one of the world's most reclusive and enigmatic leaders, presiding over a secretive and internationally isolated country.

Kim Jong-il dead

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Cardiogenic Shock

- Cardiogenic shock is a state of inadequate tissue perfusion as a result of cardiac dysfunction.
 - Acute myocardial infarction is the leading cause of cardiogenic shock.
 - The diagnosis is made by the presence of hypotension and clinical signs indicative of poor tissue perfusion.
 - Hemodynamic criteria include sustained hypotension and a reduced cardiac index in the presence of elevated pulmonary capillary occlusion pressure .
-

Diagnosis of Cardiogenic Shock

Clinical Signs

Hypotension

Oliguria

Clouded sensorium

Cool and mottled
extremities

Hemodynamic Criteria

Systolic blood pressure <90 mm Hg for >30 minutes

Cardiac index <2.2 L/min/m²

Pulmonary artery occlusion pressure >15 mm Hg

Causes of Cardiogenic Shock

Acute Myocardial Infarction

- Pump failure
 - Large infarction
 - Smaller infarction with pre-existing left ventricular dysfunction
 - Infarct extension
 - Reinfarction
 - Infarct expansion
- Mechanical complications
 - Acute mitral regurgitation secondary to papillary muscle rupture
 - Ventricular septal defect
 - Free wall rupture
 - Pericardial tamponade
- Right ventricular infarction

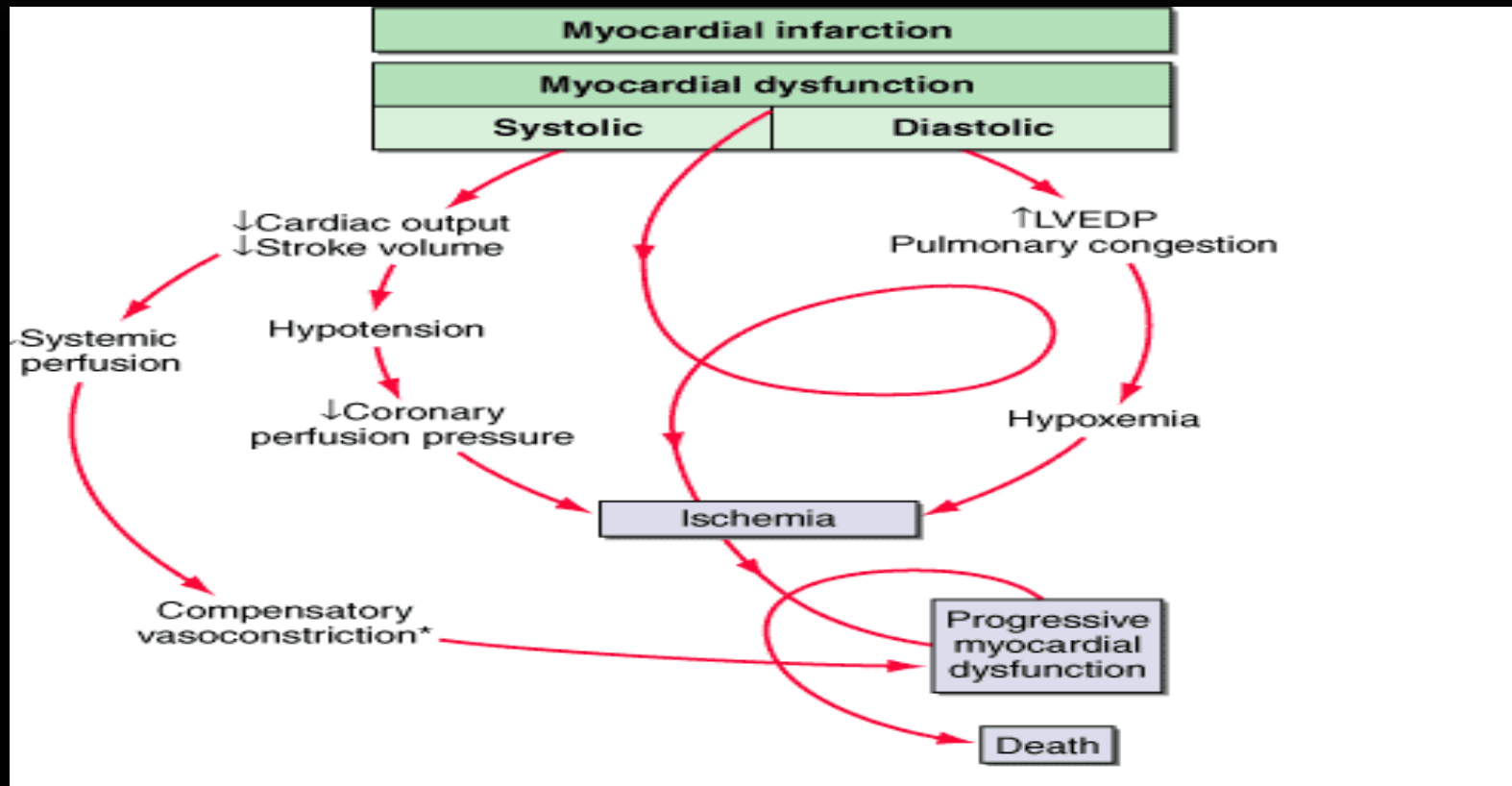
Other Conditions

- End-stage cardiomyopathy
- Myocarditis
- Myocardial contusion
- Prolonged cardiopulmonary bypass
- Septic shock with severe myocardial depression
- Left ventricular outflow tract obstruction
 - Aortic stenosis
 - Hypertrophic obstructive cardiomyopathy
- Obstruction to left ventricular filling
 - Mitral stenosis
 - Left atrial myxoma
- Acute mitral regurgitation (chordal rupture)
- Acute aortic insufficiency

Risk factors for cardiogenic shock

- Shock is more likely to develop in patients who are elderly, are diabetic, and have anterior infarction.
- Patients with cardiogenic shock also are more likely to have histories of previous infarction, peripheral vascular disease, and cerebrovascular disease.
- Decreased ejection fractions and larger infarctions also are predictors of the development of cardiogenic shock

“Downward spiral”



Cardiogenic Shock

DIAGNOSTIC

THERAPEUTIC

Initial Diagnostic Steps

- Directed history and physical examination
- EKG
- Echocardiography
- Laboratory testing
- Chest x-ray
- Pulmonary artery catheterization

Initial Management Steps

- Supplemental oxygen/mechanical ventilation
- Venous access
- EKG monitoring
- Pain relief
- Hemodynamic support
 - Fluid challenge in patients without pulmonary edema
 - Vasopressors for hypotension unresponsive to fluids

Tissue Perfusion Remains Inadequate

Inotropic agents
Intra-aortic balloon pump

Adequate Perfusion without Congestion

Adequate Tissue Perfusion with Pulmonary Congestion

Diuretics
Vasodilators

Reperfusion

PRESSORS

- Dopamine
 - <2 renal vascular dilation
 - <2-10 +chronotropic/inotropic (beta effects)
 - >10 vasoconstriction (alpha effects)
- Dobutamine
 - positive inotrope
 - Vasodilates
 - arrhythmogenic
- Norepinephrine (Levophed):
 - Vasoconstriction
 - inotropic stimulant.
- Vasopressin — vasoconstriction
 - Vasoconstriction



Hypovolemic Shock

- Hypovolemic shock can be defined as an acute disturbance in the circulation leading to an imbalance between oxygen supply and demand in the tissues, caused by a decrease in circulating blood volume.
 - The condition is lifethreatening and, if left untreated, becomes irreversible after a certain period.
 - Rapid and adequate resuscitation is mandatory to save lives. Conversely, hypovolemic shock carries a relatively favorable prognosis, if rapidly and adequately recognized and treated.
-

- Etiologic classes
 - Hemorrhage - e.g. trauma, GI bleed, ruptured aneurysm
 - Fluid loss - e.g. diarrhea, vomiting, burns, third spacing, iatrogenic
-

Blood loss and clinical signs

Parameter	I	II	III	IV
Blood loss (ml)	<750	750–1500	1500–2000	>2000
Blood loss (%)	<15%	15–30%	30–40%	>40%
Pulse rate (beats/min)	<100	>100	>120	>140
Blood pressure	Normal	Decreased	Decreased	Decreased
Respiratory rate (bpm)	14–20	20–30	30–40	>35
Urine output (ml/hour)	>30	20–30	5–15	Negligible
CNS symptoms	Normal	Anxious	Confused	Lethargic

Distributive Shock

- Results from loss of peripheral resistance
 - characterized by an overall decrease in SVR.
 - Initially, CI may be depressed, and ventricular filling pressures may be decreased. After fluid resuscitation, when filling pressures are normalized or increased, CI is usually elevated.
-

- Sepsis : septic shock is the most common form .
 - Neurogenic / spinal
 - Toxic shock syndrome
 - Anaphylaxis and anaphylactoid reactions
 - Toxin reactions – drugs, transfusions
 - Addisonian crisis
 - Myxedema coma
-

SEPTIC SHOCK

- **Physical Examination:**

Early– warm wet/ vasodilation, often adequate urine output, febrile, tachypneic.

Late – vasoconstriction, hypotension, cool, clammy skin , oliguria, altered mental status.

- **Monitor/findings:**

Early—hyperglycemia, respiratory alkylolosis, hemoconcentration, WBC typically normal or low

Late – Leukocytosis, lactic acidosis

Very Late– Disseminated Intravascular ,Coagulation & Multi-Organ System Failure

- **Hemodynamics:**

decreased CO, decreased SVR, decreased SvO₂

EGDT of Septic Shock

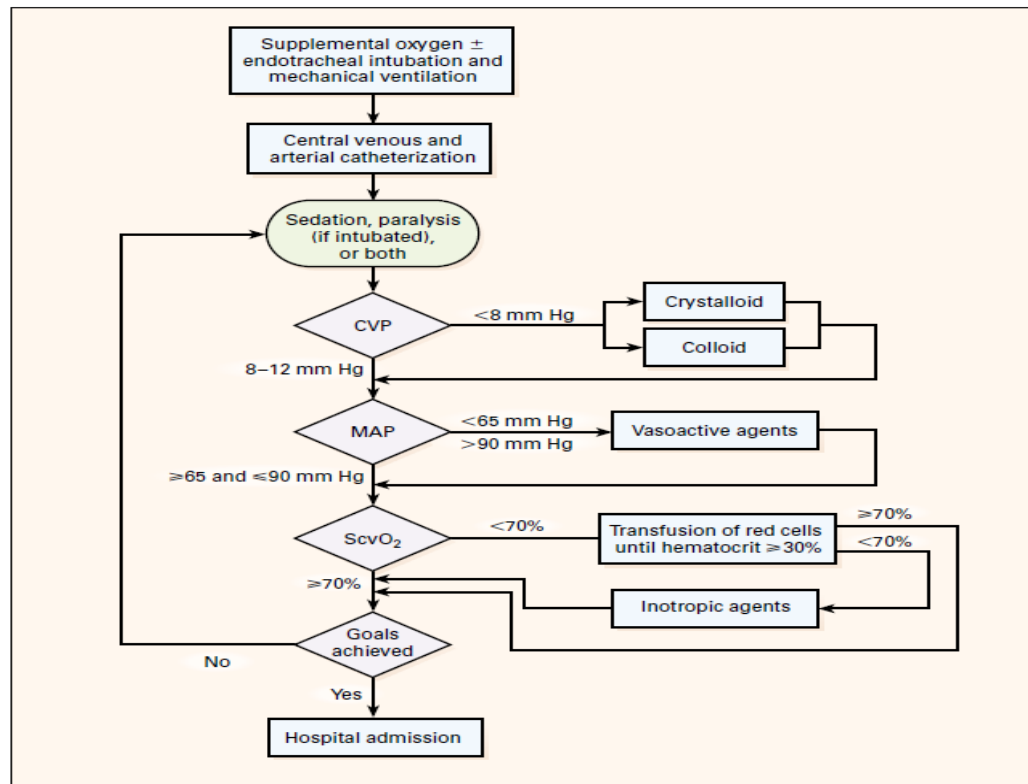


Figure 2. Protocol for Early Goal-Directed Therapy.

CVP denotes central venous pressure, MAP mean arterial pressure, and ScvO₂ central venous oxygen saturation.



Obstructive Shock

- Extracardiac obstructive shock results from an obstruction to flow in the cardiovascular circuit.
 - Pulmonary Embolism
 - Pericardial Tamponade
 - Tension Pneumothorax
 - Aortic dissection
-

MANAGEMENT

- Manage the emergency
 - Determine the underlying cause
 - Definitive management or support
-

Manage the Emergency

- How long do you have to manage this?
 - Suggests that many things must be done at once
 - Draw in ancillary staff for support!
 - What must be done?
-

Manage the Emergency

- One person runs the code!
 - Control airway and breathing
 - Maximize oxygen delivery
 - Place lines, tubes, and monitors
 - Get and run IVF on a pressure bag
 - Get and run blood (if appropriate)
 - Get and hang pressors
 - Call your senior
-

Evaluation

- Done in parallel with treatment!
 - distinguish type of shock
 - Full laboratory evaluation
 - Basic studies – CXR, EKG
 - Basic monitoring
-

Determine the Cause

- Often obvious based on history
 - Trauma most often hypovolemic
 - Postoperative most often hypovolemic
 - Debilitated hospitalized patients most often septic
 - Must evaluate all patients for risk factors for MI
-

Definitive Management

- Hypovolemic – Fluid resuscitate and control ongoing loss
 - Cardiogenic - Restore blood pressure and prevent ongoing cardiac death
 - Distributive – Fluid resuscitate, pressors for maintenance, immediate control for infection, steroids for adrenocortical insufficiency
-