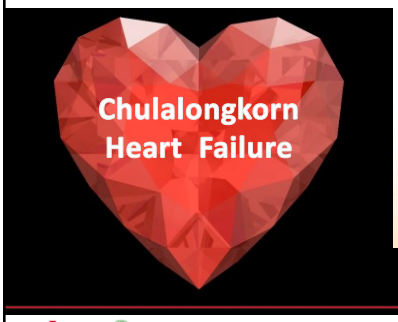
Pathophysiology of Cardiogenic shock & Heart failure



เอกราช อริยะชัยพาณิชย์
Aekarach Ariyachaipanich, MD, FACC

aekarach.a@chula.ac.th

CVS 2 3000378
4.09.2018

Agenda

1. Anatomy and physiology of the heart
2. Pathophysiology of shock
3. Pathophysiology of heart failure

Anatomy and physiology of the heart

Anatomy and physiology of the heart

1. What is the heart?
2. What is the function of the heart?
3. What is the heart consisted of?
4. What is cardiac output?
5. What govern the cardiac output?
6. What is preload, contractility and afterload?

Google what is the heart

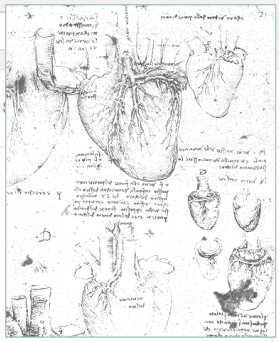
Web Images Videos News More

About 687,000,000 results (0.50 seconds)

heart
/härt/

noun

1. a hollow muscular organ that pumps the blood through the circulatory system by rhythmic contraction and dilation. In vertebrates there may be up to four chambers (as in humans), with two atria and two ventricles.
   synonyms: informal ticker
   "my heart stopped beating"

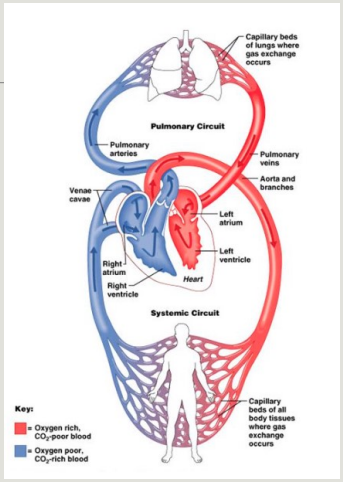


Heart is a pump

Cardiac output (L/min)

Heart rate (bpm)

Stroke volume (ml/beat)



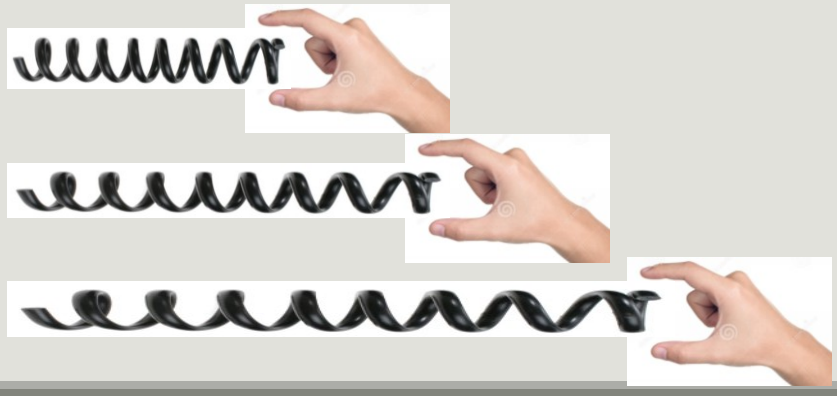
Preload

1

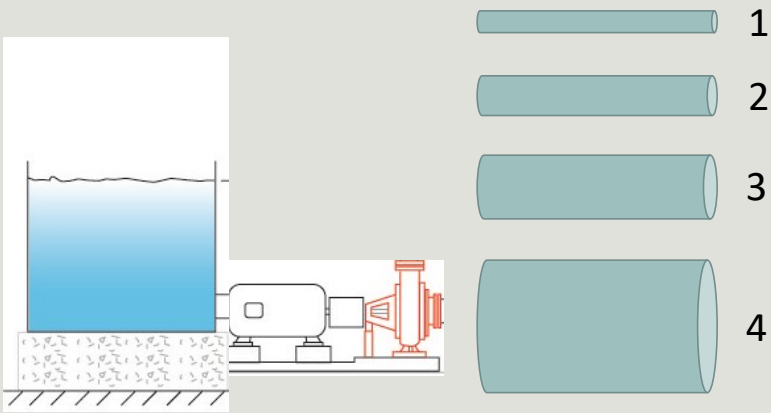
2

3

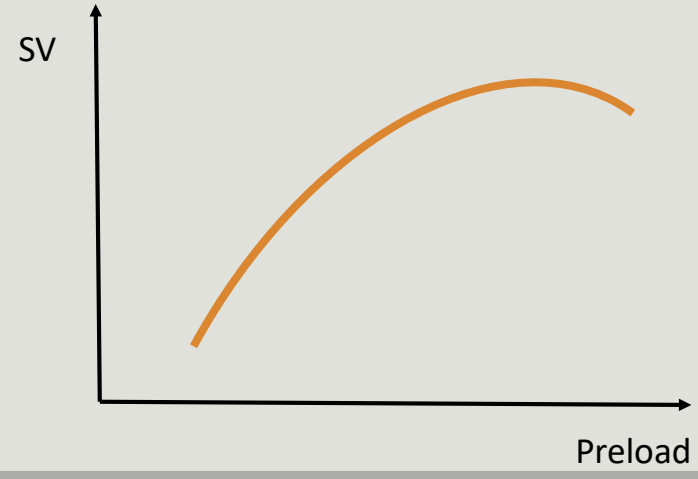
4



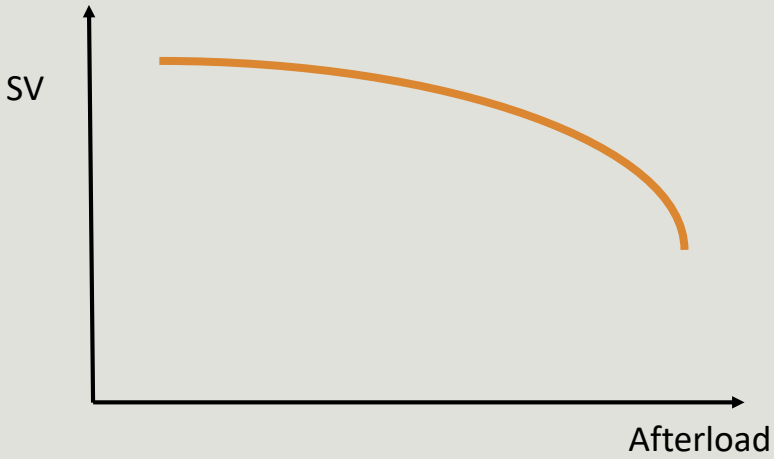
Afterload



Frank-Startling Mechanism

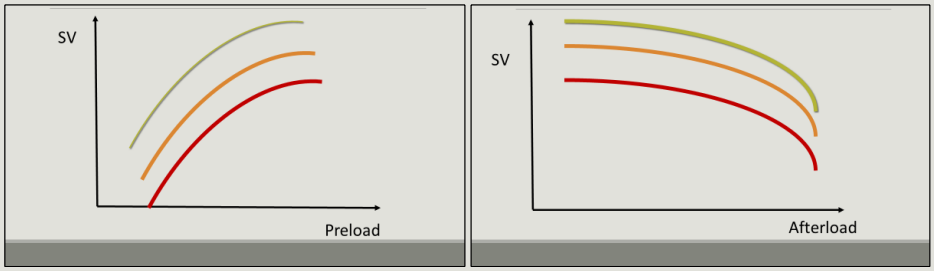


Afterload

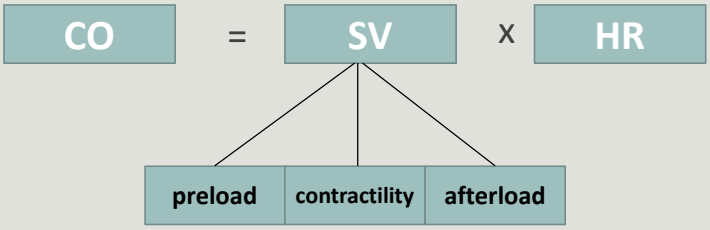


Contractility

An intrinsic property of the myocyte at a giving load



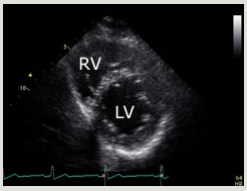
Cardiac output

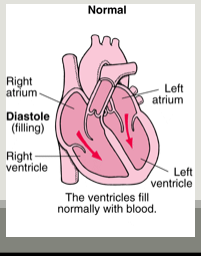


Preload

- A load (force/tension) that stretch the muscle before the initiation of contraction

- Myocardial cell length
- LV end diastolic volume --- LV size
- Pressure that streactch the heart
  - End diastolic pressure --- LVEDP
  - Atrial pressure



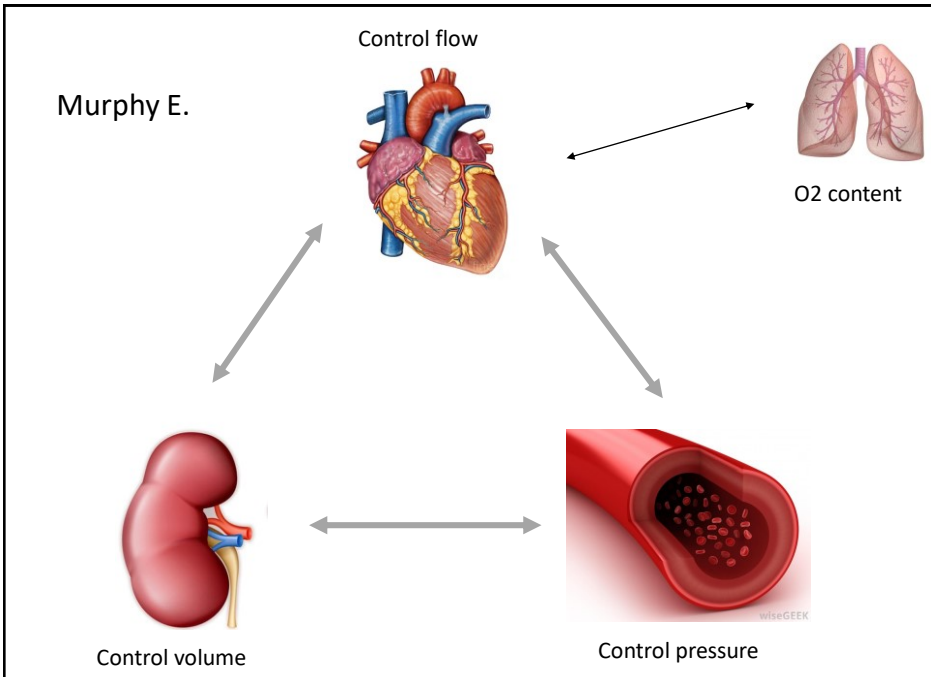


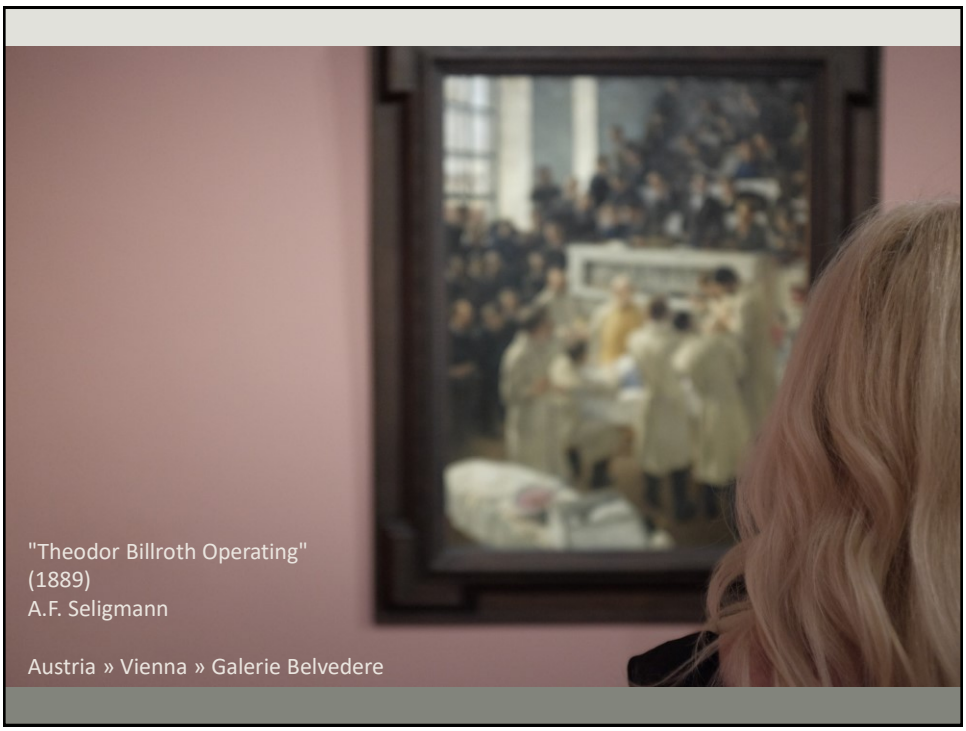
Afterload in Clinical

A "load" (resistant) that the heart has to contract against

- Vascular resistant
- Systolic blood pressure
- Vaso-constriction
- etc.

Murphy E.


Control flow
O2 content
Control volume
Control pressure


"Theodor Billroth Operating"
(1889)
A.F. Seligmann
Austria » Vienna » Galerie Belvedere

Pathophysiology of shock

Pathophysiology of shock

1. What is the shock?
2. Why shock is bad?
3. What are causes of shock?
4. What are type of shock?
5. What are S&S of shock?

Type of shock
initiation and compensatory mechanism

Type of Shock	Preload	CO	Afterload	Cause
Hypovolemic shock				
Cardiogenic shock				
Distributive shock				
Other type of shocks: Hypoadrenal, neurogenic, obstructive				

Type of shock
initiation and compensatory mechanism

Type of Shock	Preload	CO	Afterload	Cause
Hypovolemic shock	↓	~↑	↑	Blood or fluid loss (internal, external)
Cardiogenic shock	↑	↓	↑	Acute MI, acute HF Arrhythmia, cardiac tamponade pulmonary emboli
Distributive shock	~↓	↑↓	↓	Septic, anaphylaxis, inflammation, toxin
Other type of shocks: Hypoadrenal, neurogenic, obstructive				

What is shock ?

"The clinical syndrome characterize by an inadequate global tissue perfusion."

- Usually have hypotension (MAP < 60 mmHg)

Initiate by various causes

Lead to a vicious cycle of damages, due to

- Cellular dysfunction → functional and structural change.
- Multiple organ failure and death.

Adapt from harrison principles of internal medicine 18th edition

Hypoperfusion: Lead to a vicious cycle of damages

Cellular

- ATP depletion
- Aerobic to anaerobic
- Abnormal membrane function
- Cell dysfunction, swelling, death
- Inflammatory response
- Hematologic response

Multiorgan involments

- Renal failure
  - Acute kidney injury
- Liver failure
  - Ischemic hepatitis, shock liver
- Respiratory distress or failure
- Cardiac depression
- DIC

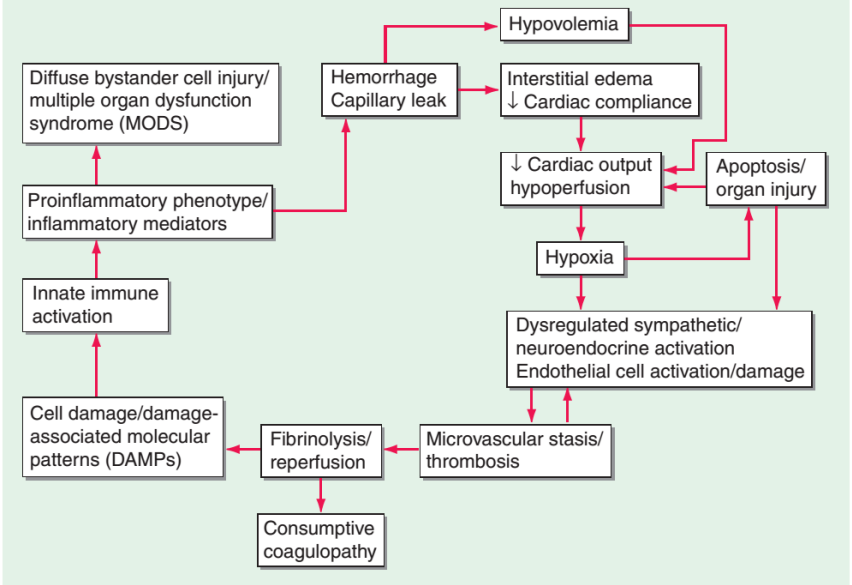
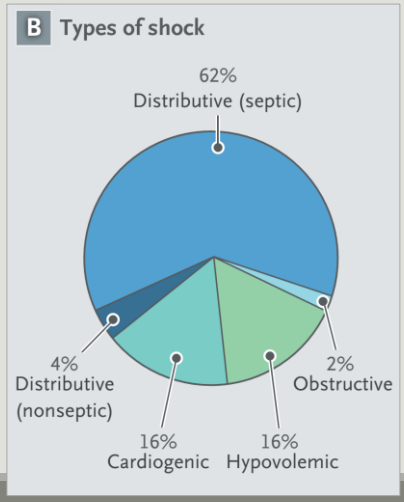


Figure 270-1 Shock-induced vicious cycle.

Type of shock



NEJM 2013

Type of shock
initiation and compensatory mechanism

Type of Shock	Preload	CO	Afterload	Cause
Hypovolemic shock				**Poor perfusion:** Δ mental status, tachycardia, ↓ BP, ↓ urine, ↑Cr, ↑Lactic acid, etc.
Cardiogenic shock				**Elevated preload:** ↑ JVP, (+) ascites, edema, (+) crepitations **Low preload:** dry mucosa, low JVP, skin turgor, orthostatic hypotension
Distributive shock				**Elevatred afterload:** cold skin, pale, ↑SVR **Low afterload:** warm skin, low SVR

Other type of shocks: Hypoadrenal, neurogenic, obstructive

Treatment

Type of Shock	Preload	CO	Afterload
Hypovolemic shock	↓	~↑	↑
Cardiogenic shock	↑	↓	↑
Distributive shock	~↓	↑↓	↓

Reverse the cause(s)
- In a timely fashion

Support and prevent further end organ damage
- Restore perfusion, reverse the physiology

ICU:

Fluid resuscitation: Crystalloid > colloid, ~~Cardiogenic shock~~

Circulatory support: Inotrope, vasopressor medications, Devices

Ventilation support: O2 support, Mechanical Ventilator/ Endotracheal tube

Invasive monitor: Arterial line, PA catheter (Swan-Ganz), Foley cath

Portrait of Père Paul (The Chef) – 1882
Claude Monet.

Pathophysiology of heart failure

Heart failure

A complex syndrome caused by cardiac abnormality resulted in typical symptoms and signs such as dyspnea, orthopnea, fatigue, leg edema, etc.

A syndrome of mal-adaptation – ”Adverse remodeling”

Chronic – Neurohormonal activation

Acute – Hemodynamic alterations

Pathophysiology

1. Structure changes
   - Macro
     - Concentric LVH, thick, sarcomeres added in parallel
     - Eccentric LVH, dilate, sarcomeres added in series, spherical
     - Reduced EF, mitral valve regur
   - Micro
     - Increase fibrosis,
     - Cal Handling (Ryanodine, SERCA2a, Phospholamban
     - Cardiac metabolism (staving: FFA → glycolysis)
     - Beta-adrenergic receptor
     - Fetal gene, miRNA
2. Hemodynamics
   - Impair contraction, impair relaxation
   - Elevated filling pressure, low CO
3. Neurohormonal
   - RAAS
   - sympathetic

Adverse remodeling

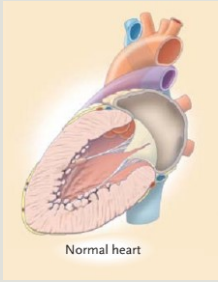
TABLE 25-2 Overview of Left Ventricular Remodeling

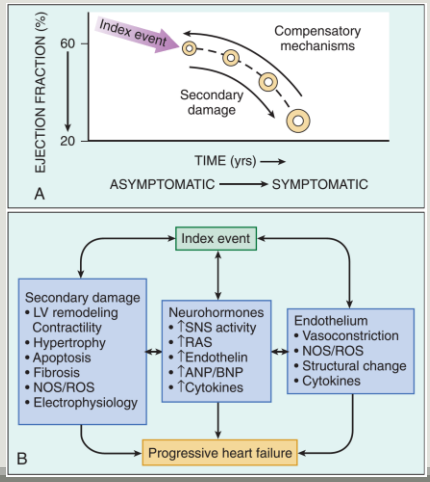
Alterations in Myocyte Biology
Excitation-contraction coupling
Myosin heavy chain (fetal) gene expression
Beta-adrenergic desensitization
Hypertrophy
Myocytolysis
Cytoskeletal proteins

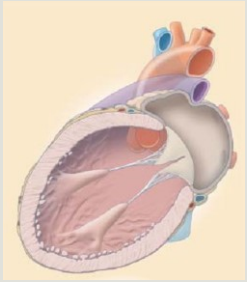
Myocardial Changes
Myocyte loss
  Necrosis
  Apoptosis
  Autophagy
Alterations in extracellular matrix
  Matrix degradation
  Myocardial fibrosis

Alterations in Left Ventricular Chamber Geometry
LV dilation
Increased LV sphericity
LV wall thinning
Mitral valve incompetence

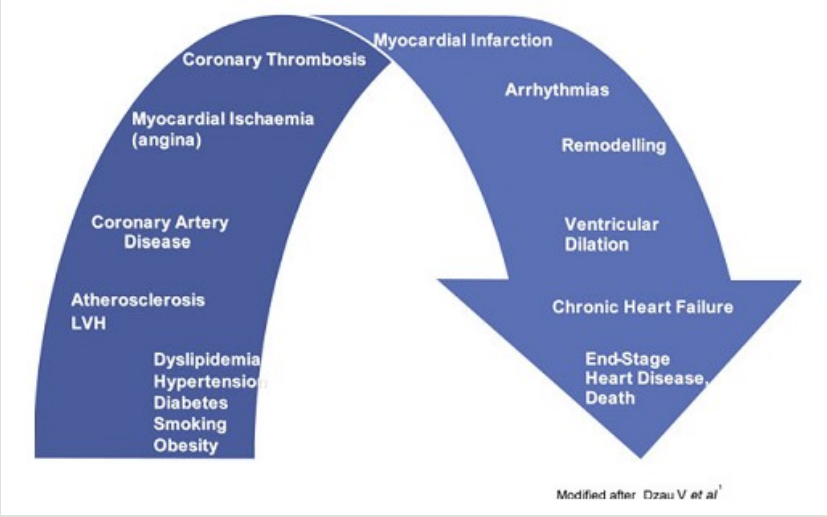
Pathophysiology
Remodeling



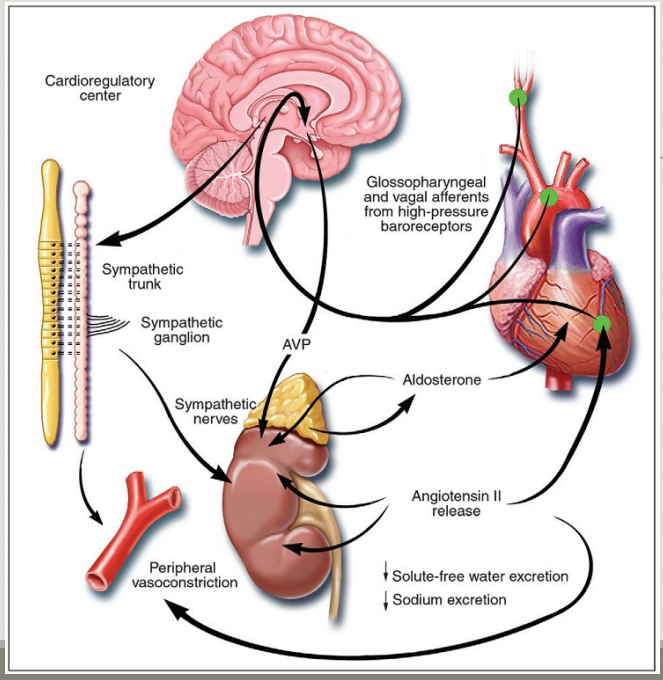


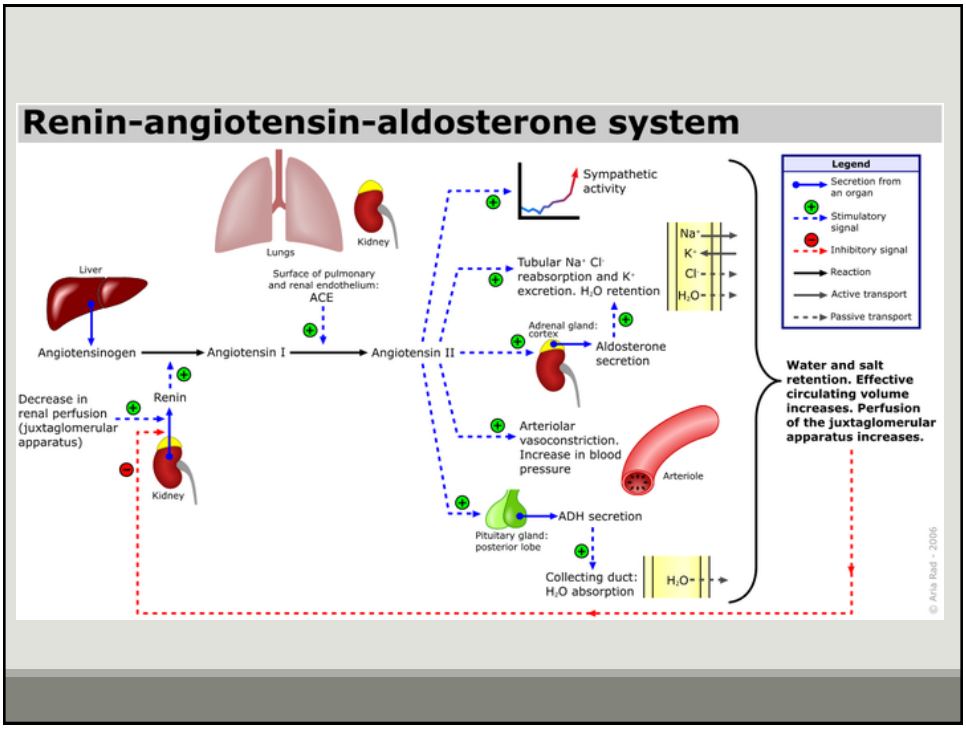


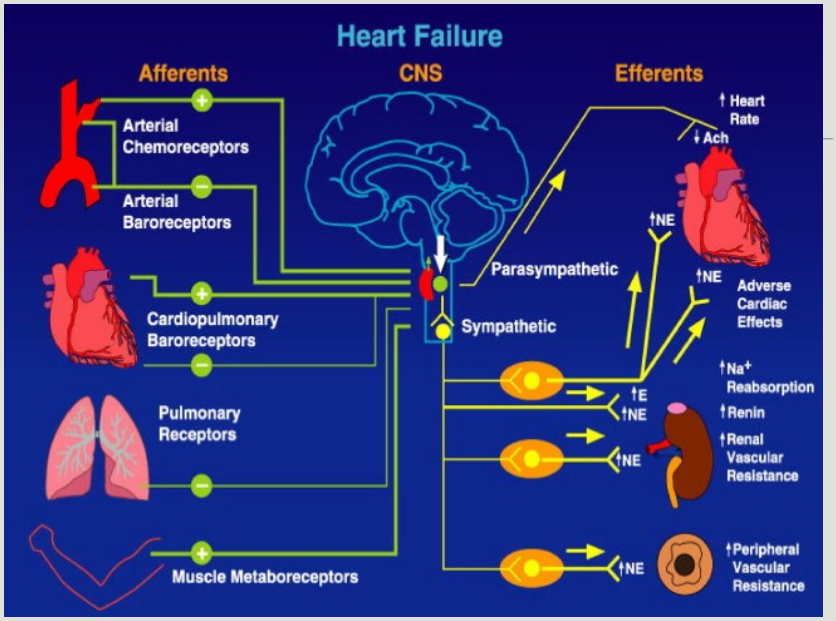
The Cardiovascular Continuum



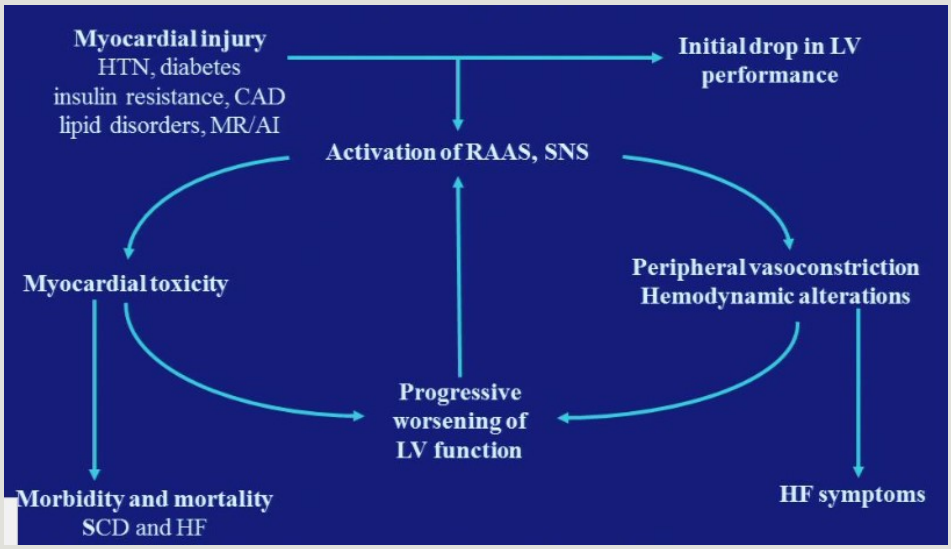
European Heart Journal 2008;10:Supp F17–F21


Cardioregulatory center
Glossopharyngeal and vagal afferents from high-pressure baroreceptors
Sympathetic trunk
Sympathetic ganglion
AVP
Aldosterone
Sympathetic nerves
Angiotensin II release
Peripheral vasoconstriction
Solute-free water excretion
Sodium excretion


Renin-angiotensin-aldosterone system
Lungs
Kidney
Liver
Surface of pulmonary and renal endothelium: ACE
Angiotensinogen
Angiotensin I
Angiotensin II
Renin
Decrease in renal perfusion (juxtaglomerular apparatus)
Kidney
Sympathetic activity
Tubular Na+ Cl- reabsorption and K+ excretion. H2O retention
Adrenal gland: cortex
Aldosterone secretion
Arteriolar vasoconstriction. Increase in blood pressure
Arteriole
ADH secretion
Pituitary gland: posterior lobe
Collecting duct: H2O absorption
Water and salt retention. Effective circulating volume increases. Perfusion of the juxtaglomerular apparatus increases.
Legend
Secretion from an organ
Stimulatory signal
Inhibitory signal
Reaction
Active transport
Passive transport
© Ania Rad - 2006

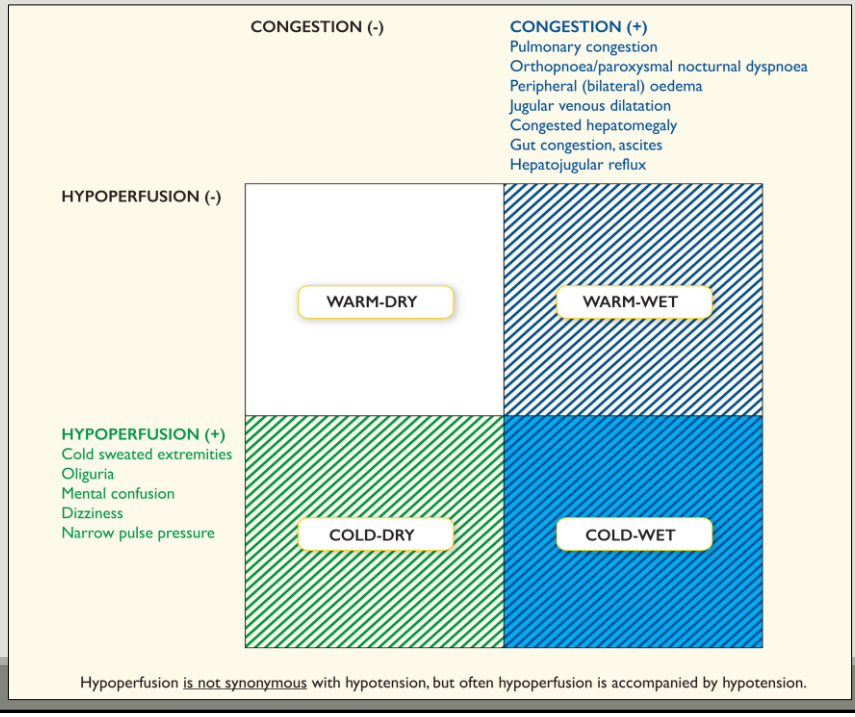


J Am Coll Cardiol 2009;54:375–85.



Classification of HF

Chronicity Acute Chronic	Stage A, B, C, D	EF rEF (< 40%) pEF (≥ 50%) mrEF (40-50)	Hemodynamic profile Wet - Dry Cold - warm
NYHA fn class I, II, III, IV	Etiology Ischemic cause Non-ischemic cause	involvement LV RV Both	phenotype Dilated Hypertrophic Restrictive
Endo / myo / epi	Backward / Forward failure	Low / High output	Systolic / diastolic failure



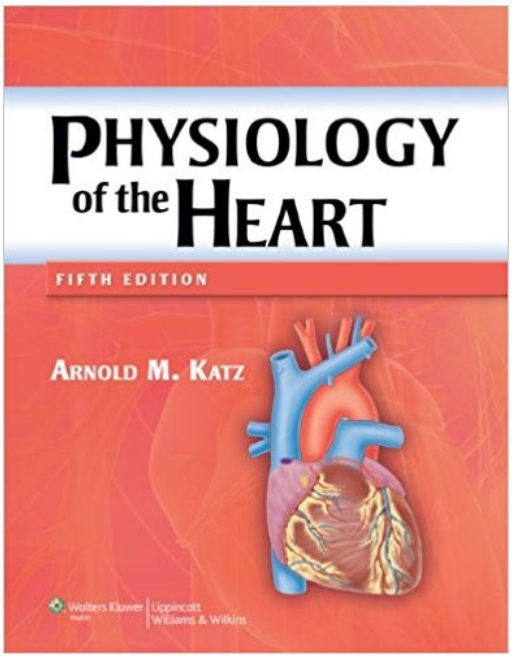
Treatment

Gen:
- Treat the cause
- Self-care e.g. weight monitor, ↓ salt intake
- Diuretics to control volume status

Chronic
- Betablocker
- ACEI / ARB
- Aldosterone blocker - spironolactone
- Angiotensin receptor, neprilysin inhibitor(ARNI) - Valsartan/sacubitril
- Other meds: Ivabradine, HDZ, ISDN, digoxin
- CRT - Cardiac resynchronize therapy (special pacemaker)
- ICD - Implantable cardioverter Defibrillator

Acute
- Aggressive diuresis, vasodilator, inotrope

End-staged HF
- Heart transplant, mechanical circulatory support, palliative care



Thank you

Aekarach.a@chula.ac.th

จุฬาลงกรณ์
ภาวะหัวใจล้มเหลว
และอายุรศาสตร์การปลูกถ่ายหัวใจ

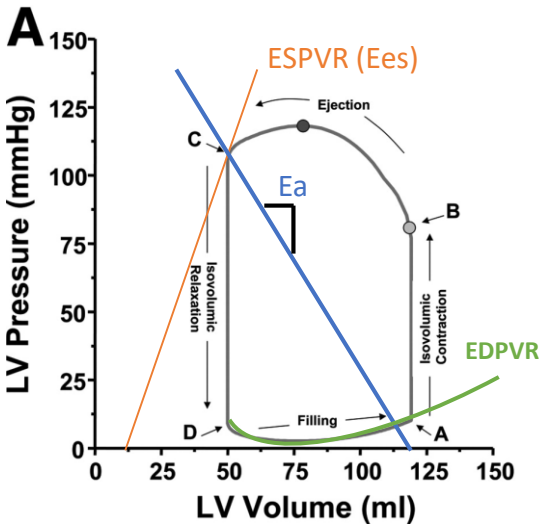
ศูนย์โรคหัวใจ
โรงพยาบาลจุฬาลงกรณ์
สภากาชาดไทย

Back up slide

Chulalongkorn
HEART
FAILURE
& TRANSPLANT CARDIOLOGY

	Action	Usual dose	C[1]	A[2]	Note
Epinephrine	α1 β1 β2	0.01-0.1mcg/kg/min 1 mg iv bolus q 3 mins	↑↑↑↑	↑↑↑	Low dose = more β. (like dobutamine) High dose = more α. (like norepi) Use: ACLS, anaphylaxis, S/E: splanchnic vasoconstrict.
Norepinephrine	α1 β1 β2	0.01-3 mcg/kg/min	↑↑↑	↑↑↑↑	Potent vasoconstriction. Moderate ↑CO. ~HR effect (reflex bradycardia from increased MAP. Use: Septic shock.
Dopamine **Low** **Moderate** **High**	DA α1 β1 β2 DA α1 β1 β2 DA	0.5 - 2 mcg/kg/min 2-10 mcg/kg/min 10-20 mcg/kg/min	~ ↑↑ ↑↑	↓ ↑ ↑↑↑	Precursor to norepi but less α , more β effect. Dose-dependent effects. Dose is varied pt to pt. Use: Septic shock, 2nd-line alternative to norepinephrine.
Dobutamine	β1 β2 (α1)	2- 20 mcg/kg/min	↑↑	↓↓	Not a vasopressor. Inotrope with a vasodilation. The net effect = ↑CO + ↓SVR, may not ↓ BP. Use: HF, cardiogenic.
Milrinone	PDE inh	0.375 – 0.75 mcg/kg/min	↑↑	↓↓↓	Similar to dobutamine more vasodilator, ↓PA Use: HF, cardiogenic.
Isoproterenol	β1 β2	2-10 mcg/min	↑	↓↓↓	Prominent chronotropic. Prominent vasodilation. Use: Bradycardia
Phenylephrine	α1	0.5-10 mcg/kg/min	0	↑↑↑	Pure vasoconstriction. May decrease SV.
Vasopressin	V_1	0.04 unit/min	0	↑↑↑	Pure vasoconstriction. Use: 2nd -line in refractory vasodilatory shock. . S/E: coronary, mesenteric ischemia, skin necrosis. ↓Na and pulm vasoconstriction

Pressure volume Loop of the LV



A: MV close
B: AV open
C: AV close
D: MV open

Ees: end-systolic elastance (ESPVR: End systolic PV Relationship)

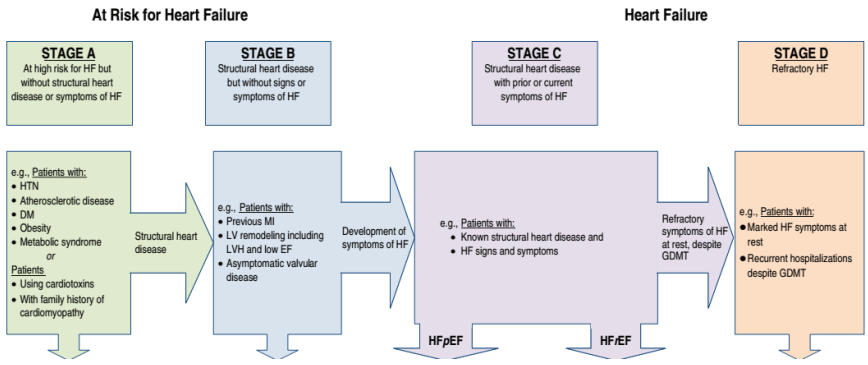
Ea: Arterial elastance

EDPVR: End diastolic PV Relationship

Cause of cardiogenic shock

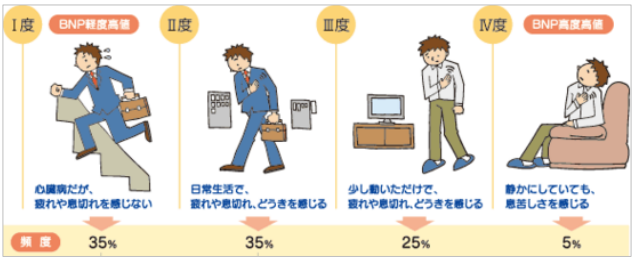
Etiologies of Cardiogenic Shock or Pulmonary Edema
Acute myocardial infarction/ischemia
LV failure
VSR
Papillary muscle/chordal rupture—severe MR
Ventricular free wall rupture with subacute tamponade
Post-cardiac arrest
Post-cardiotomy
Refractory sustained tachyarrhythmias
Acute fulminant myocarditis
End-stage cardiomyopathy
Left ventricular apical ballooning
Takotsubo's cardiomyopathy
Hypertrophic cardiomyopathy with severe outflow obstruction
Aortic dissection with aortic insufficiency or tamponade
Pulmonary embolus
Severe valvular heart disease
Critical aortic or mitral stenosis
Acute severe aortic or MR
Toxic-metabolic
Beta-blocker or calcium channel antagonist overdose
Other Etiologies of Cardiogenic Shock[b]
RV failure due to:
Acute myocardial infarction
Acute coronary pulmonale
Refractory sustained bradyarrhythmias
Pericardial tamponade
Toxic/metabolic
Severe acidosis, severe hypoxemia

Stage of HF



S&S of HF

- Non-specific
- Dyspnea from increased breathing drive
  - Reduction in exercise capacity (NYHA II-IV)
  - Orthopnea, PND
- Wt gain, leg swollen, fatigue, early satiety, N/V, confusion
- Apical shift, S3, S4, ↑ JVP, (+) HJR, crepitations, ascites, edema



Hypoperfusion: Autonomic responses

- Sympathetic nervous system
  - Baroreceptor, adrenal gland
  - NE, epinephrine, dopamine, and cortisol release
  - Vasoconstriction, ↑ HR, ↑ contractility, ↑ BP
- Renin-angiotensin-aldosterone system
  - Water and Na absorption, vasoconstriction
  - ↑ blood volume and ↑ BP
- ADH (vasopressin), cortisol,

Sample

- A 55 yo M with hx of HTN, DM presents with "crushing" substernal CP, diaphoresis, hypotension, tachycardia and cool, clammy extremities
- An 81 yo F from a nursing home presents to the ED with altered mental status. She is febrile to 39.4, hypotensive with a widened pulse pressure, tachycardic, with warm extremities
- A 68 yo M with hx of HTN and DM presents to the ER with abrupt onset of diffuse abdominal pain with radiation to his low back. The pt is hypotensive, tachycardic, afebrile, with cool but dry skin

Pathophysiology