Myocardial Infarction - heart muscle cell death resulting from disruption of blood flow in a coronary artery accompanied by:

- crushing chest pain
- \( \uparrow \) BP initially, followed by an \( \uparrow \) BP resulting from activation of the sympathetic nervous system that serves to \( \uparrow \) SV, HR, arterial vasoconstriction

- 53 heart sounds resulting from abnormal blood flow through the heart chambers sound like flutters or rumblings
- Arrhythmias
- Pericardial friction rub resulting from rubbing of the heart wall against the pericardial sac.
- Presence of cardiac enzymes in the blood, released from dying heart muscle cells.
  - Creatine Kinase (CK)
  - Lactate dehydrogenase (LDH)

Blood levels can be used to clinically assess the extent of heart muscle damage.

2 primary coronary arteries:

- Right Coronary Artery (RCA) - supplies blood to the muscle of the right side of the heart (RH, RV, SA node, AV node), muscle of posterior side of the heart, and posterior part of the interventricular septum.
Left coronary Artery (LCA) supplies blood to muscle of the left side of the heart (LA, LV) and interior part of the interventricular septum.
MI's are named and described relative to the area of the LV they affect.

Fig. 23-21 Site of myocardial infarction (MI) and vessel involvement. Ao, Aorta; PA, pulmonary artery; PV, pulmonary vein; LV, left ventricle; RV, right ventricle; IVC, inferior vena cava; RA, right atrium; SVC, superior vena cava; LA, left atrium. (Modified from Stevens, A. & Lowe, J. [1995]. Pathology. St. Louis: Mosby.)
Treatment for MI involves limiting the damage to the heart muscle achieved by:

- Administration of O₂ to increase O₂ content in the blood that may get through the occlusion.
- Aspirin is given to prevent further occlusion of the coronary artery by reducing platelet adhesion.
- Administration of thrombolytic drugs (ex. streptokinase or tissue plasminogen activator) to break up blood clots.
- Heparin may be given to prevent further clot formation.
Nitroglycerin is given in a sublingually placed capsule. Acts as a potent, fast-acting, vasodilator.

Acts to:
- reduce peripheral arterial resistance, reducing workload on the heart + O2 the heart muscle needs, resulting in minimizing damage to the heart muscle
- Also causes vasodilation of coronary arteries → increasing blood flow to the heart muscle, minimizing heart muscle damage

Β blockers + ACE inhibitors may be given to promote vasodilation and the β blockers will also reduce
the response of the heart to the increase in sympathetic activity.

- Plasma or isotonic saline are given as an IV to keep BPP and prevent cardiogenic shock.

Once the patient is stabilized, then a percutaneous transluminal coronary angioplasty (s.t.k. "Balloon Angioplasty") can be performed.
Catheter is inserted into a peripheral artery (usually the femoral artery).

Catheter with uninflated balloon

Catheter with inflated balloon

Infused balloon pushes blockage open

**Ballooon Angioplasty**

30-40% of the time re-occlusion of the artery will occur within 6 months. Stents can be used to improve the success rate.
In cases where balloon angioplasty is not successful, coronary artery bypass surgery is necessary.
B. View of heart after bypass surgery

- Vein grafts
- Left subclavian artery
- Left internal mammary artery
- Graft to artery beyond blockage
Pericarditis - inflammation of the pericardial sac

Caused by:
- MI
- Autoimmune disease
- Heart surgery
- Most common cause is infection of the pericardial sac by a bacteria or virus

Characteristic signs:
- Sharp, stabbing chest pain that may radiate to the back
- Pericardial friction rub
- Pericardial effusion resulting in a buildup of fluid inside the pericardial sac → cardiac tamponade - squeezing down on
The heart preventing normal filling of the heart chambers with blood. → \( VCO \) → cardiogenic shock + death

Constrictive pericarditis - repeated inflammation of the pericardial sac causes buildup of scar tissue that is not as compliant as normal pericardial sac tissue preventing stretching of the pericardial sac as the heart fills with blood and expands → \( VCO \)

Treatment depends on cause

- If due to infection
  Antibiotics are given to fight the infection + aspirin to relieve the pain
If tamponade occurs then pericardiocentesis can be performed to drain fluid from pericardial sac.

For constrictive pericarditis cardiac glycosides (ex. digitalis) are given to increase cardiac contractility (force of contraction) which increases SV $\rightarrow$ $\text{A CO}$.

If the constriction is severe, then the scar tissue can be surgically removed.
Heart Value Dysfunctions

4 heart valves

Tricuspid — between RA + RV

Bicuspid (mitral) — between LA + LV

Pulmonary — between pulmonary trunk + RV

Aortic — between aorta + LV

These values prevent the backflow of blood in the heart

Heart valve dysfunctions are most commonly the result of inflammation of the valve due to rheumatic heart disease or infectious endocarditis.
Rheumatic heart disease (RHD) is typically triggered by a streptococcal throat infection.

The strep bacteria release cell membrane antigens (CMA) that can bind to mural tissues, muscle tissues, synovial joint tissues, and heart valve tissues.

Immune response is initiated against the tissue that has the CMA bound to it → damage to the tissue.

Infectious endocarditis — inflammation of the lining of the heart chambers
most commonly caused by infection of the endocardium by strep or staphylococcal bacteria → damage to heart valves

Two types of heart valve dysfunctions:

- **Heart valve stenosis** → value doesn't open all the way resulting in blocking flow of blood out of the chamber

- **Heart valve regurgitation** → value doesn't close all the way allowing blood to flow back into the chamber it was pushed out of.
In a stenosis, the chamber that the blood is being pushed out of has to work harder to overcome the increased resistance created by the narrowed valve orifice → hyper trophy of the muscle of the heart chamber

Ex Aortic stenosis
Aortic valve doesn't fully open when LV contracts

↓

To maintain normal CO & BP
Sympathetic nervous sys. stimulates the LV to contract with greater force

↓

Increased workload causes the muscle of the LV to hypertrophy
- The hypertrophied muscle needs more O2
- The hypertrophied muscle doesn't contract as efficiently
- The hypertrophied muscle doesn't stretch as easily (less compliant)

In regurgitation, the failure of the valve to close allows blood to leak back into the ventricle. This increases the volume of blood the chamber has to pump per beat. Thus, the chamber expands to accommodate increased volume of blood. Also, the heart muscle of the chamber hypertrophies as a result.
of the increased workload

Ex. Aortic regurgitation

Some of the blood the LV ejects when it contracts leaks back into the LV from the aorta.

\[\downarrow\]

Results in volume overload of the LV (LV receives blood from LA + aorta).

\[\downarrow\]

LV dilates to accommodate the volume overload.

\[\downarrow\]

has to contract with greater force to maintain a normal CO + BP.

Muscle of LV hypertrophies due to
Increased workload.

Value dysfunctions are normally initially diagnosed using cardiac auscultation. Followed by echocardiography.

Treatment of value dysfunctions:

Value dysfunctions worsen over time so eventually the heart will fail unless surgery is done to repair or replace the valve.

Heart failure—failure of the heart as a pump results in inadequate perfusion of the tissues with the O2 and nutrients needed.
Can be caused by any condition that affects the heart's ability to supply O2 and nutrients to the tissues:

- Hypertension
- MI
- Valve dysfunction
- Diseases of the heart muscle (myocarditis)

Congestive heart failure (CHF) is most common type.

CHF is the failure of the left side of the heart

Divided into two categories:
- Systolic heart failure
Inability of the heart to generate adequate CO as a result of decreased left ventricular contractility → Ejection fraction (fraction of blood ejected from the LV per beat, normal 60-70% of blood received from LA)

In early stages body compensates

↓ CO

Stimulates sympathetic nervous system

Stimulates RAAS system

↓ BP

↓ Natriuresis in kidneys

↓ blood osmolality

↓ arterial vasocostriction

↑ heart contractility

↑ CO

↑ BP

↑ fluid intake

↑ GADH

↑ arterial vasocostriction

↓ Net reabsorption in blood
Eventually these compensatory mechanisms fail resulting in the back up of blood from the LV into the pulmonary circulation.

Diastolic heart failure—

results from a decrease in compliance of the LV

LV isn't able to stretch to receive the normal blood volume from the LA

Again resulting in back up of blood into the pulmonary circulation.
Principle sign of CHF is pulmonary edema disrupting normal gas exchange in the lungs.

Pressure generated by heart moves blood through the vessels, and is responsible for movement of fluid + materials between blood in the capillaries + tissues.
**Fig. 15-18  Fluid exchange at the capillary**

\[ \pi = \text{Colloid osmotic pressure} \]

\[ P_H = \text{Capillary hydraulic pressure} \]

**Arterial end**

Net movement of fluid + solutes out

Net filtration

\[ P_H > \pi \]

\[ \pi > P_H \]

**Venous end**

Net absorption

Net flow out = 3 L/day

Net movement of fluid + solutes in

Results from greater conc. of blood than interstitial fluid, created by pressure of blood pushing on walls of capillary.
In CHF, the back up of blood into the pulmonary cire. raises the hydraulic pressure on the venous end of the capillaries.
So, less fluid & solutes move back into capillaries on the venous end.
Results in accumulation of fluid in the lung tissue. → reduced gas exchange in the lungs.
Causing:
— Dyspnea
— Cyanosis (bluish color of skin due to low content of blood)
— Acidosis (due to accumulation of CO₂ in the blood)
Treatment for CHF

Directed at helping the heart compensate.

- Diuretics to reduce pulmonary edema

- Inotropic drugs
  ex. digoxin
  to increase cardiac contractility

- Vasodilators
  ex. ACE inhibitors
  to reduce peripheral arterial resistance

- If necessary mechanical support for the heart
  Ex. Ventricular Assist Device
New Hope For Advanced-Stage Heart Failure Patients
The HeartMate II Left Ventricular Assist System (LVAS) is a small, quiet, next-generation blood pump shown to restore hemodynamic function and improve patient outcomes and quality of life.\textsuperscript{1-7} Through the rotary action of a single-moving part, the HeartMate II LVAS can provide long-term circulatory support\textsuperscript{3} by pumping blood from the heart to the body at up to 10 liters per minute.

The HeartMate II received CE Mark approval in November, 2005 based on the results of its Phase I clinical trial.

HeartMate II LVAS animation with narration

Success Factors in Hemodynamic Restoration Therapy
Successful Hemodynamic Restoration Therapy (HdRT) with the HeartMate II LVAS hinges upon six interdependent factors:

1. **Positive Clinical Outcomes** including low thrombosis, low hemolysis, and low infection.\textsuperscript{1,2,4,5,7}

2. **Reliability**\textsuperscript{8} made possible by precision engineering such as advanced, patented bearing design, a single-moving part, and back-up controller electronics.

3. **Ease of Implantation** facilitates treatment of a broad patient population, and a small (400 gm) device allows small pocket and shorter surgical times.\textsuperscript{9}

4. **Improved Patient Quality of Life** and functional class allows patients to return to daily life at home.\textsuperscript{6,7,9} Patients typically do not feel or perceive the VAD's presence due to its small size and light weight.\textsuperscript{6}

5. **Ease of Patient Management** facilitated by a thin, flexible driveline designed to reduce infections, simple user interface on peripherals, and standardized equipment shared with the HeartMate XVE LVAS.

6. **Thoratec Support** offers unrivaled education and training with exceptional clinical, technical, and reimbursement assistance.

The HeartMate II LVAS is implanted in the chest to support the pumping function of the heart. An external, belt-worn System Controller and battery are attached to the implanted pump via a thin, flexible, percutaneous cable.