

The Heart

The Heart

The heart is a fist size pump that drives the blood in the arteries and veins throughout the body

It is somewhat conical in shape

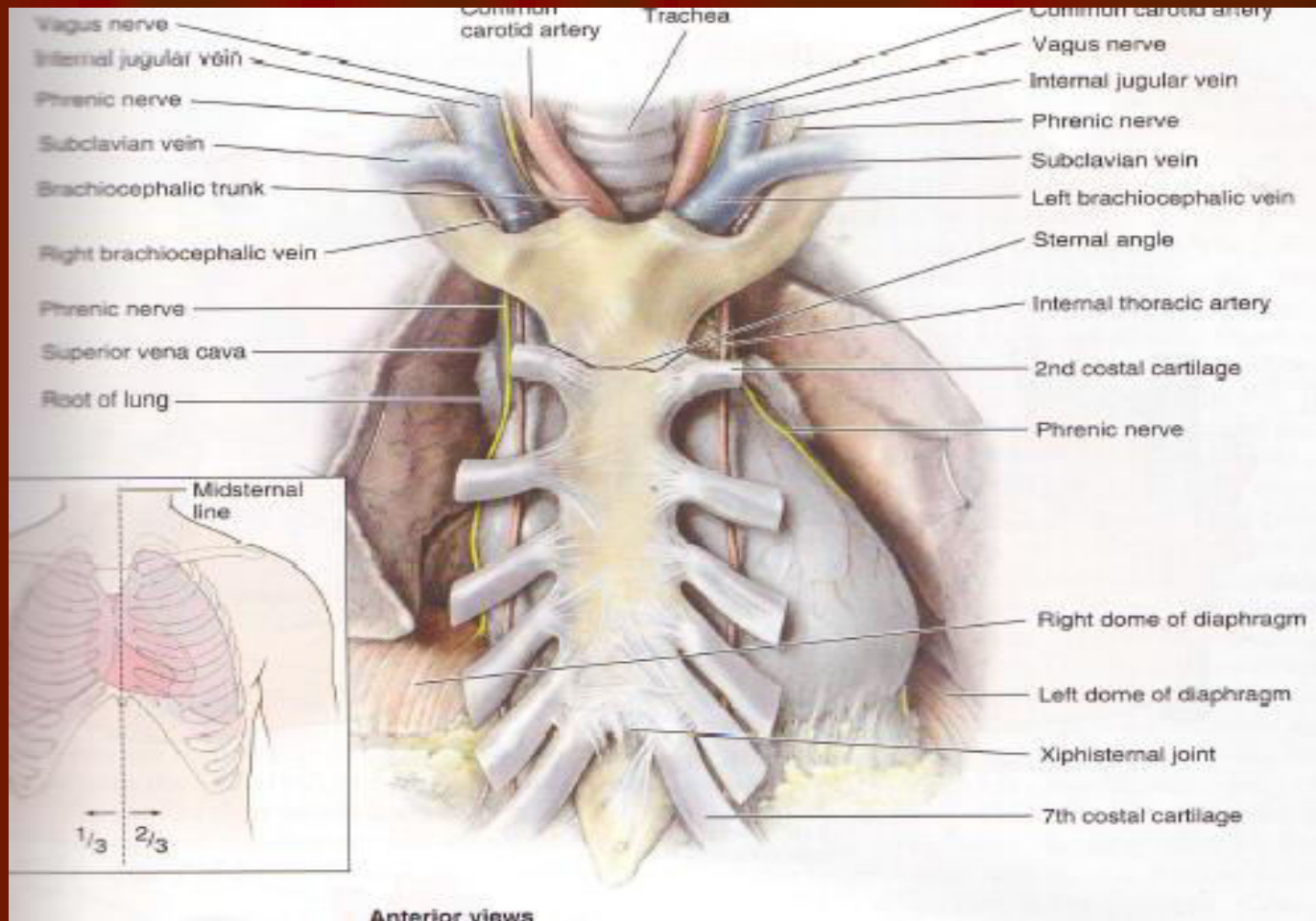
Its "base" lies upward and posteriorly, is made largely by the atria

Its "apex" is made by the tip of the left ventricle

It rests on the central tendon of the diaphragm

It is kept in its place by its pericardial attachments and the great vessels that enter into and emanate from its chambers

It weighs about 300 grams



Location and general anatomy of the heart

The Heart

The heart is made of three layers

Pericardium

Fibrous outermost

Parietal, adherent to the fibrous layer

Epicardium (visceral), envelops the muscle layer and adherent to it

Accumulation of blood or fluid in the pericardial sac can restrict cardiac filling and subsequently cardiac output (cardiac tamponade)

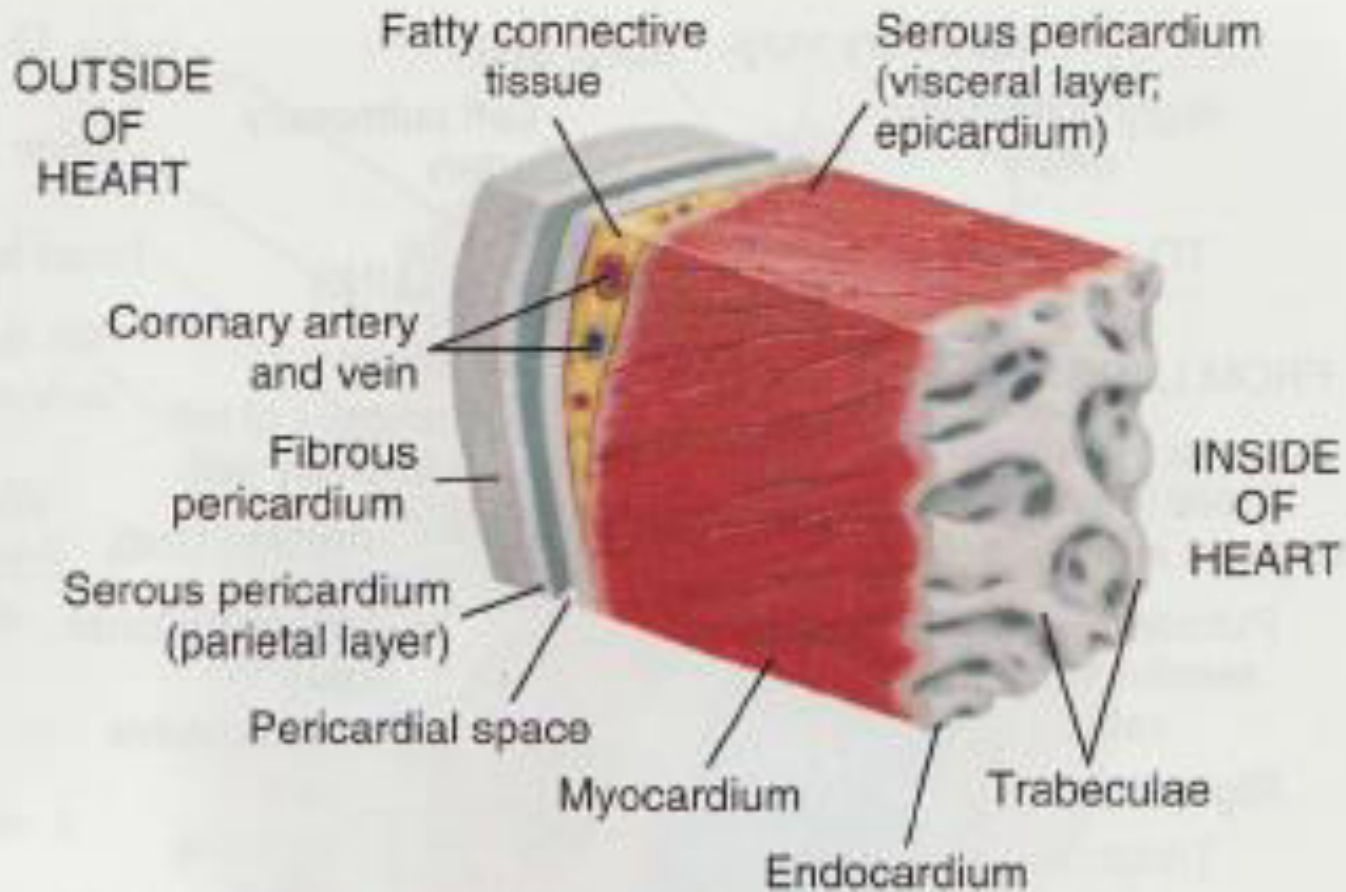
Myocardium

The contractile layer responsible for the pumping action

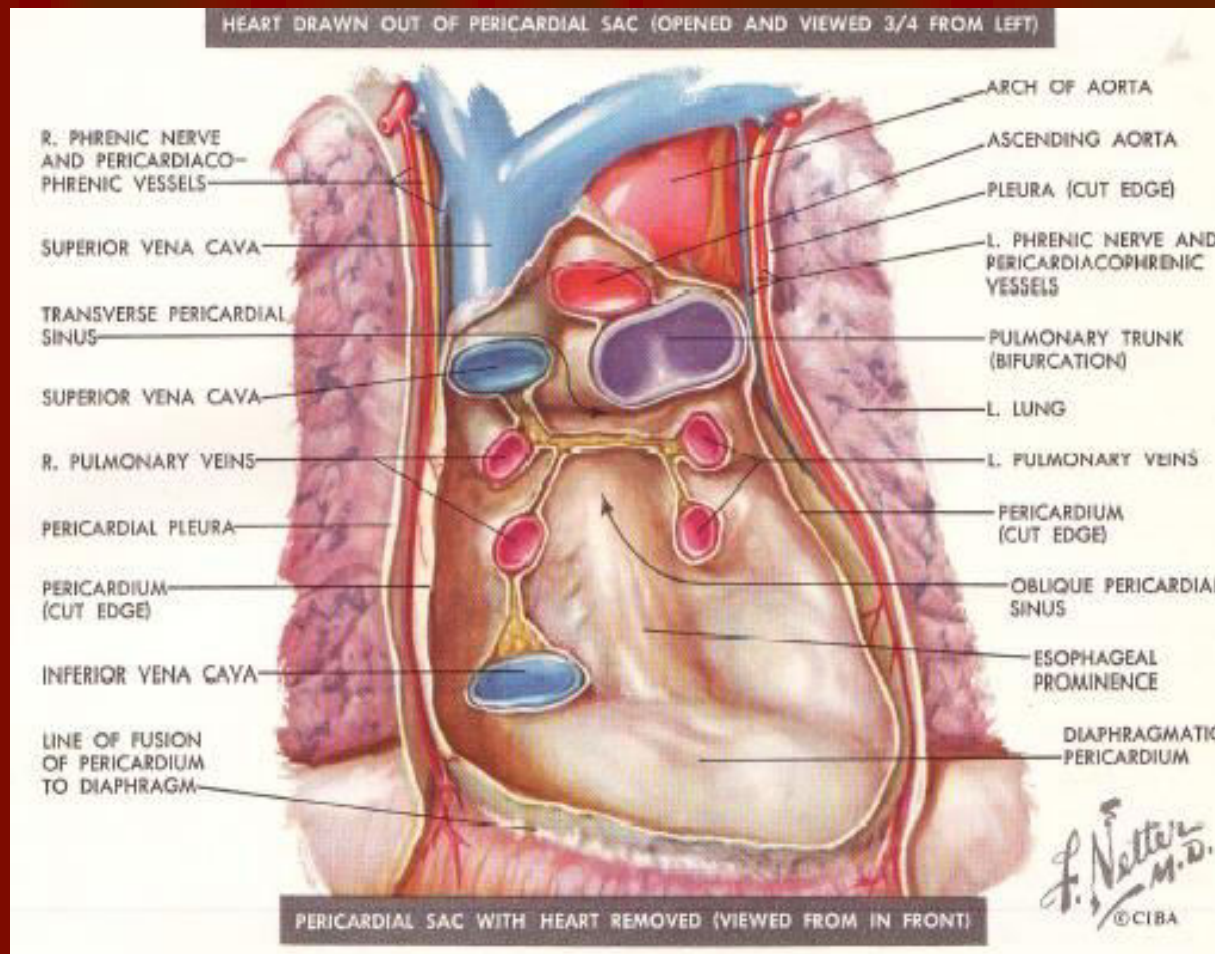
Endocardium

The inner lining of the cavities, extends to form the “valves”

A fibrous skeleton separates the atria from the ventricles and provides attachment to the cardiac muscle



Structure of the wall of the heart



The pericardium and the great vessels

The Heart

The Pericardium

It functions as a protecting layer around the heart

It contains a minimal amount of serous fluid that facilitates and lubricates the cardiac contraction

It helps anchoring the heart in place

It prevents the sudden distension of the heart chambers

The Heart

Gross Anatomy and Function

Two large veins collect the blood (venous return) from the body and pour it into the right atrium (RA)

The superior vena cava (SVC) drains the blood from the head and neck

The inferior vena cava (IVC) collects the blood from the rest of the body

The RV pumps the blood to the lungs for gas exchange

Each lung sends its oxygenated blood to the left atrium (LA) through a pair of pulmonary veins (a total of 4)

There are no valves between the left atrium and the pulmonary capillaries

Therefore pulmonary capillary pressure reflects left atrial pressure

The LA sends the blood to the LV, and the LV pumps it into the rest of the body through the aorta

The Heart

Gross Anatomy and Function

The heart is made up of four cavities (chambers)

Two small chambers: right atrium (RA) and left atrium (LA), lie posterior and superior to two larger ones, the ventricles

The two atria are separated by a dividing interatrial "septum" (IAS)

Each atrium has an ear like appendage (auricle) that protrudes toward the corresponding great vessel

The atria form the "base" of the heart

The atria are "receiving" chambers

The ventricles are the pumping chambers

The atria normally contribute about 15% - 20% of the cardiac output

The Heart

Gross Anatomy and Function

Two large chambers: right and left ventricles (RV & LV) are separated by an interventricular septum (IVS)

The ventricles lie below the atria

The tip of the left ventricle forms the "apex" of the heart

The ventricles are pumping chambers, therefore they are thicker walled

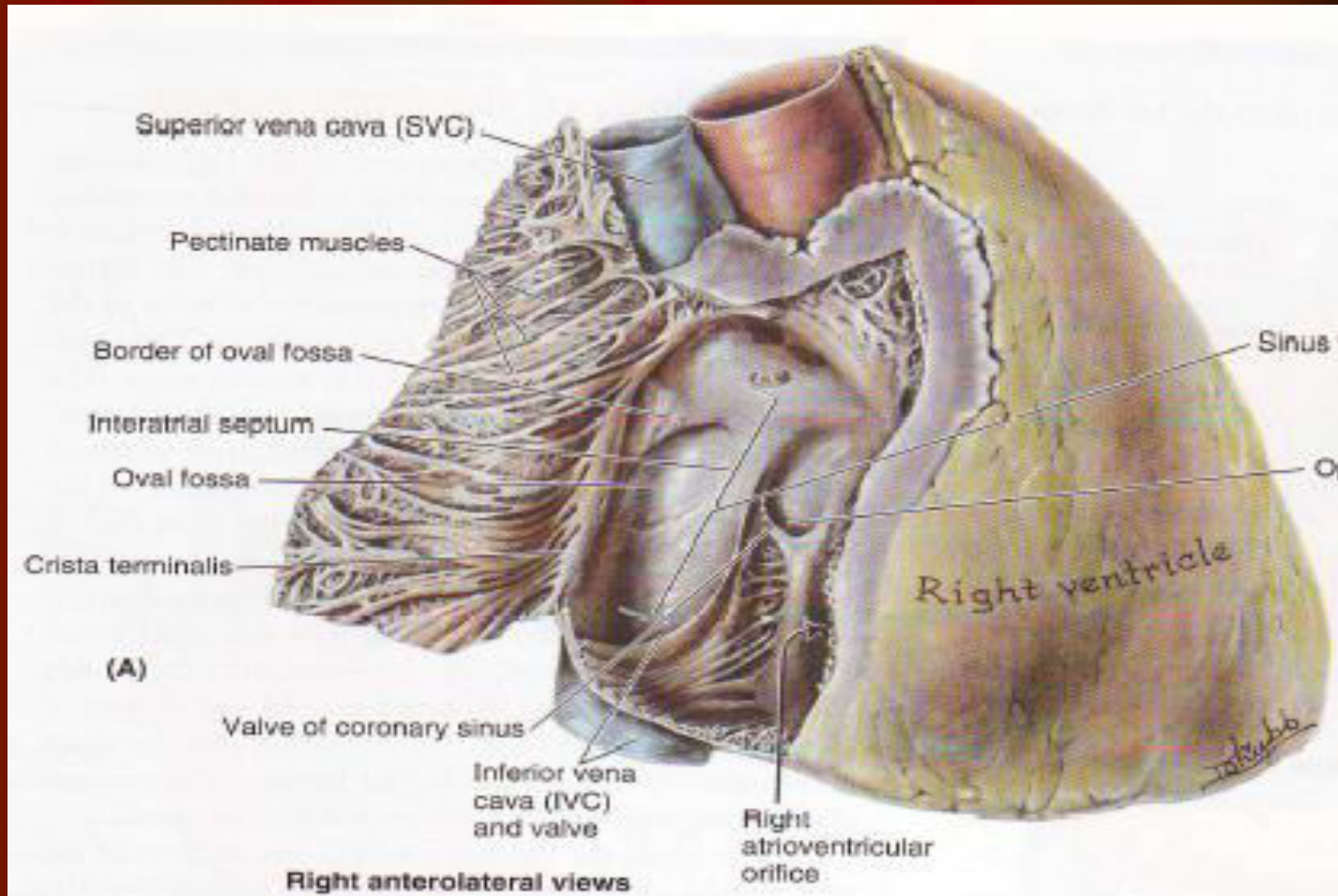
The left ventricle is thicker than the right

The right atrium and ventricle are separated by an endocardial reflection, a "valve", made of three leaf like structures, the tricuspid valve (TV)

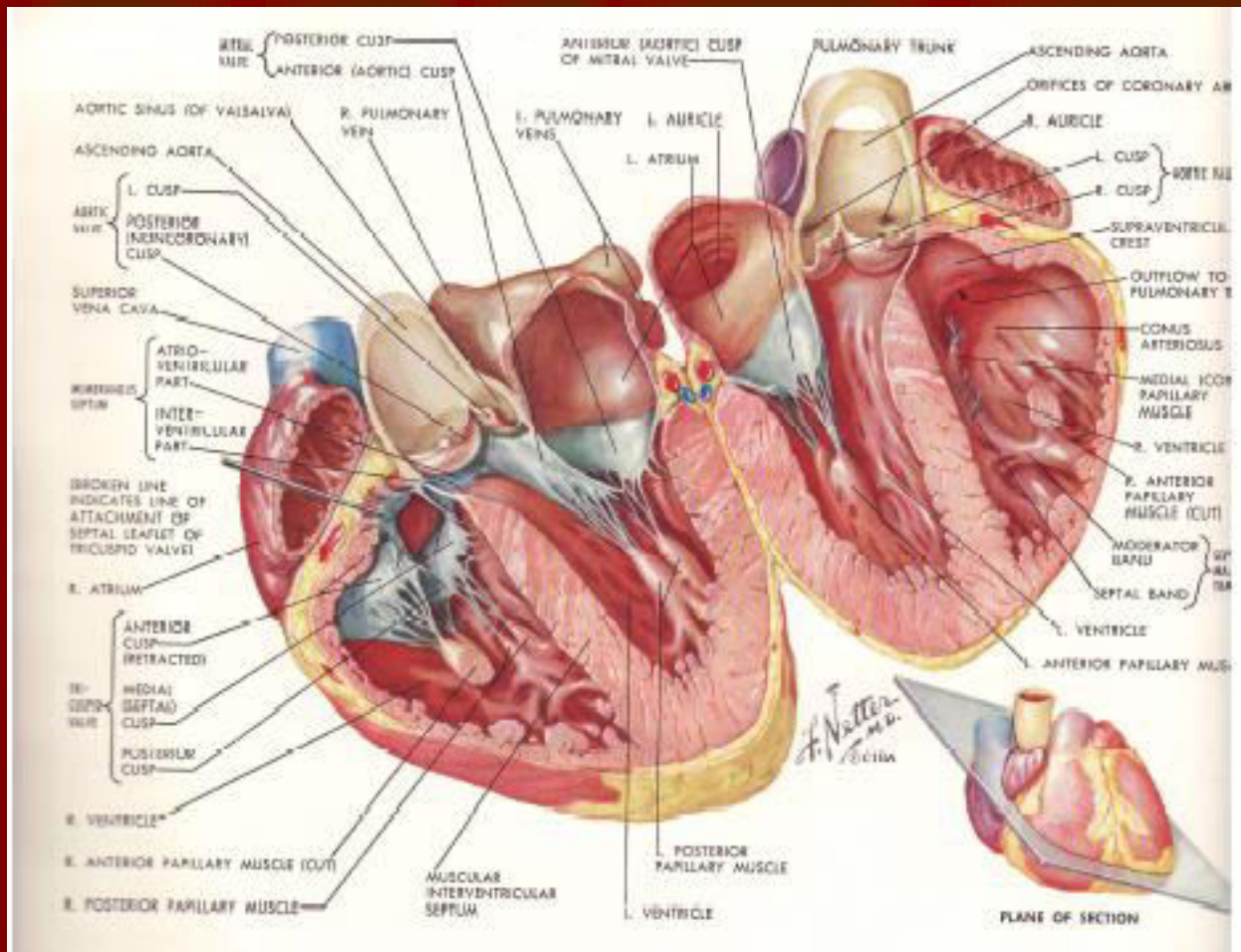
The left atrium and ventricle are separated by a valve made of two leaflets, the mitral valve (MV)

The AV valves are made of "leaflets" while the pulmonary and aortic valves are made of "cusps"

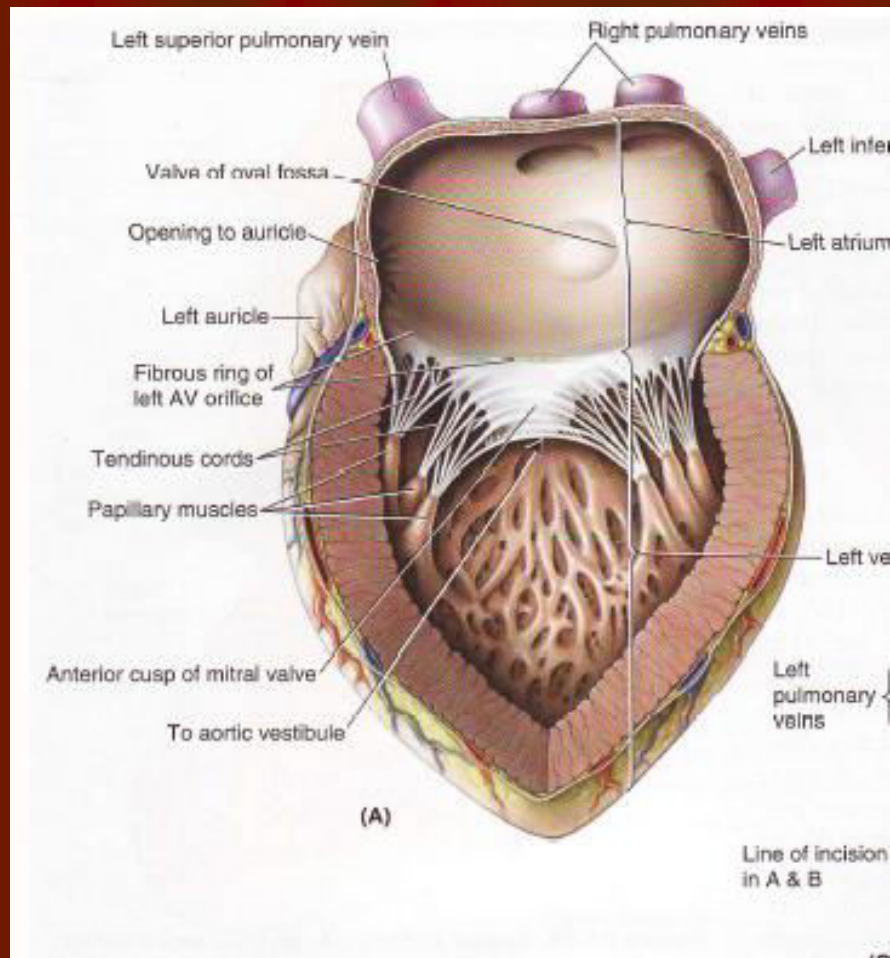
All the valves are attached to the cardiac skeleton



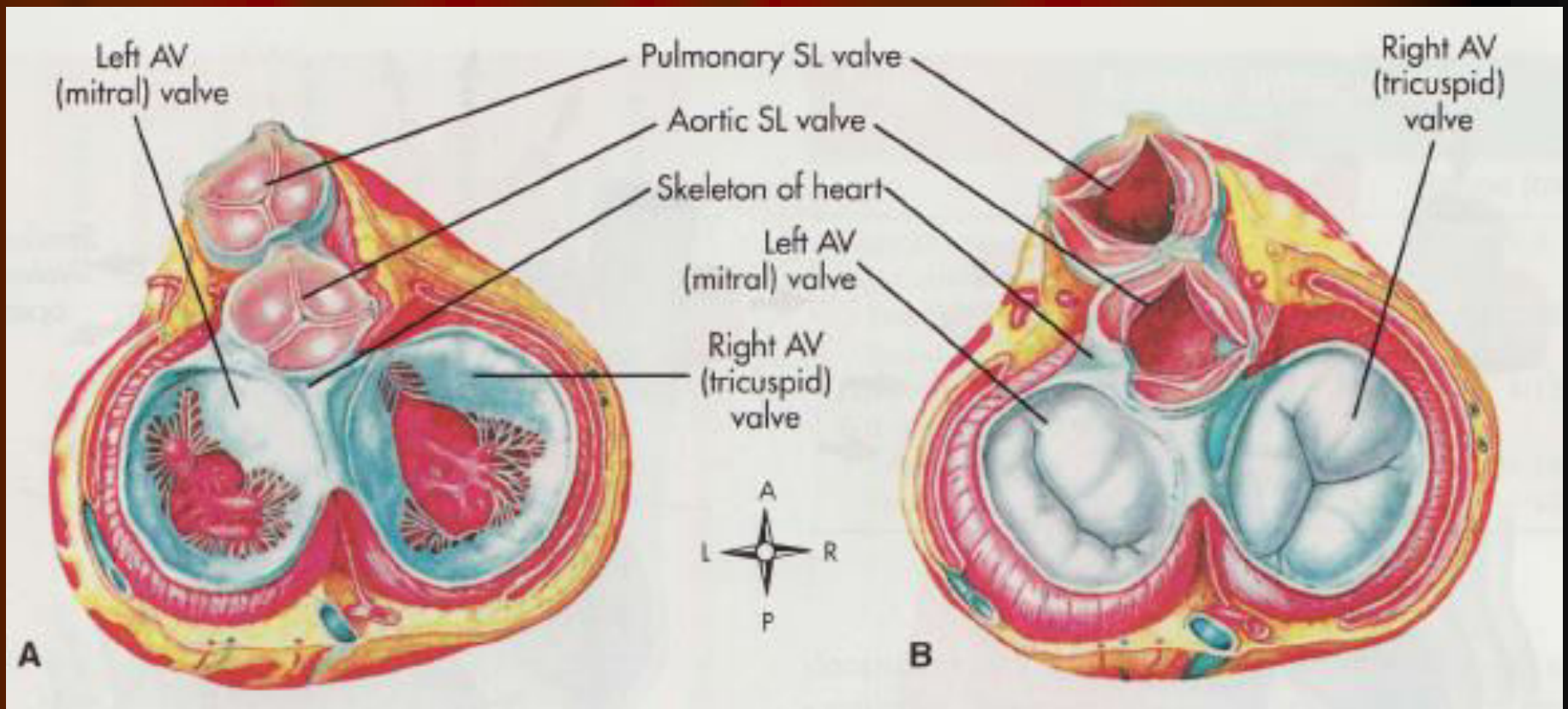
The right atrium



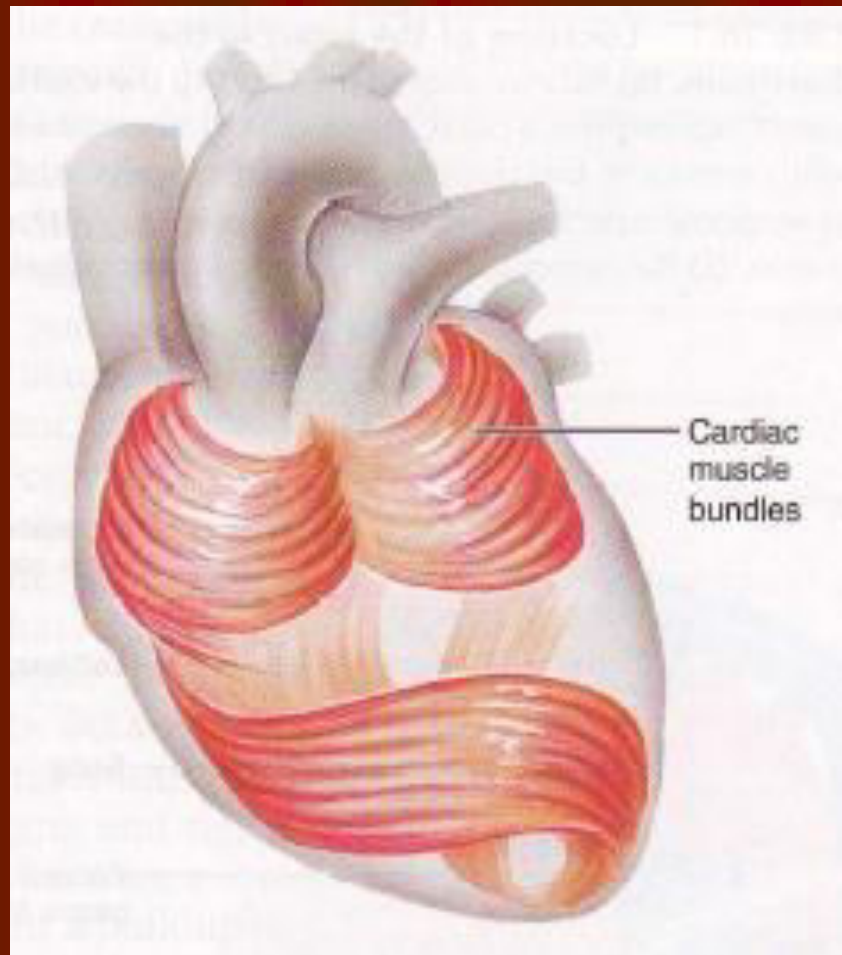
The cardiac chambers



The left atrium and ventricle



The heart valves during diastole (A) and systole (B)



The general arrangement of the cardiac muscle

The Heart

The Circulation

Blood is collected by the SVC and IVC and delivered to the RA

The RA sends the blood through the TV to the RV

The RV pumps the blood through the PV and the PA to the lungs

Gas exchange takes place in the lungs

The lungs send the oxygenated blood to the LA through 4 pulmonary veins, two for each lung

The LA delivers the blood through the MV to the LV

The LV pumps the blood through the AV into the AO to the rest of the body, including the heart muscle

The Heart

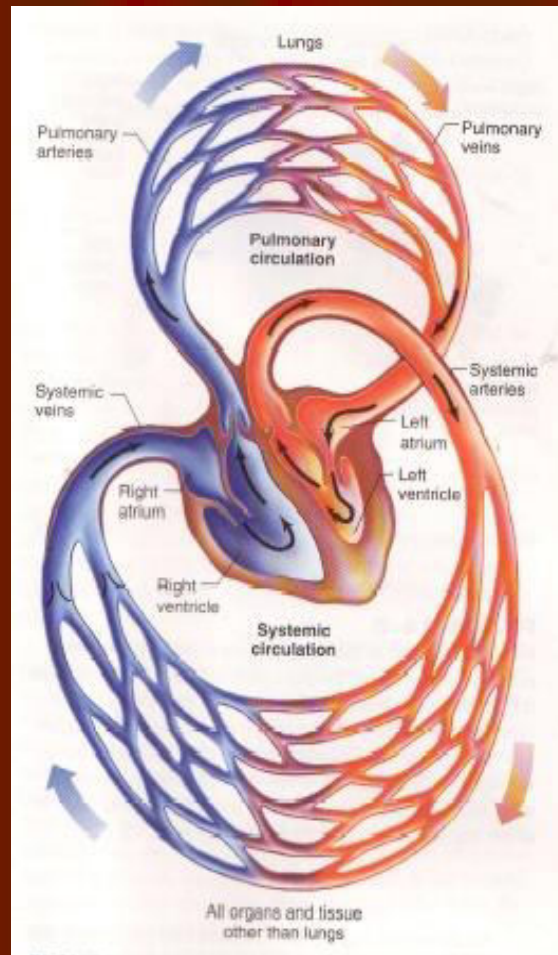
Gross Anatomy and Function

The right ventricle pumps the blood to the lungs through the pulmonary artery (PA)

A valve at the root of the pulmonary artery, the pulmonary valve (PV) prevents the blood from dropping back (regurgitating) into the ventricle

The left ventricle pumps its blood to the rest of the body through the aorta (AO)

A valve at the root of the aorta, the aortic valve (AV) prevents regurgitation back into the left ventricle



Pulmonary and systemic circulation

The Heart

Gross Anatomy and Function

Myocardial contraction is called "systole"

After each contraction the chambers relax "diastole"

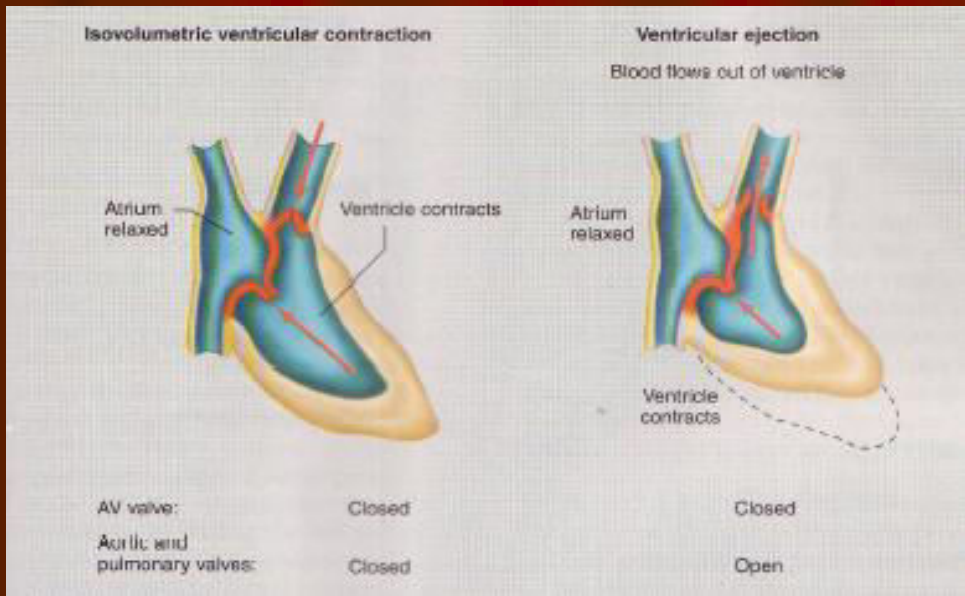
The atria contract and relax together and the ventricles do the same

At the time the atria contract the ventricles relax and vice versa

Atrial systole propels the blood from the atria to the ventricles

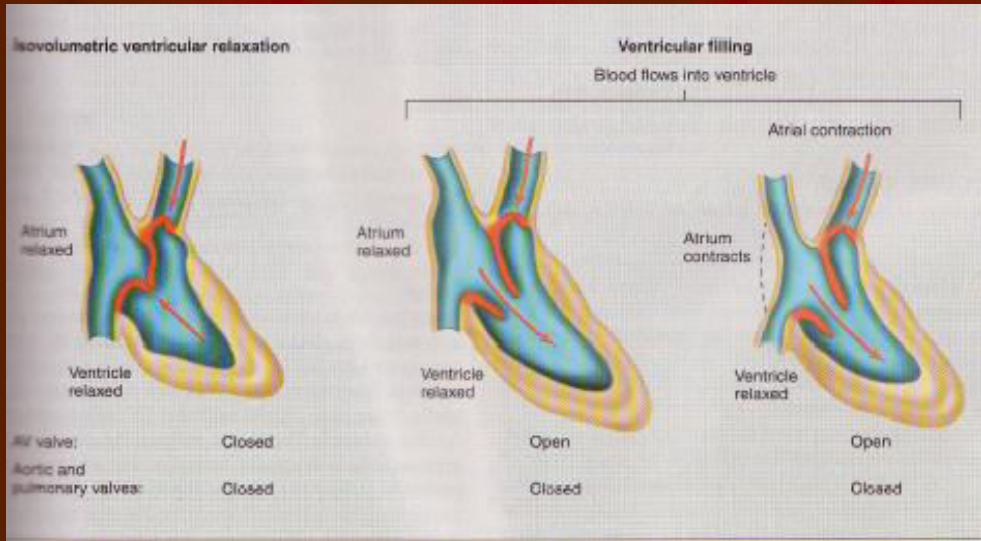
The atria then relax (go in "diastole") and the ventricles go into systole
sending the blood to the PA and the AO

Regurgitation of blood from ventricles to atria is prevented by the TV and
the MV



Systole

The cardiac cycle



Diastole

The Heart

Gross Anatomy and Function

The right ventricle can cope with volume sending it a short distance

The left ventricle copes better with pressure sending the blood to the rest of the body

The Heart

Gross Anatomy and Function

TV and MV competence is maintained by cord like structures (chordae tendineae)

These cords are attached on one side to the ventricular surface of the valve, and to the other side to the tips of nipple like protrusions of the ventricular myocardium (papillary muscles)

Papillary muscles contract during systole preventing the prolapse of the AV valves into the atria

The Heart

Gross Anatomy and Function

Atrial systole helps to propel the blood from the atria but is not essential for the adequate output of blood from the ventricles

Atrial systole contributes about 20% of the cardiac output (CO)

This contribution becomes important in cases of heart failure

The terms systole and diastole, when used without chamber designation, indicate ventricular contraction and relaxation

The Heart

Gross Anatomy and Function

The aortic and pulmonic valves are of the semilunar types

Aortic and pulmonic valve closure is affected by the fall of the blood column in the corresponding vessel during early diastole

This downward pressure forces the three components (cusps) of the valve to coapt preventing regurgitation into the ventricles

Ventricles do not eject all the blood they accumulate during diastole, the end diastolic volume (EDV)

The difference between EDV and the volume ejected during systole, the end systolic volume (ESV) is the "stroke volume" (SV)

Therefore $SV = EDV - ESV$

The ratio SV/EDV is normally about 60%

This is referred to as the "ejection fraction" (EF)

The Heart

The Myocardium

The cardiac muscle is striated, shorter and thicker than the skeletal muscle

Cardiac cells branch and are interlock at "intercalated discs"

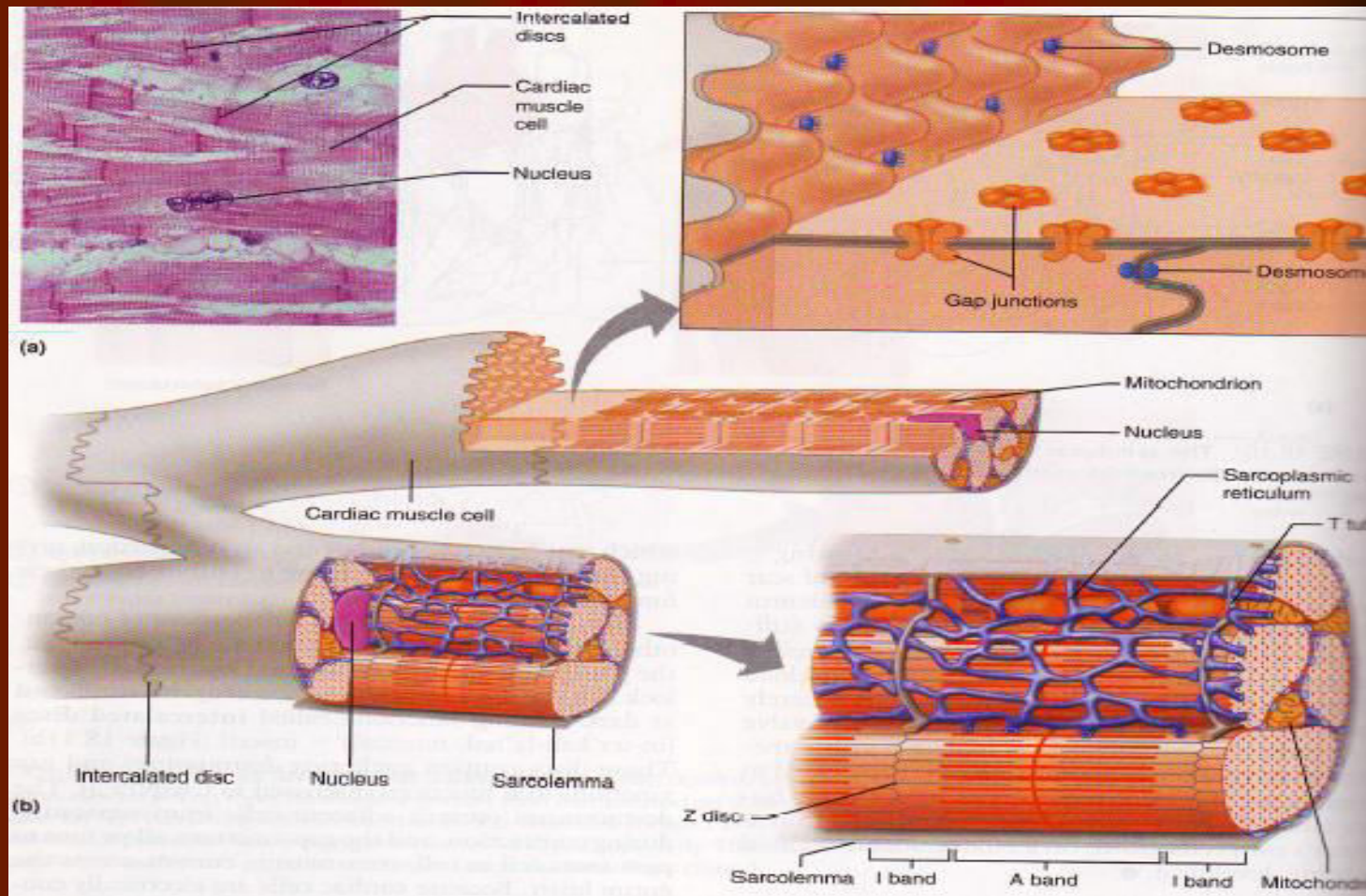
Each cell has pale central nucleus and large mitochondria

Loose connective tissue surrounds the muscle, it carries the blood supply and connects them to the fibrous skeleton that anchors the muscle

Dense bodies "desmosomes" in the intercalated discs hold the cells together during contraction

Gap junctions exist between cells to allow the passage of ions and the action potential

Cardiac muscle contracts and relaxes as a unit



Structure of the cardiac muscle

The Heart

The Myocardium

The contractile element of the muscle are fibres arranged in filaments

They are of two types

Thick fibres "myosin"

Thin fibers "actin"

The two types overlap longitudinally

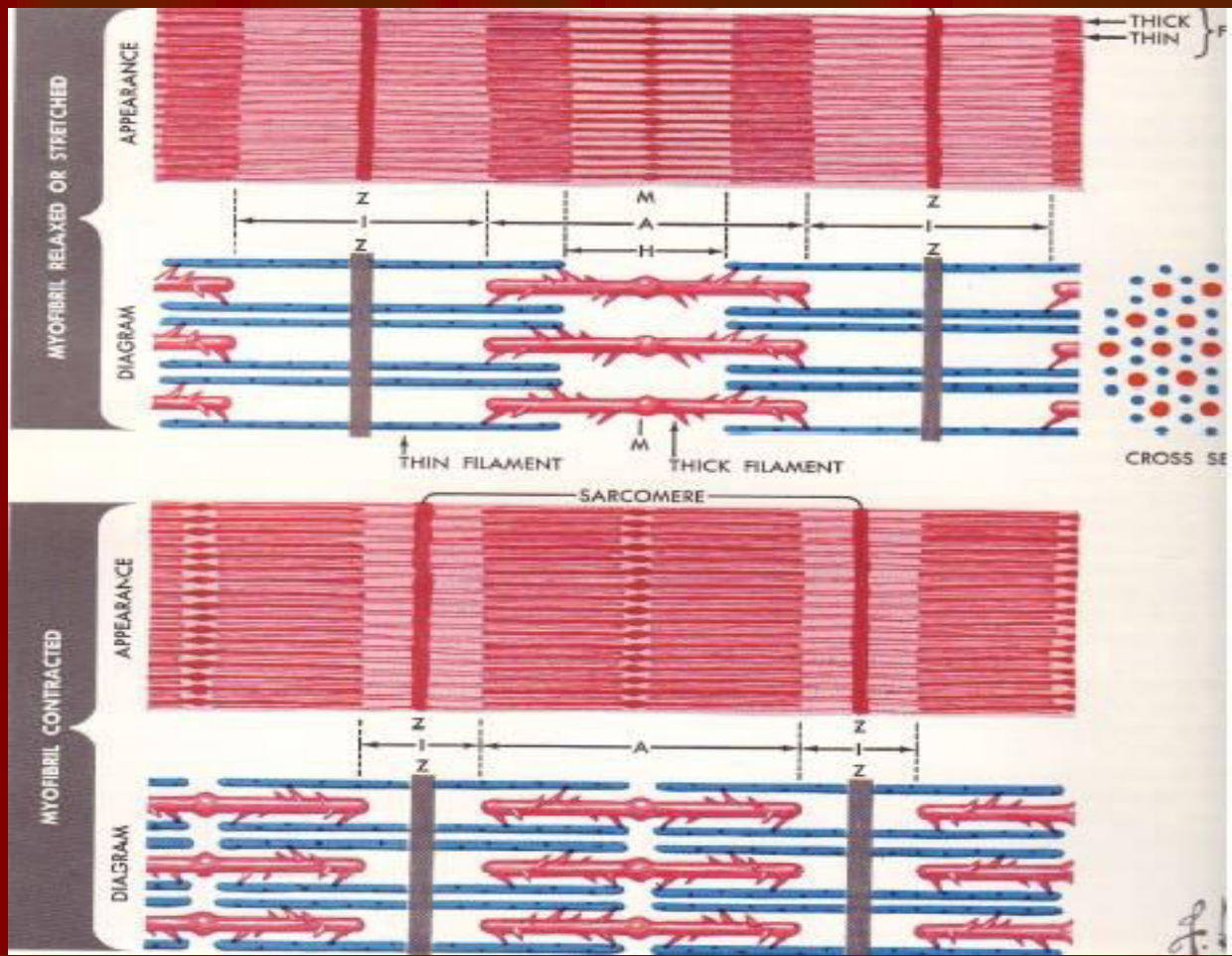
A bundle of filaments forms a "sarcomere"

The filaments are covered with cell membrane "sarcolemma"

The myocardium exhibit "banding" : Z, A, M, and I bands

Sarcomeres are surrounded by a network of channels, the sarcoplasmic reticulum

Sarcoplasmic reticulum is attached to invaginations of the sarcolemma (T tubes) that allow the transfer of Ca^{++} to the fibrils



The structure of the myocardium

The Heart

The Myocardium

Myosin filaments lie in the middle between Z bands

Actin filaments are made of

- Actin units

- Troponin

- Tropomyosin

Each myosin fiber is attached to several troponin molecules on every one of the actin fibers

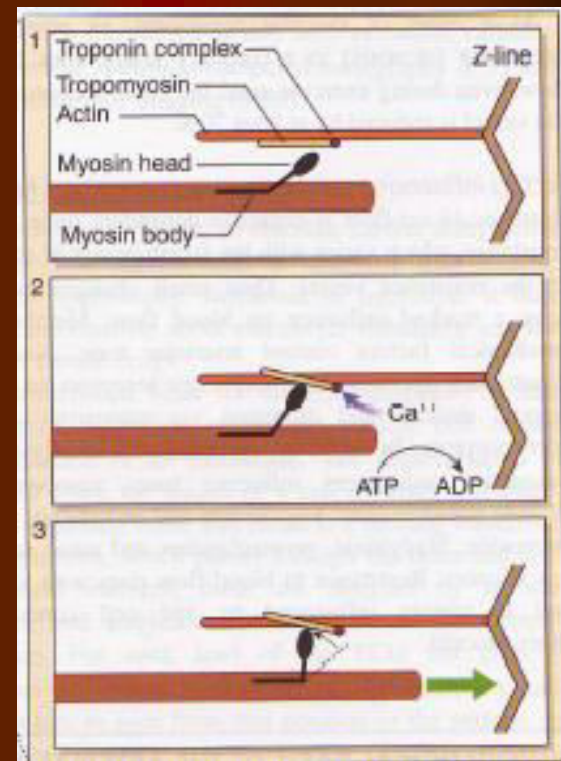
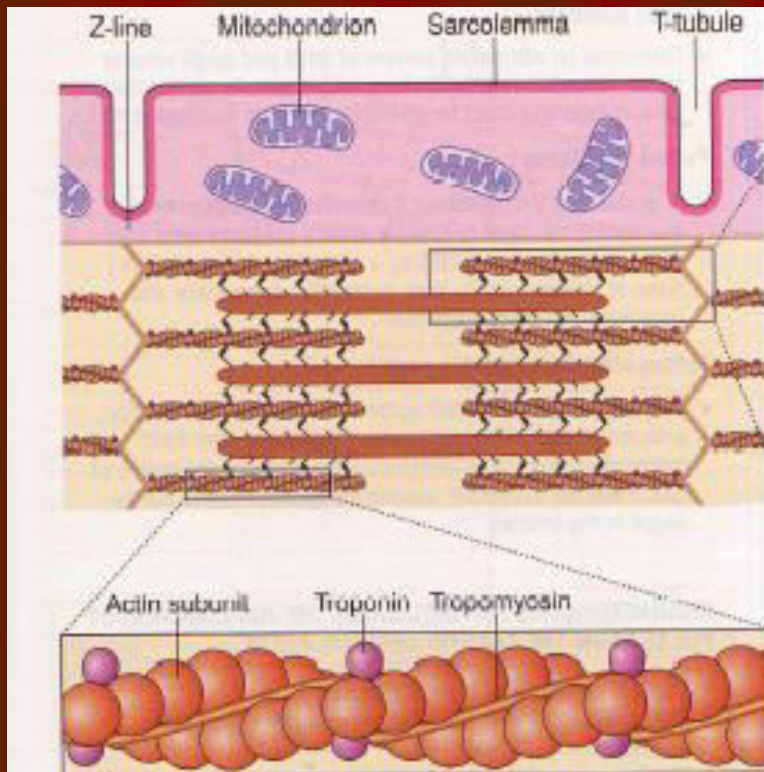
Ca⁺⁺ unblocks actin/myosin binding sites, myosin attaches to tropomyosin

Myosin head tilts pulling the Z lines closer

Each wave of depolarization is followed by an absolute refractory period during which no depolarization can take place

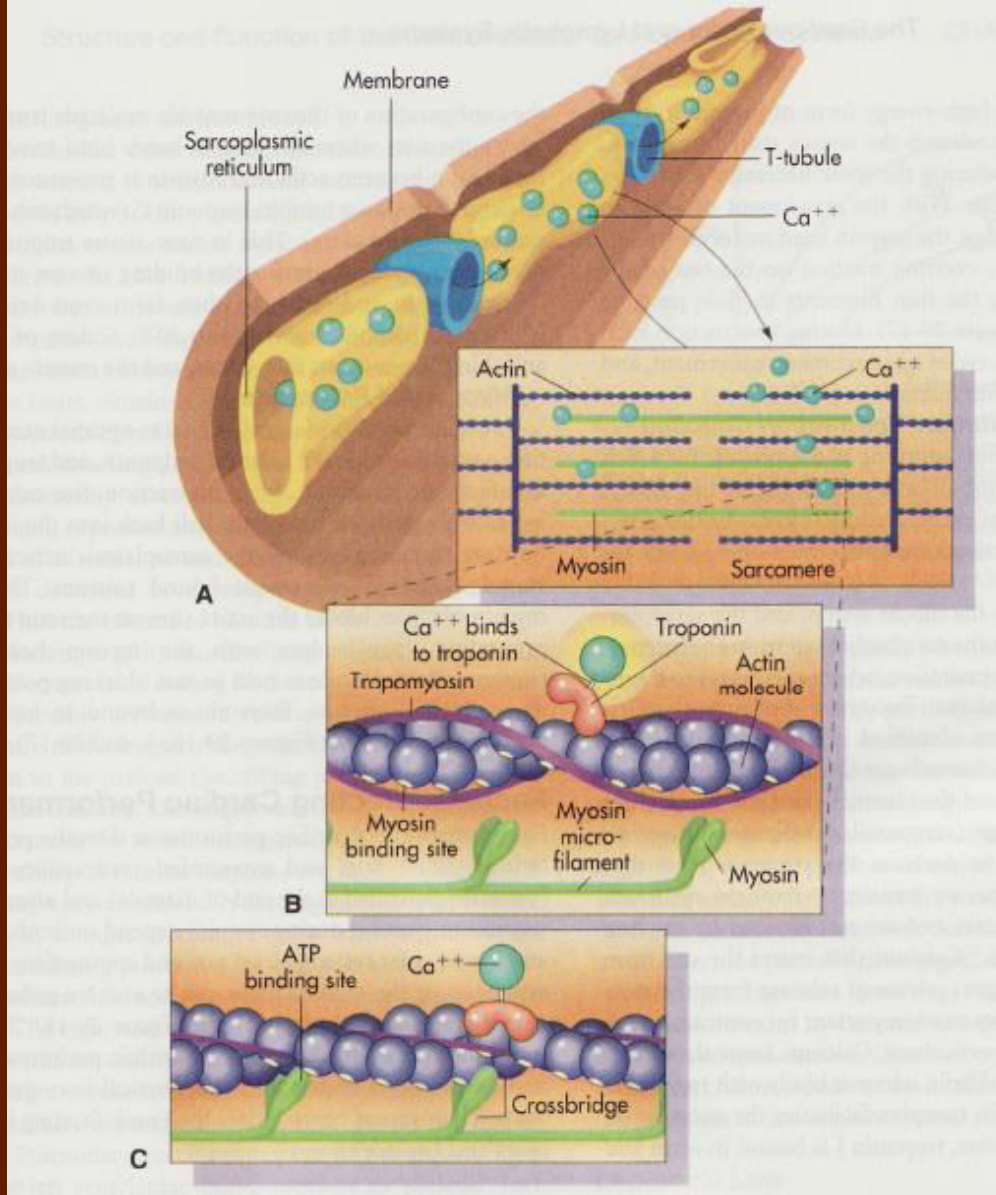
- The refractory period is equal to the length of cardiac muscle contraction

- This guards against tetanic contraction of the cardiac muscle

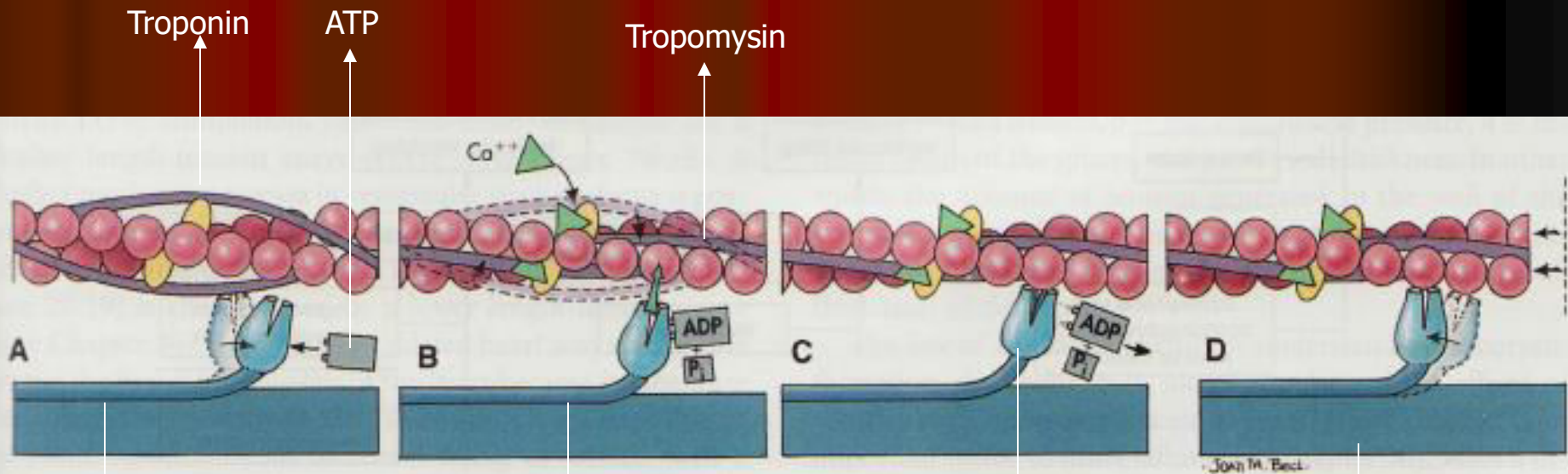


Myosin actin interaction, myocyte shortening

Following actin/myosin interaction, Ca^{++} uptake pumps remove Ca^{++} from the sarcoplasm back into the sarcoplasmic reticulum



Mechanism of muscle contraction



Myosin head resting
ATP binds and
transfers energy

Ca^{++} flux binds to troponin
shifting tropomyosin

Myosin cross bridge
binds to binding site
on thin filament, ADP
moves away

Energy stored from (A)
allows myosin head to
move back to original
position

Cardiac muscle contraction

The Heart

The Coronary Circulation

The heart muscle gets its arterial supply from two main arteries that arise from the base of the aorta

The left main coronary artery divides into

Anterior descending, runs along the IVS to the apex of the LV, and

Circumflex, turns around the LV and supplies its lateral wall and the LA

The right coronary descends inferiorly, supplies the RV, SA node

It divides into two

Marginal artery runs along the inferior border of the RV, and

Posterior interventricular artery that supplies the IVS and anastomoses with the anterior descending at the apex

The Heart

The Coronary Circulation

Three cardiac veins form on the epicardium

- The great cardiac vein along the anterior descending artery

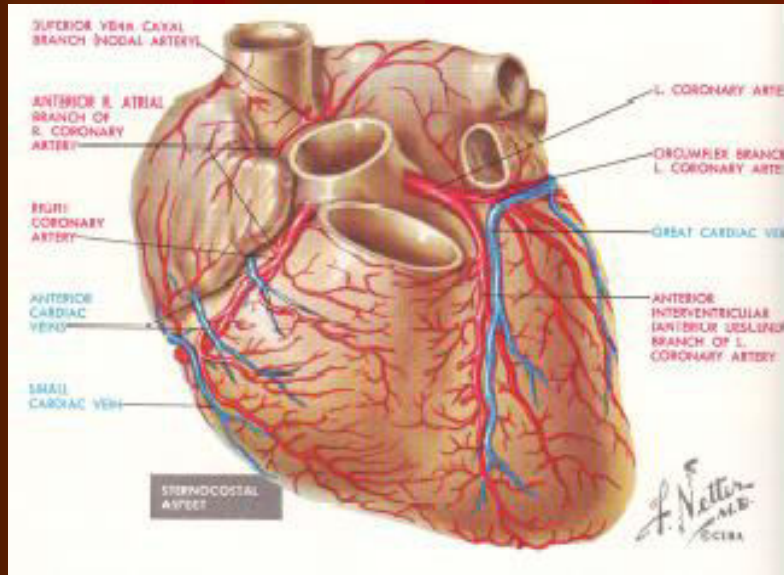
- The middle cardiac vein along the posterior descending artery

- The small cardiac vein along the marginal branch of the RCA

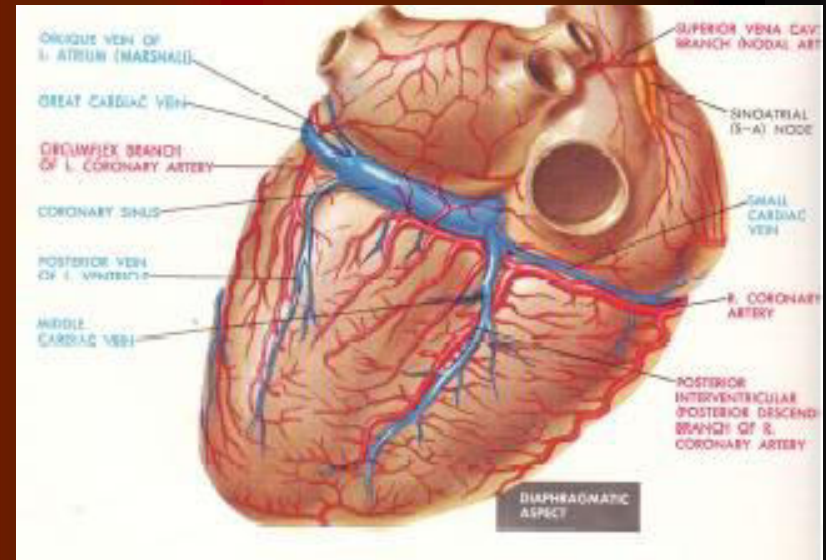
All major three veins drain in the coronary sinus which opens in the RA

Small anterior cardiac veins drain directly into the RA

Other "thebesian veins" also drain directly into the cardiac chambers

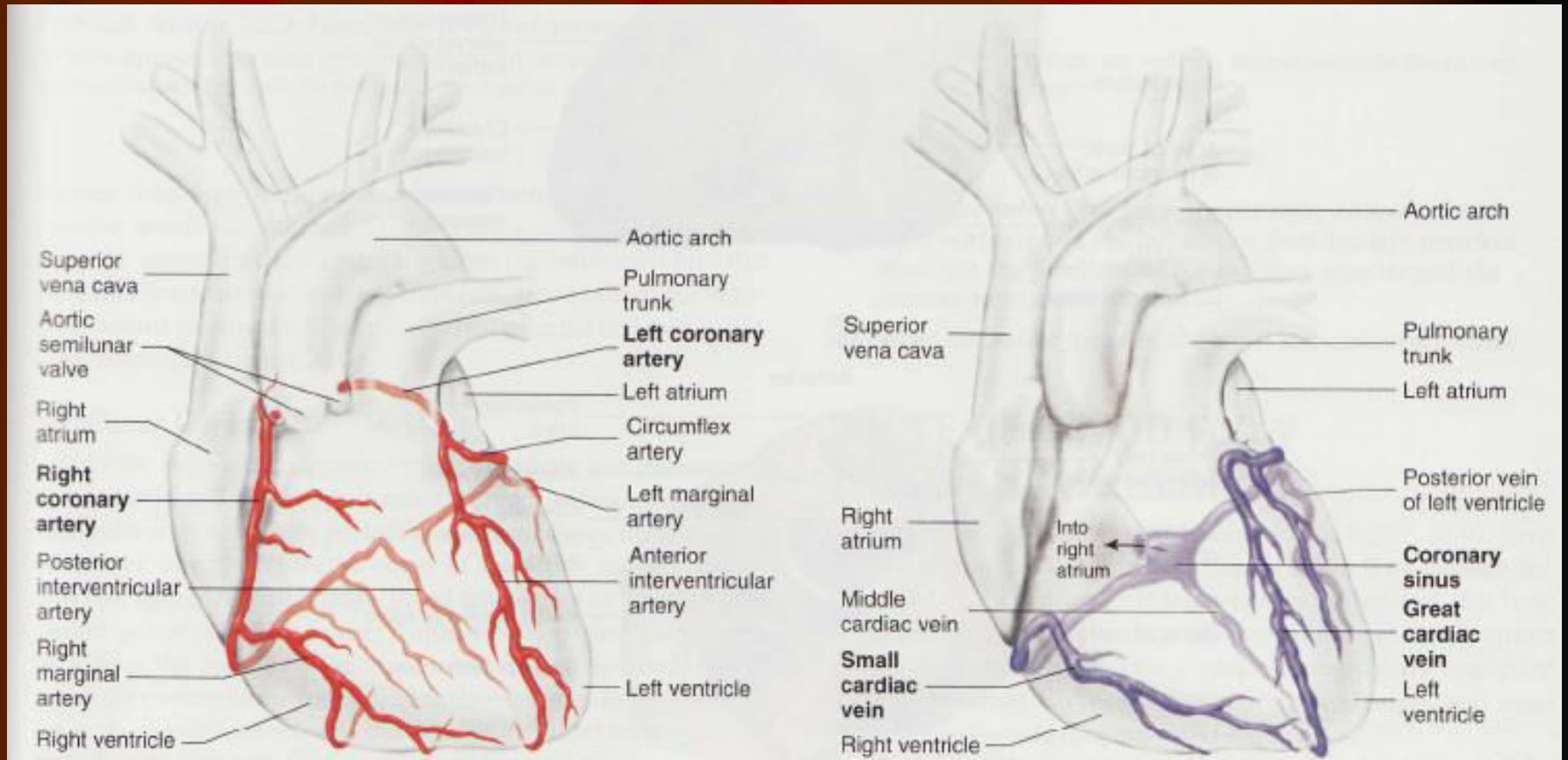


Anterior view



Posterior view

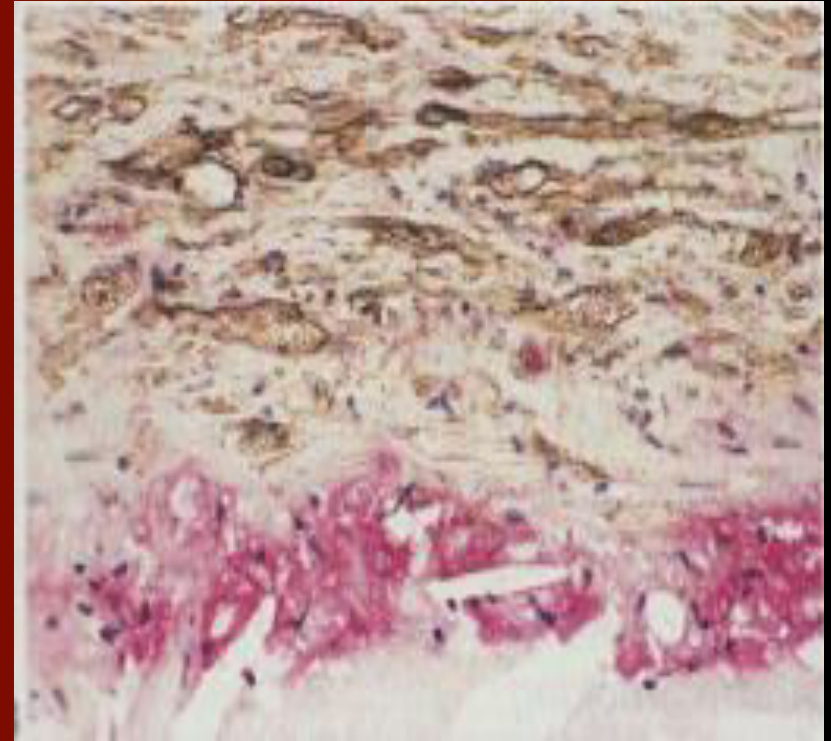
The coronary arteries and veins



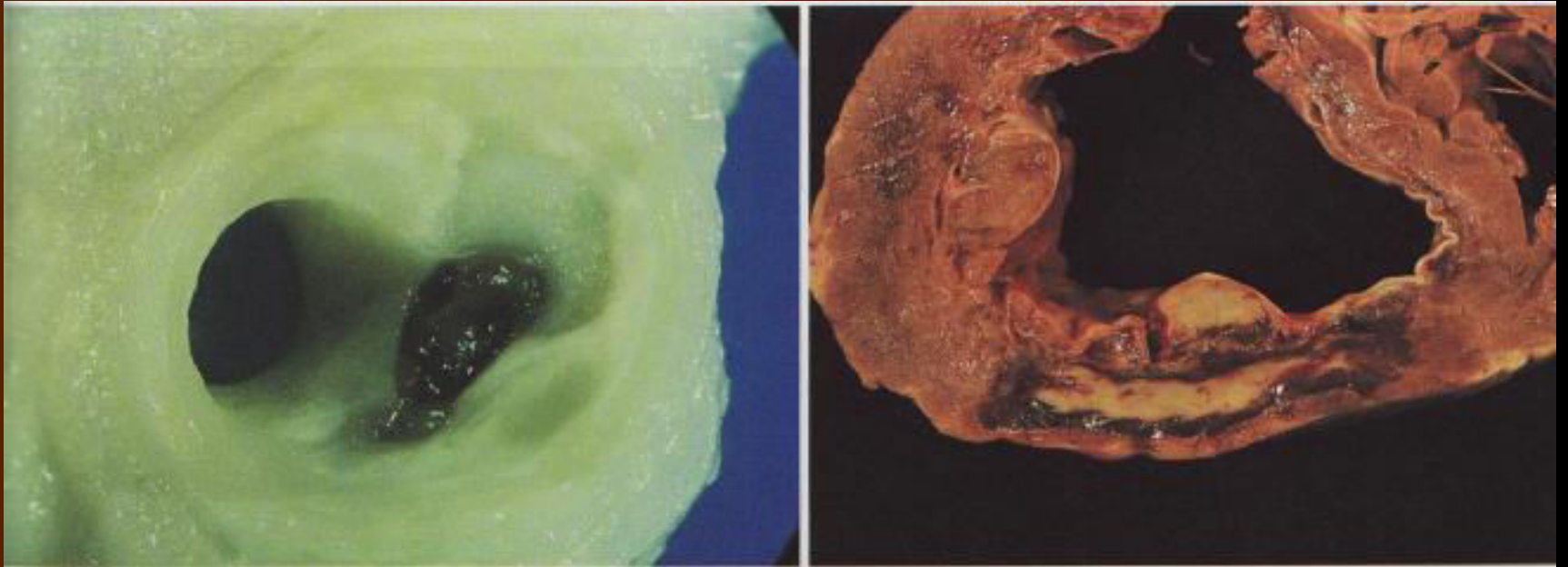
The coronary circulation



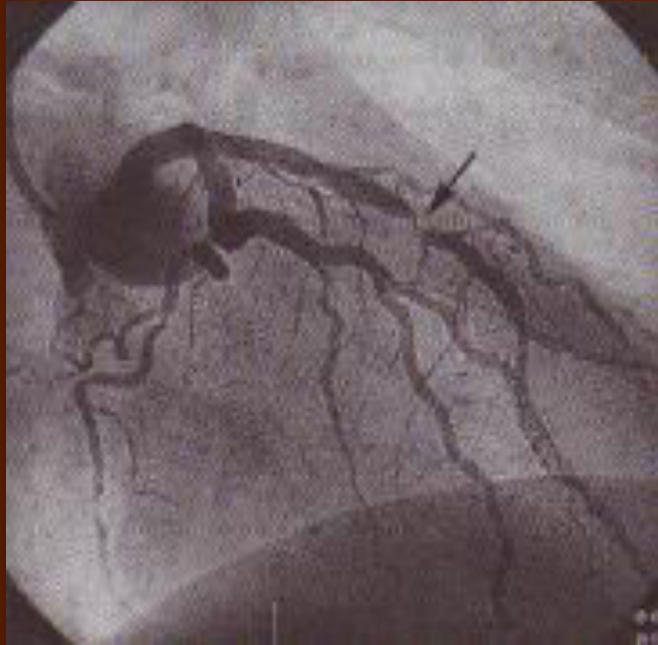
Coronary artery plaque



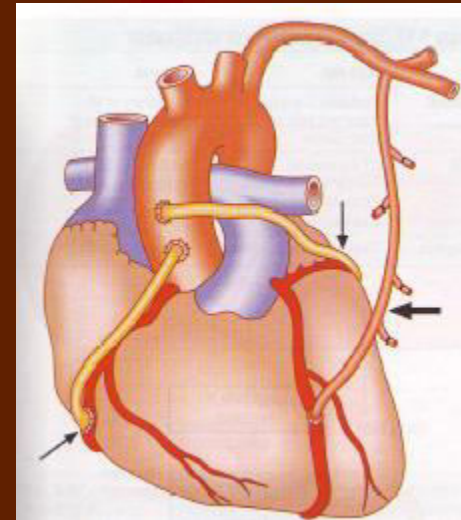
Atheromatous plaque



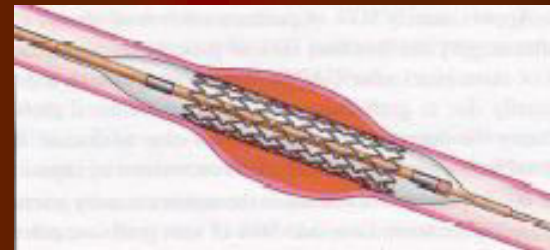
Atheromatous plaque disruption and myocardial infarction



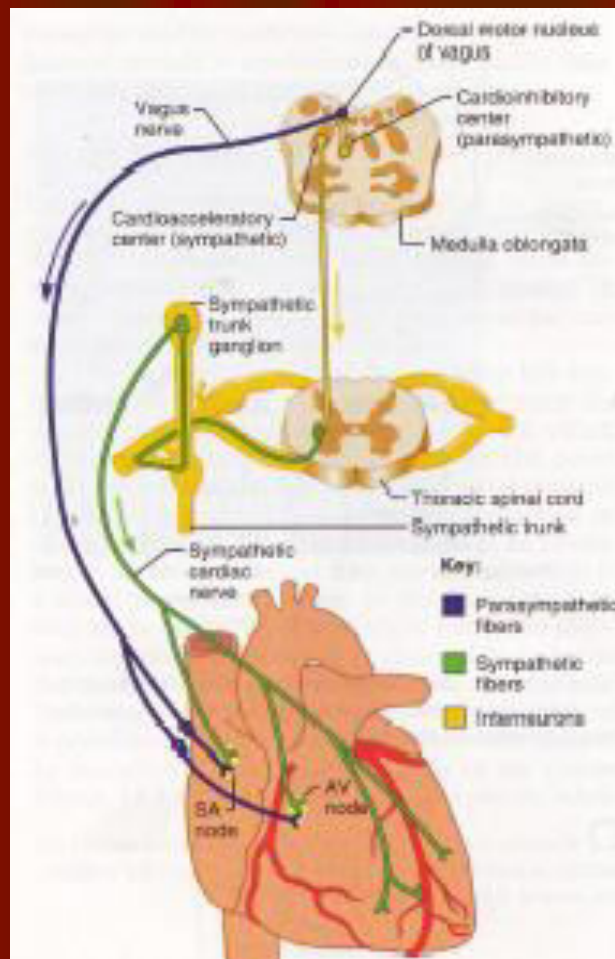
Coronary angiogram showing stenosis of the LAD



Coronary bypass surgery



Angioplasty and stenting



Autonomic innervation of the heart

The Heart

The Conduction System

The conduction system is the electric wiring of the heart

Its function is to synchronize the sequential contraction of the atria followed by the contraction of the ventricles

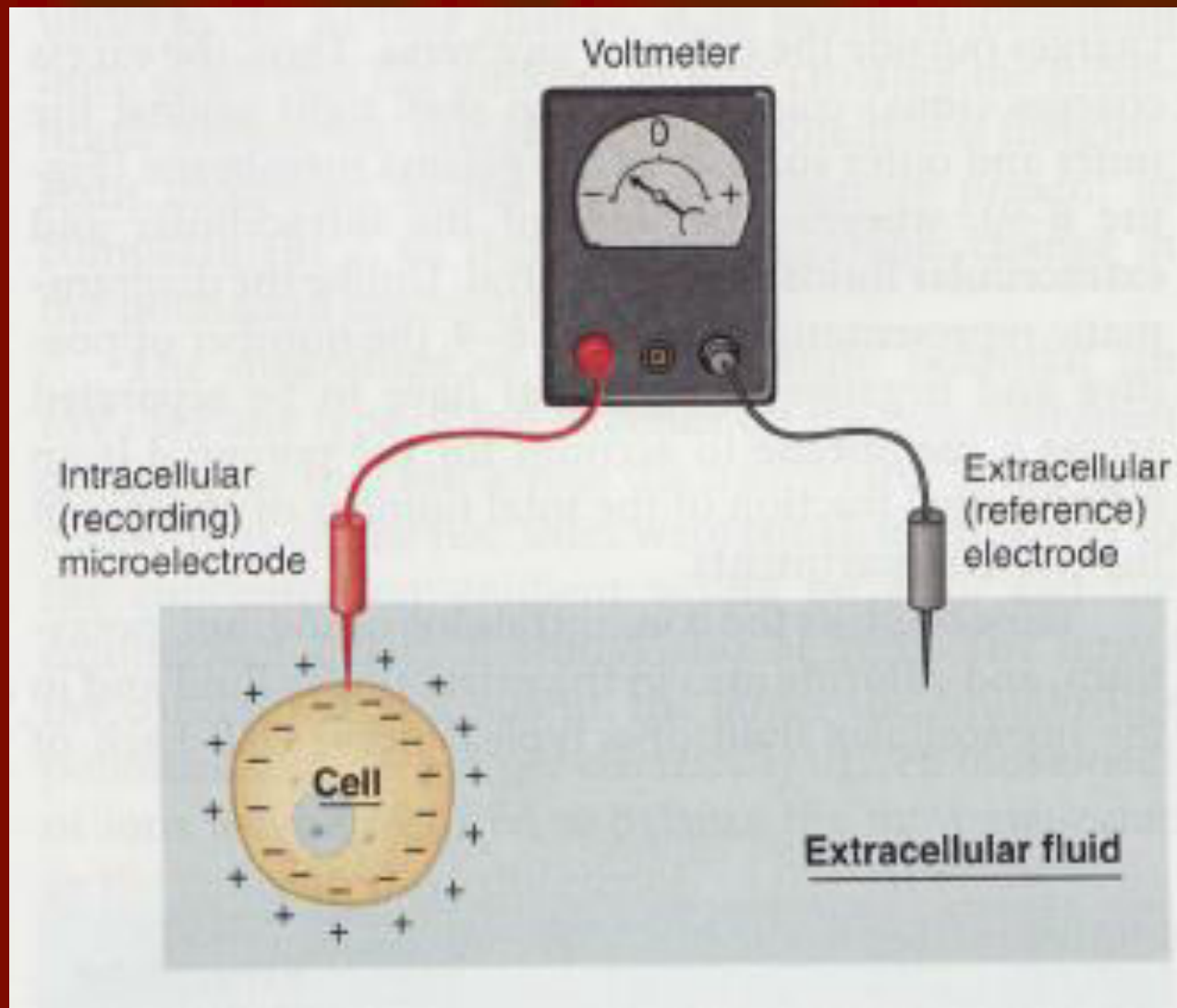
It is made of specialized cells with unstable resting membrane potential that allows spontaneous repolarization and depolarization

Repolarization is the building up of an electric difference between the inside and the outside of the cell membrane

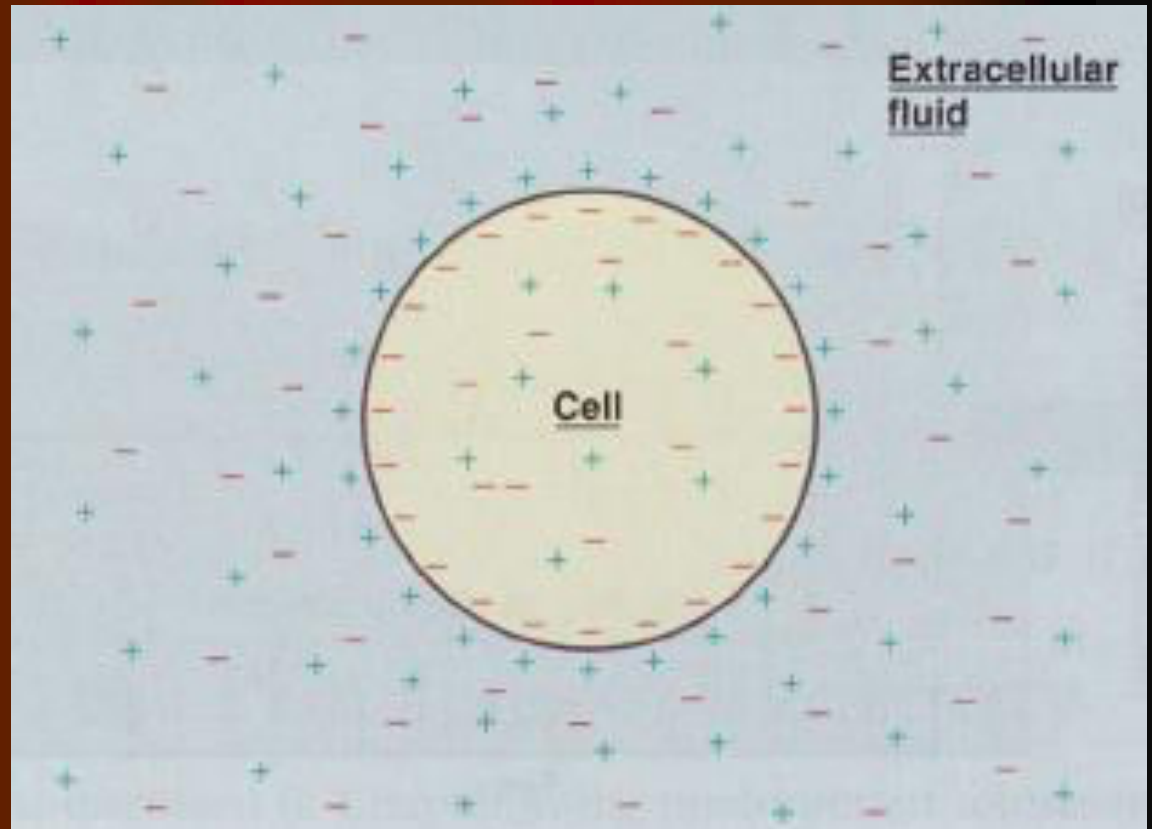
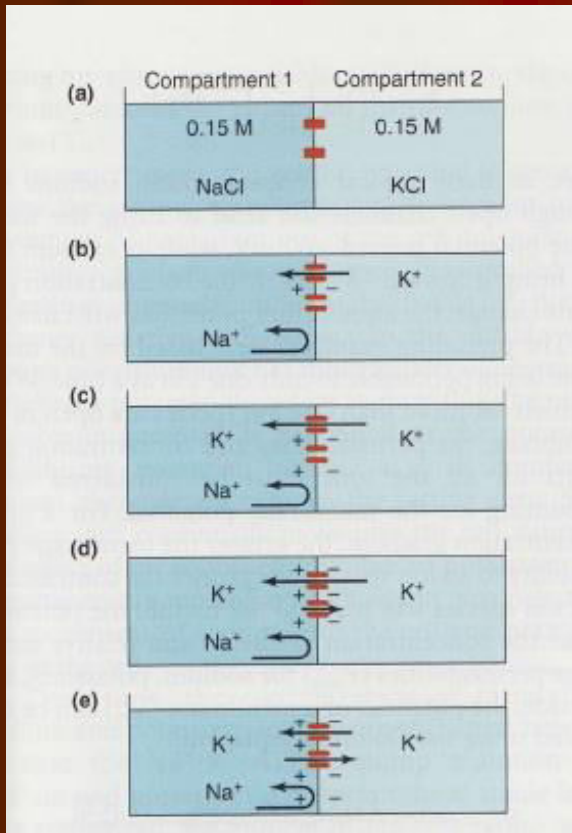
Depolarization is the return of the two sides of the membrane to electric neutrality

Polarization is affected by the selective movement of ions across the membrane

This process requires pump action and energy



Resting membrane potential



Creation of electric potential across the cell membrane through selective ion diffusion

The Heart

Conduction System

Sequential systole of the atria followed by the ventricles is the result of depolarization of the myocardial cell membrane

Gap junctions between cells allow the spread of the action potential

The initial excitation of a myocardial cell allows the excitation of all the cells

The Heart

The Conduction System

Depolarization cycle

K^+ channels close, this leads to increased movement of Na^+ into the cell

The cell membrane then becomes less negative

A less negative cell membrane allows Ca^{++} channels to open, Ca^{++} rushes in

Ca^{++} rush brings the membrane potential to zero (depolarized)

Ca channels then close and K channels open increasing the negativity (repolarization)

The Heart

The Conduction System

The conduction system Initiates and spreads action potential (an electric current) to cardiac muscle fibers

The spread (conduction) takes place through specialized cardiac muscle

Action potential consists of depolarization and repolarization cycles

Depolarization depends on the flux of Na^+ and Ca^{++} into the cell through their specific gates

Ca^{++} gates open and close slower than Na^+ gates

Repolarization occurs as a result of the closure of Ca^{++} and opening of K^+ gates

The cardiac muscle has the ability to depolarize and repolarize autonomically

A refractory period takes place during depolarization/repolarization

The Heart

The Conduction System

The cardiac muscle has the ability to depolarize and repolarize autonomically

A refractory period takes place during depolarization/repolarization

The cardiac muscle can not depolarize during the absolute refractory period

And can depolarize under stronger stimulation during the relative refractory period

The refractory period is longer in the cardiac than the skeletal muscle

This is because there is a ' plateau phase that follows cardiac muscle depolarization before repolarization is complete

The refractory period prevents the tetanic contraction of the cardiac muscle

The Heart

The Conduction System

Different cardiac muscles have different rates of depolarization and repolarization

The specialized muscles of the conduction system have faster depolarization/repolarization rates than the rest of the cardiac muscle

The cells of the sinoatrial node have the fastest rate in the conduction system

The sinoatrial node (SAN) therefore sets the pace for the rate of cardiac muscle contraction

The SAN is therefore called the “pacemaker” under normal conditions

The Heart

The Conduction System

Anatomy

The conduction system is made of

- Sinoatrial node (SAN) located near the orifice of the SVC

- Specialized atrial bundles exist

- Atrioventricular node is located at the base of the right atrium

- Common bundle (Bundle of His)

- Bundle of His branches run in the IVS and divides into a left and a right "bundle branch"

- Purkinje fibers emanate from the bundle branches

The Heart

The Conduction System

Normally, the SAN rate of depolarization is faster than the rest of the myocardium

The SAN "sets the pace" for the heart rate, it is the normal "pacemaker"

The rate generated is termed "sinus rhythm"

Conduction through the AVN is slow to allow for the completion of atrial systole before the ventricles contract

If the SAN fails, the AVN takes over, it is inherently slower than the SAN

It generates AV nodal rhythm, simply called "nodal rhythm"

If the AV node also fails, the ventricular muscle takes over, its rhythm is slower than the nodal, and it is referred to as "idioventricular rhythm"

The Heart

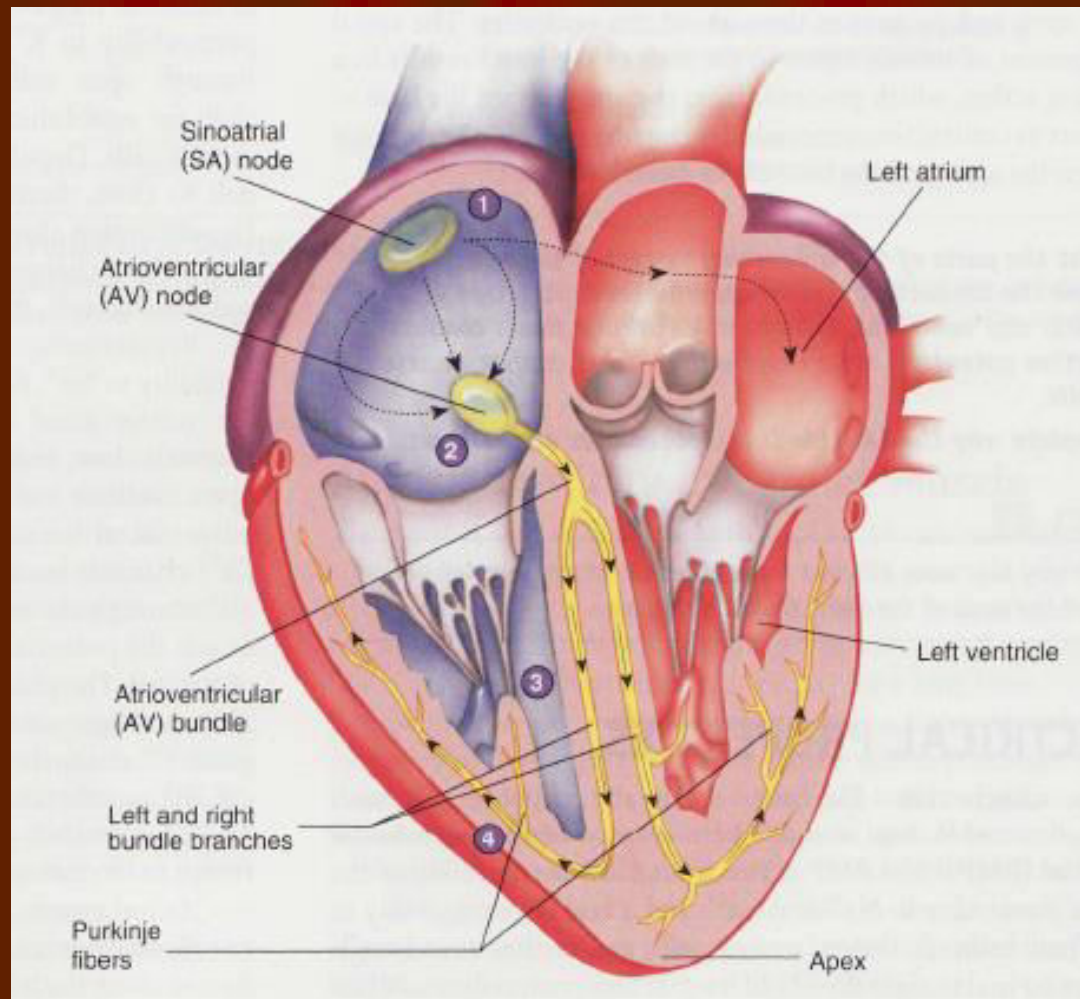
The Conduction System

The Action potential spreads from one muscle to the other through the gap junctions between the cells

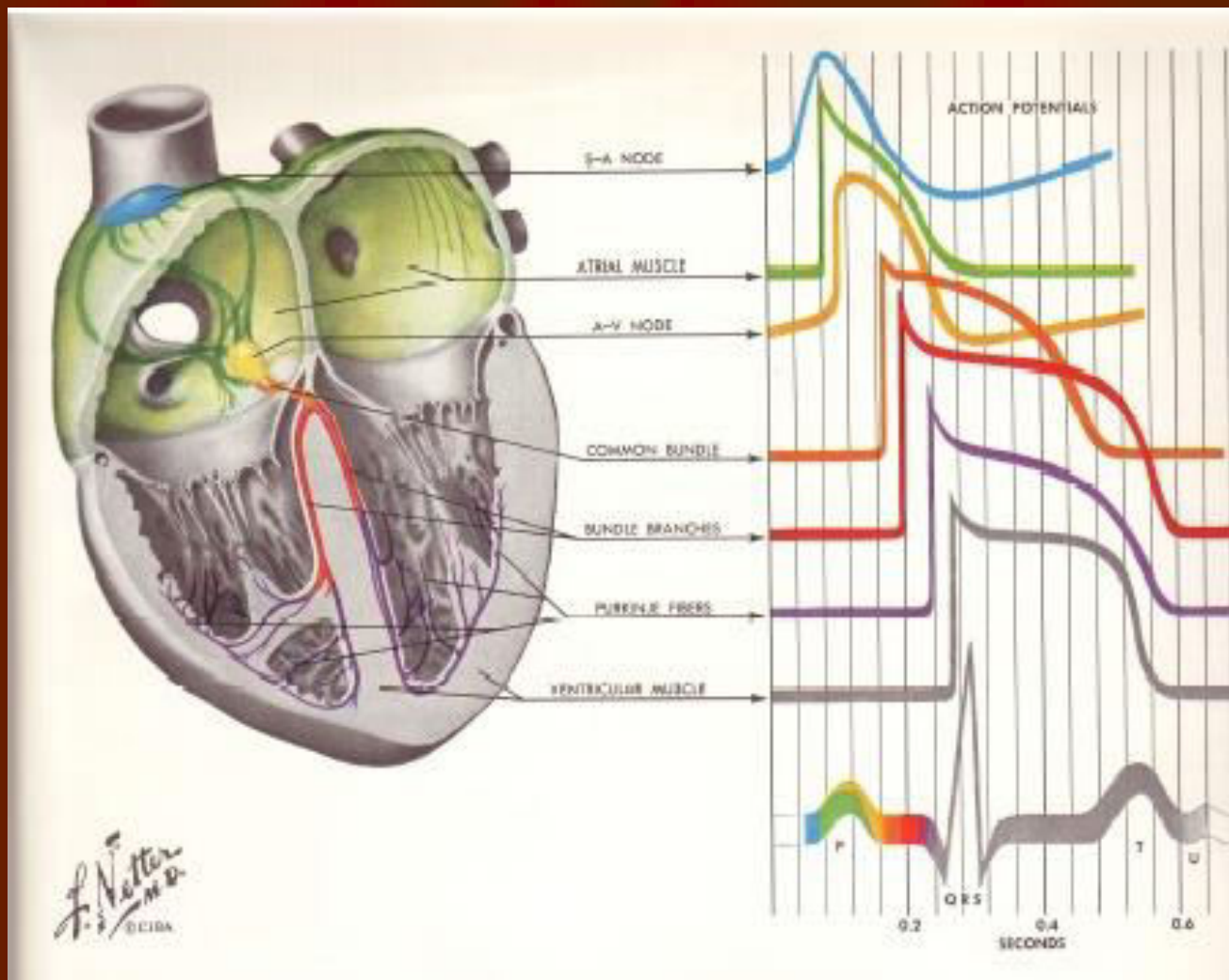
During and following an action potential, the cardiac muscle goes into a "refractory period" during which an excitable membrane can not be re-excited

The refractory period prevents the myocardium from going into tetanic contractions

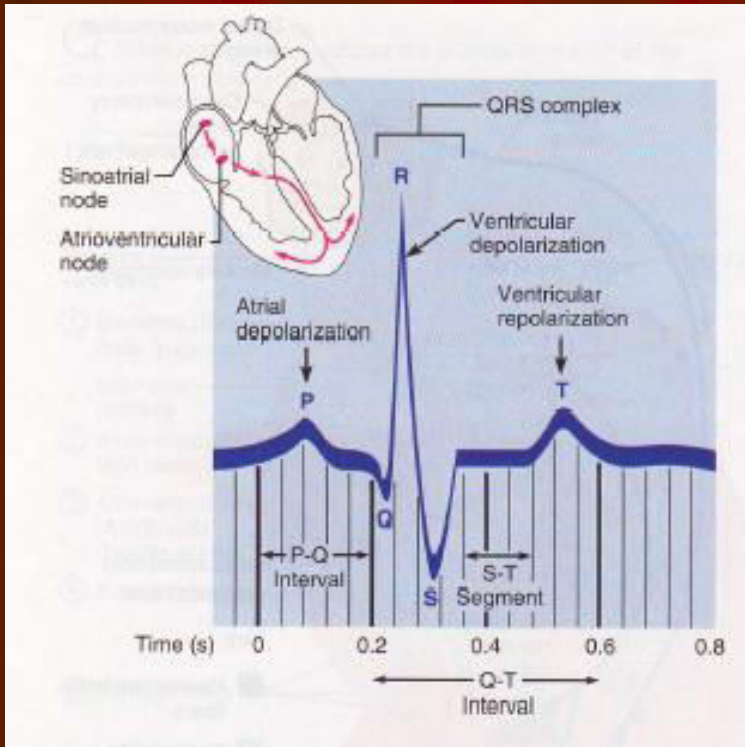
When the conduction between the atria and the ventricle is impaired the condition is termed "heart block", this could be partial or complete



The anatomy of the conduction system

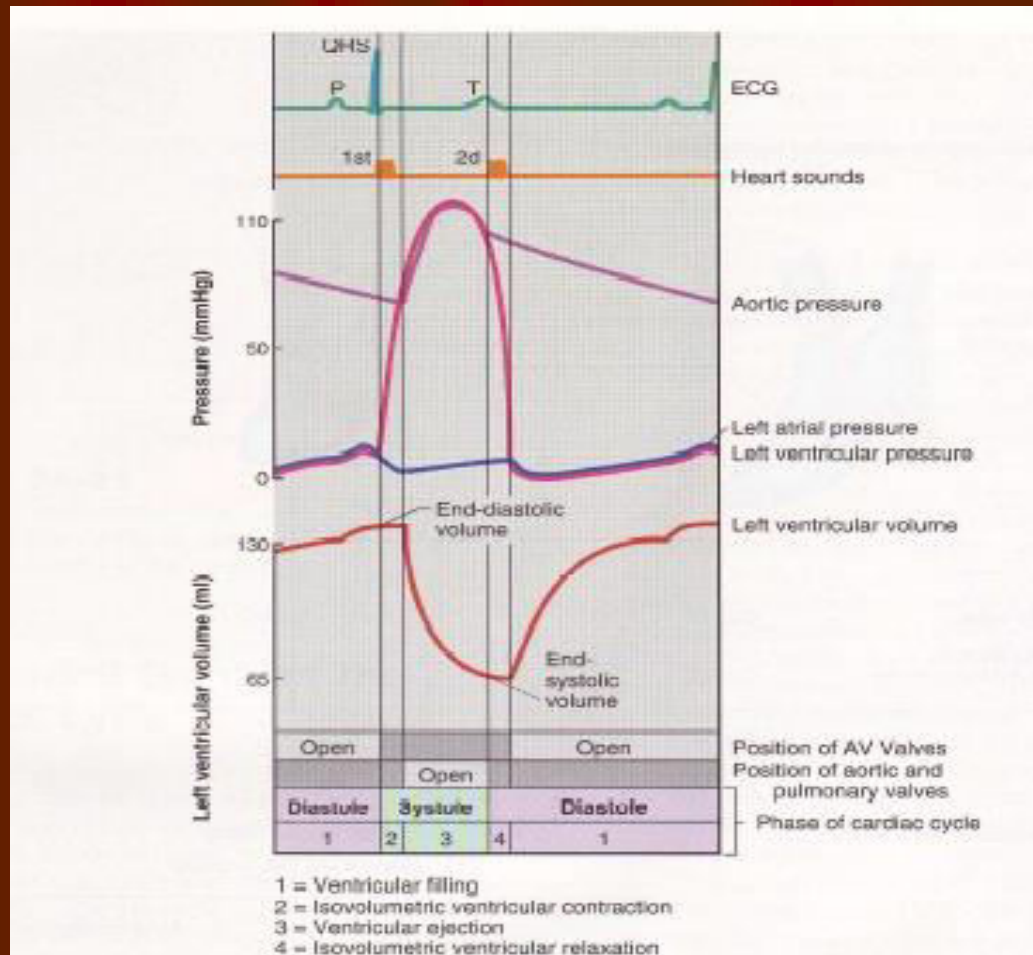


The conduction system



Each wave of depolarization is followed by an absolute refractory period during which no depolarization can take place. The refractory period is equal to the length of cardiac muscle contraction. This guards against tetanic contraction of the cardiac muscle.

The EKG



Events during the cardiac cycle

Systole and diastole in this diagram refer to the ventricles and not the atria

The Heart

Cardiac Output (CO)

The cardiac output is the volume of blood delivered to the circulation in one minute, i.e. the heart rate (HR) multiplied by the volume ejected with each heart beat [called the stroke volume (SV)]

Therefore $CO = HR \times SV$

Cardiac output depends on

The amount of blood returning to the heart (also called "preload")

Cardiac contractility which determines the amount of blood ejected during every ventricular contraction, the stroke volume (SV)

Heart failure is the inability of the CO to meet the metabolic demands of the body

The Heart

Cardiac Output (CO)

The normal cardiac output is 3 L/m²/ min

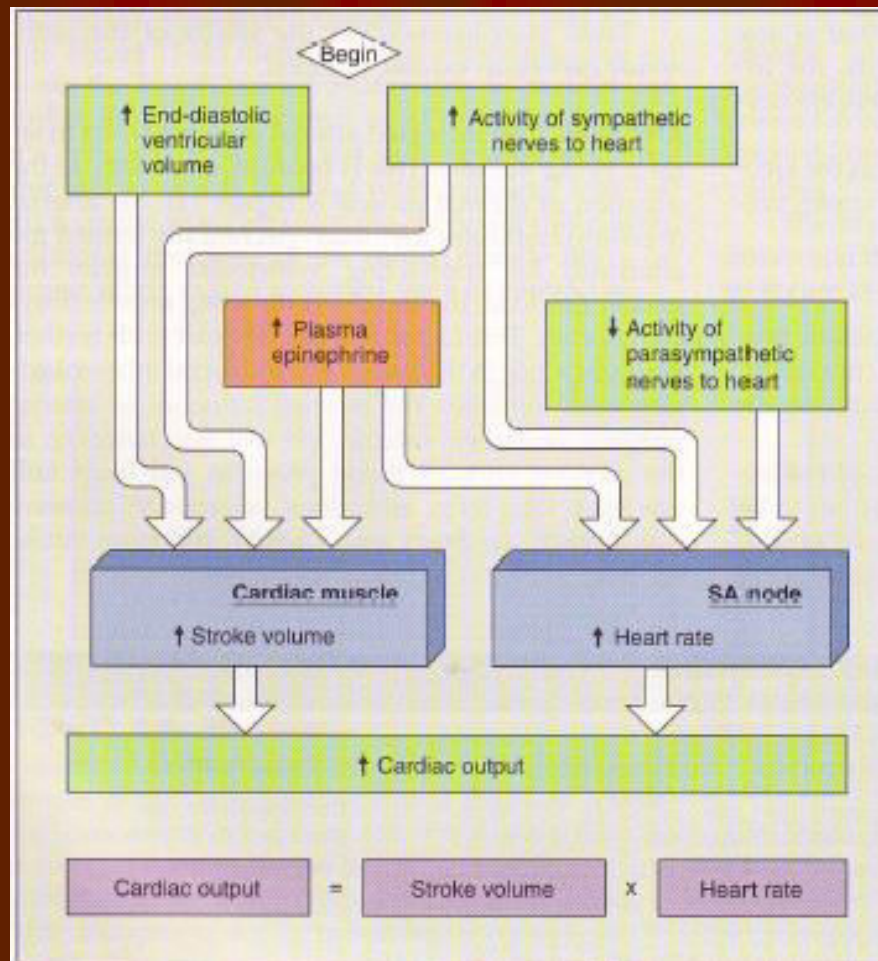
Its purpose is to supply adequate amounts of O₂ to the tissues

Normally, CO provides 3 – 4 times the amount of O₂ consumed

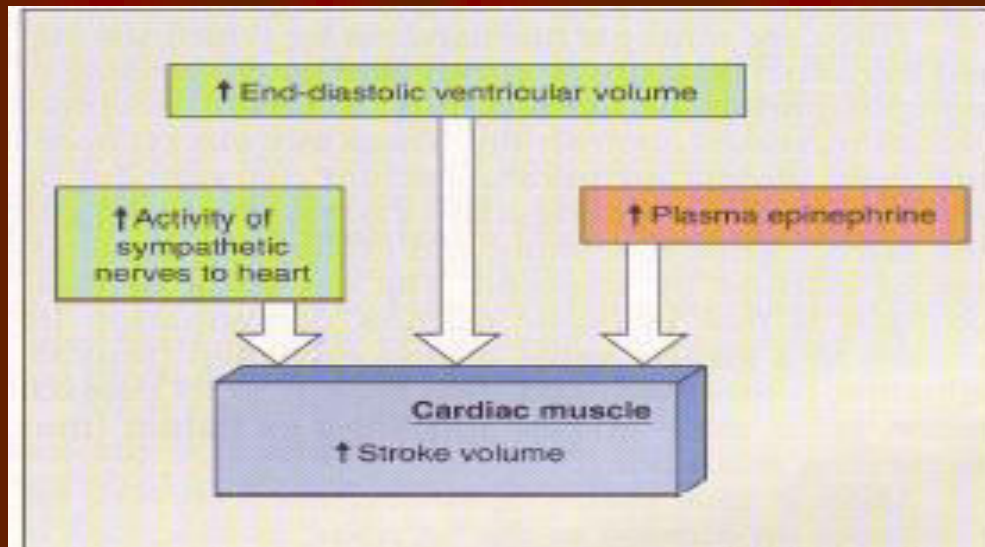
If the need for O₂ increases or decreases chemoreceptors adjust the CO proportionately

The adjustment takes place through increasing the heart rate and contractility

Clinically, the urine output, skin temperature brain function are indices of adequacy of CO



Factors affecting cardiac output



Control of stroke volume

The Heart

Cardiac Output (CO)

The Ejection Fraction

Ventricles do not eject all the blood they accumulate during diastole, the end diastolic volume (EDV)

The difference between EDV and the volume ejected during systole, the end systolic volume (ESV) is the "stroke volume" (SV)

Therefore $SV = EDV - ESV$

The ratio SV/EDV is normally about 55% to 60%

This is the "ejection fraction" (EF)

Reduced cardiac contractility results in a lower EF

The Heart

Cardiac Output (CO)

The Frank-Starling Law

The more stretched the cardiac muscle the stronger its contraction until an optimal length is reached after which further stretching will weaken the force of contraction

The amount of myocardial stretch is decided by the preload

The Heart

Cardiac Output (CO)

Factors Affecting the Heart Rate

Sympathetic stimulation increases SAN discharge through the effect of noreadrenalin on the β receptors, it also increases the cardiac contractility

Parasympathetic stimulation reduces the SAN rate

There is no parasympathetic innervation to the ventricles

Bradycardia allows for a larger EDV

Extreme tachycardia and extreme bradycardia reduce CO; the first through reducing the SV, and the second through reducing HR

The Heart

Cardiac Output (CO)

Cardiac Reflexes

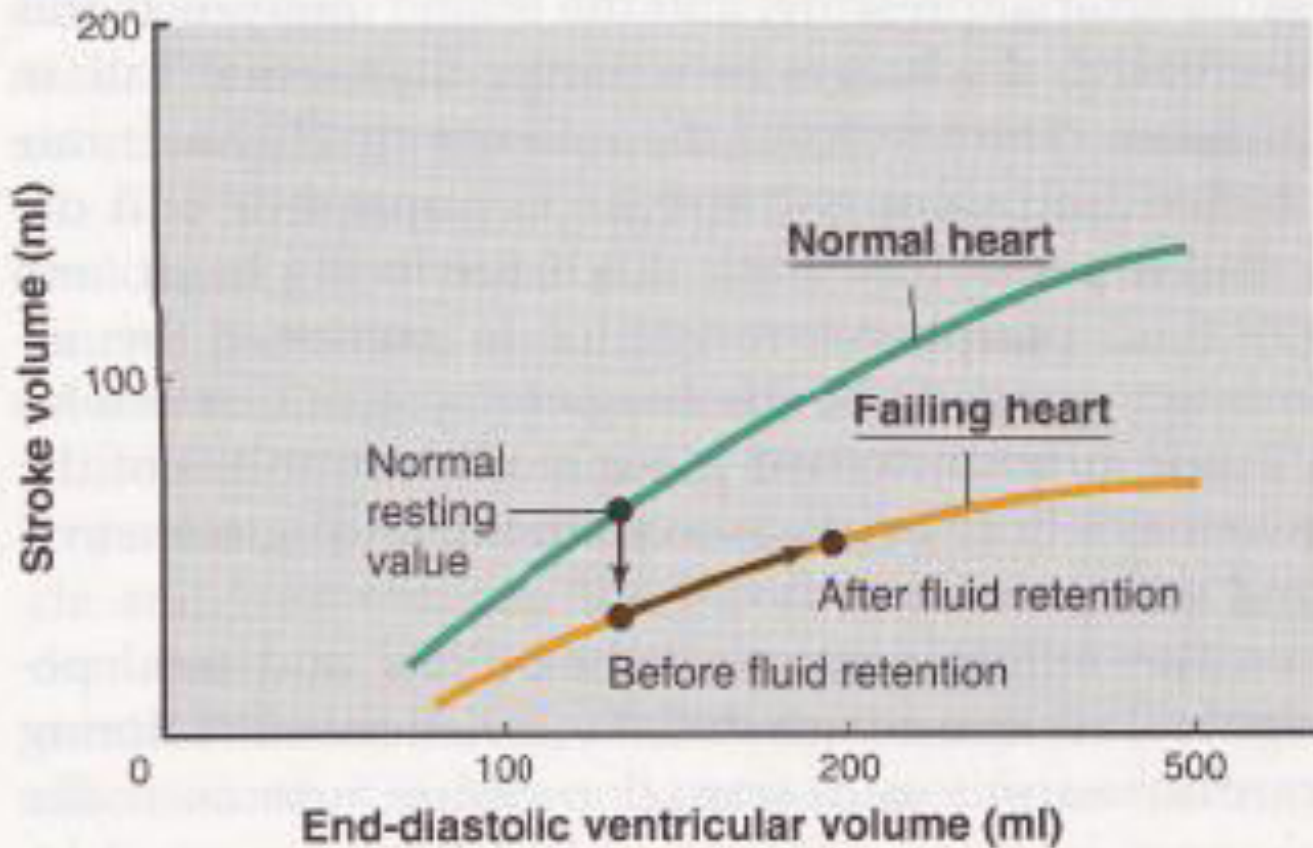
Carotid body receptors reduce the heart rate in response to hypertension and increases it in response to hypotension

Bainbridge reflex stretching the right atrial wall produces tachycardia

Adrenaline and thyroxine induce tachycardia

Ca⁺⁺ injections augment cardiac contraction, excessive Ca⁺⁺ stops the heart in systole

K⁺ injections lead to heart block and cardiac arrest in diastole



How does the failing heart compensate for the loss of contractility?

The Heart

Diastolic and Systolic Dysfunction

Reduced compliance of the RV results in a rapid rise of its pressure with additional volume

This leads to a reduced EDV compared to a state of normal compliance at a given pressure

Low EDV results in a low SV by RV, and consequently by LV

In pure diastolic dysfunction, RV contractility remains normal

The right ventricle does not have to pump the blood too far

The RV is a volume pump

The Heart

Systolic Dysfunction

Unlike the RV, LV has to pump the blood for a long distance and against higher resistance, the LV is a pressure pump

Systolic dysfunction results from myocardial damage due to chronically increased after load (systemic hypertension)

Myocardial damage and changes in the LV geometry result in a \downarrow SV at any given EDV, i.e. \downarrow ejection fraction

Baroreceptors discharge rate drops leading sympathetic stimulation, \uparrow HR, \uparrow PR, and \uparrow angiotensin II that leads to fluid retention and \uparrow venous pressure causing edema in the lower limbs

When the LV fails to pump all the volume it receives from RV, edema develops in the lungs