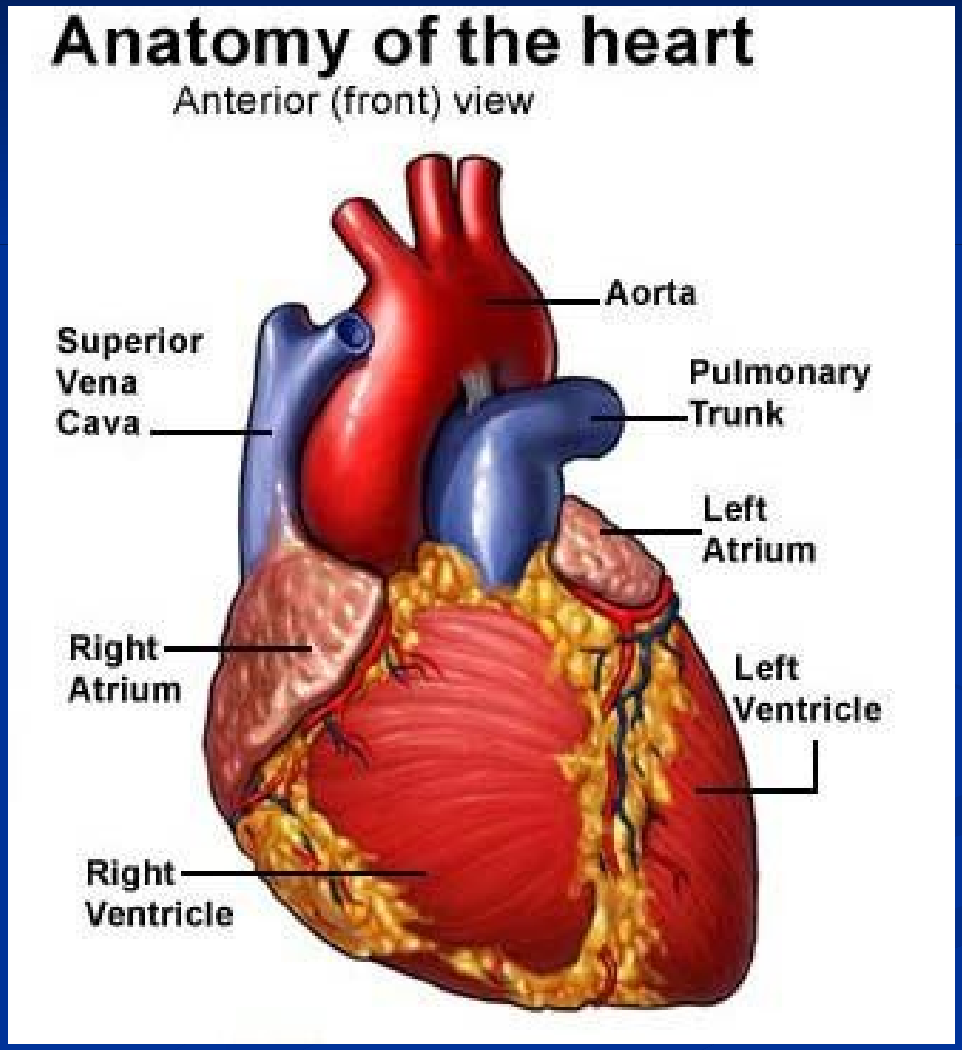
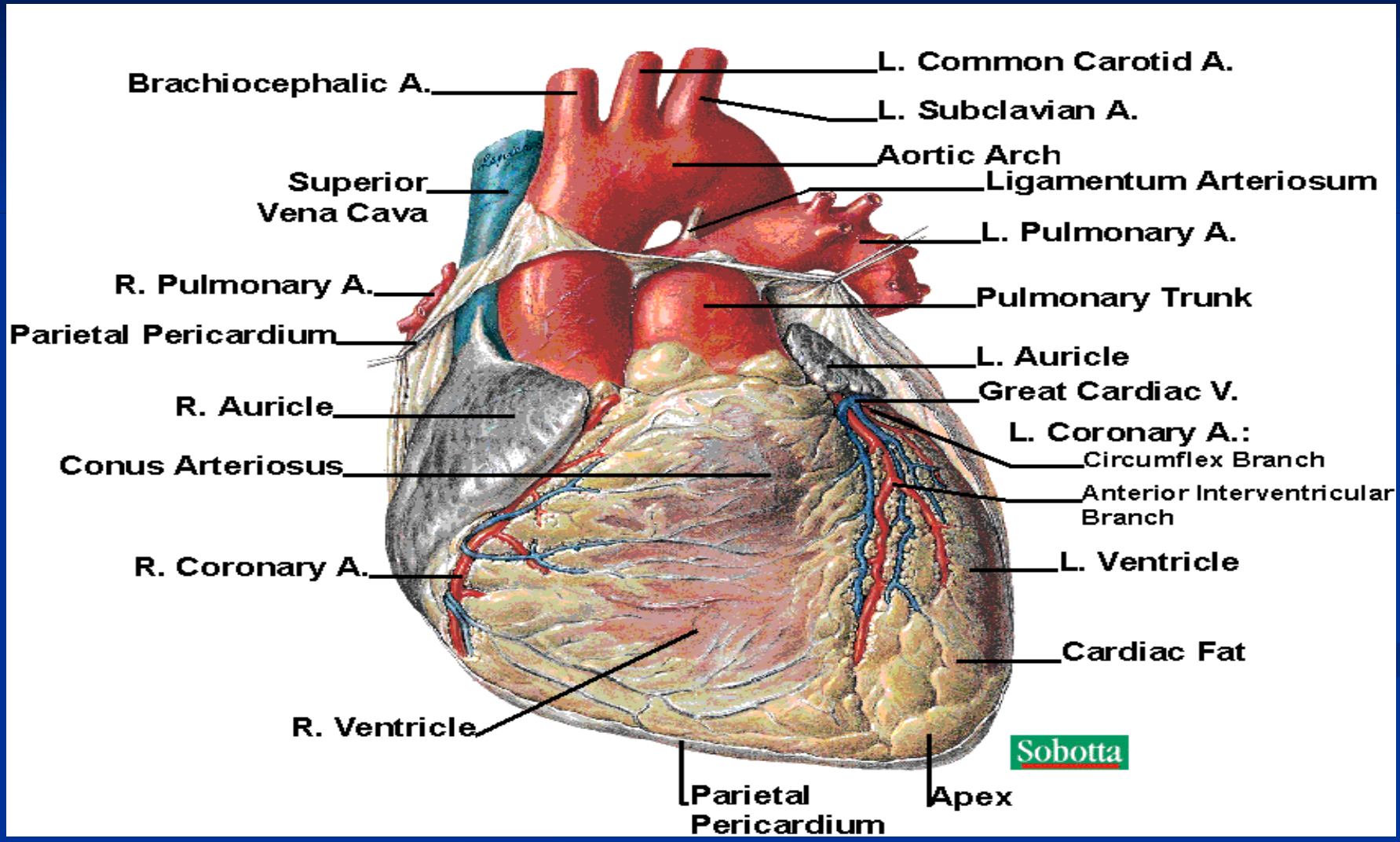
Cardiac anatomy. Cardiac physiology. Atherosclerosis. IHD-CABG

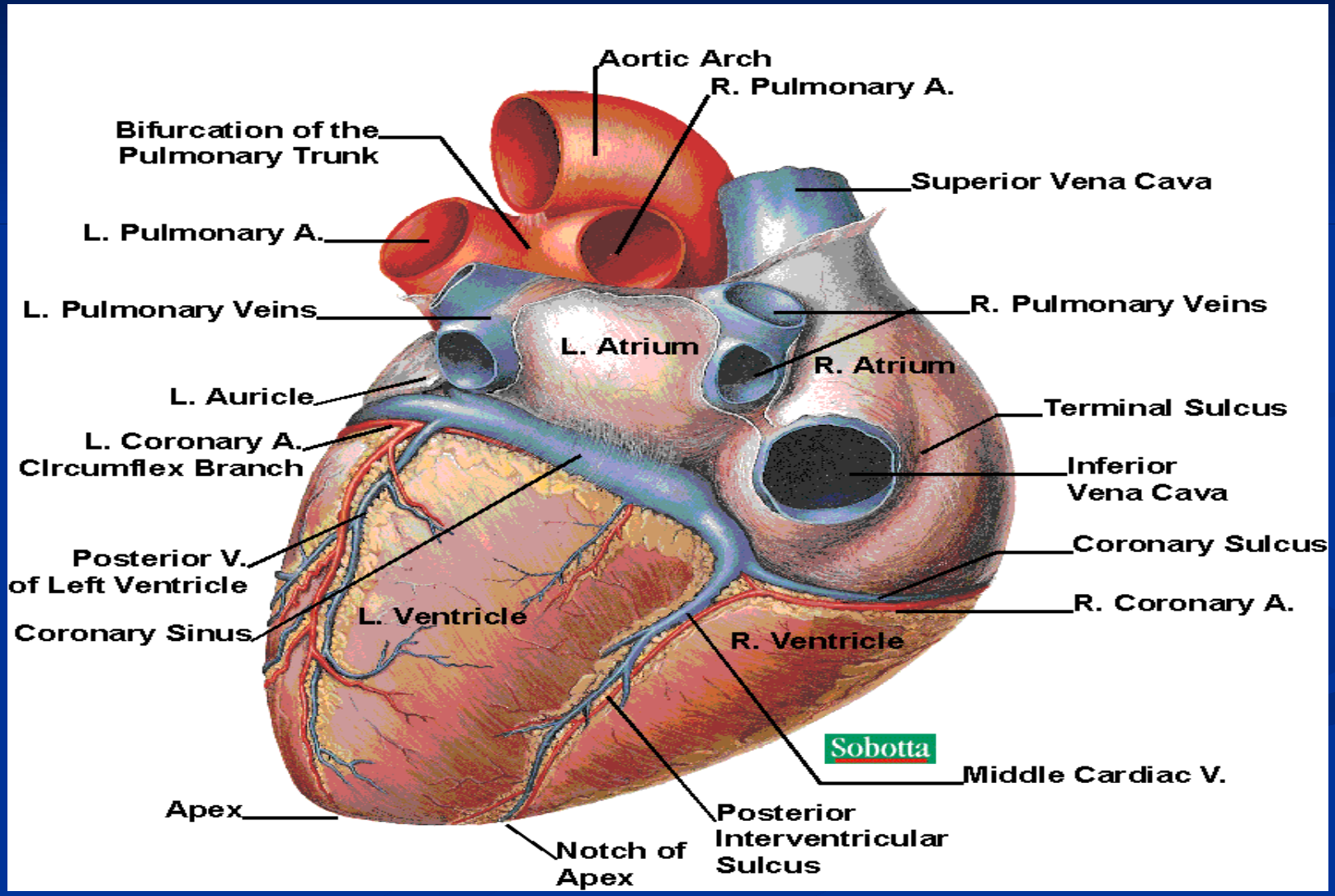
Sotiris Papaspyros

Gross cardiac anatomy

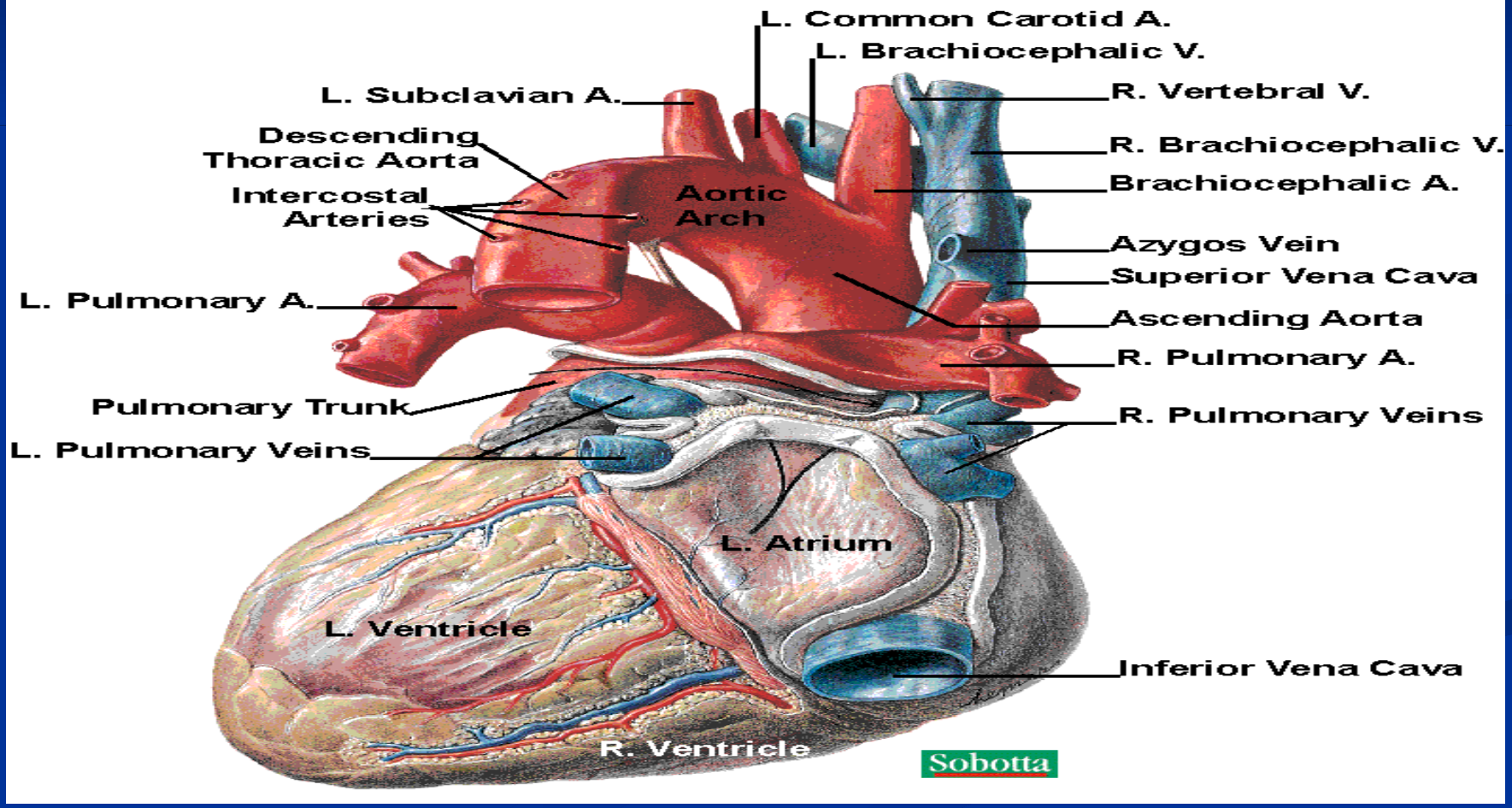


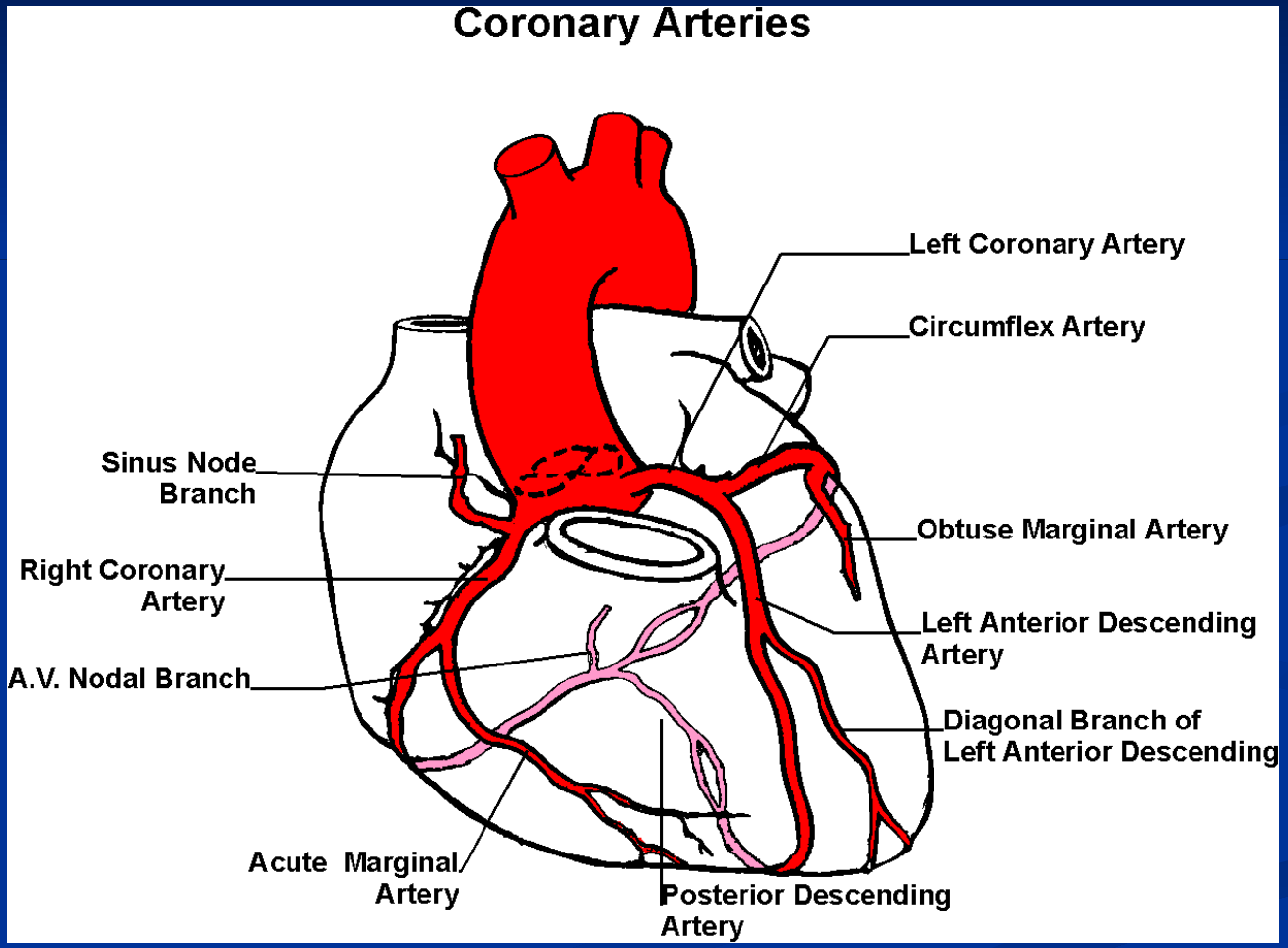
Anatomy


Brachiocephalic A.
L. Common Carotid A.
L. Subclavian A.
Aortic Arch
Superior Vena Cava
Ligamentum Arteriosum
L. Pulmonary A.
R. Pulmonary A.
Pulmonary Trunk
Parietal Pericardium
L. Auricle
Great Cardiac V.
R. Auricle
L. Coronary A.:
Circumflex Branch
Conus Arteriosus
Anterior Interventricular Branch
R. Coronary A.
L. Ventricle
Cardiac Fat
R. Ventricle
Sobotta
Parietal Pericardium
Apex

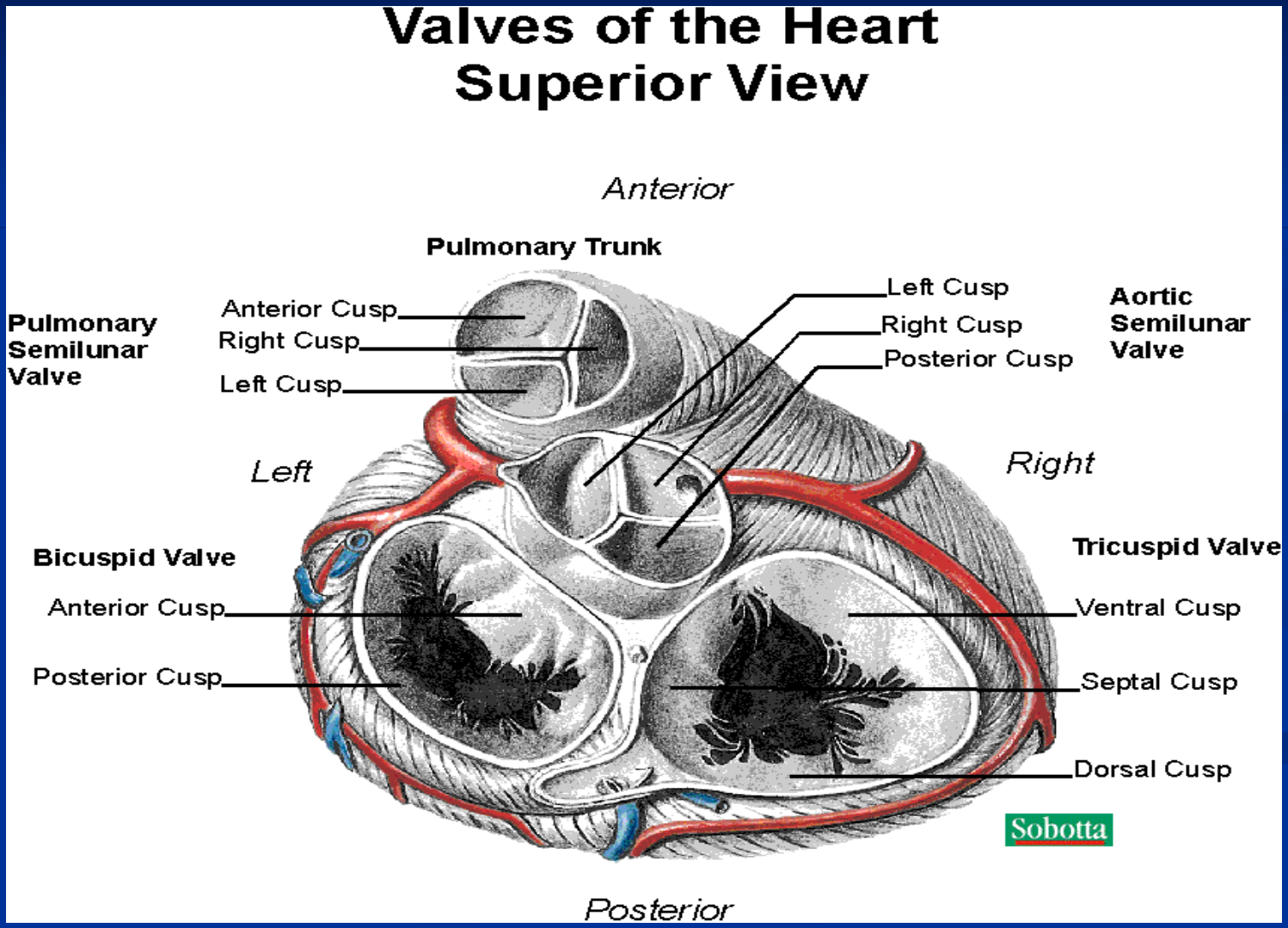

Aortic Arch
R. Pulmonary A.
Bifurcation of the Pulmonary Trunk
Superior Vena Cava
L. Pulmonary A.
L. Pulmonary Veins
R. Pulmonary Veins
L. Atrium
R. Atrium
L. Auricle
Terminal Sulcus
L. Coronary A. CIrcumflex Branch
Inferior Vena Cava
Posterior V. of Left Ventricle
Coronary Sulcus
L. Ventricle
R. Coronary A.
Coronary Sinus
R. Ventricle
Sobotta
Middle Cardiac V.
Apex
Posterior Interventricular Sulcus
Notch of Apex

The Heart with Great Vessels
Posteroinferior View



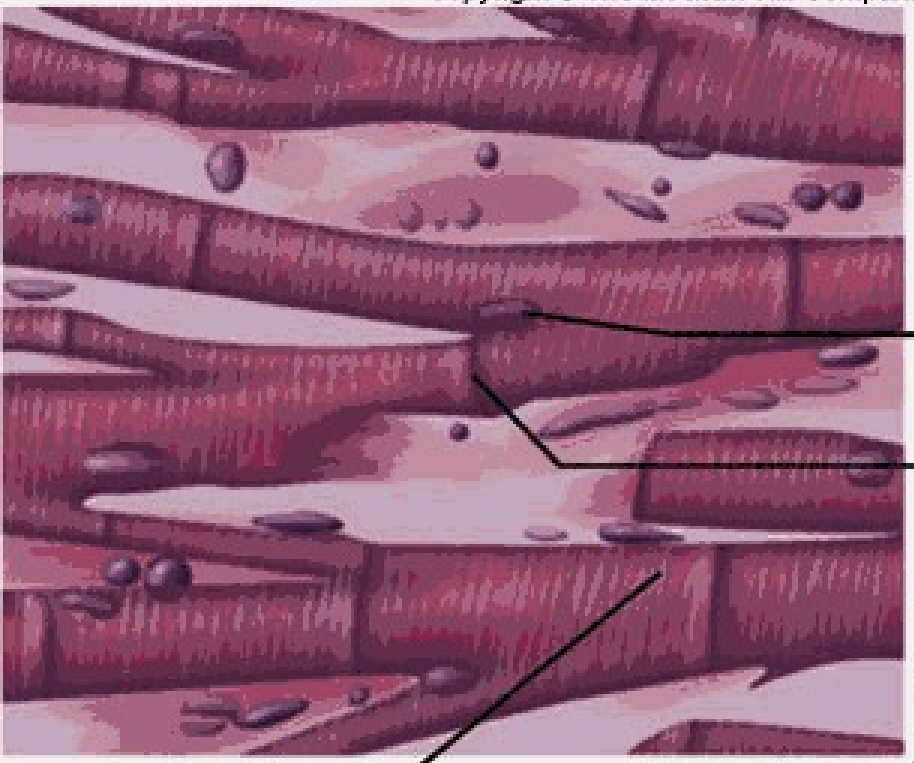

Coronary Arteries
Left Coronary Artery
Circumflex Artery
Sinus Node Branch
Obtuse Marginal Artery
Right Coronary Artery
Left Anterior Descending Artery
A.V. Nodal Branch
Diagonal Branch of Left Anterior Descending
Acute Marginal Artery
Posterior Descending Artery

Valves of the Heart
Superior View



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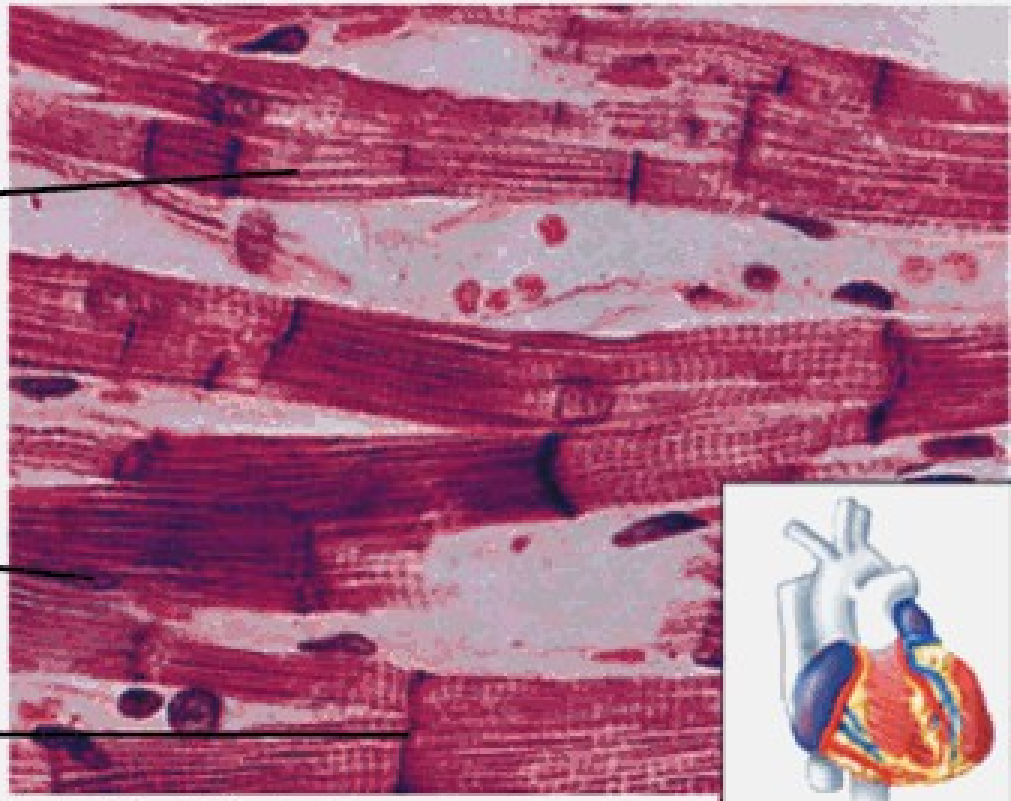
Cardiac Muscle Tissue



Nucleus

Intercalated disk

Striations



Nucleus

Intercalated disk

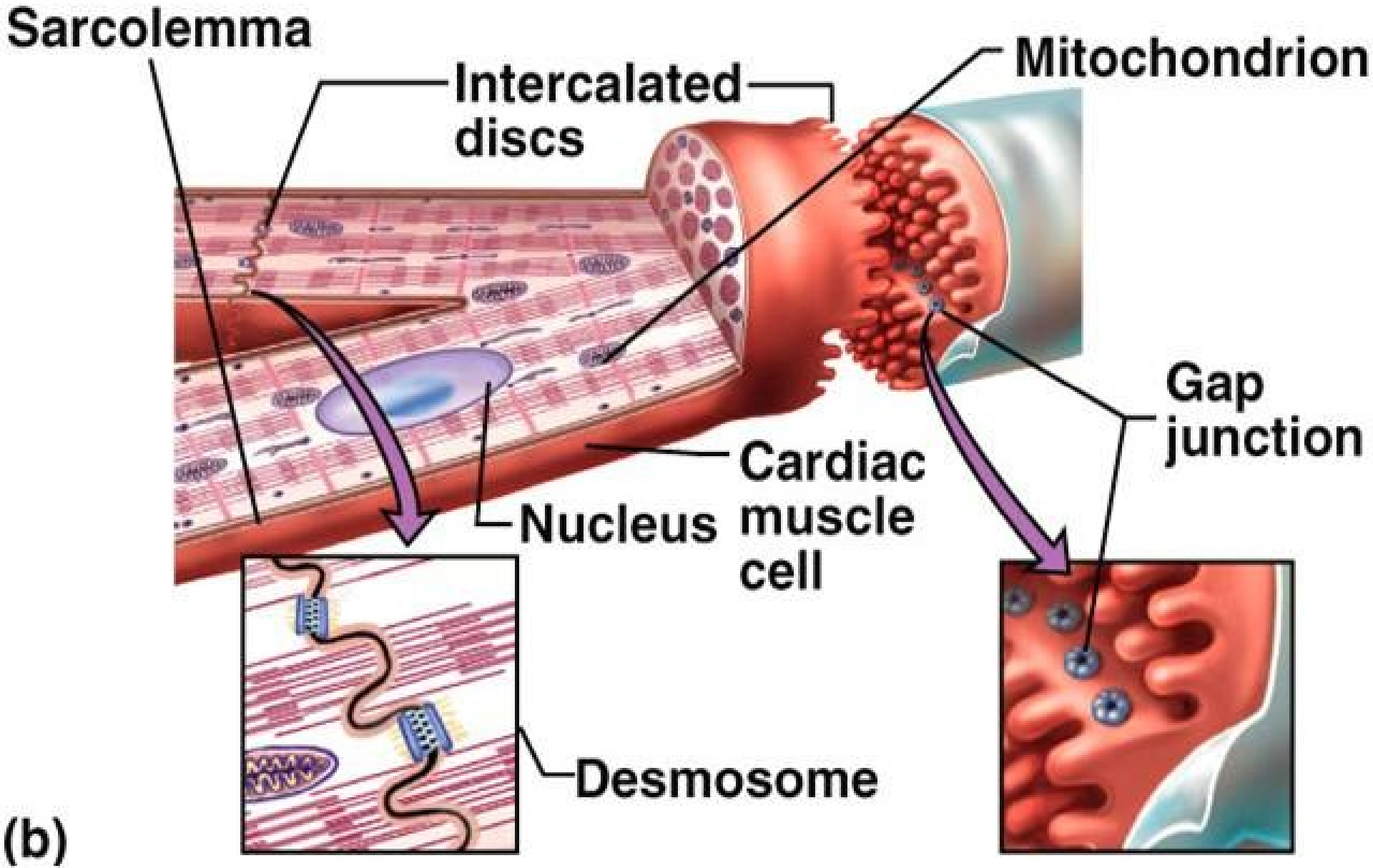
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Characteristics-Cardiac muscle as a Syncytium

- Striated.
- Typical myofibrils that contain actin-myosin filaments.
- Intercalated discs (gap junctions).

Intercalated discs

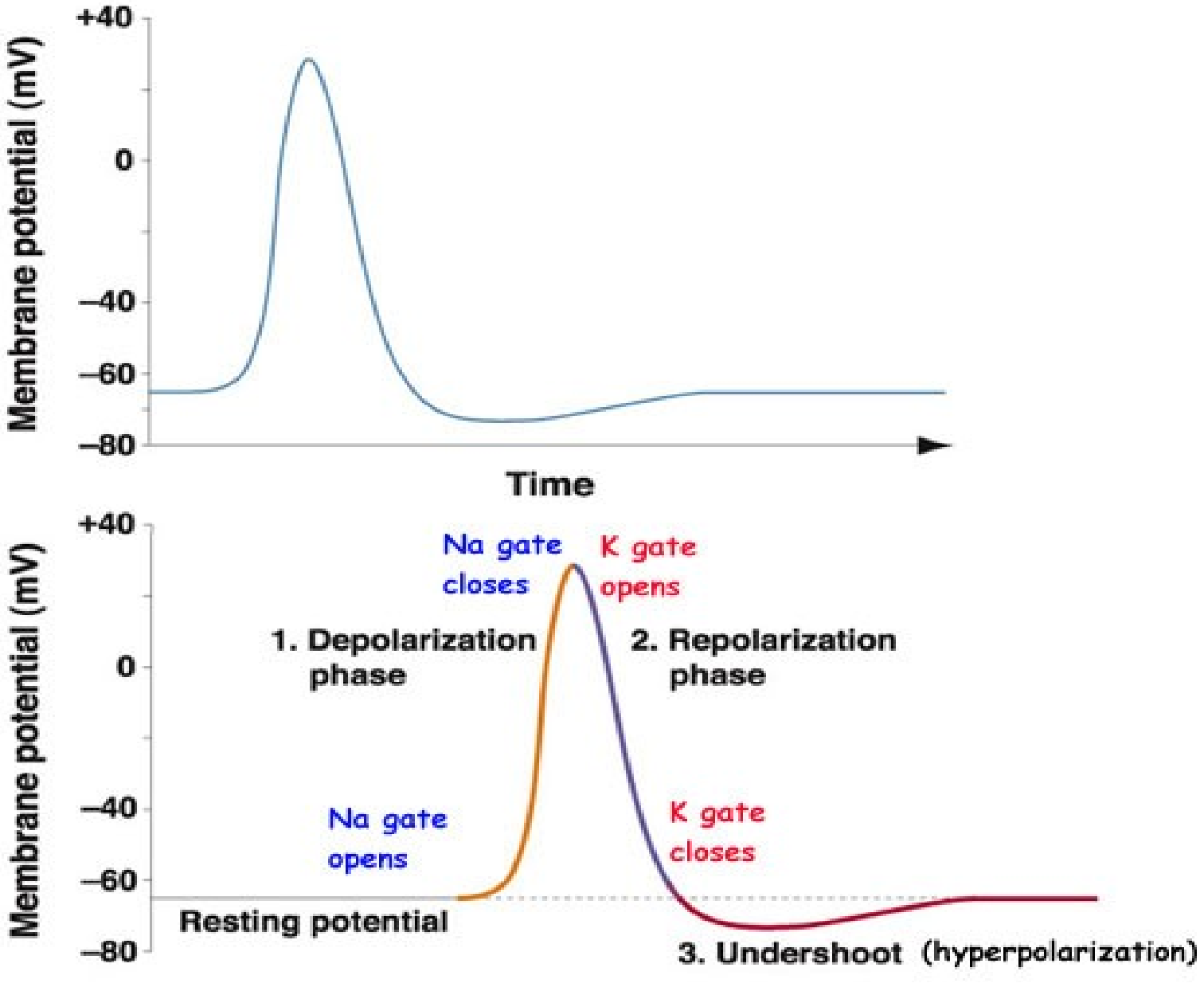
- Cell membranes separating cardiac muscle cells from one another.
- Electrical resistance through the discs is 1/400 of the resistance through the outside membrane because of:
- The cell membranes fuse with each other forming gap junctions (allow totally free diffusion of ions).


Sarcolemma
Intercalated discs
Mitochondrion
Gap junction
Cardiac muscle cell
Nucleus
Desmosome
(b)

Two functional syncytiums

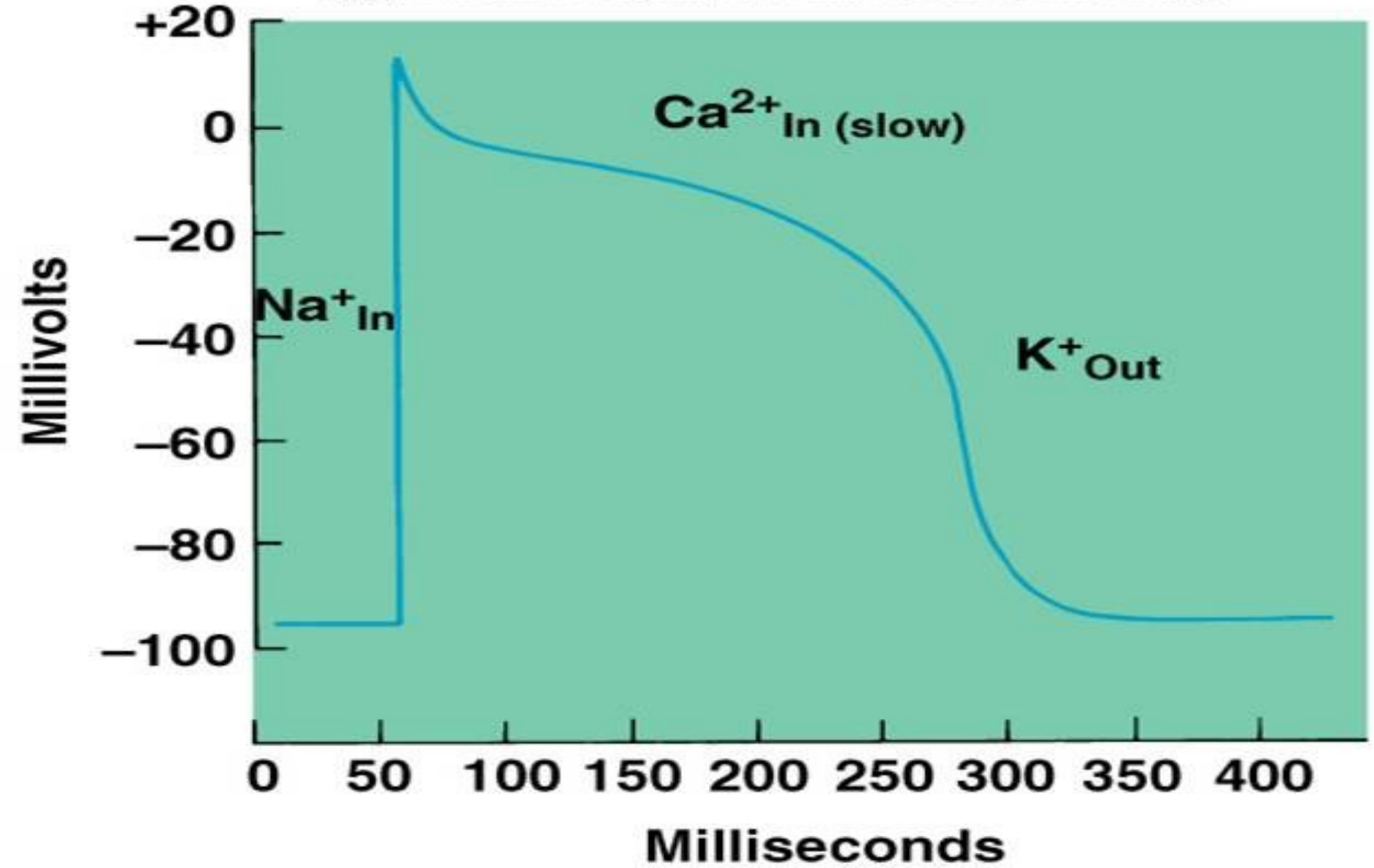
- Atrial syncytium (walls of the atria) and ventricular syncytium (walls of the ventricles).
- Fibrous tissue separates them so potentials are normally conducted only by way of a specialized conductive system (the A-V bundle).
- The atria contract a short time ahead of the ventricles. (allows effective pump action).

Action Potentials



Cardiac muscle action potential

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Fast Na channels

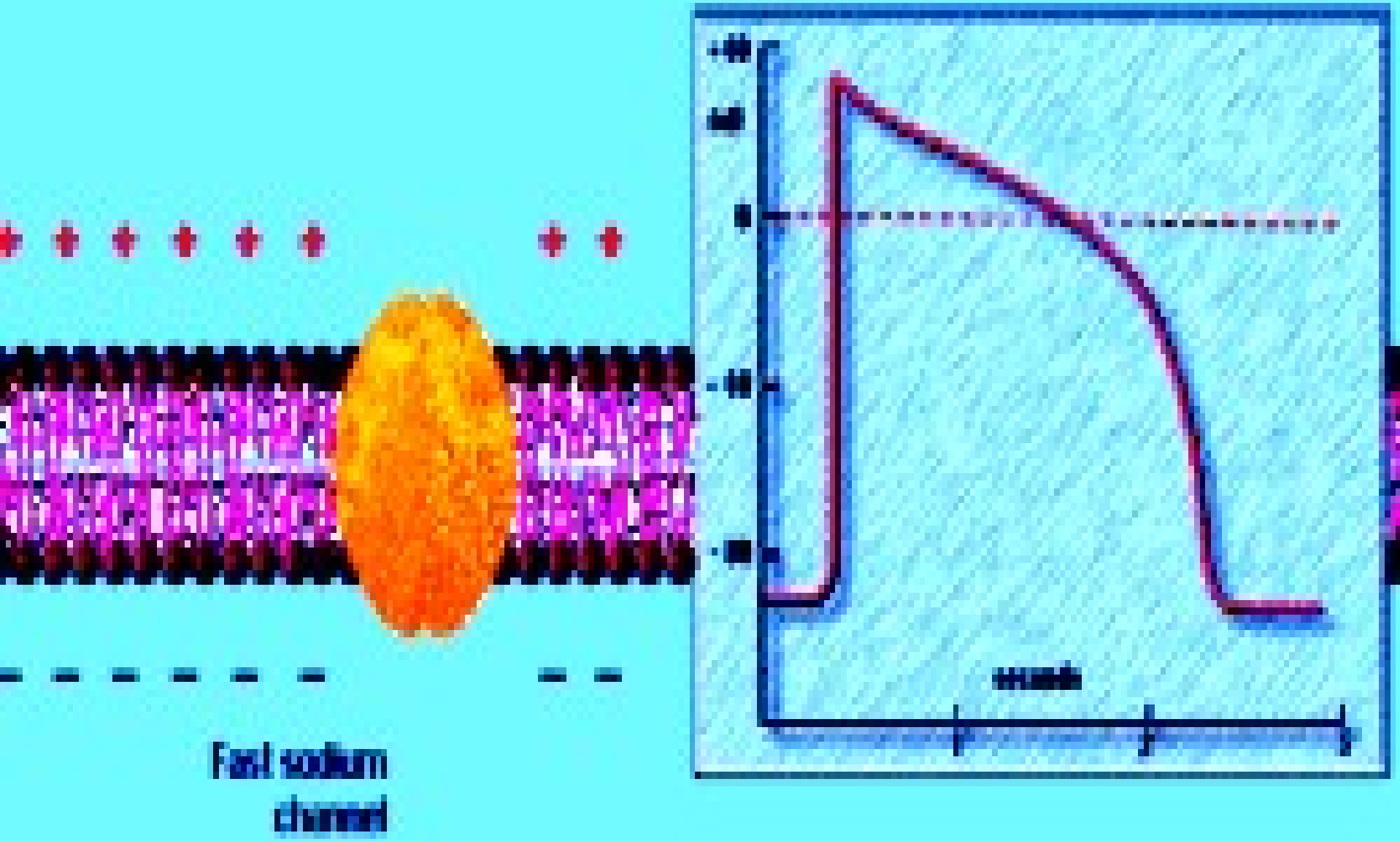
- Remain open for only a few 10,000ths of a second and then abruptly close.
- Allow tremendous amounts of Na ions to enter the cell.

Slow ca channels

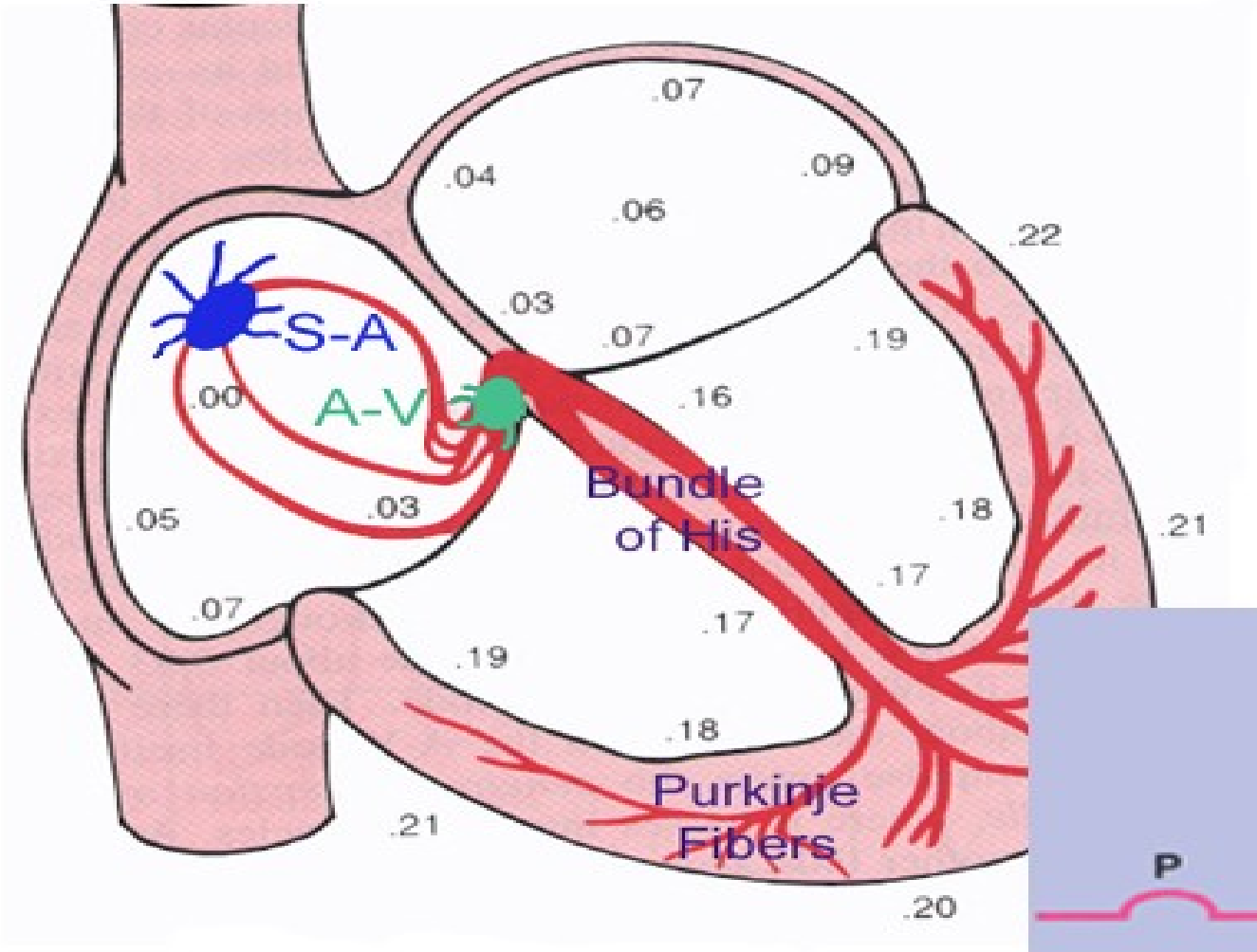
- Slower to open.
- Remain open for several tenths of a second.
- A large quantity of Ca and Na flows through causing:

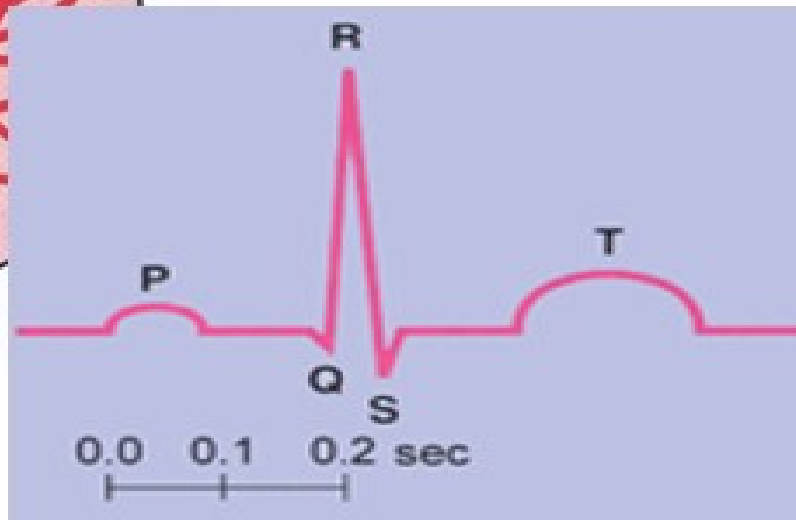
1. The plateau.
2. Help to excite the muscle contractile process.

Depolarization - Initial



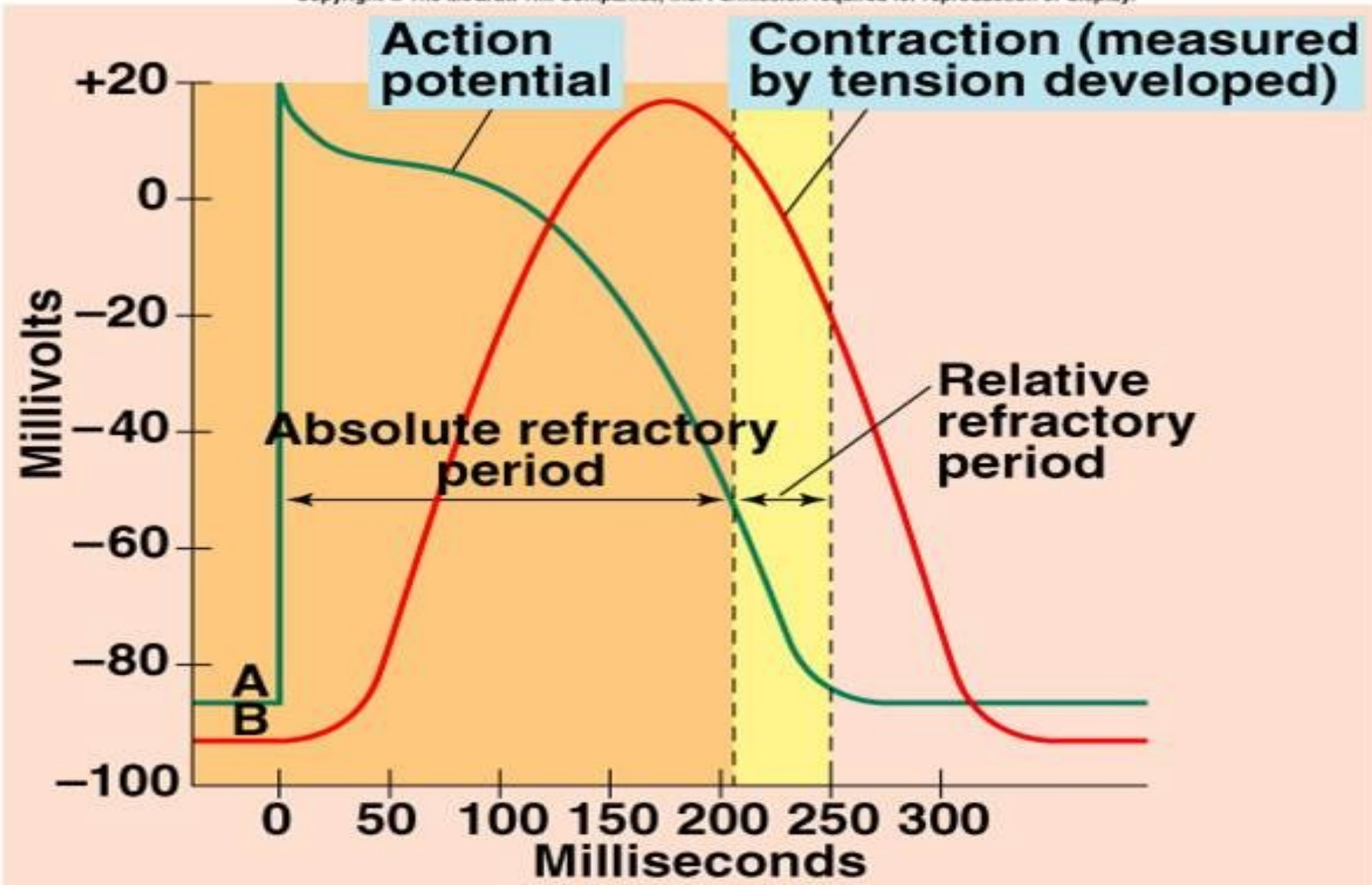
Velocity of signal conduction

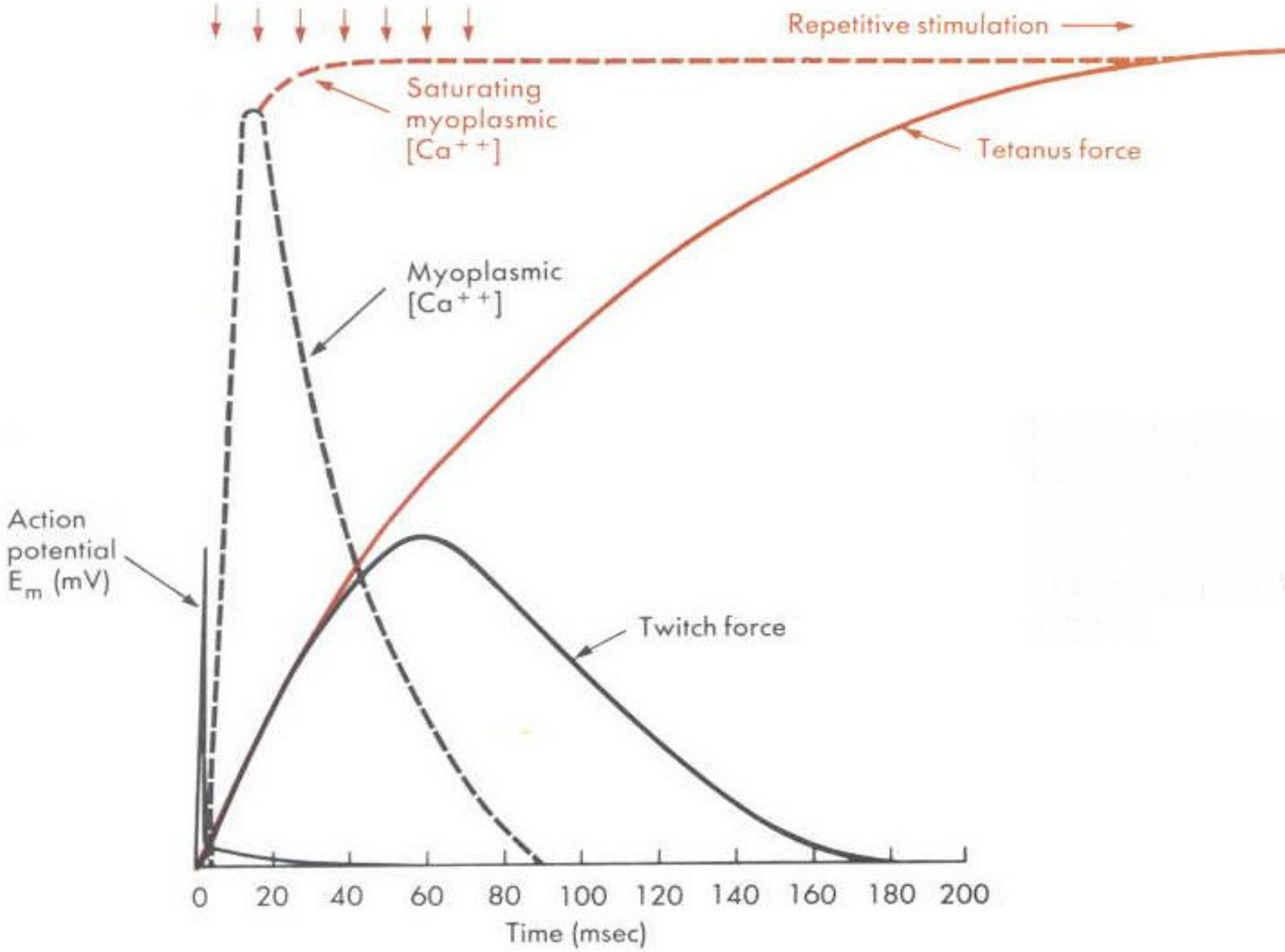




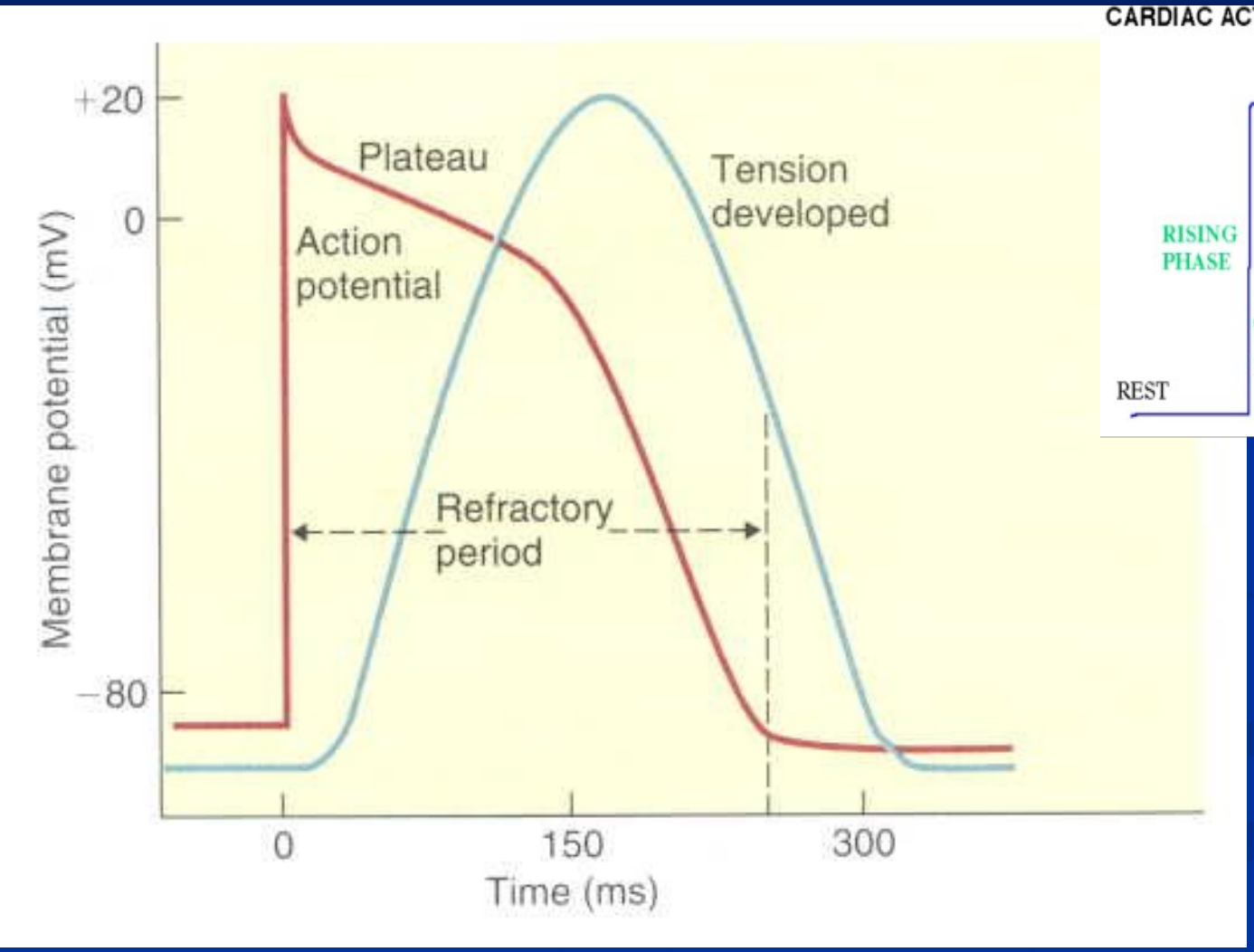
Refractory period

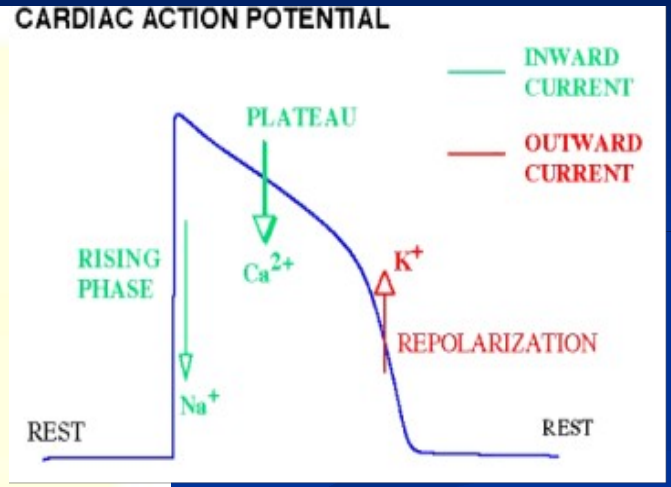
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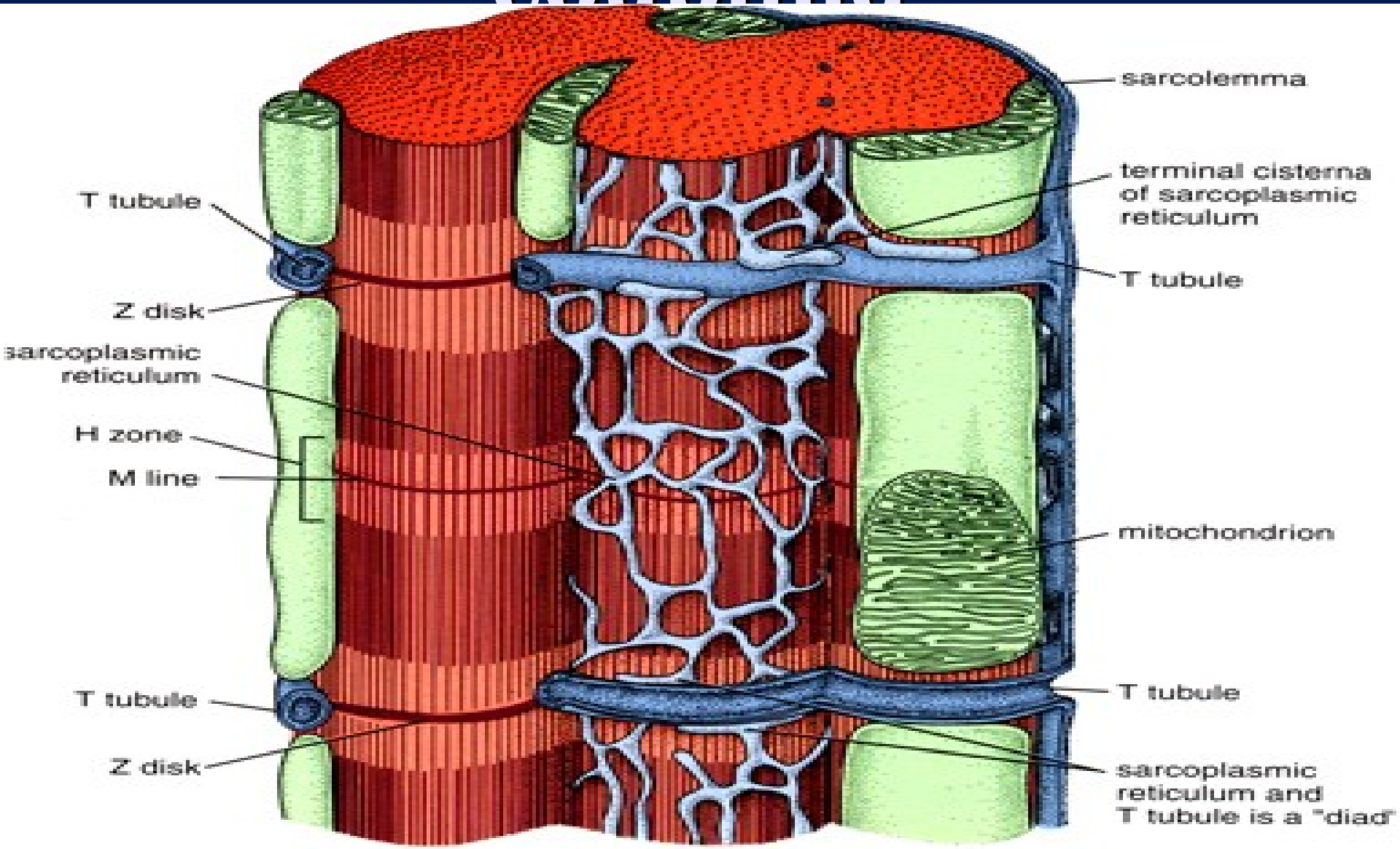

Repetitive stimulation
Saturating myoplasmic [Ca++]
Tetanus force
Myoplasmic [Ca++]
Action potential E_m (mV)
Twitch force
0 20 40 60 80 100 120 140 160 180 200
Time (msec)

Electrical: action potential

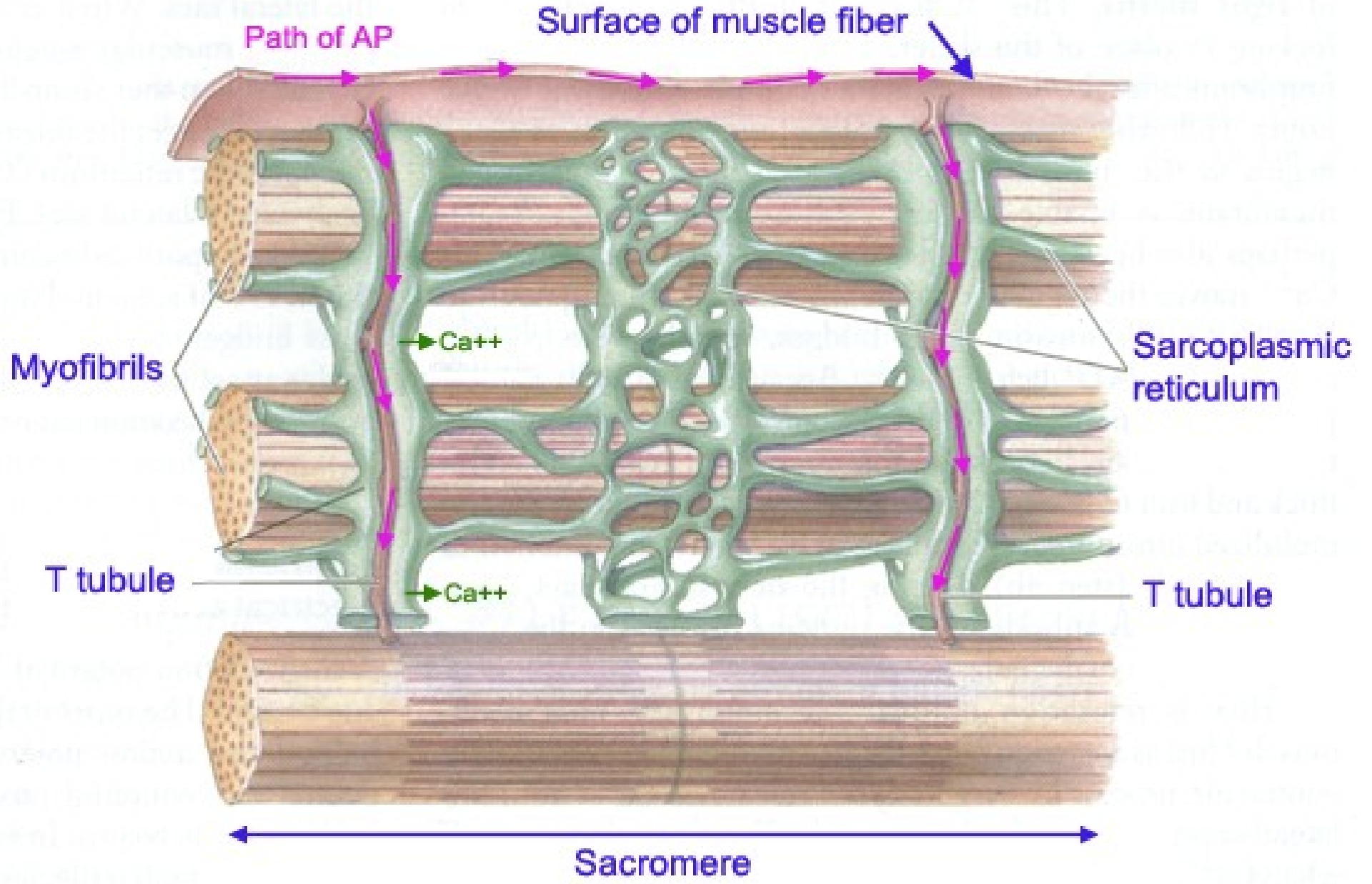


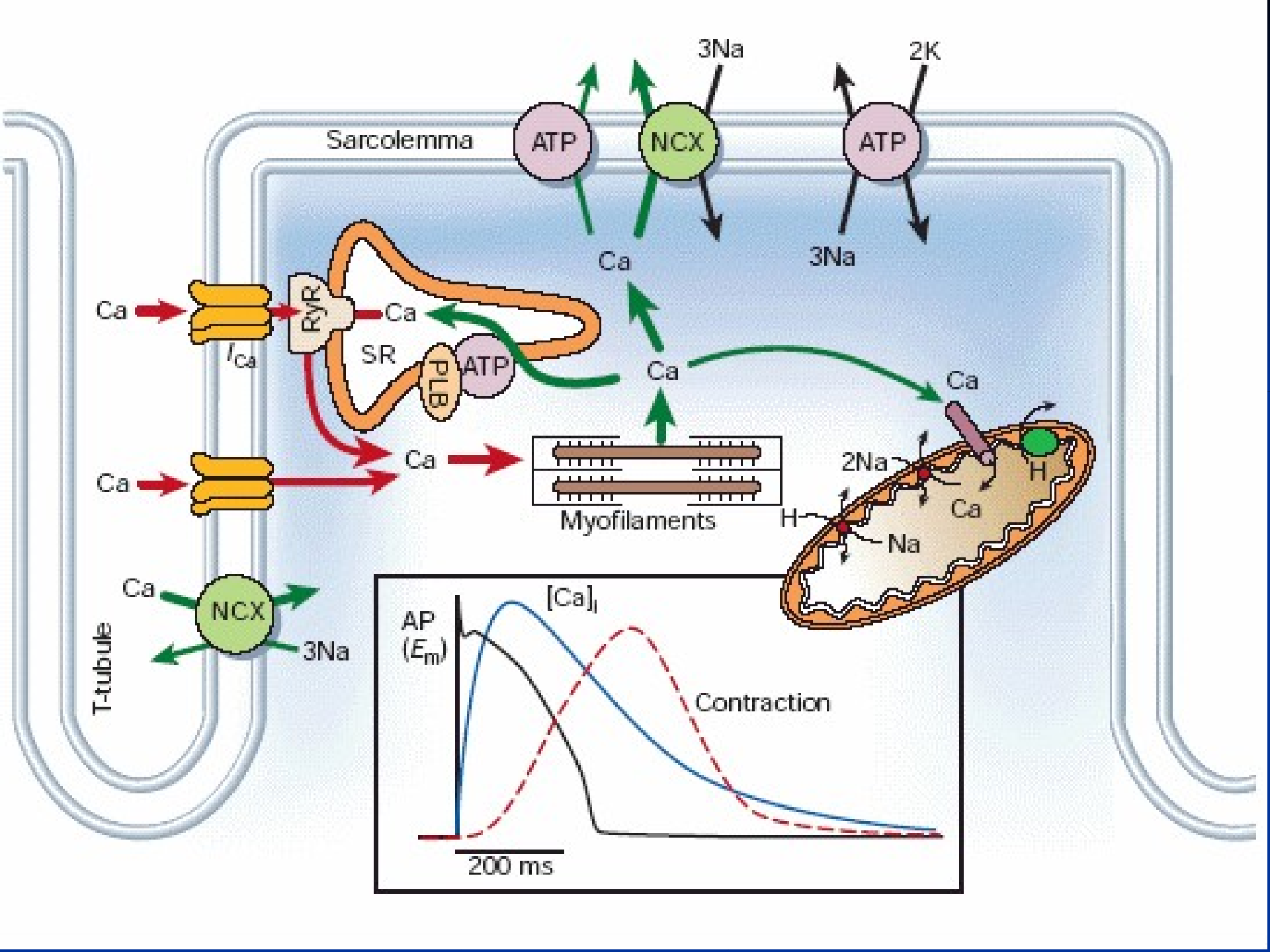


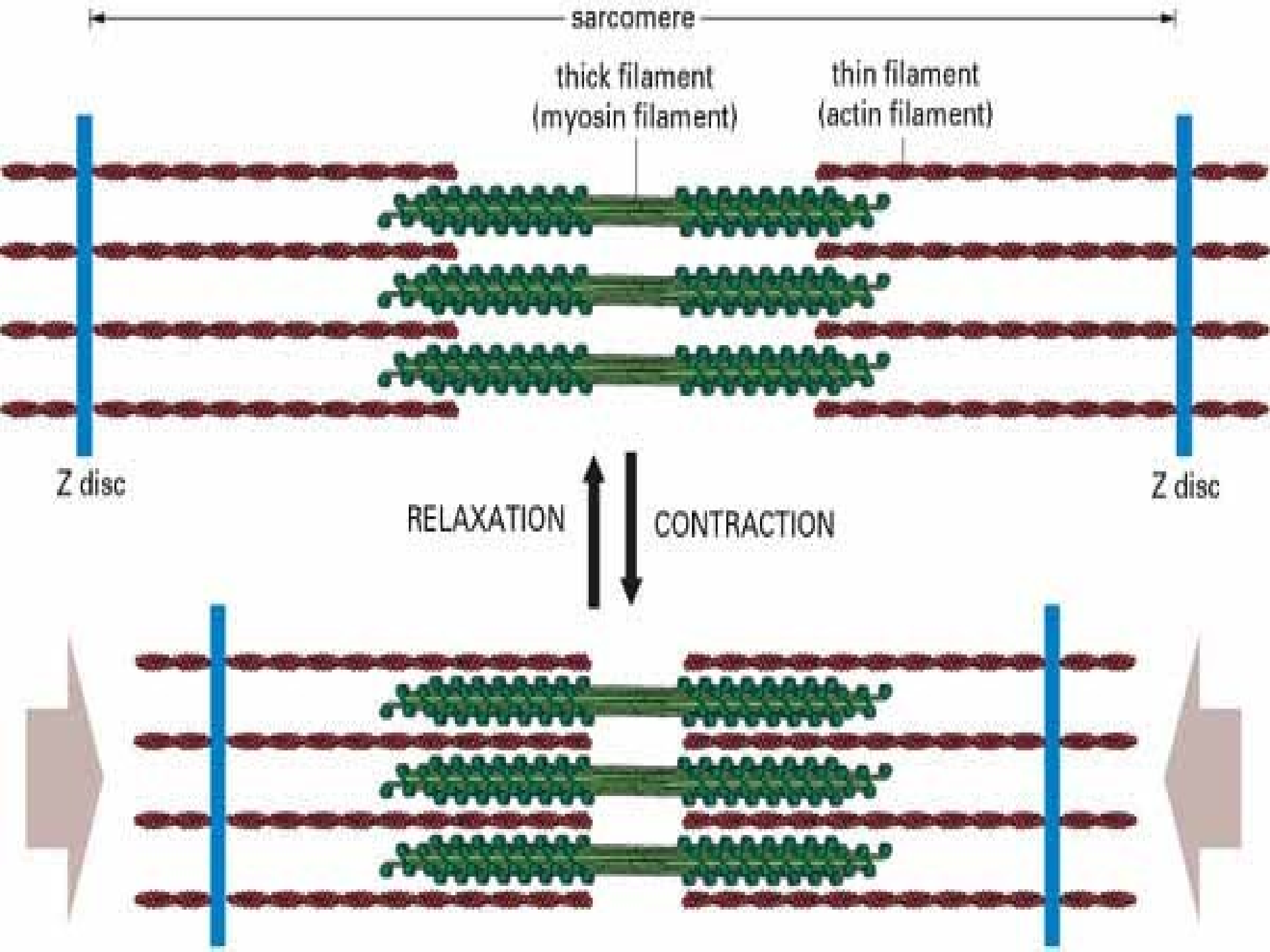
Excitation-contraction coupling

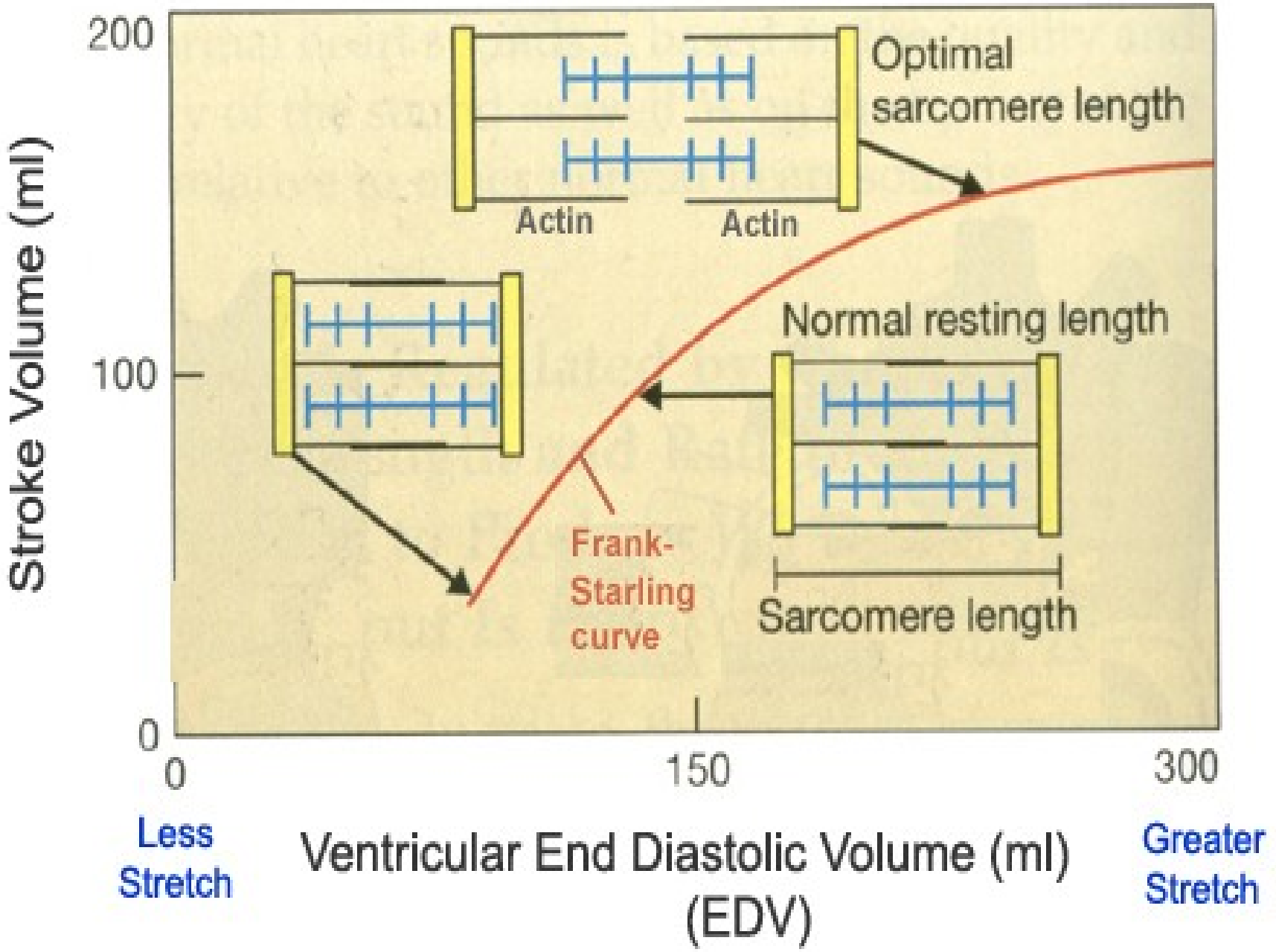


Role of Action Potential and Ca++ in Muscle Contraction



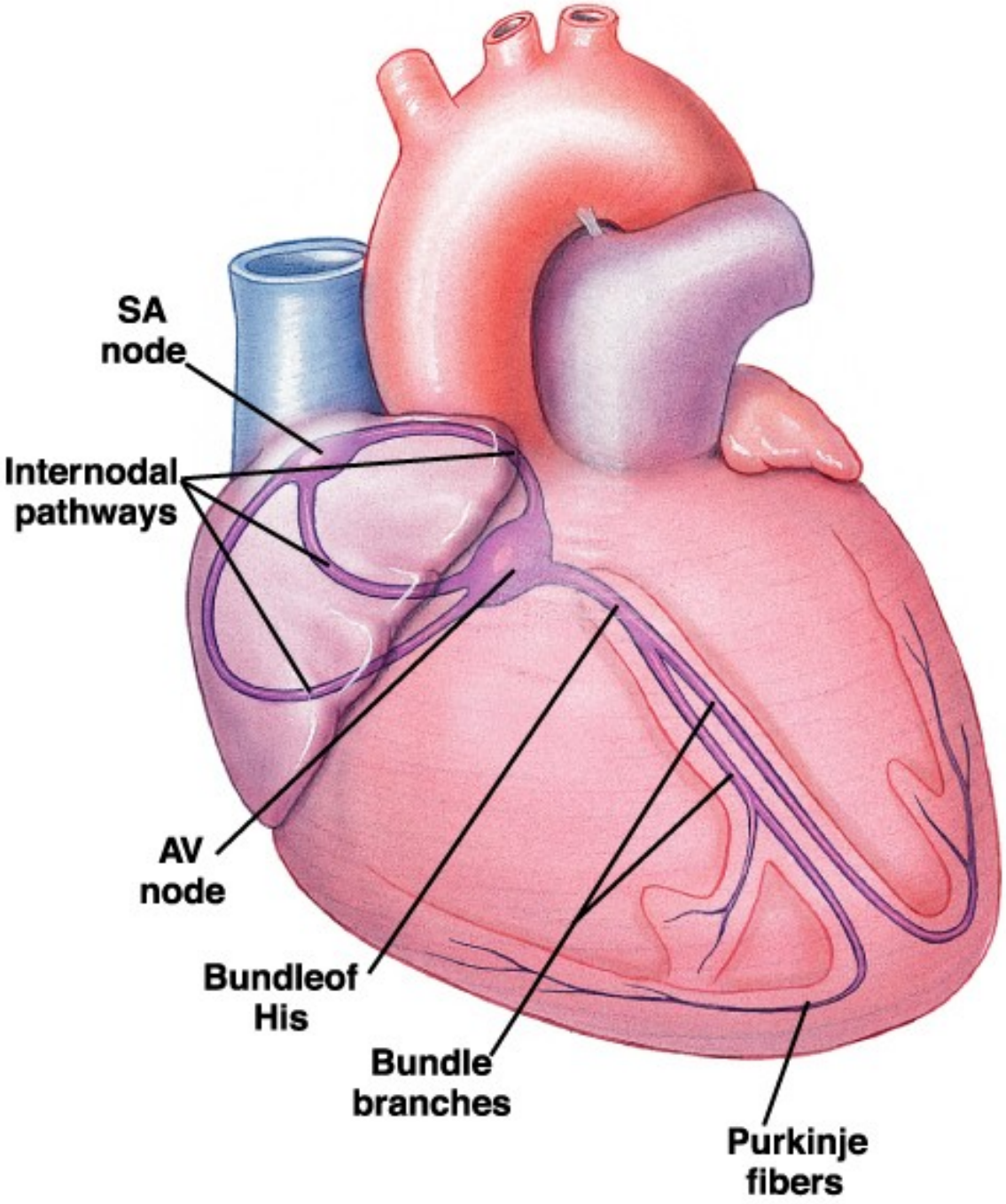

3Na
2K
Sarcolemma
ATP
NCX
ATP
3Na
Ca
Ca
RyR
Ca
SR
PLB
ATP
Ca
Ca
Ca
Ca
Myofilaments
2Na
H
Ca
H
Na
Ca
NCX
3Na
T-tubule
AP
(E_m)
$[Ca]_i$
Contraction
200 ms

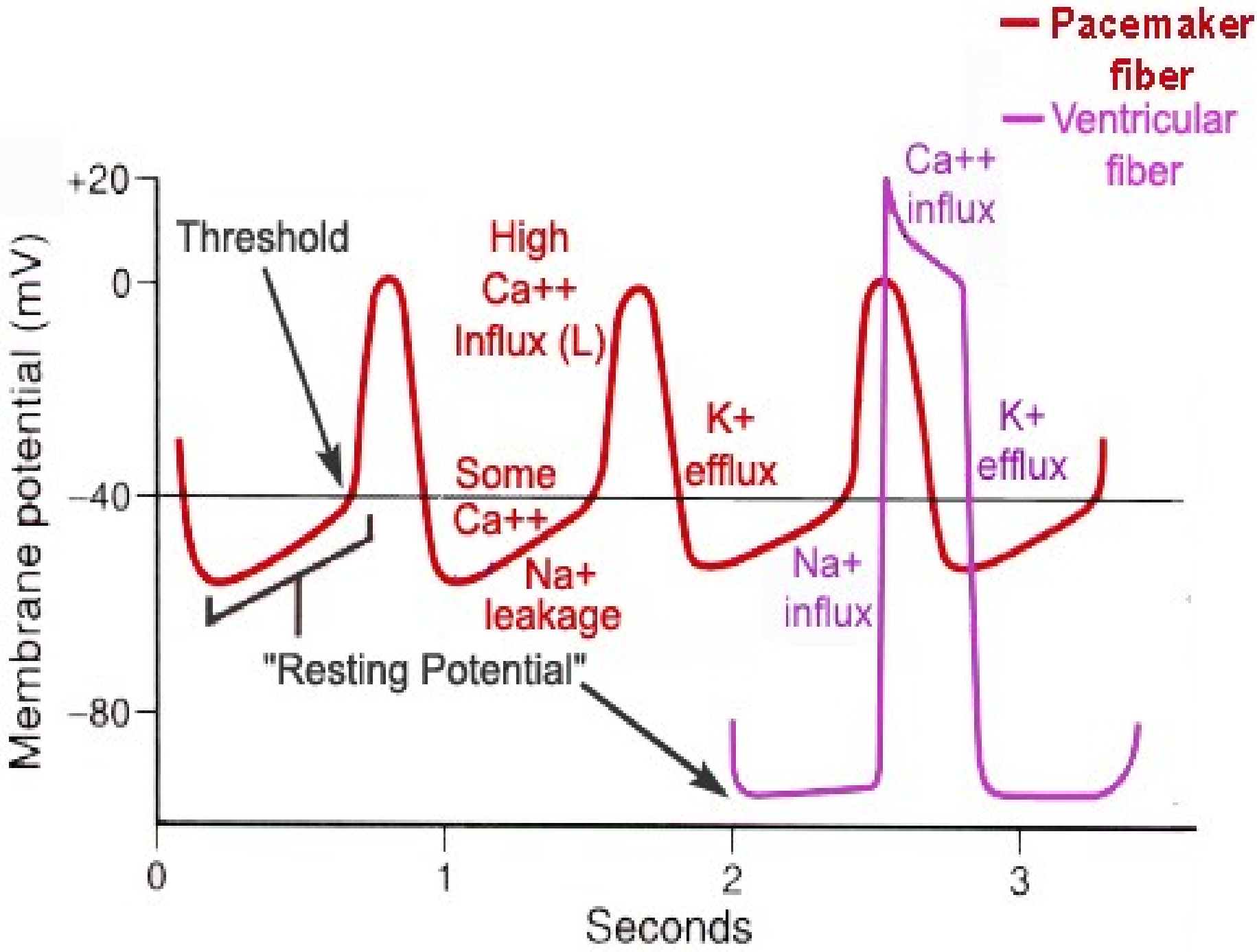

sarcomere
thick filament
(myosin filament)
thin filament
(actin filament)
Z disc
Z disc
RELAXATION
CONTRACTION


200
100
0
Stroke Volume (ml)
Optimal
sarcomere length
Actin
Actin
Normal resting length
Frank-
Starling
curve
Sarcomere length
0
150
300
Less
Stretch
Ventricular End Diastolic Volume (ml)
(EDV)
Greater
Stretch

Duration of contraction and effect of heart rate

- The duration of contraction of cardiac muscle is mainly a function of the duration of the action potential-about 0.2 sec in atrial muscle and 0.3 in ventricular muscle.
- At 72beats/min the period of contraction is about 40% of the cycle. At 3x normal this period is about 65% and the heart does not remain relaxed long enough to allow complete filling.


SA
node
Internodal
pathways
AV
node
Bundleof
His
Bundle
branches
Purkinje
fibers


Pacemaker fiber
Ventricular fiber
Membrane potential (mV)
+20
0
−40
−80
Threshold
High Ca++ Influx (L)
Some Ca++
Na+ leakage
K+ efflux
Ca++ influx
Na+ influx
K+ efflux
"Resting Potential"
0
1
2
3
Seconds

Cardiac output

- The volume of blood pumped by the heart per minute.

$$CO = HR \times SV$$

$$BP = CO \times SVR$$

- *At rest, CO is 5lt/min*
- *In exercise it may rise to 35 L/min.*
- *CI=CO/BSA=2.5-4.2 L/min/m2).*

The CO is pivotal in maintaining arterial BP (BP=CO X SVR) and oxygen delivery.

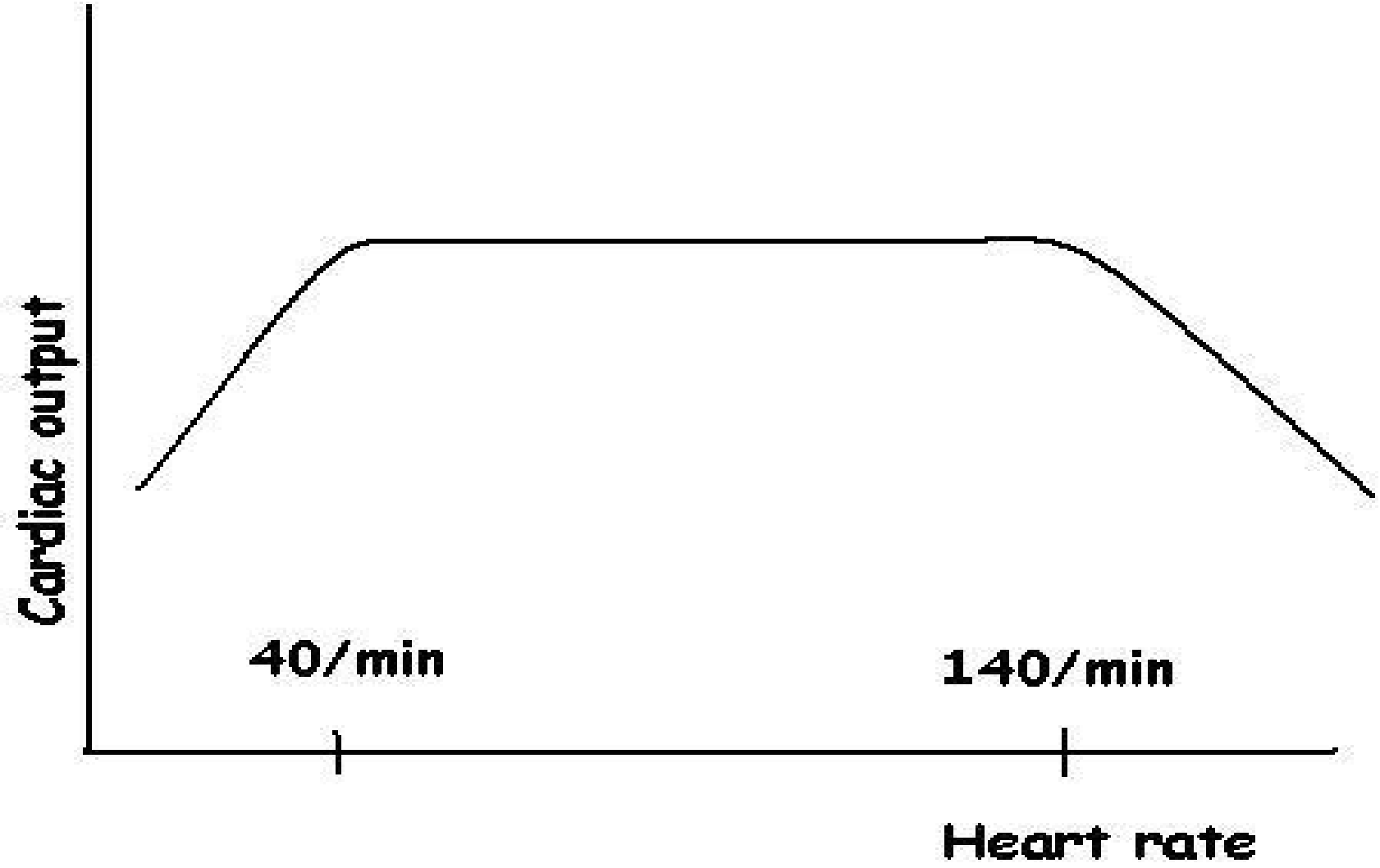
- *DO2= CO x Hb x 1.31 x % Sat*

mlO2/min lt/min gr/lt mlO2/grHb

Distribution of CO

- Heart 5%
  Brain 14%
  Muscle 20%
  Kidneys 22%
  Liver 25%
  Rest of the body 14%

HR effect on CO (Treppe effect)



Stroke Volume

Stroke volume: the volume of blood ejected by the ventricle with a single contraction.

SV = EDVol – ESVol

EF=SV expressed as a percentage.

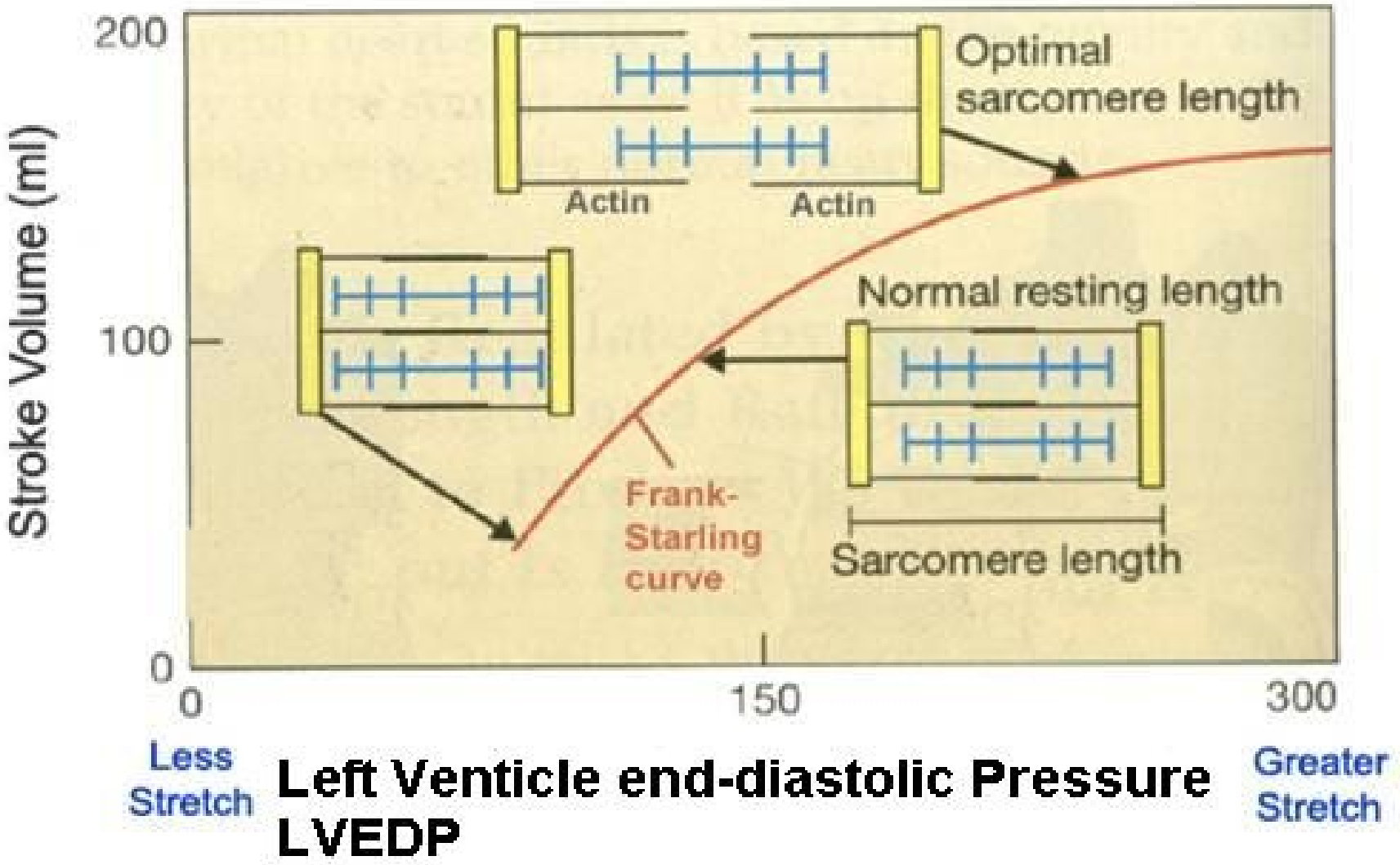
Determinants of SV:

1. Preload
2. Afterload
3. Contractility

Preload

1. ***Preload**: The initial length of the muscle fibre before contraction.*
   - *The filling pressure of the ventricles.*
   - *Described by the Frank-Starling curve.*
   - *In clinical practice CVP(RV) and PAWP(LV) are used. (Several limitations).*
     - *In normal individuals, CVP correlates with filling pressures of both right and left atria, this is not true in the following disease states:*
     - *Left ventricular failure with pulmonary oedema*
       Interstitial pulmonary oedema
       Chronic pulmonary disease
       Valvular heart disease

The Frank-Starling curve

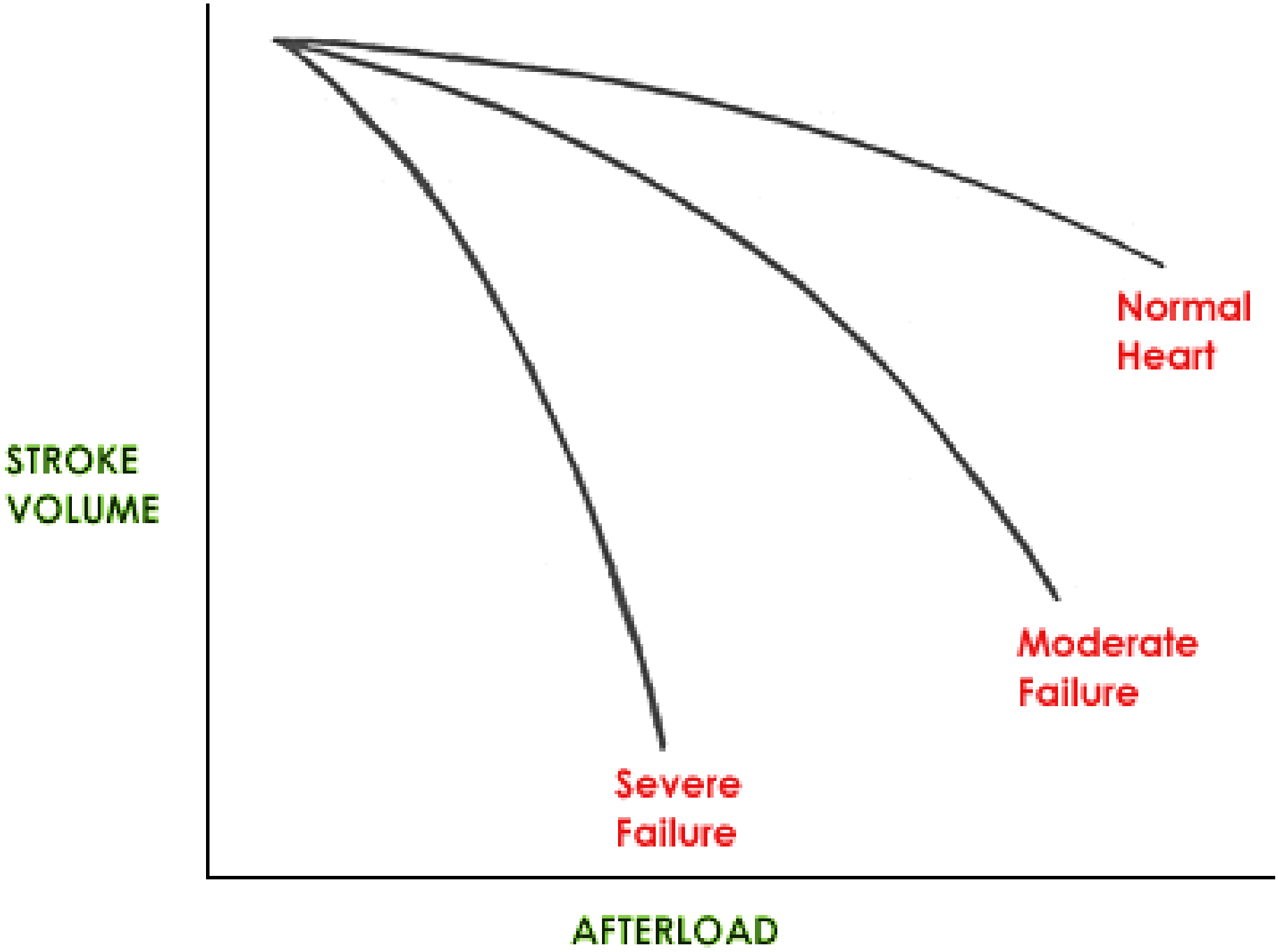


Frank-Starling law

- In isolated myocytes the tension developed during contraction is dependent on the initial length of the muscle fibre.
- Laplace's law: $P=2T/R$
- For an isolated muscle fibre it translates into a relationship between SV and LVEDP

Afterload

2. **Afterload:** The tension developed in the ventricular wall during systole.
   - Affected by preload, SVR, and external pressure (for example Positive Pressure Ventilation)
   - How forcefully the ventricles have to contract to eject blood.


STROKE VOLUME
Normal Heart
Moderate Failure
Severe Failure
AFTERLOAD

3. *Contractility:* *The amount of mechanical work that is done for a given preload and afterload.*

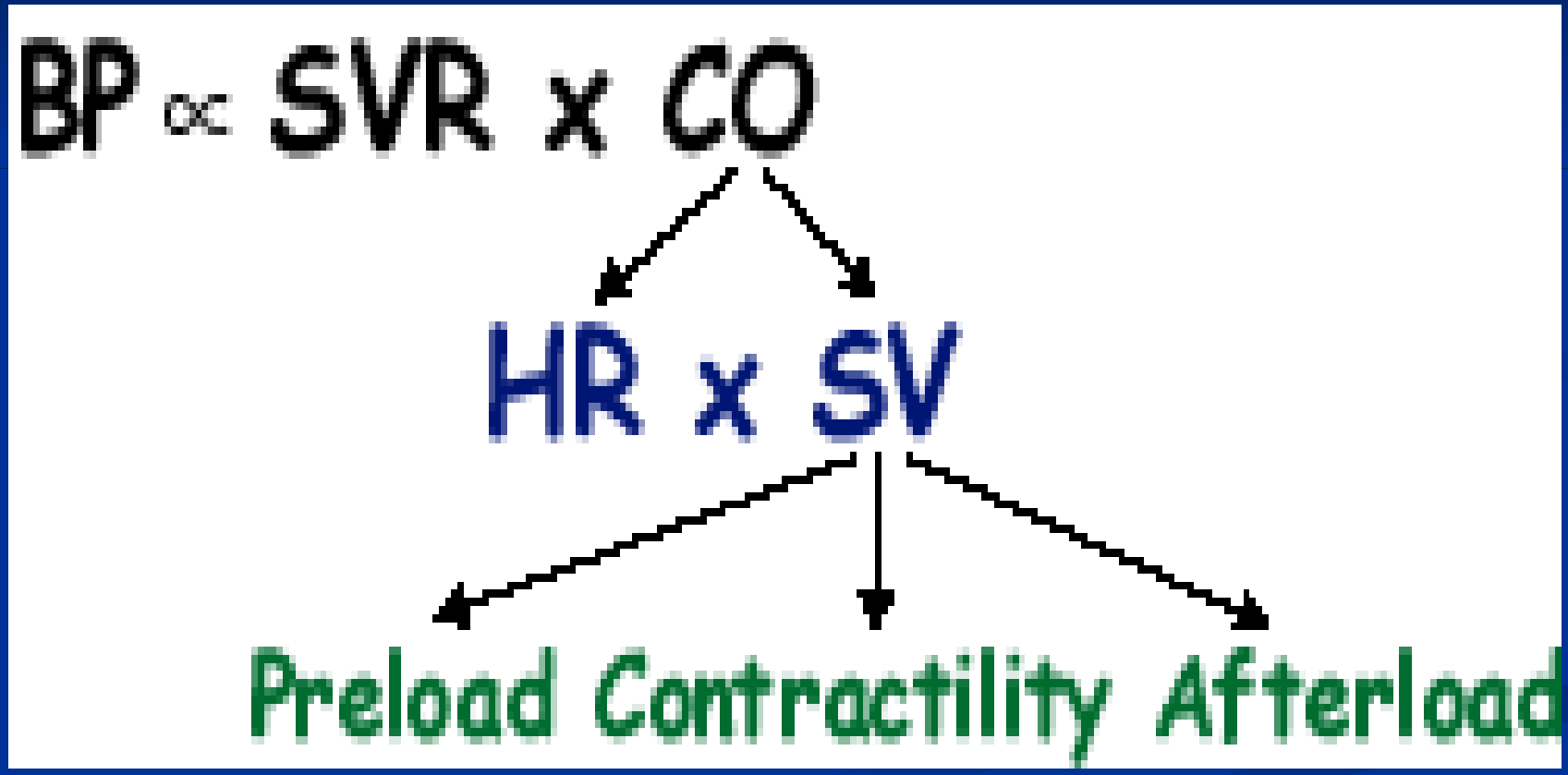
Increased contractility

1. *Sympathetic stimulation*
2. *Positive inotropes*

Reduced contractility

1. *Parasympathetic stimulation*
2. *Negative inotropes (b-block)*
3. *Ischaemia/Infarction*
4. *Hypoxaemia*
5. *Acidocis*
6. *HypoCalcaemia*

Haemodynamics



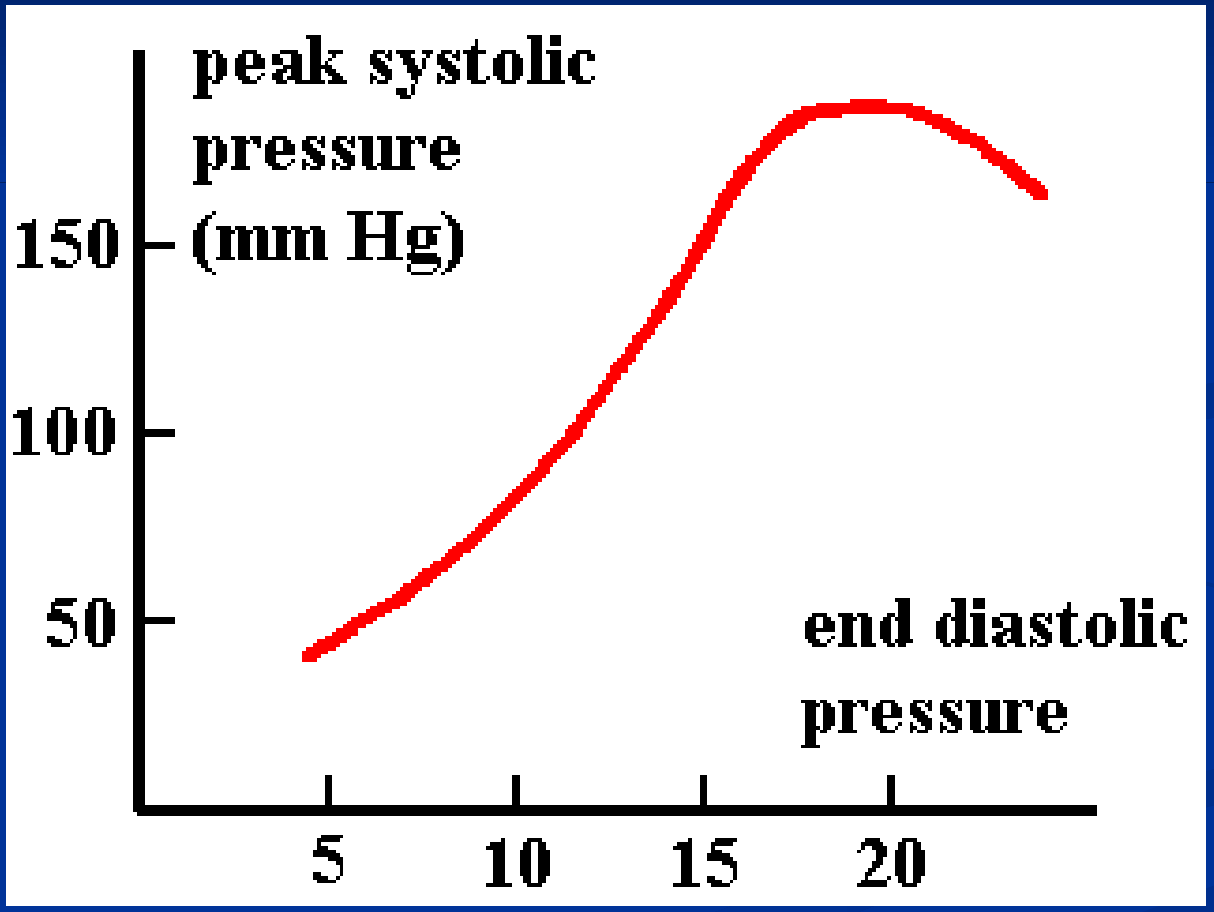
Clinical application

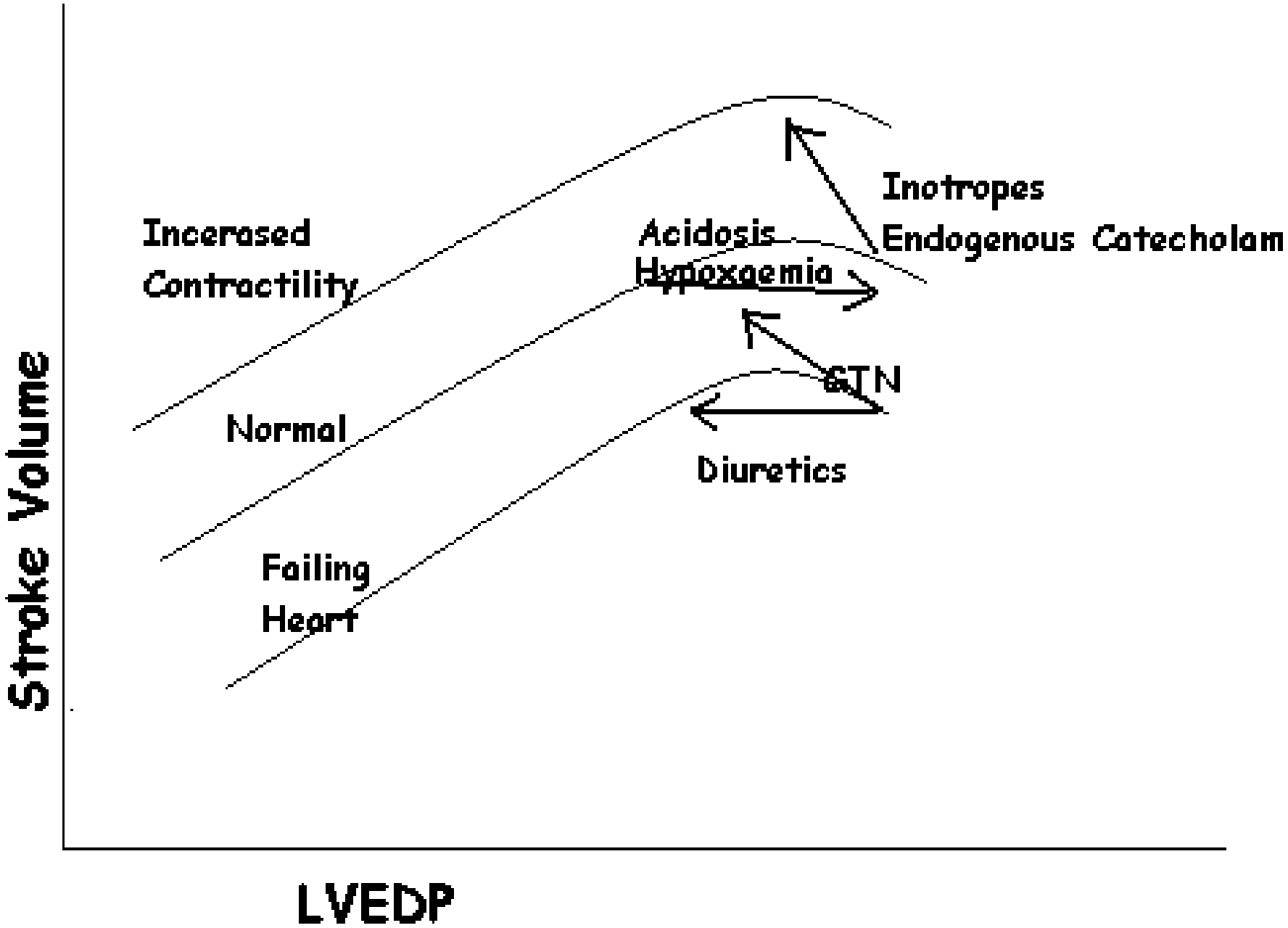
- *In low CO states leading to hypoperfusion, the*

1. *HR,*
2. *preload,*
3. *afterload and*
4. *contractility should be addressed- **in that order**.*

(No point trying to pharmacologically improve the contractility of a heart that is either too empty or too distended to eject).

Starling's Law




Stroke Volume
LVEDP
Incerased
Contractility
Normal
Failing
Heart
Acidosis
Hypoxaemia
Inotropes
Endogenous Catecholam
GTN
Diuretics

Methods of measuring CO

- Invasive

Dilution/ thermodilution techniques

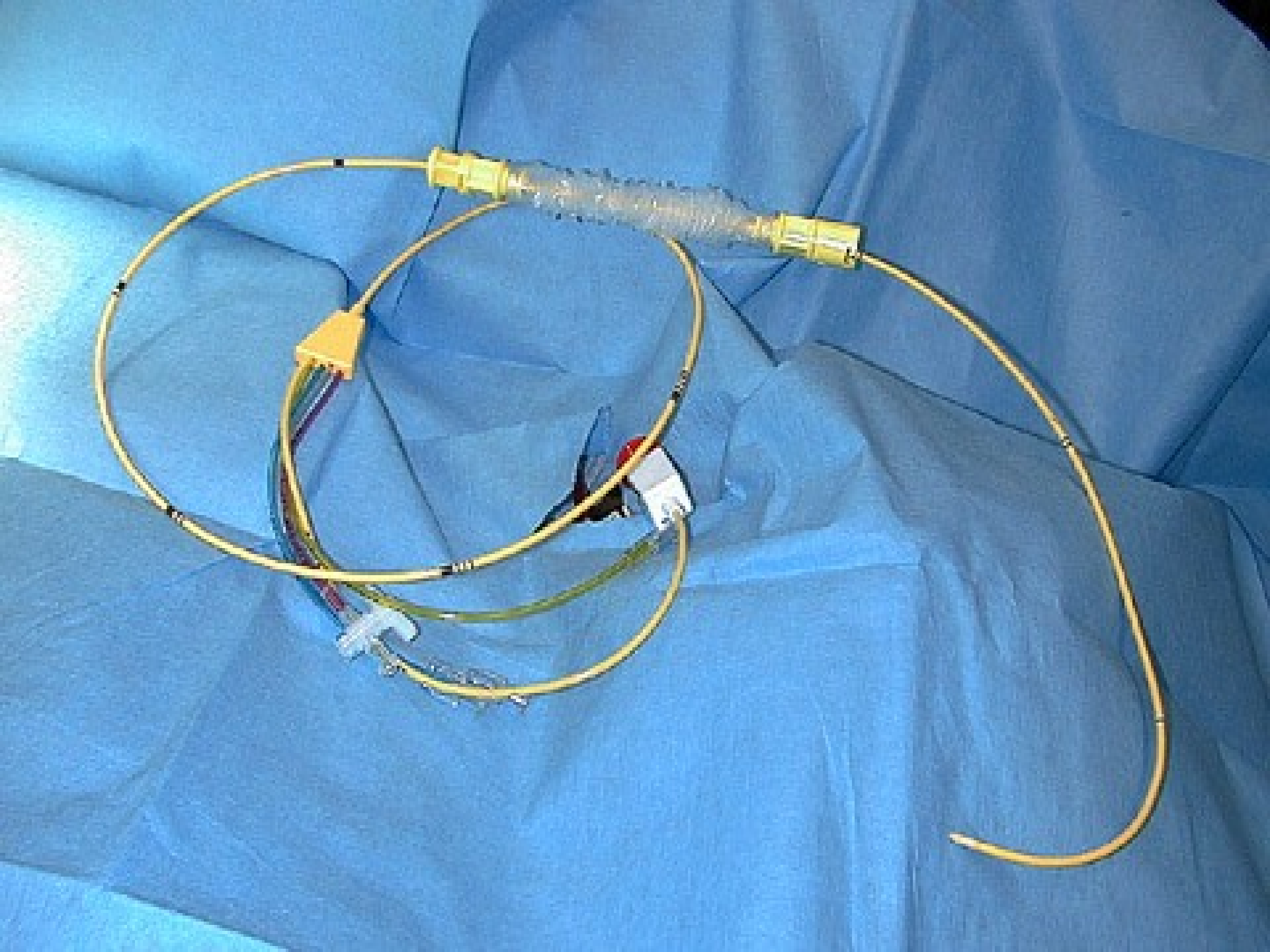
- Non invasive

TOE

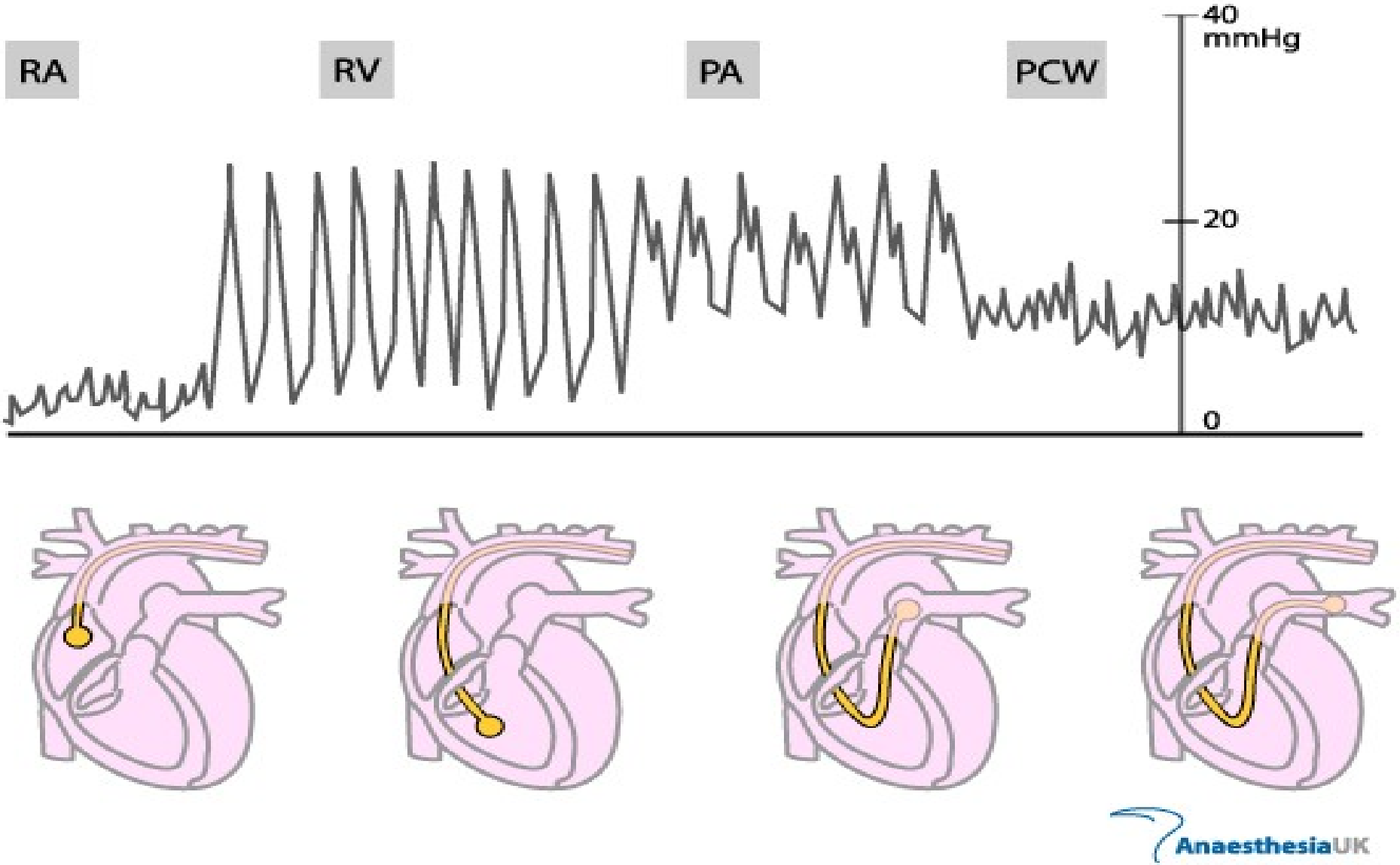
Transthoracic Echo

PA catheter

- Proximal lumen: 25 cm from tip, lies in right atrium, measures central venous pressure (CVP).
- Distal lumen: at tip of catheter, lies in a branch of the pulmonary artery, connected to a pressure transducer.
- Balloon lumen: permits introduction of 1.5 ml of air into the balloon at the distal tip.
- Thermistor lumen: bead situated 4 cm from the tip of the catheter and measures temperature.



Characteristic intracardiac pressure wave forms during passage through the heart



AnaesthesiaUK

Complications

1. Arrhythmias on insertion
2. Knotting of catheter in right ventricle
3. Balloon rupture
4. Pulmonary infarction
5. Infection

The Fick principle

- Fick described the following relationship in the 19th century:

  $Q = M / (V - A)$

- Q is the volume of blood flowing through an organ in a minute.
- M the number of moles of a substance added to the blood by an organ in one minute,
- V and A are the venous and arterial concentrations of that substance.
- This principle can be used to measure the blood flow through any organ that adds substances to, or removes substances from, the blood. The heart does not do either of these but the CO equals the pulmonary blood flow, and the lungs add oxygen to the blood and remove carbon dioxide from it.

Fick's method

PVein[O2]=200ml/lt

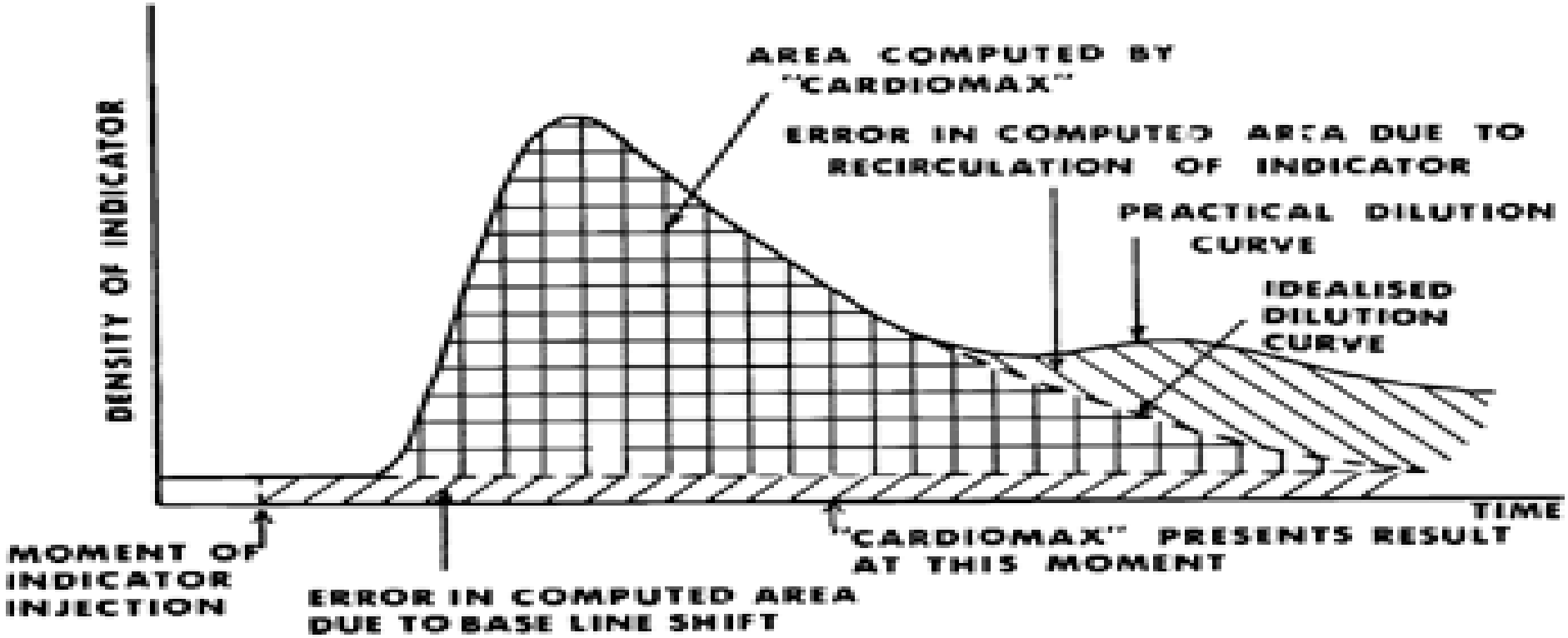
PArtery[O2]=150ml/lt

At rest the blood takes 250ml/min of O2 from the lungs.

CO (at rest)=5lt/min

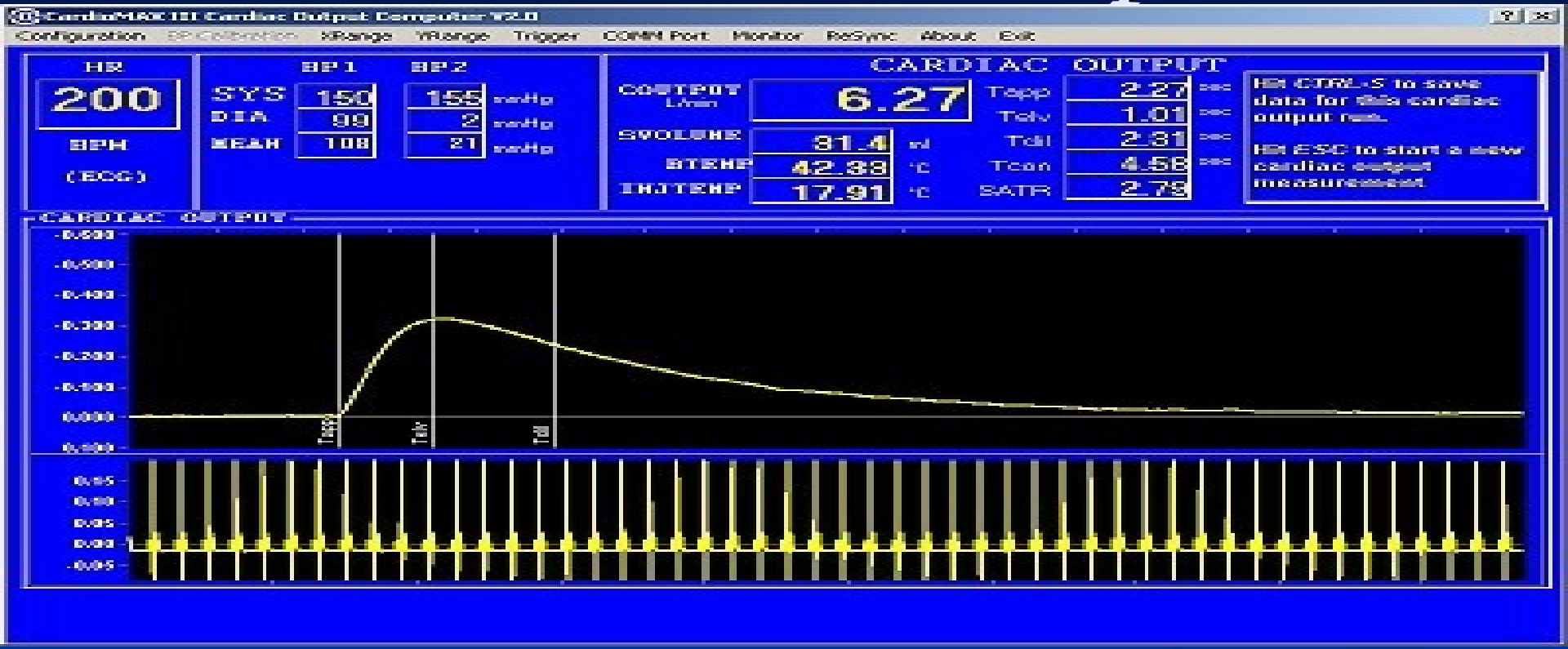
Adolf Fick 1829-1901

Dilution techniques



- *A known amount of dye is injected into the pulmonary artery, and its concentration is measured peripherally. Indocyanine green is suitable due to its low toxicity and short half-life. A curve is achieved, which is replotted semi-logarithmically to correct for recirculation of the dye. CO is calculated from the injected dose, the area under the curve (AUC) and its duration. (Short duration indicates high CO).*

Thermodilution technique



- *5-10 ml cold saline injected through the port of a pulmonary artery catheter. Temperature changes are measured by a distal thermistor. A plot of temperature change against time gives a similar curve to the dye curve (but without the second peak).*
- *Calculation of CO is achieved using the Stewart-Hamilton equation.*
- *Application of this equation assumes three major conditions; complete mixing of blood and indicator, no loss of indicator between place of injection and place of detection and constant blood flow.*

Measured values

- **CVP**: Central Venous Pressure 1-6 mm Hg (reflects right atrial pressure).
- **PAP**: Mean Pulmonary Artery Pressure Systolic 15-30mm Hg, Diastolic 6-12mm Hg.
- **PCWP**: Pulmonary Capillary Wedge Pressure 6 - 12mm Hg. Estimates left atrial heart pressure and left ventricular end diastolic pressure.
- **CO**: Cardiac Output 3.5 - 7.5 L/min
- **Sv02**: Mixed venous partial pressure of oxygen (70 - 75%). Drawn from the end of the pulmonary artery catheter. Used to calculate how well oxygen is extracted by the tissues.

Non-invasive (TOE)

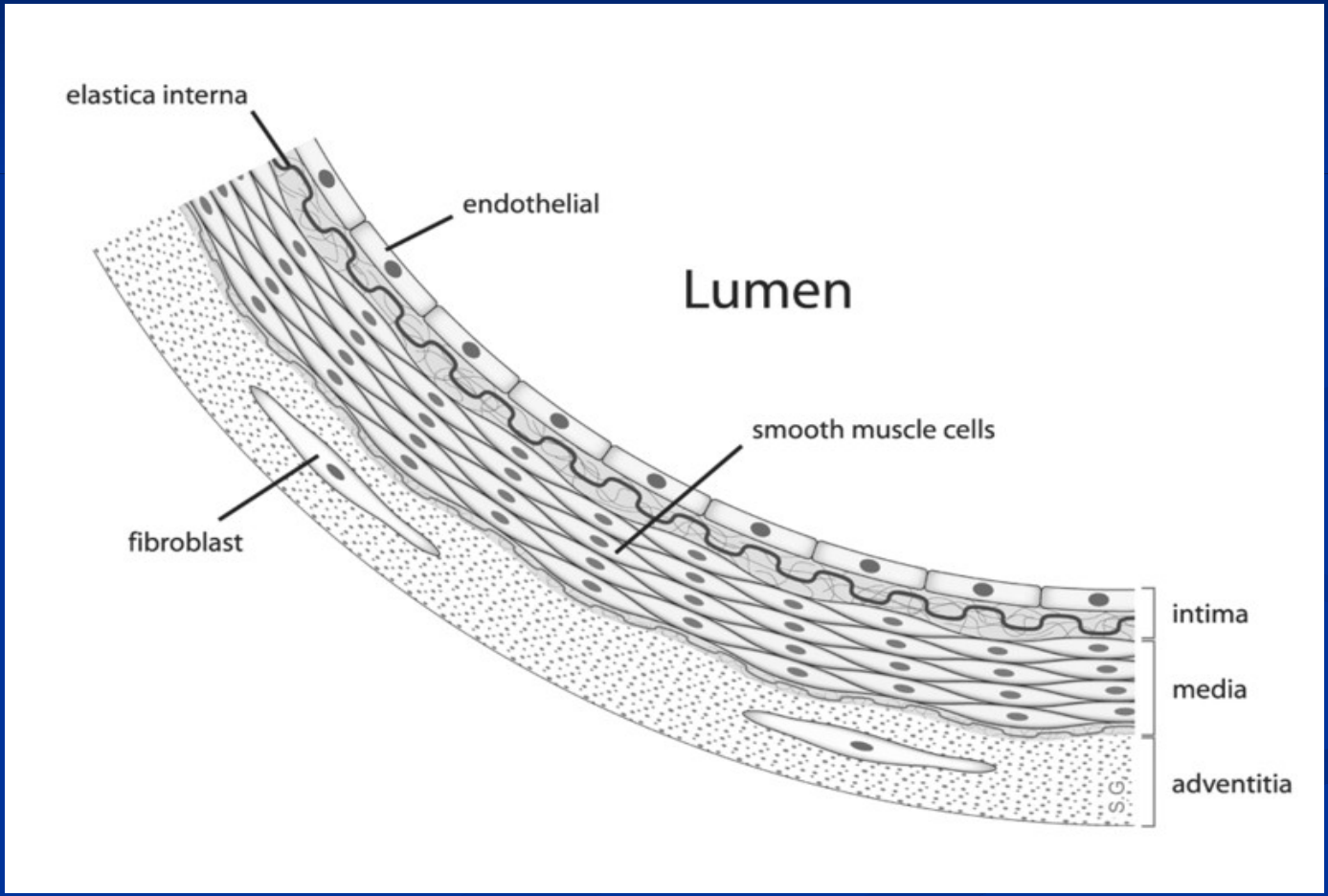
- *These machines transmit an ultrasonic vibration into the body and record the change in the frequency of the signal that is reflected off the red blood cells.*
- *Doppler techniques measure **velocity**, not flow; the flow could be obtained by integrating the signal over the cross-sectional area of the vessel.*
- *Transoesophageal echocardiography (TOE) can measure the **length of blood in the ascending aorta in unit time.** This is multiplied by the cross-sectional area of the aorta to give stroke volume.*

Ischaemic Heart Disease

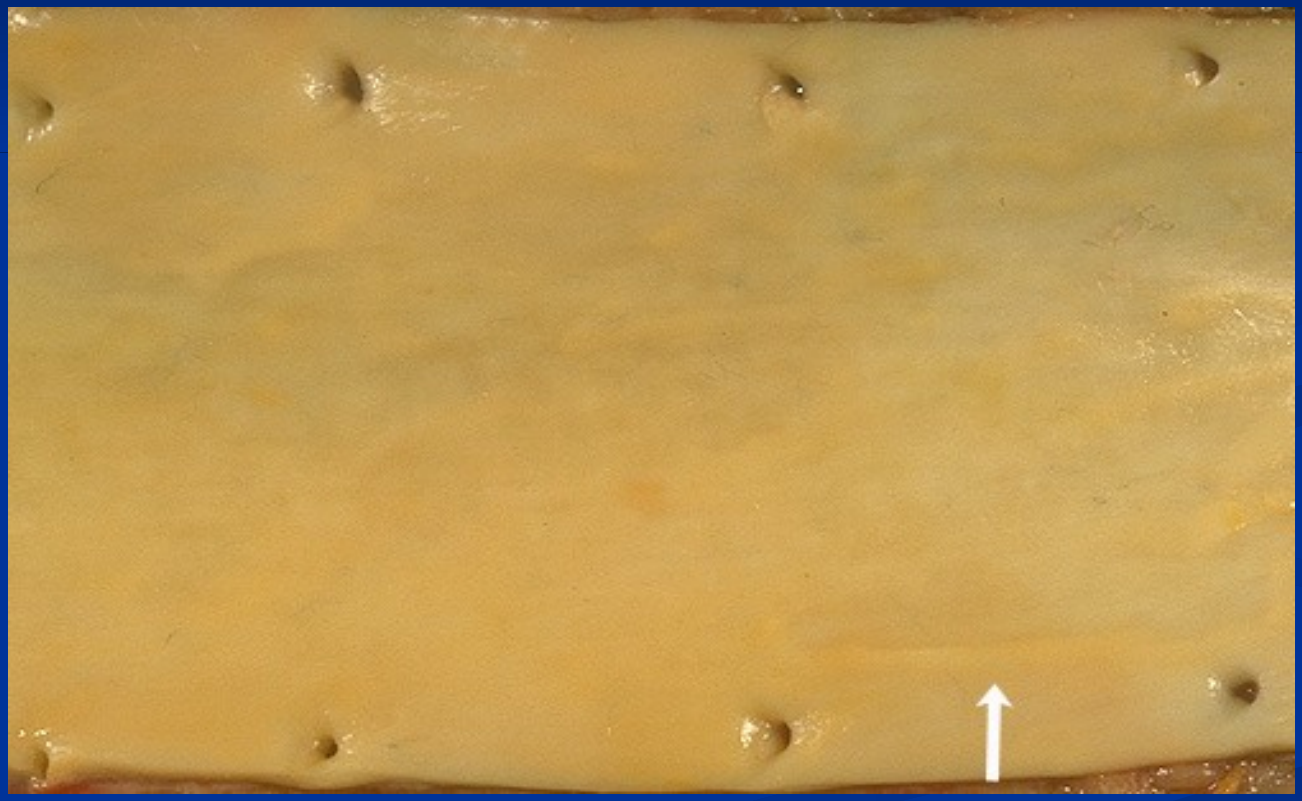
Normal Arterial Anatomy

- **Intima** – single layer endothelium + connective tissue
- Internal elastic lamina separates intima from media
- **Media** – mainly smooth muscle (aorta – elastic)
- **Adventitia** – fibrous connective tissue

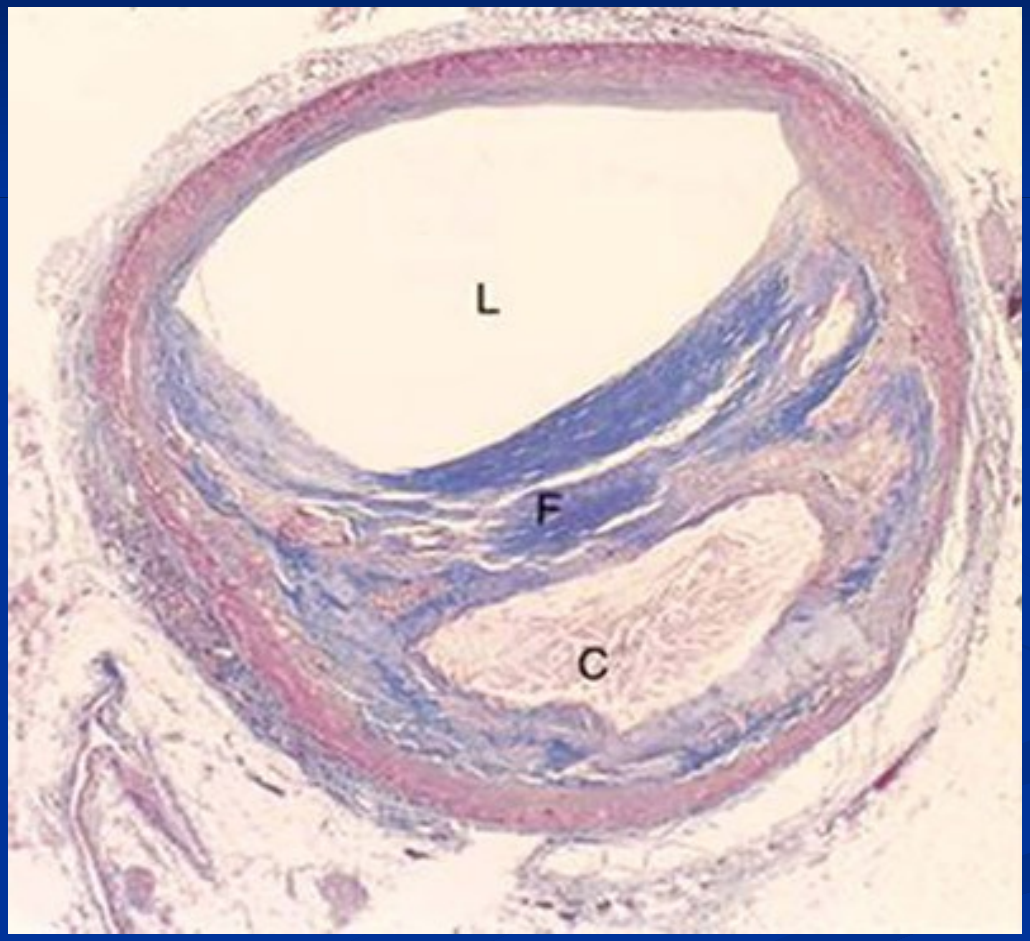
Normal Arterial Anatomy



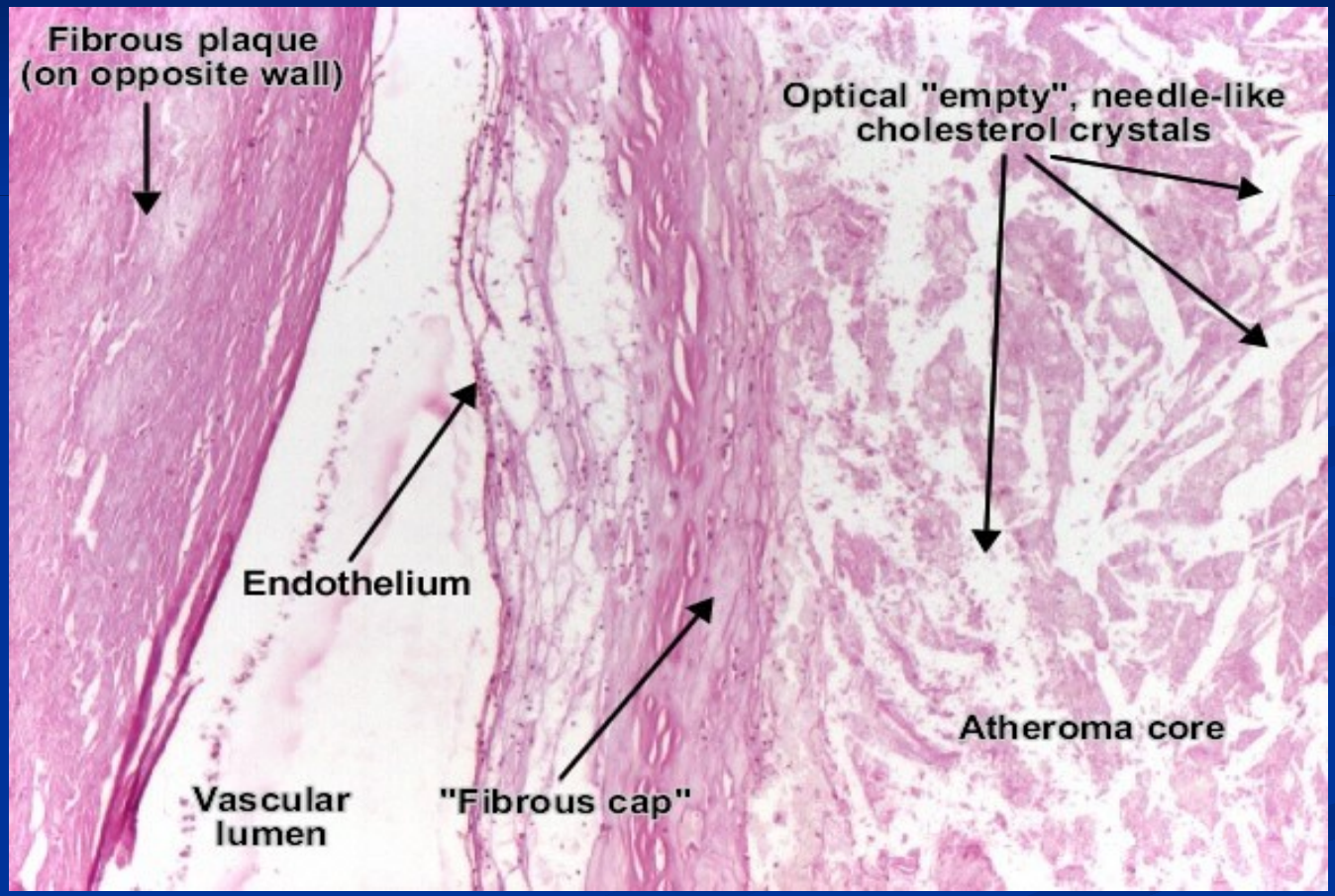
Fatty streaks



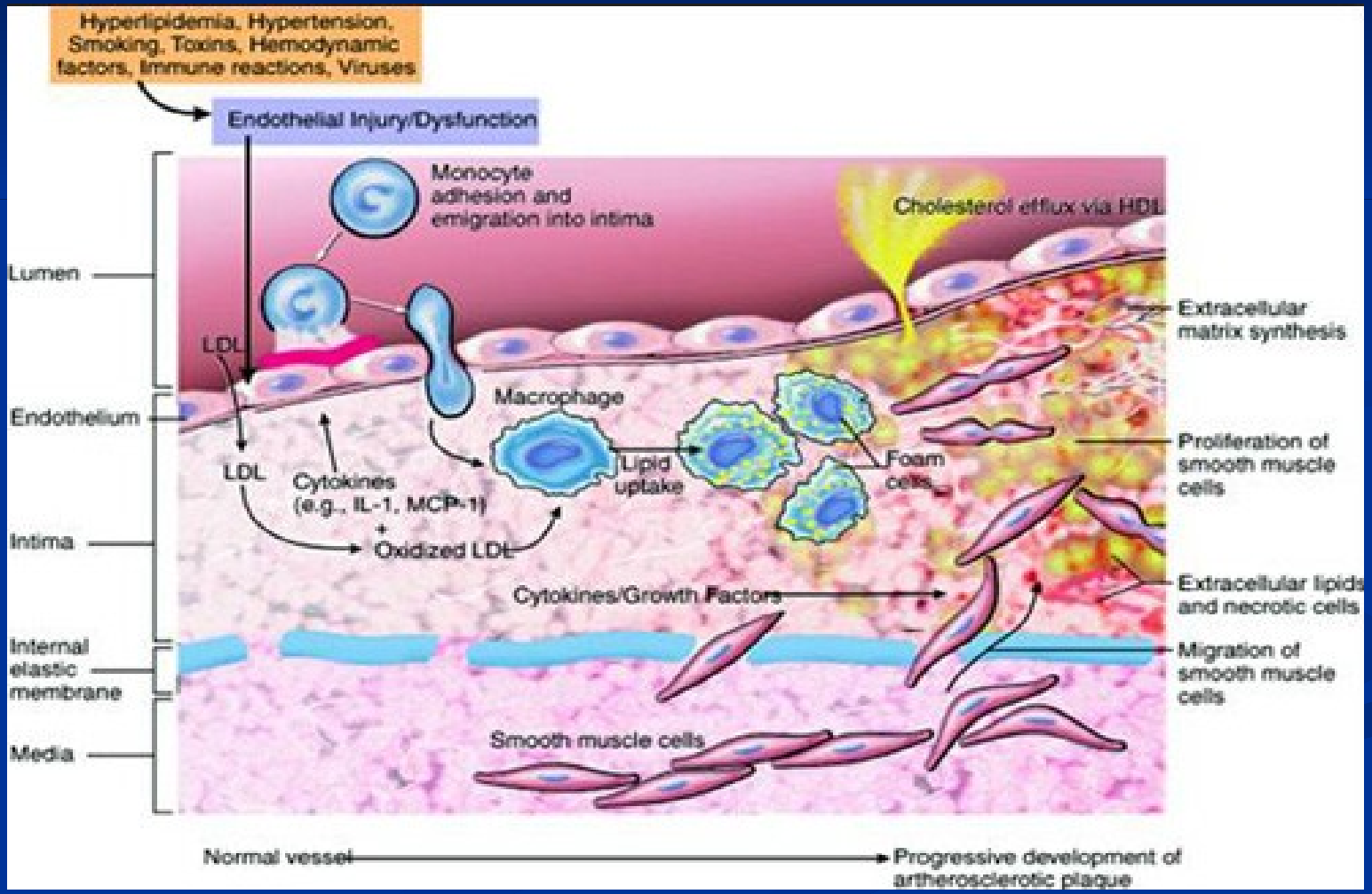
Advanced plaque in X-section



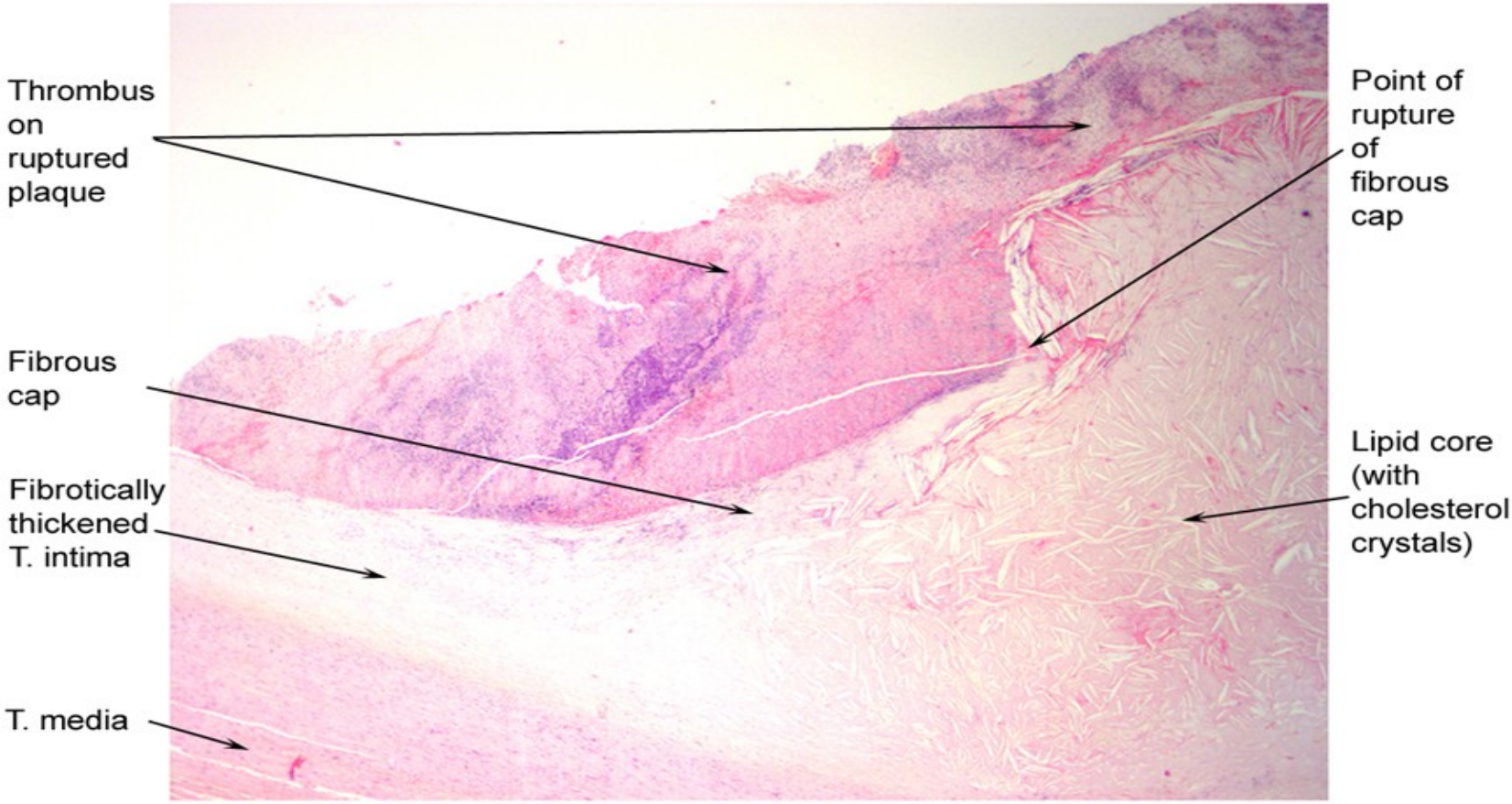
Advanced plaque



Smooth muscle cell effects



Atherosclerotic plaque rupture



Clinical manifestation of plaque pathology

- Plaque erosion/minor fissure
- Endothelial dysfunction
- Vascular remodelling
- Organised thrombus
- Residual stenosis
  - Chronic angina
  - Peripheral vascular disease

Clinical manifestation of plaque pathology 2

- Plaque erosion/fissure
- Platelet activation & aggregation
- Inflammatory cell migration
- Transient occlusion
- Platelet emboli

- Unstable angina
- Sudden death
- Stroke
- Acute ischaemic PVD

Clinical manifestation of plaque pathology 3

- Plaque erosion/fissure
- Platelet activation & aggregation
- Inflammatory cell migration
- Temporary occlusion (<2 hours)
- Endogenous thrombolysis
  - Non-Q wave MI
  - Stroke

Clinical manifestation of plaque pathology 4

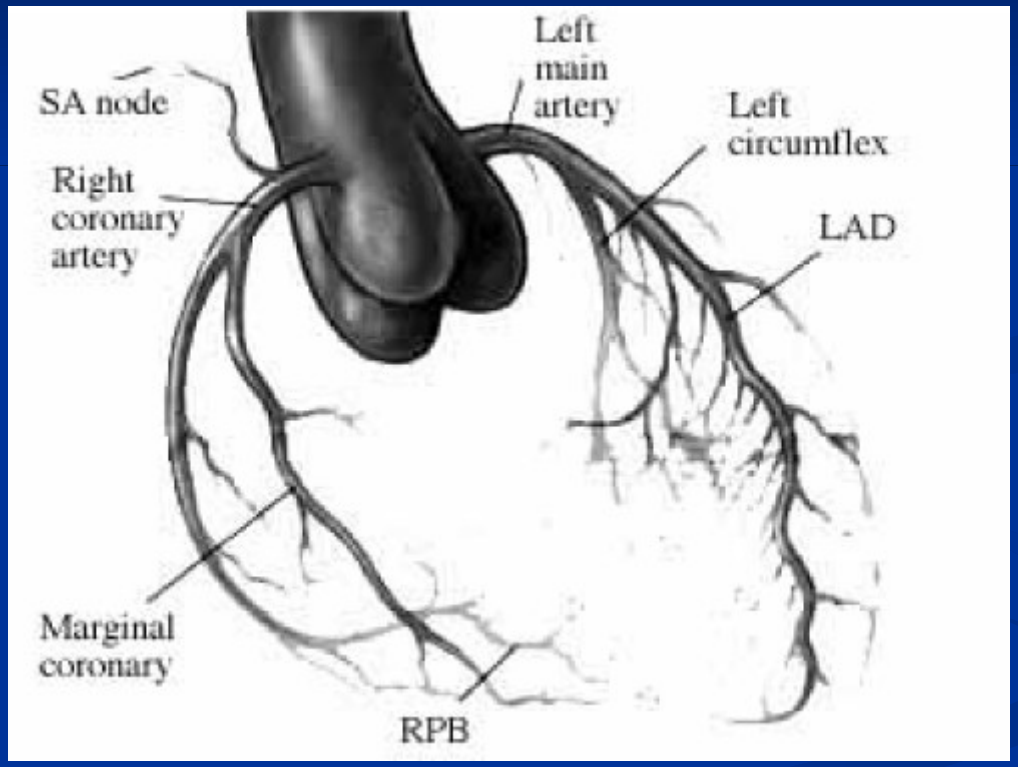
- Plaque rupture/fissure
- Highly thrombogenic substrate
- Occlusive fibrin rich thrombus
- Abrupt cessation perfusion
- Myocardial necrosis
  - Q-wave or STEMI
  - Sudden death

Atheromatous plaque with haemorrhage

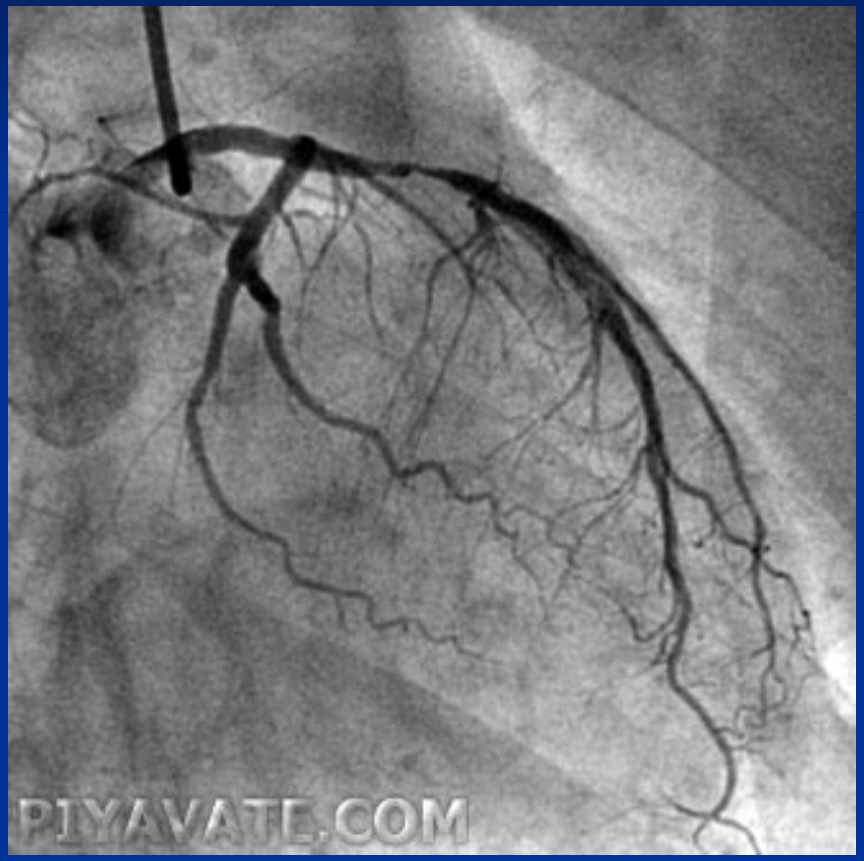


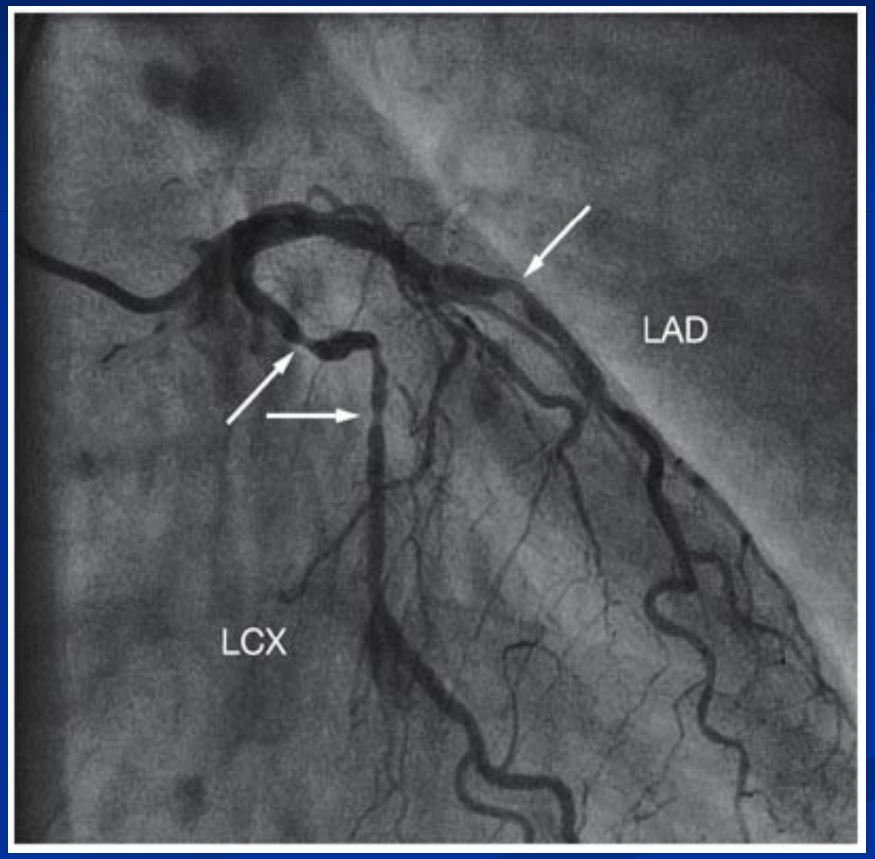
Risk factors

- Hypertension
- Diabetes
- Family history
- Dislipidaemia
- Smoking

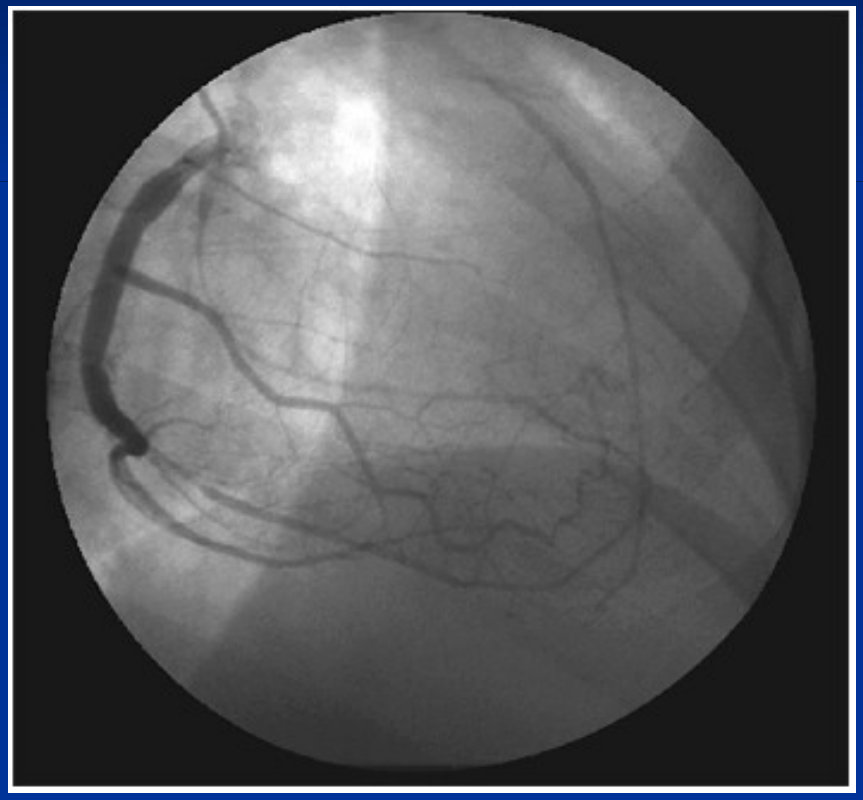

SA node
Left
main
artery
Left
circumflex
Right
coronary
artery
LAD
Marginal
coronary
RPB

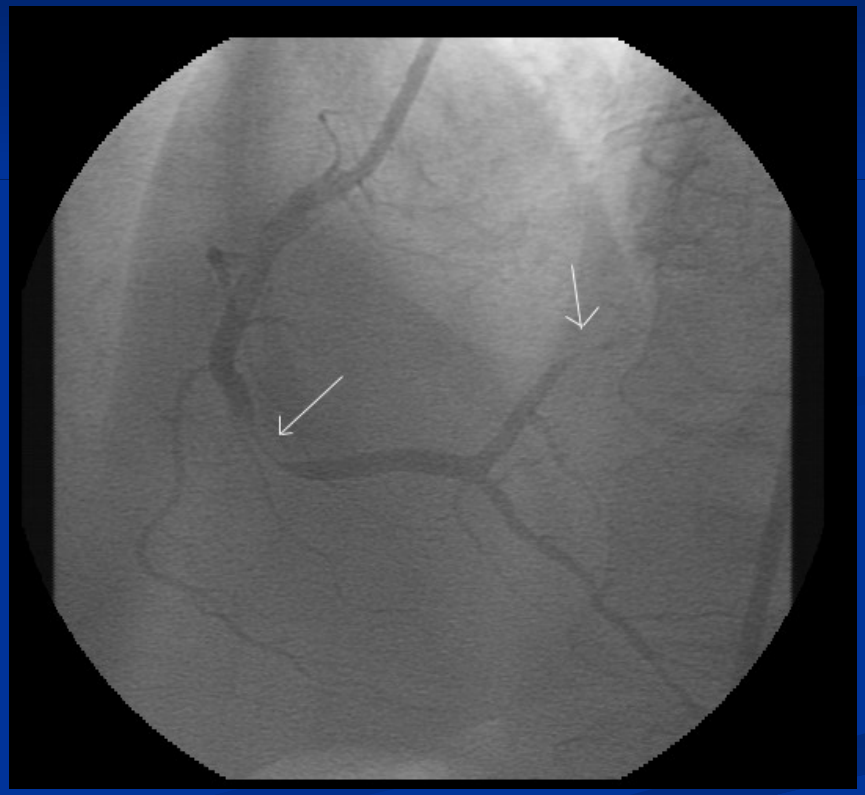
Angiograms





Angiogram





Indications

- CABG is the preferred treatment with:
- Disease of the left main coronary artery (LMCA). LMCA disease is associated with sudden death; therefore, lesions of the LMCA are sometimes referred to as *widow makers*.
- Disease of all three coronary vessels (LAD, LCX and RCA).
- Diffuse disease not amendable to treatment with a PCI.
- CABG is the likely the preferred treatment with other high-risk patients such as those with severe ventricular dysfunction (i.e. low ejection fraction), or diabetes mellitus.

Alternative treatments

- Several alternative treatments for coronary artery disease exist. They include:
- Medical management (statins, antihypertensives, smoking cessation, tight blood sugar control in diabetics)
- Percutaneous coronary intervention (PCI)

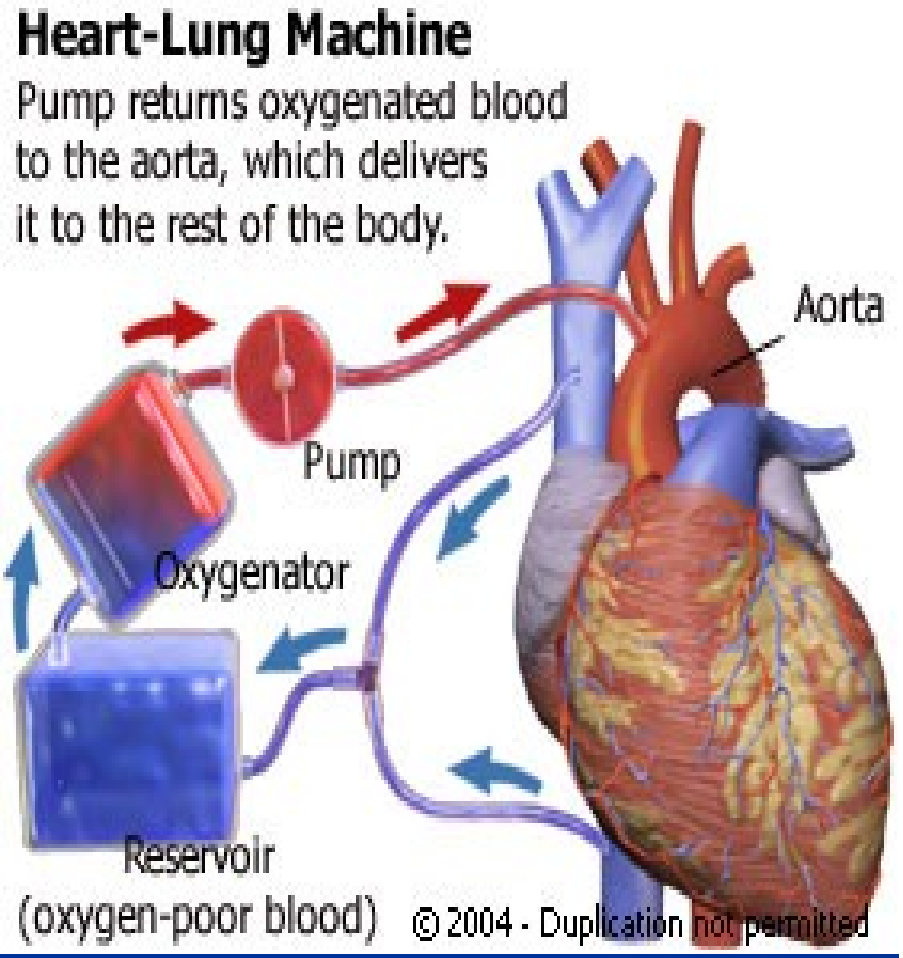
Procedure

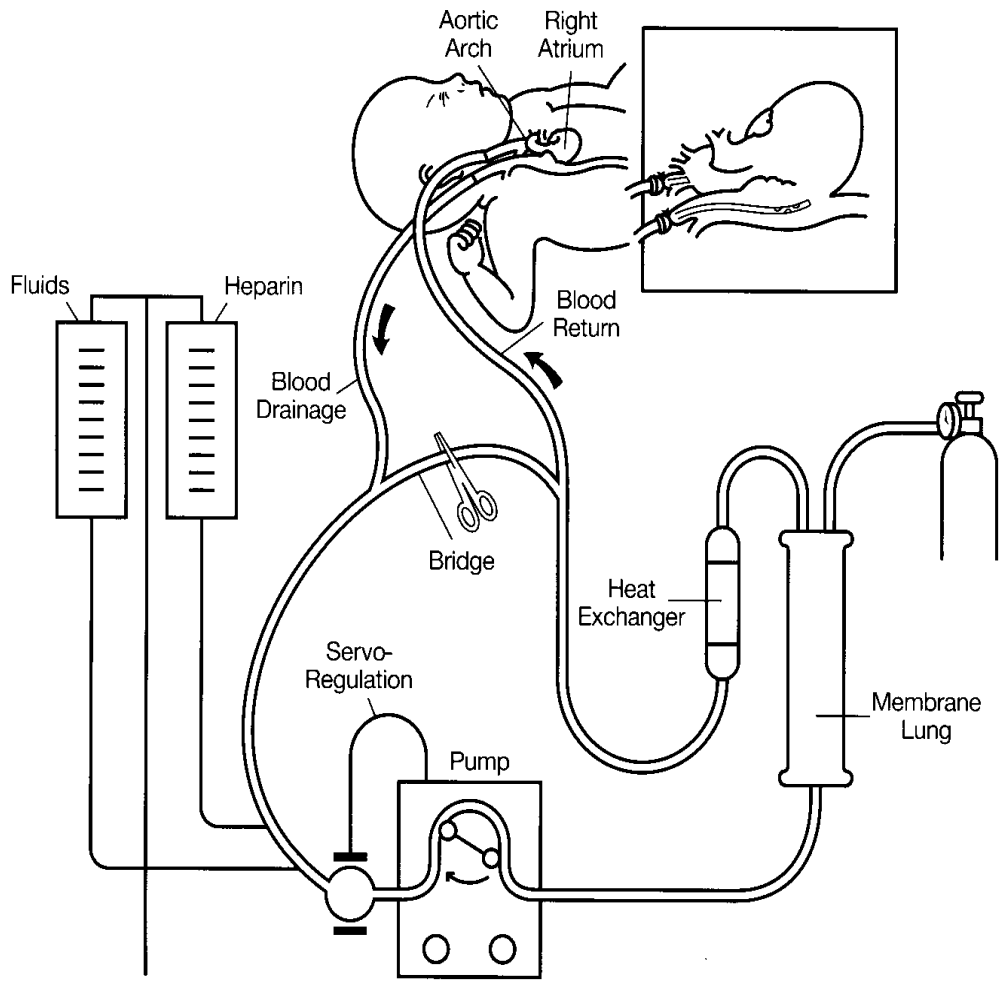
1. The patient is brought to the operating room and moved onto the operating table.
2. An anesthetist places a central and an arteria lines and injects an induction agent (usually propofol) to render the person unconscious.
3. Intubation and mechanical ventilation.
4. The chest is opened via a median sternotomy and the heart is examined by the surgeon.
5. The bypass grafts are harvested - frequent conduits are the internal thoracic arteries, radial arteries and saphenous veins. When harvesting is done, the patient is given heparin to prevent the blood from clotting.
6. In the case of "off-pump" surgery, the surgeon places devices to stabilize the heart.
7. If the case is "on-pump", the surgeon sutures cannulae into the heart and instructs the perfusionist to start cardiopulmonary bypass (CPB). Once CPB is established, the surgeon places the aortic cross-clamp across the aorta and instructs the perfusionist to deliver cardioplegia to stop the heart.
8. One end of each graft is sewn onto the coronary arteries beyond the blockages and the other end is attached to the aorta.
9. The heart is restarted.
10. Protamine is given to reverse the effects of heparin.
11. The sternum is wired together and the incisions are sutured closed.
12. The person is moved to the intensive care unit (ICU) to recover. After awakening and stabilizing in the ICU (approximately 1 day), the person is transferred to the cardiac surgery ward until ready to go home (approximately 4 days).

Conduits used for bypass

- The choice of conduits is highly surgeon and institution dependent.
- internal thoracic artery (LITA0 is grafted to the Left Anterior Descending artery and a combination of other arteries and veins is used for other coronary arteries.
- The right internal thoracic artery (RITA),
- the great saphenous vein from the leg and the
- radial artery from the forearm are frequently used.
- The right gastroepiploic artery from the stomach is infrequently used given the difficult mobilization from the abdomen.

CPB circuit


Heart-Lung Machine
Pump returns oxygenated blood to the aorta, which delivers it to the rest of the body.
Aorta
Pump
Oxygenator
Reservoir
(oxygen-poor blood)
© 2004 - Duplication not permitted


Aortic Arch
Right Atrium
Fluids
Heparin
Blood Return
Blood Drainage
Bridge
Heat Exchanger
Servo-Regulation
Membrane Lung
Pump

CPB machine



Complications/Risks

- Postperfusion syndrome , a transient neurocognitive impairment associated with cardiopulmonary bypass. Some research shows the incidence is initially decreased by Off-pump coronary artery bypass, but with no difference beyond three months after surgery. A neurocognitive decline over time has been demonstrated in people with coronary artery disease regardless of treatment.
- Nonunion of the sternum; internal thoracic artery harvesting devascularizes the sternum increasing risk.
- Myocardial infarction due to hypoperfusion, early graft occlusion, or graft failure.
- Late graft stenosis, particularly of saphenous vein grafts due to atherosclerosis causing recurrent angina or myocardial infarction.
- Acute renal failure due to hypoperfusion.
- Stroke, secondary to aortic manipulation or hypoperfusion.