# Diagnostic approaches in cardiology

## I – Haemodynamics
## II - Arrhythmias
## III - Myocardial ischemia

Jan Živný
Department of Pathophysiology
jzivny@LF1.cuni.cz

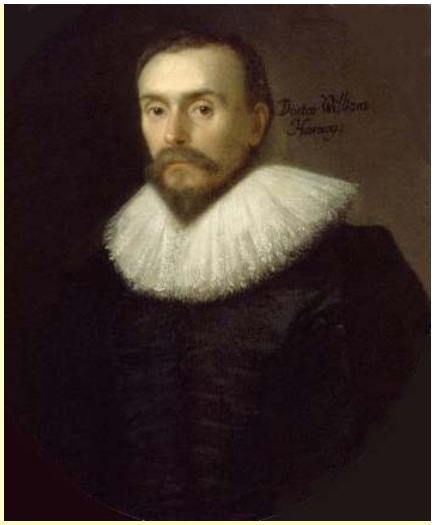
# I - Hemodynamics

Jan Živný
Department of Pathophysiology
jzivny@LF1.cuni.cz

# Outline

- Introduction to cardiac disorders
- Blood pressure evaluation
  - non-invasive and invasive measurement of BP
  - Case report (hypertension)
- Evaluation of blood volume
  - imaging methods

# William Harwey (1578-1657)



- Discovery of blood circulation and heart function (published 1628)
- Disproved Galen theory of blood circulation in veins (alternating back and forth)
- This theory was fully accepted after discovery of capillaries (Marcello Malpighi - 1661).

MARCELLO MALPIGHI.
From an engraving of the oil-painting by A. M. Tolar, presented to the Royal Society by Malpighi.

# Cardiovascular system diseases

- Hypertension
- Arrhythmia
- Diseases of endo-, myo-, peri-cardium, vessel wall
- Valve diseases
- Inherited cardiac and vascular defects

- Ischemia

# Principles of hemodynamics

- **Blood flow**
  - The cardiovascular system (CVS) transports blood (**volume**) between individual CVS compartments
  - Blood **pressure** is necessary to form pressure gradient between heart and the periphery.

- **To maintain blood flow**
  - sufficient blood volume
  - to overcome the peripheral resistance

# Ohm's law

- Q (flow) = $\Delta$P (pressure gradient) / R (resistance)

- Blood pressure depend on
  - **Cardiac output**
  - Blood volume
  - **Resistance**
  - Blood viscosity

# Short term effect of pressure and volume insufficiency

- Low cardiac output and/or decreased pressure gradient
- Ischemia – organ and tissue hypoxia

# Short term effect of pressure and volume overload

- Endothelial damage
- Edema

# Long term effect of pressure and volume overload

Changes in heart and vessels anatomy

- heart muscle dilatation
- heart muscle hypertrophy
- Increase in vessel resistance
  - organ X systemic,
  - temporary X permanent

# Functional assessment of cardiovascular system

- Measurement of blood pressure

- Measurement and evaluation of blood volume and blood volume distribution

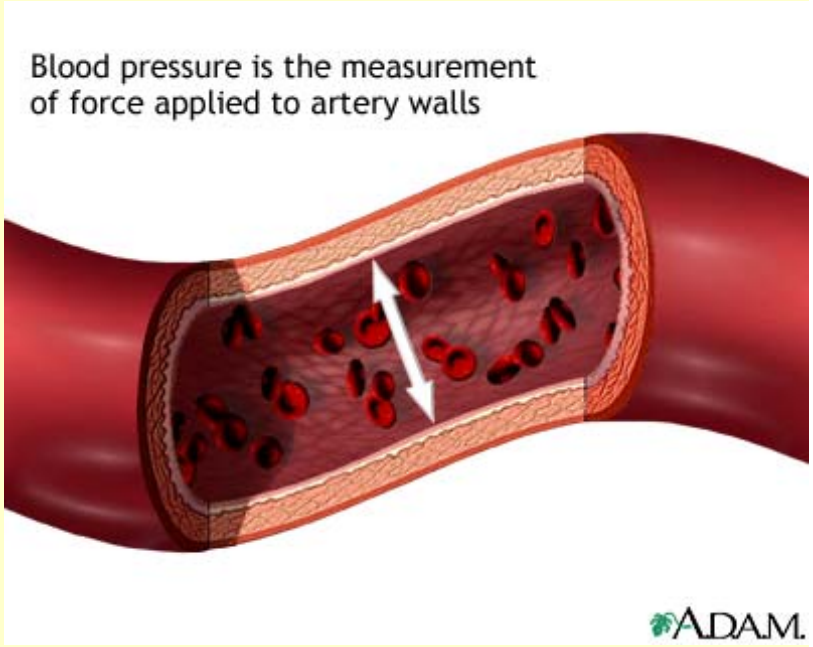
# Symptoms and Signs of Cardiovascular Diseases

- Chest pain or discomfort
- Dyspnea
  - abnormally uncomfortable awareness of breathing
- Palpitations
  - uncomfortable awareness of beating of the heart
- Syncope
- Peripheral edema
- Intermittent vascular claudication
  - cramping pain in the lower extremity (calf) caused by poor circulation of the blood during exercise

# Pressure

# Blood Pressure

- Measured in millimeters of mercury (or kPa), within the major arterial system of the body
- Systolic pressure
  - maximum blood pressure during contraction of the ventricles
- Diastolic pressure
  - minimum pressure recorded just prior to the next contraction

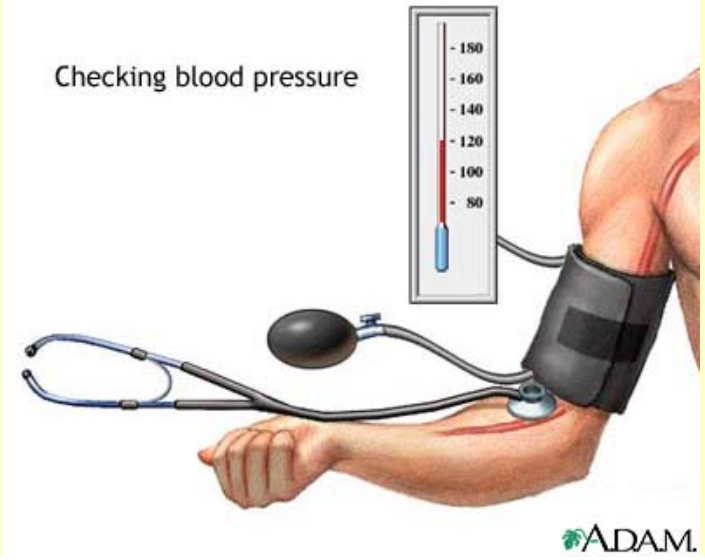


# Indications for BP measurement

- Screening for hypertension
- Assessing a person’s suitability for a sport or certain occupations
- Estimation of cardiovascular risk
- Determining for the risk of various medical procedures

# Non-invasive blood pressure measurement

- Usually taken with the patient seated using standard blood pressure cuff
- Orthostatic hypotension examination:
  - by checking the patient in the lying and standing positions
    - Systolic blood pressure should not drop more than 10 mm Hg
    - Diastolic pressure should remain unchanged or rise slightly

# Systemic BP

- Systolic: heart and aorta function
- Diastolic: peripheral resistance

- Mean pressure
- Pressure amplitude

- Hypertension X Hypotension

# Interpretation of Blood Pressure Measurements (JNC7 2003)

| **Diastolic pressure (mm Hg)** | **Category** |
|---|---|
| <85 | Normal |
| 80-89 | Prehypertension |
| 90 - 99 | Stage I |
| >100 | Stage II |
| **Systolic pressure (mm Hg)** | **Category** |
| < 119 | Normal |
| 120-139 | Prehypertension |
| 140-159 | Stage I |
| >160 | Stage II |

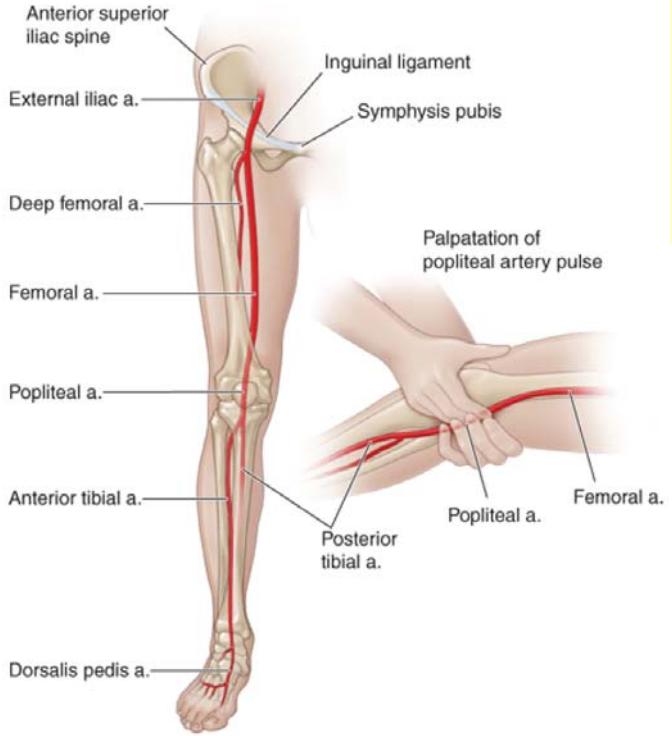
**Isolated systolic hypertension (when diastolic < 89) and systolic > 140 mmHg**

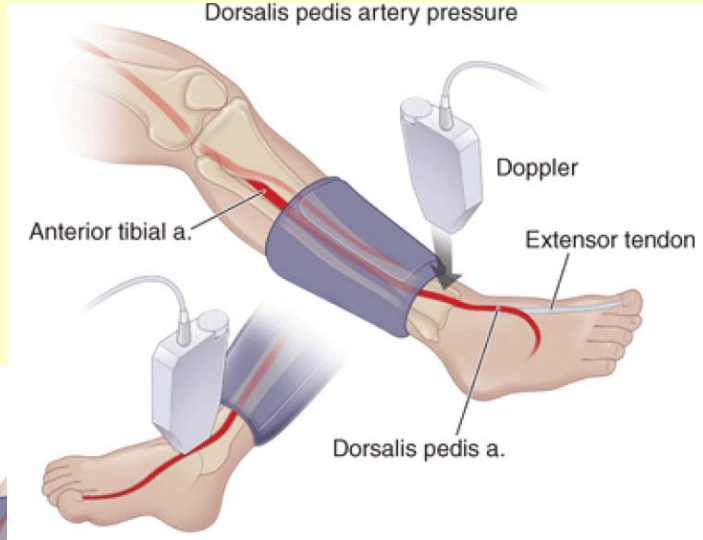
# The progression of essential hypertension

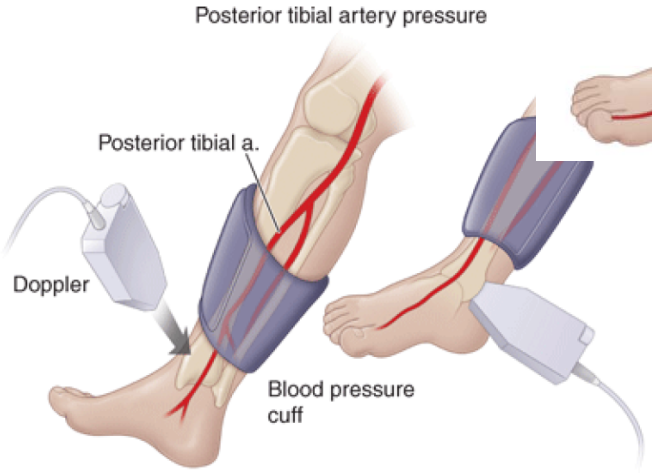
- Prehypertension
  - in persons aged 10-30 years (by increased cardiac output)
- Early hypertension
  - in persons aged 20-40 years (in which increased peripheral resistance is prominent)
- Established hypertension
  - in persons aged 30-50 years
- Complicated hypertension
  - in persons aged 40-60 years

# Arterial pulse

- Peripheral arterial pulses that should be assessed:
  - subclavian, brachial, radial, ulnar, femoral, popliteal, dorsalis pedis, posterior tibial



# Arterial pulse

- *Pulsus paradoxus*

# Arterial pulse

- *Pulsus paradoxus*
  - refers to a fall in systolic pressure >10 mmHg with inspiration
  - palpable at the brachial or femoral artery when the pressure difference exceeds 15 mmHg
  - Cause:?

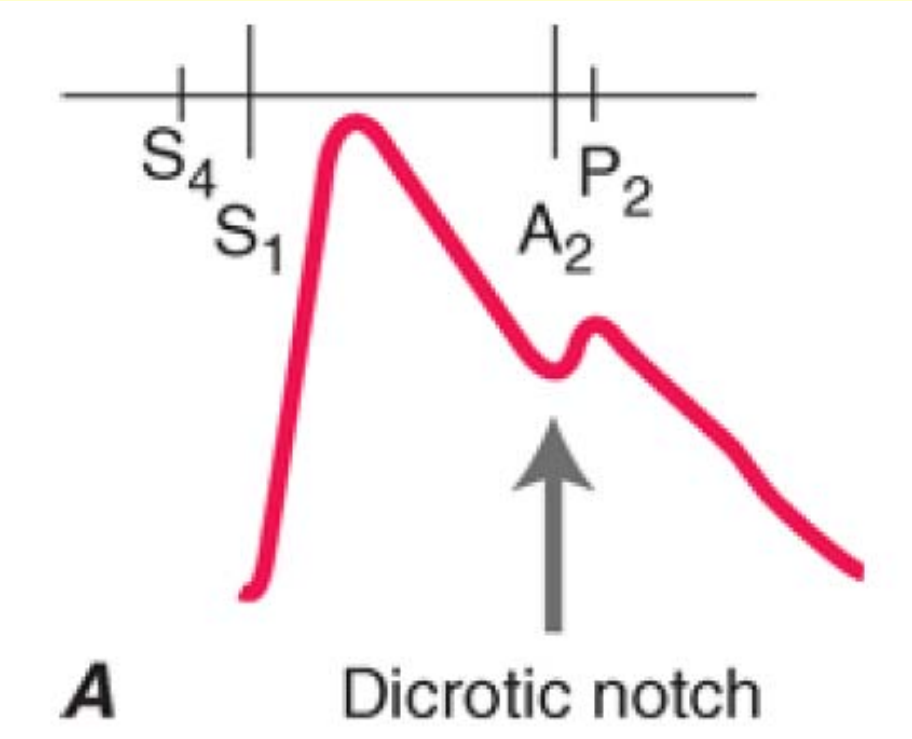
# Arterial pulse

- *Pulsus paradoxus*
  - refers to a fall in systolic pressure >10 mmHg with inspiration
  - palpable at the brachial or femoral artery when the pressure difference exceeds 15 mmHg
  - Cause:
    - **cardiac causes** (pericardial tamponade, cardiogenic shock)
    - **pulmonary causes** (massive pulmonary embolism, severe obstructive lung disease, tension pneumothorax)
    - **non-pulmonary and non-cardiac causes** (severe hypovolemia, hemorrhagic shock, anaphylactic shock).

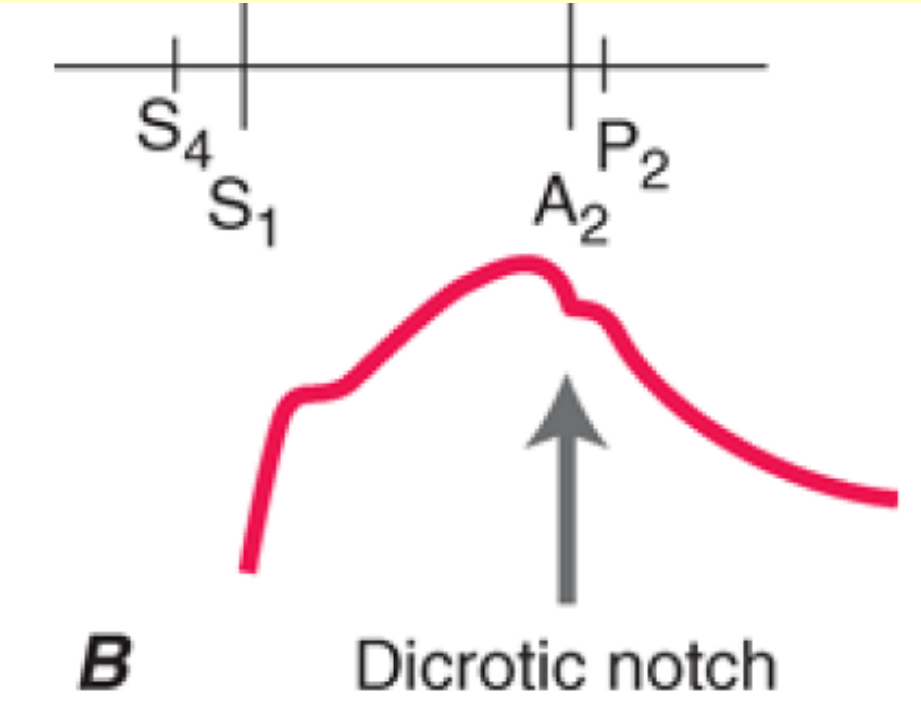
# Arterial pulse

- *Pulsus alternans*
  - is defined by beat-to-beat variability of pulse amplitude independent of the respiratory cycle.
  - Cause:
    - is thought to be due to cyclic changes in intracellular calcium and action potential duration
    - severe left ventricular systolic heart failure

# Diagrams of the configurational changes in carotid pulse

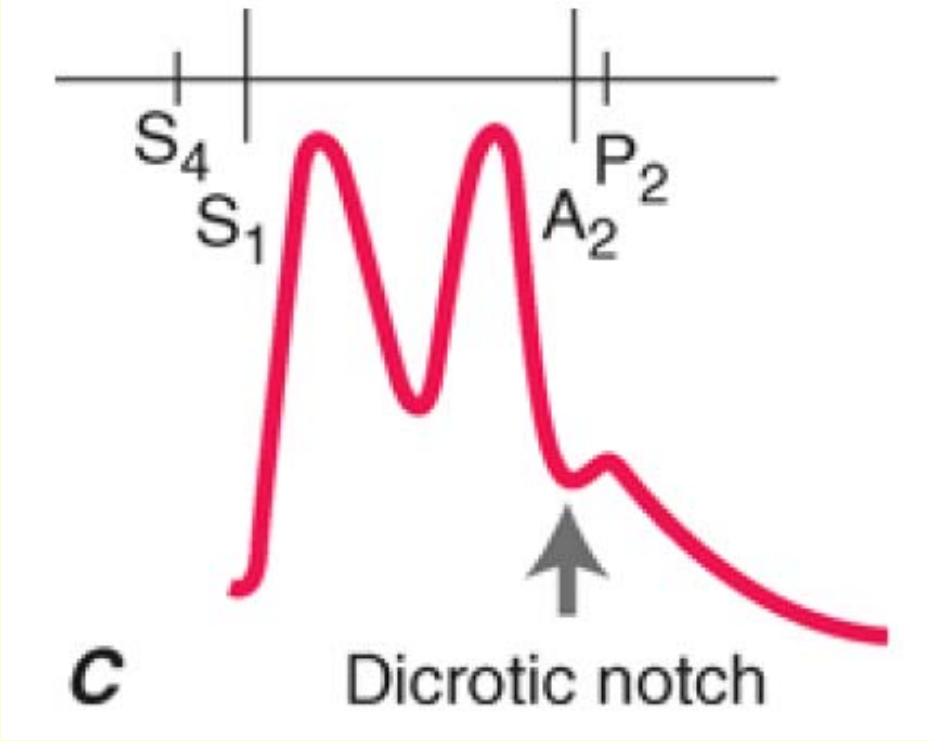



- Normal

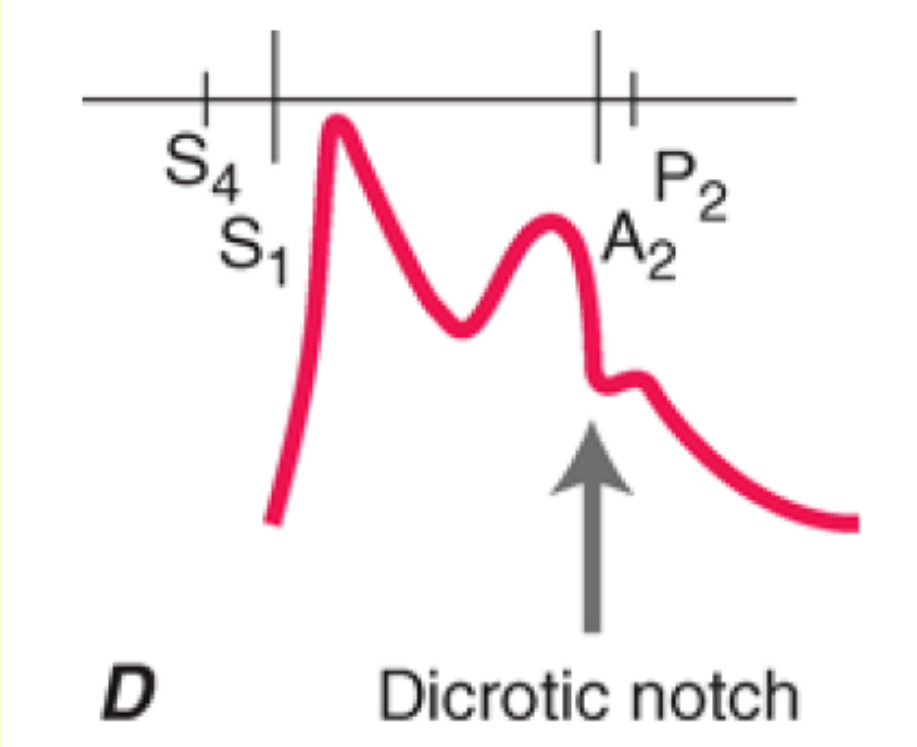



- Aortic stenosis
  Anacrotic pulse with slow upstroke to a reduced peak

# Diagrams of the configurational changes in carotid pulse



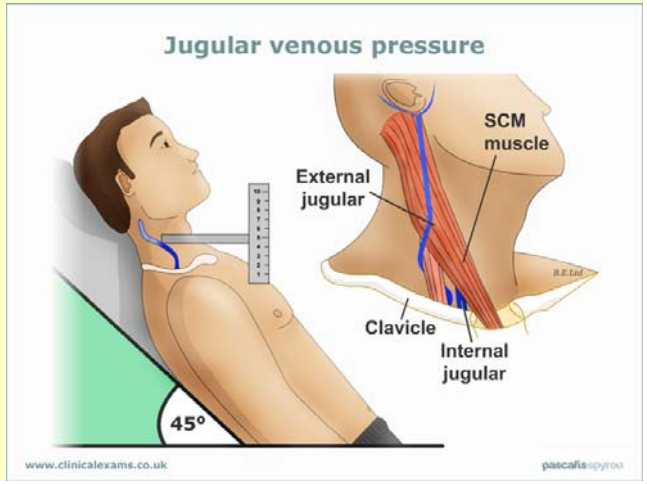

- ***C.*** Bisferiens pulse with two peaks in systole. In patients with severe aortic regurgitation (rare).




- ***D***. Bisferiens pulse in hypertrophic obstructive cardiomyopathy

# Jugular Venous Pressure

- To estimate the volume status
- Venous pressure is measured as the vertical distance between the top of the jugular venous pulsation and the sternal inflection point
- A distance >4.5 cm at 30° elevation is considered abnormal



# Jugular Venous Wave Form

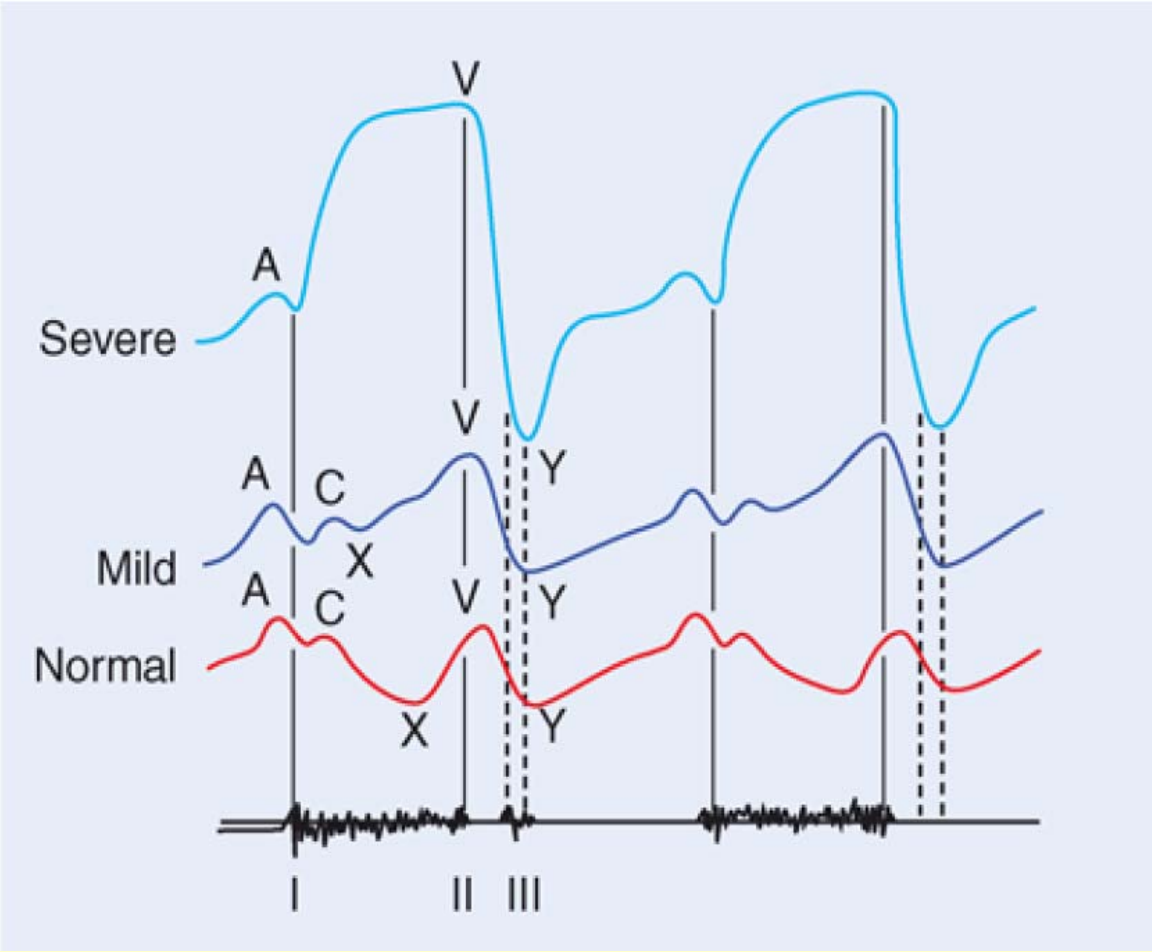
**A:** right atrial presystolic contraction after the ECG P wave

**C:** carotid pulsation and/or an early systolic increase in right atrial pressure

**X:** atrial pressure fall

**V:** atrial filling during ventricular systole

**Y:** descent corresponds to the fall in right atrial pressure after tricuspid valve opening




**Tricuspid regurgitation**

# Invasive measurement of BP

- Pressure measurements in the individual vessels and separate heart cavities
- Wedge pressure measurements
- Evaluation of pressure gradients
- Evaluation of cardiac output

Also possible to:

- Evaluate blood for oxygen saturation
- Injection of contrast dyes for angiography
- Biopsy

# Invasive measurement of BP

- Heart catheterization
- Werner Forsmann
  - 1926 – first heart catheterization through cubital vein (of himself) using flexible urinary catheter
  - forced to leave the Berliner Charité Hospital for self-experimentation and later for not meeting scientific expectations
  - joined Nazi party – medical officer (major) – during WW II captured – from 1945 worked as lumberjack and later as country physician
  - 1956 – Nobel prize (shared with André Frédéric Cournand and Dickinson W. Richards from Columbia University)

# Heart catheterization

- Goal:
  - detailed hemodynamic and anatomic assessment of the heart and coronary arteries
- Vascular access
  - Right heart
    - femoral or jugular vein
  - Left heart
    - femoral artery
    - brachial or radial artery (pts. with arterial disease that involves the abdominal aorta, iliac, or femoral vessels)

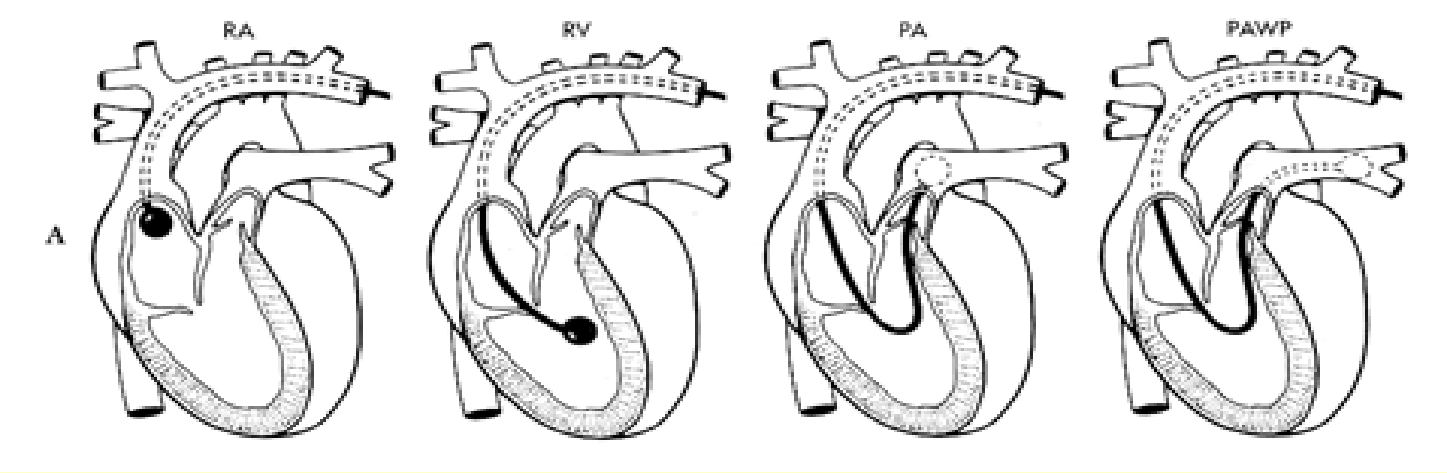
# Indications for right heart catheterization

- no longer a routine part of diagnostic cardiac catheterization

- unexplained dyspnea
- valvular heart disease
- pericardial disease
- right and/or left ventricular dysfunction
- congenital heart disease
- suspected intracardiac shunts

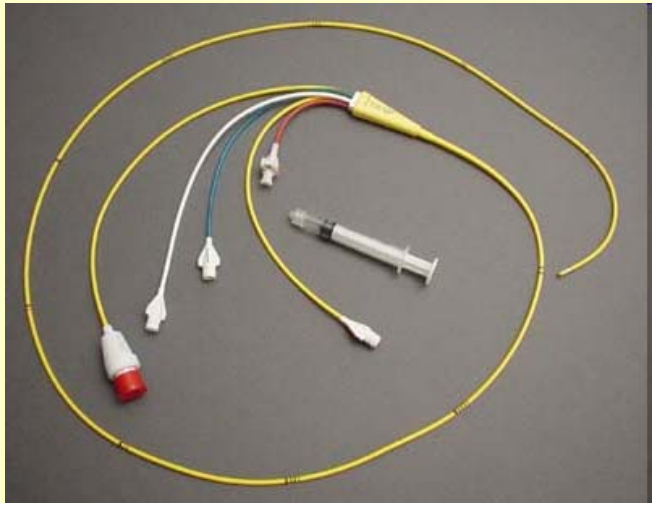
# Right heart catheterization

- Swan-Ganz catheter position in heart
  - Superior vena cava (SVC)
  - Right atrium (RA)
  - Right ventricle (RV)
  - Pulmonary artery (PA)
  - Pulmonary (artery) wedge pressure (PAWP) *or* PCWP – pulmonary capillary wedge pressure

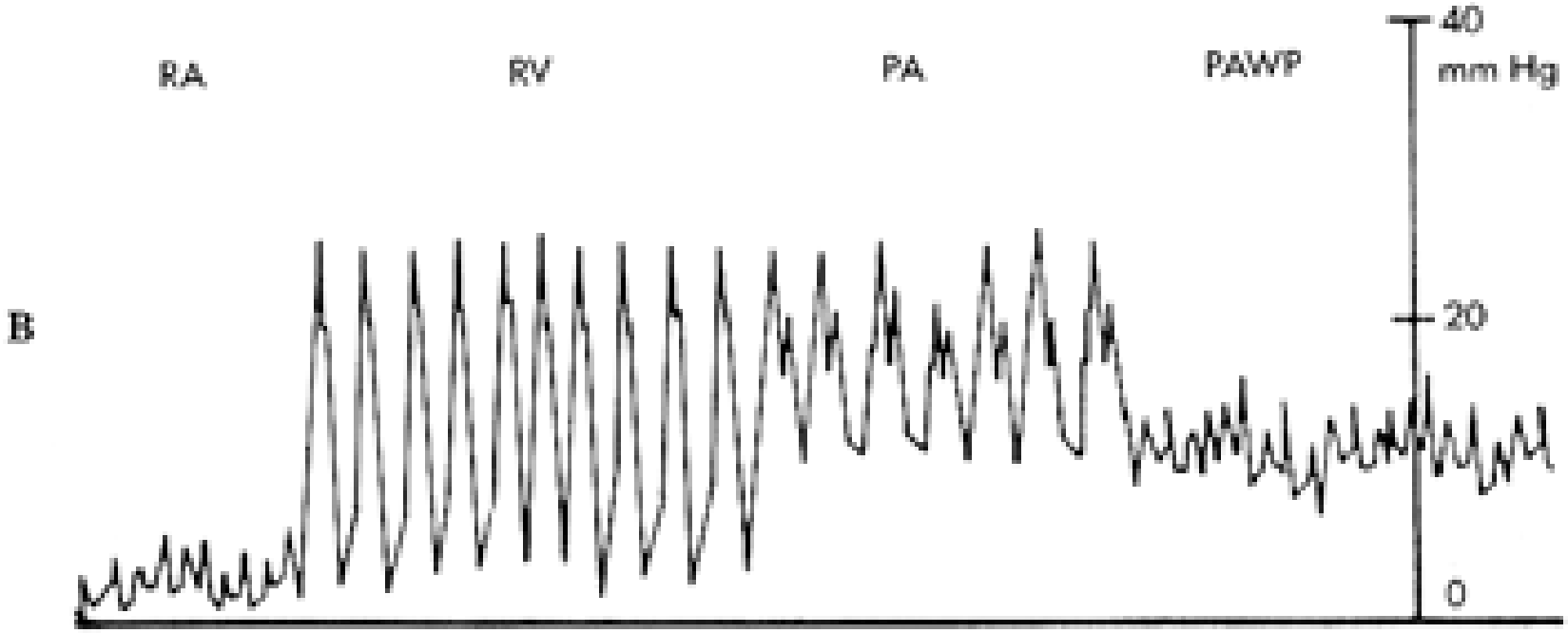


# Pulmonary artery (Swan-Ganz) catheter

- single catheter 110 cm in length containing four lumina
- Constructed of flexible, radio-opaque polyvinyl chloride
- 10 cm increments are marked in black
- Latex balloon of 1.5 mL capacity is at the distal end



# Pressure tracing during catheterization by Swan-Ganz catheter




right atrium – RA; right ventricle (RV); a. pulmonalis (PA); Pulmonary artery wedge pressure (PAWP)

# Central venous pressure (CVP)

- The pressure of blood in vena cava or right atrium
- Normal values: 2-8 mm Hg
- Monitoring of **systemic volume** filling

- CVP indirectly indicates the efficiency of the heart's pumping action (if not tricuspidal stenosis)

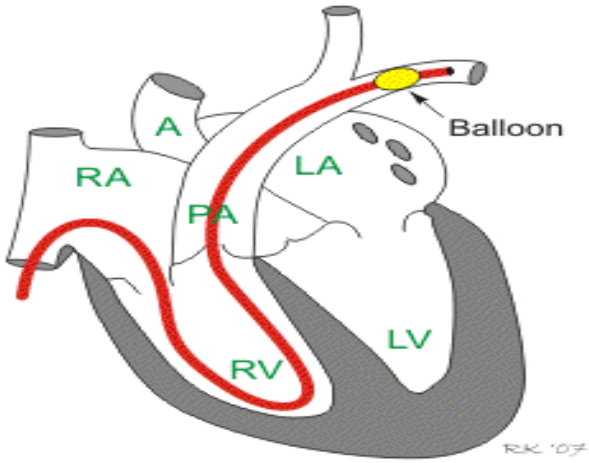
# Central venous pressure (CVP)

- Increased:
  - **Hypervolemia**
  - **Right heart failure**
  - **Tricuspidal stenosis**
  - Cardiac tamponade
  - Other non cardiovascular causes (forced exhalation, tension pneumothorax, pleural effusion)
- Decreased
  - **Hypovolemia**
  - Deep inhalation
  - Distributive shock

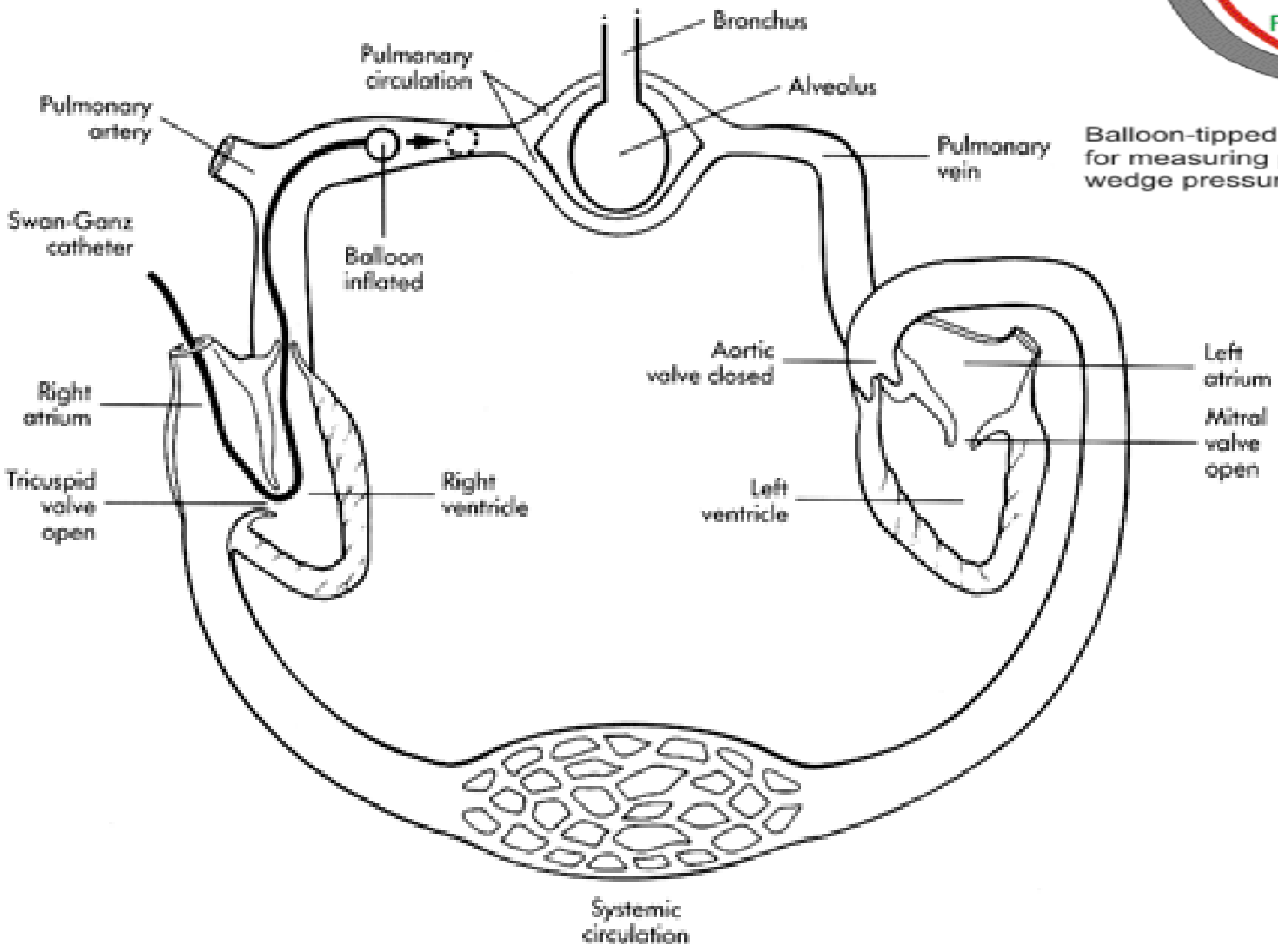
# Pulmonary artery pressure

- Systolic pressure is 15 to 30 mmHg
- Diastolic pressure is 0 to 8 mmHg
- Mean pressure is 9 to 17 mmHg (normal < 20 mmHg)

# Pulmonary (artery) capillary wedge pressure

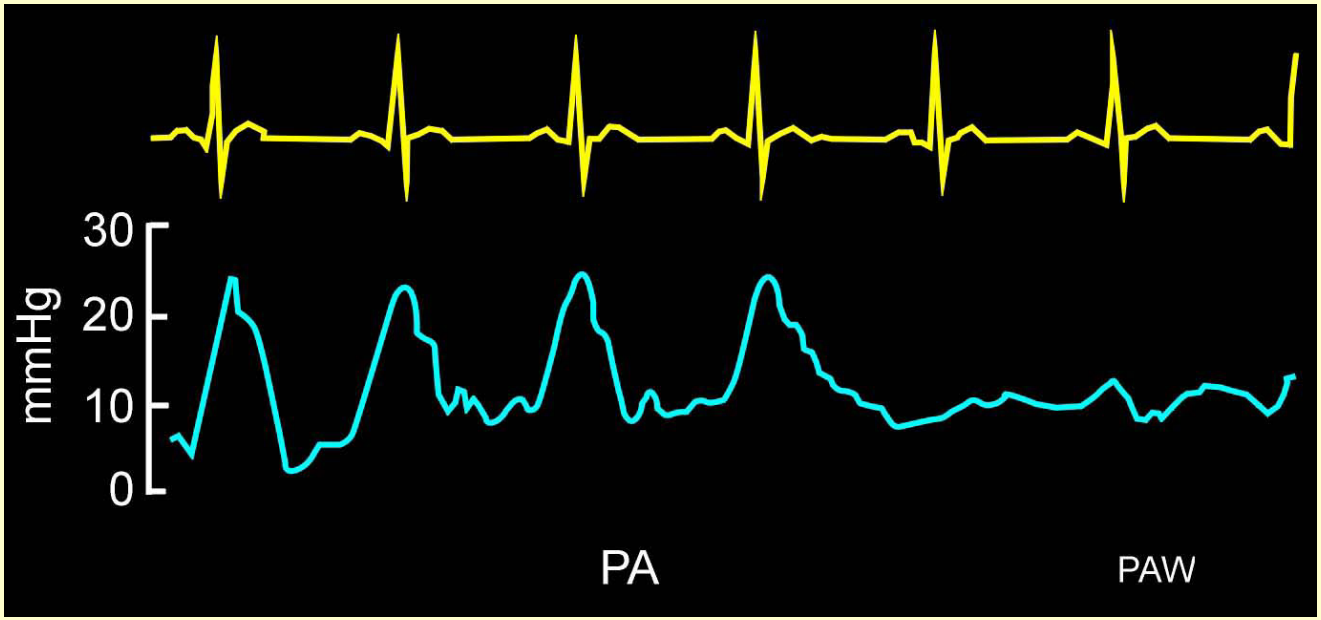



Balloon-tipped, Swan-Ganz catheter for measuring pulmonary capillary wedge pressure (PCWP).



# Pulmonary artery (capillary) wedge pressure (PAWP or PCWP)

- Transmitted pressure of left atrium
- Depends on the filling (preload) and on properties of myocardium (compliance) of left ventricle



# PAWP

- Is a reflection of the **left atrial pressure (**LAP) because
  - There are no valves between the pulmonary capillaries and the left atrium
  - During diastole, when mitral valve is open, the PAWP reflects left ventricular end-diastolic pressure (volume)

# Why to measure PAWP?

- Asses the left heart function in a critically ill patient and a patient with cardiovascular disease

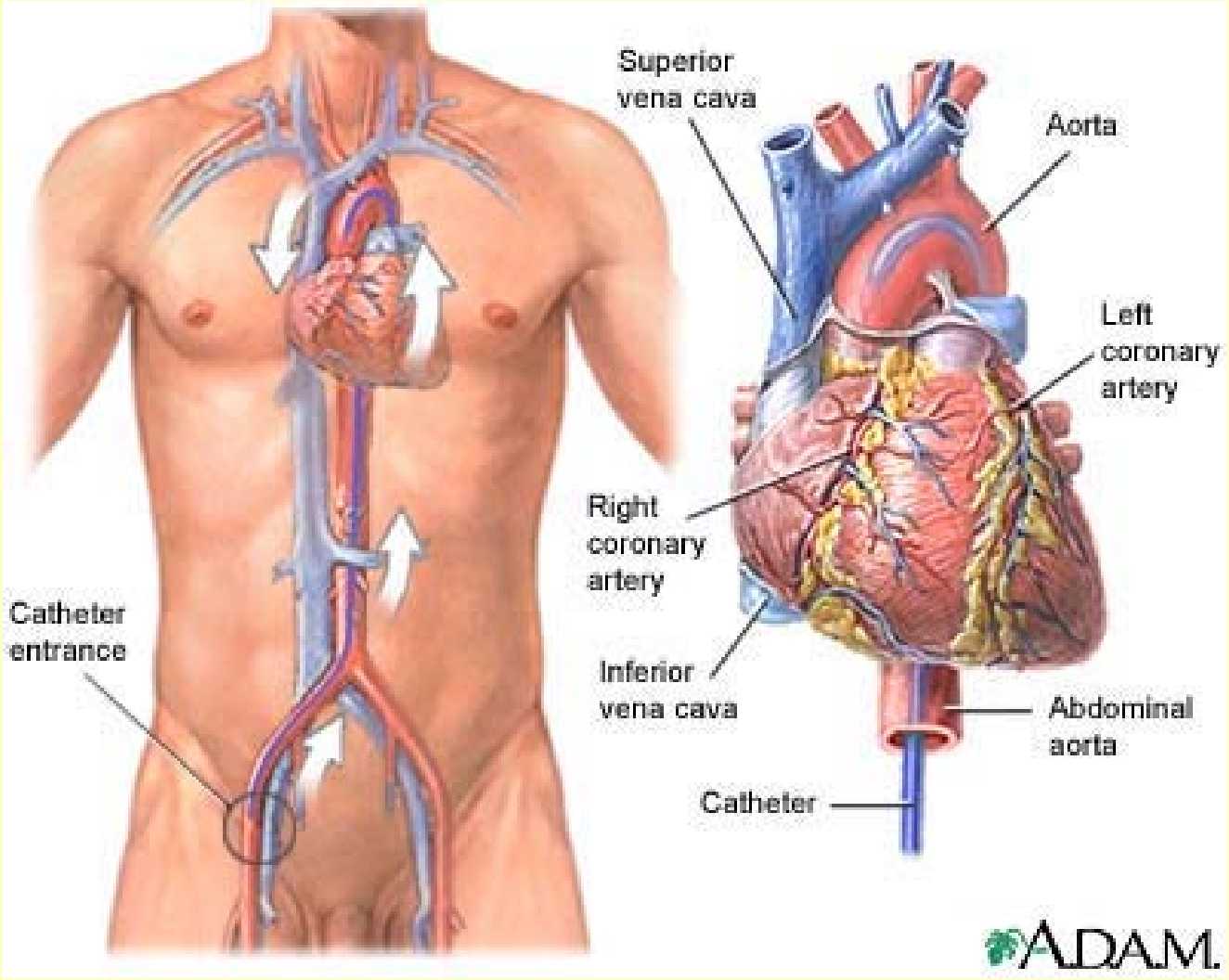
# Increase of PAWP

- Left ventricular failure
- Mitral valve stenosis
- Aortic valve stenosis and regurgitation
- Mitral regurgitation

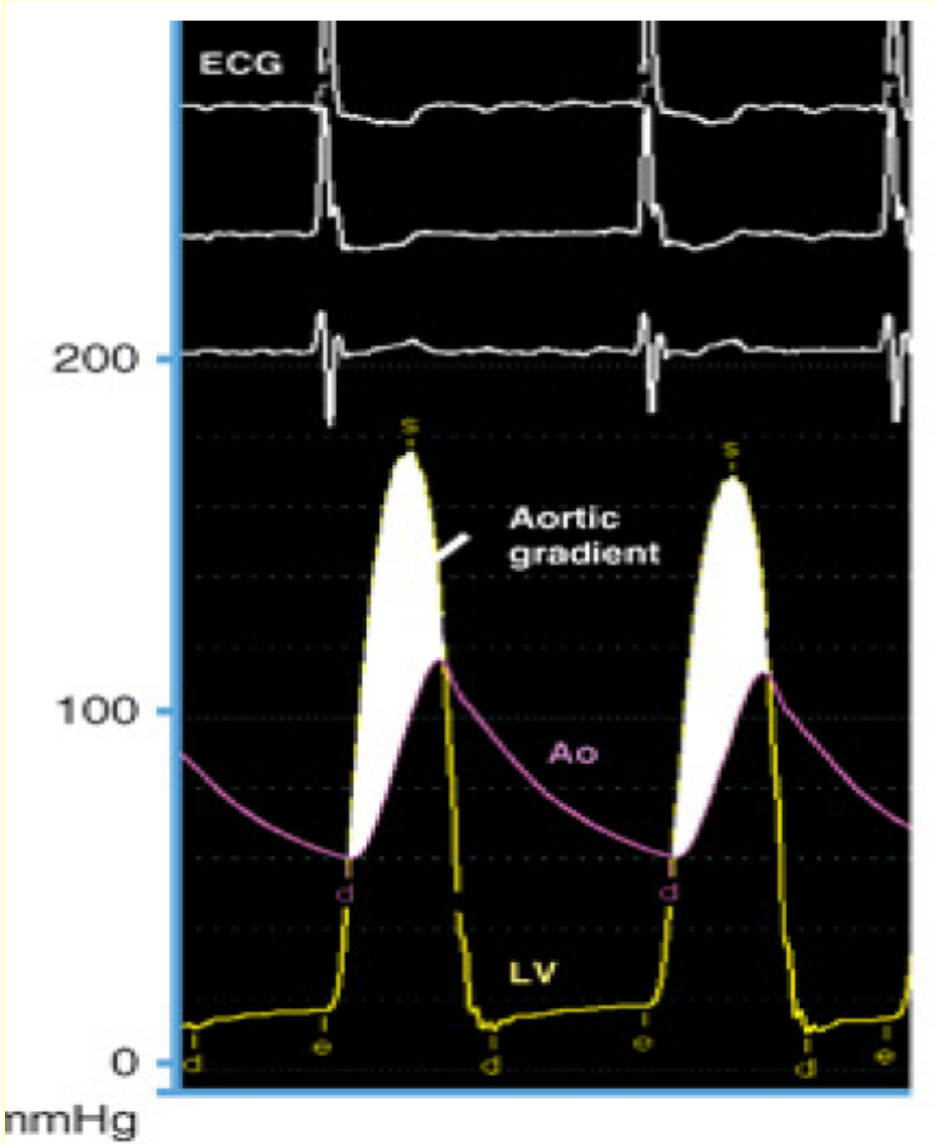
- > 20 mmHg is likely to be cause of pulmonary edema
- Evaluating blood volume status (12-14 mmHg)
  - therapy of hypotensive shock

# Left hert catheterization

# Left heart catheterization



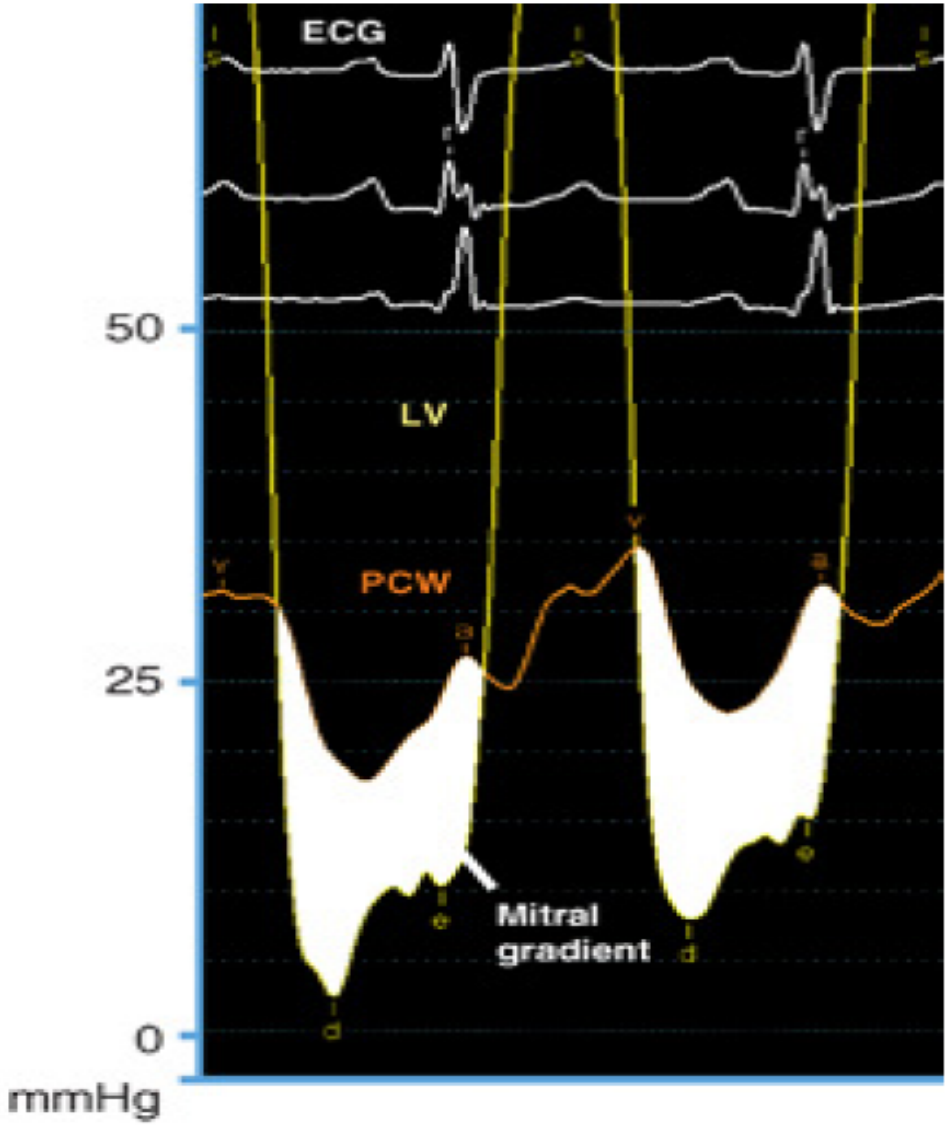
# Severe aortic stenosis




- Simultaneous recording of left ventricular (LV) and aortic (Ao) pressure
- 62-mmHg mean systolic gradient (shaded area)

Left ventricle
Peak systolicic 90–140 mmHg
End diastolic 5–12 mmHg
Aorta
Peak systolic 90–140 mmHg
End diastolic 60–90 mmHg

# Severe mitral stenosis




- Simultaneous recording of LV and pulmonary capillary wedge (PCW) pressure
- 14-mmHg mean diastolic gradient (shaded area)

*Normal*
*mean PCW* ~ 4–12 mmHg
*LV end diastolic* ~ 5–12 *mmHg*

# Complications of heart catheterization

- **Complications of cannulation**
  - Arterial (carotid, subclavia) and vein puncture
    - Haematoma, haemothorax, pleural effusion
  - Nerve injury (brachial plexus, stellate ganglion)
  - Emboli (air, catheter insertion)
- **Complications of catheter insertion**
  - Cardiac perforation, dysrhythmia
  - Knotting
  - Valve injury (Tricuspid, pulmonary)
- **Complications of catheter presence**
  - Thrombosis, thromboembolisin (pulmonary infarction)
  - Infection, endocarditis, sepsis
  - Pulmonary artery rupture

# CASE - Hypertension

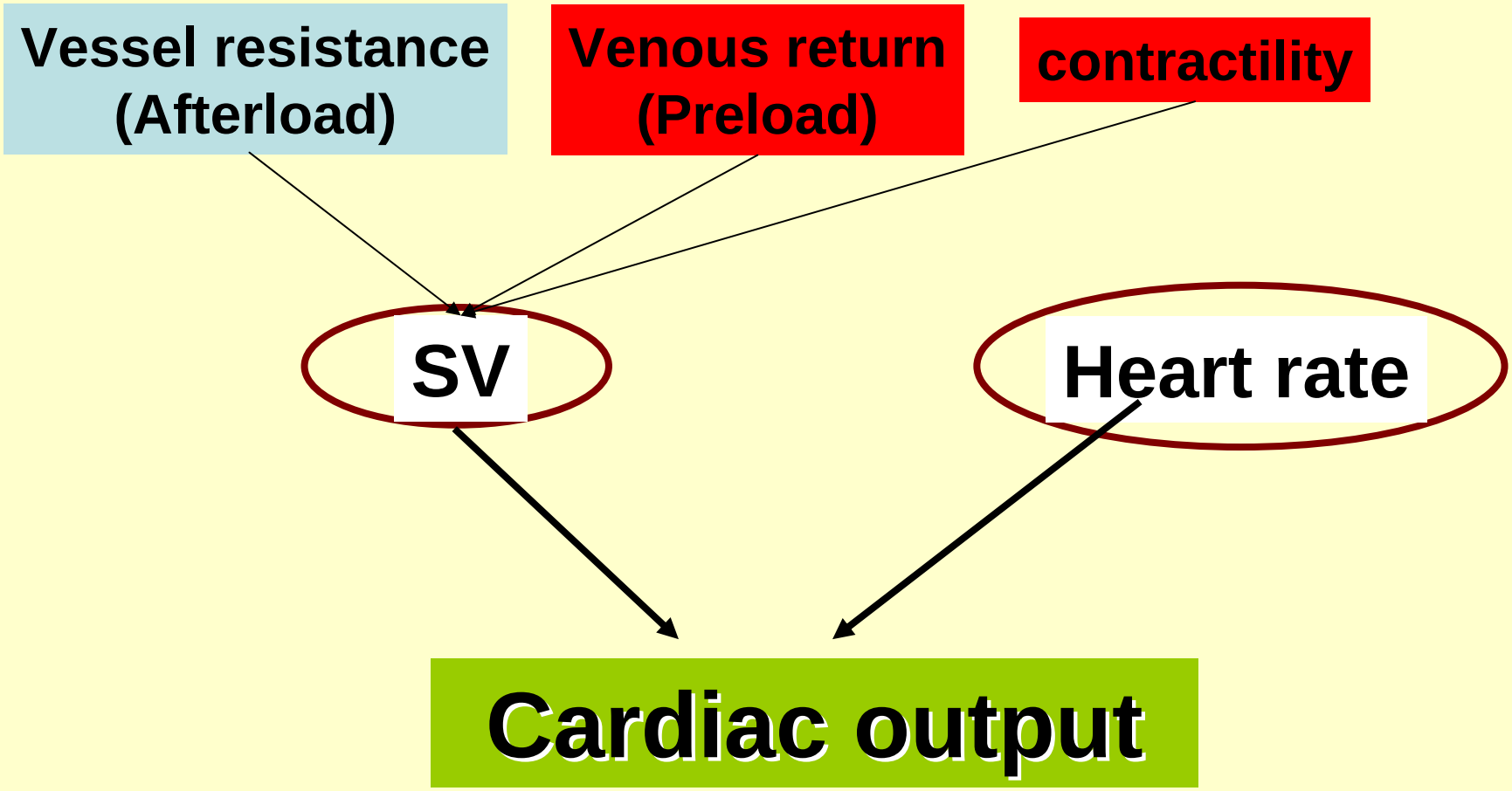
# To remember

- Systemic arterial blood pressure
- Central venous pressure
- Pulmonary Artery (Capilary) Wedge pressure (PAWP or PCWP or PWP)
- Arterial hypertension and target organ damage

# Volume

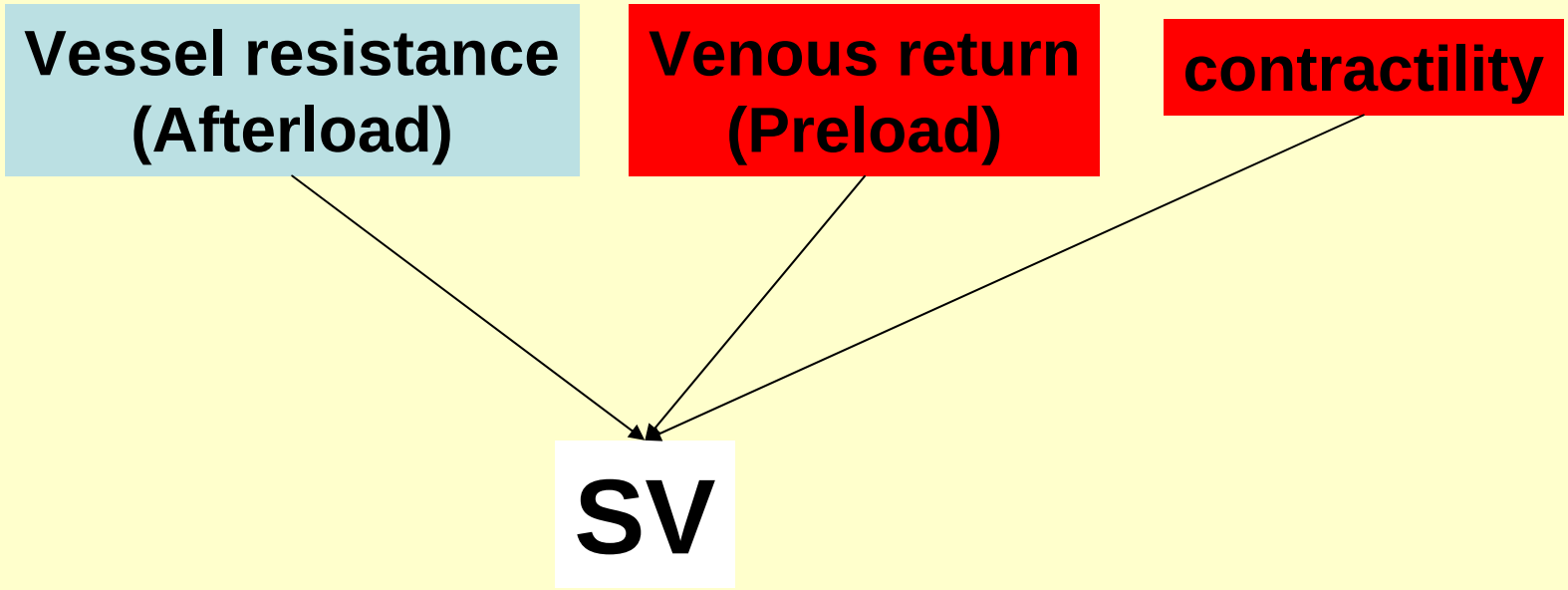
# Cardiac output

**Is determined by heart rate and stroke volume**

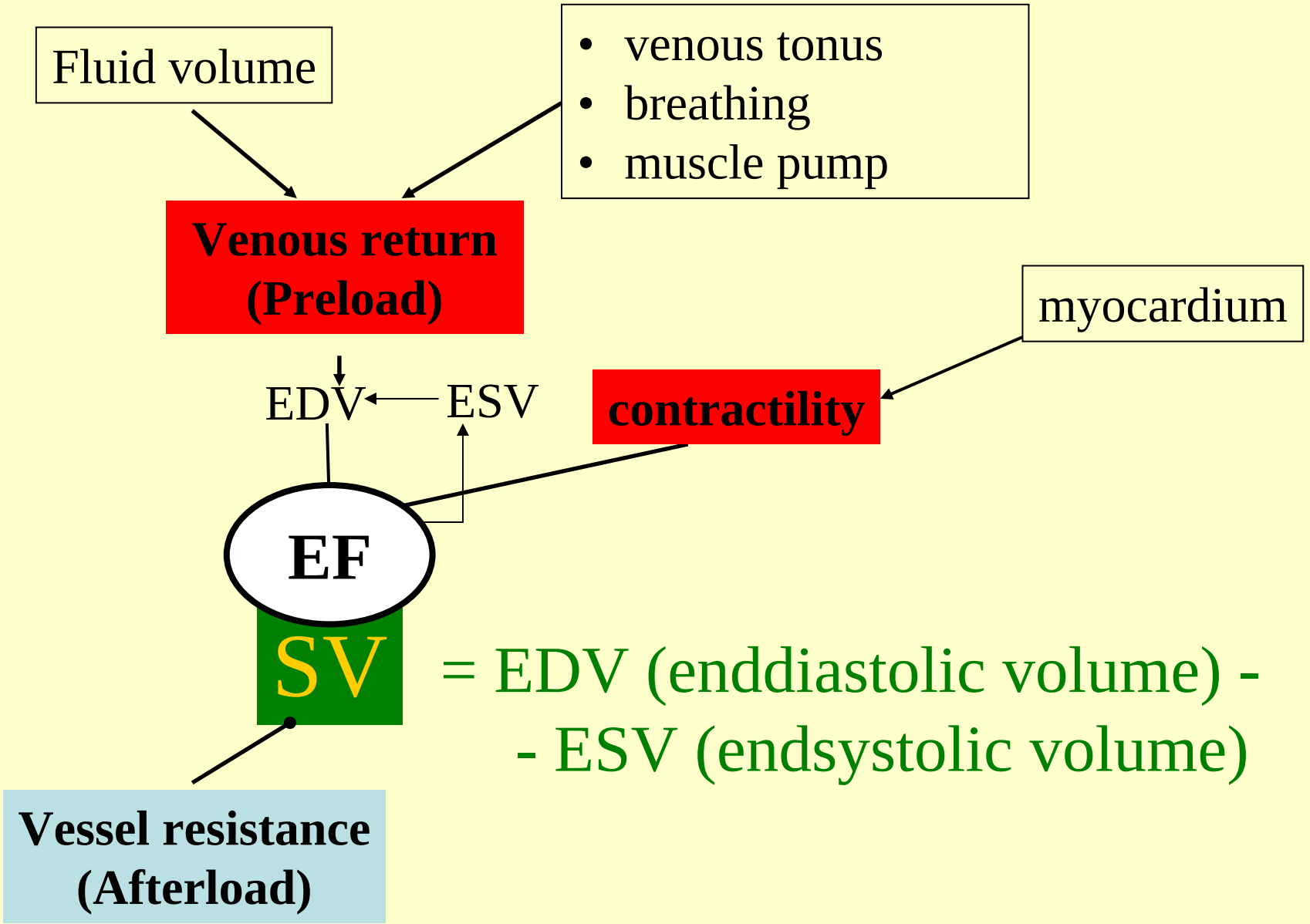


# Stroke (systolic) volume (SV)

- Volume of blood pumped by the right/left ventricle of the heart in one contraction



# Stroke Volume (SV)



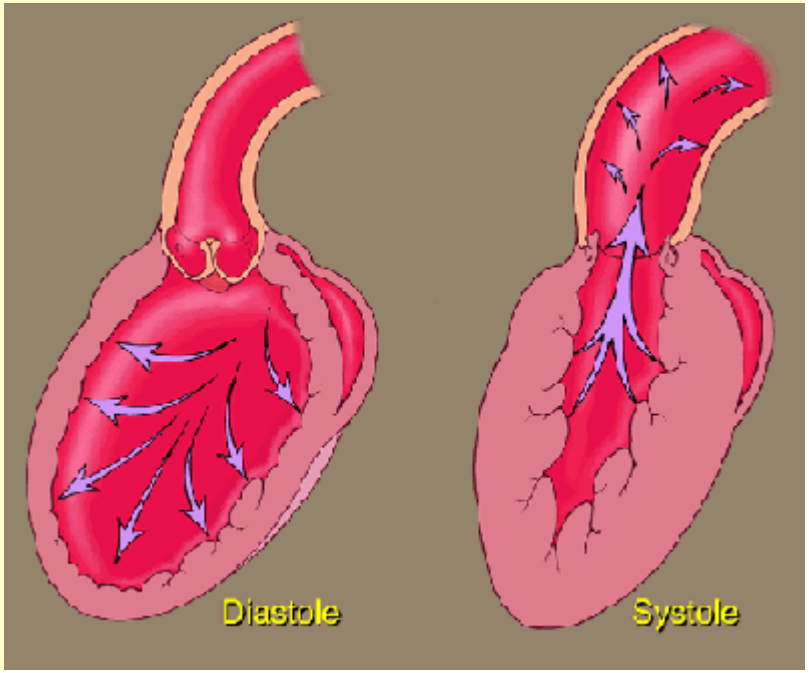

• Depends on: preload, afterload, contractility

# Ejection fraction (EF)

- Fraction of blood pumped out of a ventricle with each heart beat

- EF = SV / EDV = (EDV-ESV) / EDV

SV: stroke volume
EDV: endiastolic volume
ESV: endsystolic volume



# Ejection fraction (EF)

- Basic parameter for evaluation of the **systolic function** of the heart

- Decreased:
  - Decreased contractility (Coronary heart disese, heart failure)
  - valvular diseases (regurgitation or stenosis)
- Increased:
  - hypertrophic cardiomyopathy

# Ejection fraction (EF)

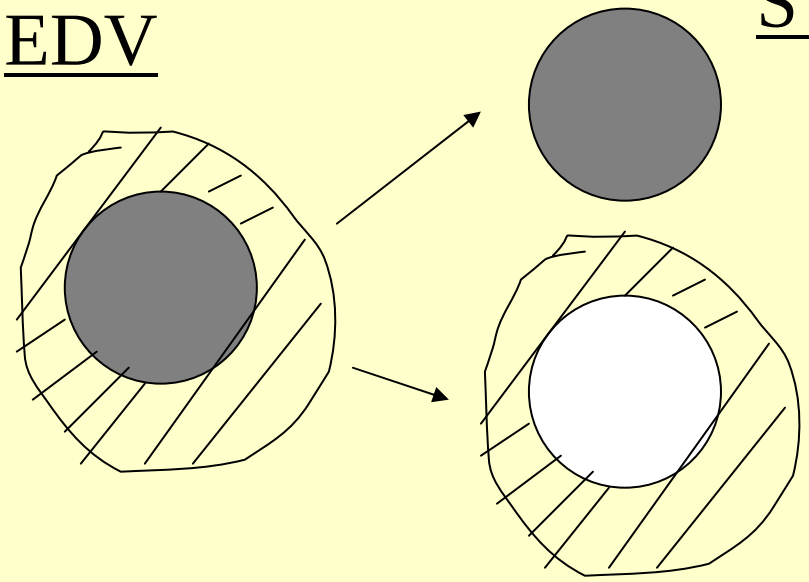
- Normal values:
  - 50–55 % and more
  - 40 % and less in systolic dysfunction

- Measurement:
  - most commonly by ***echocardiography***

# Ejection fraction (EF)

## Normal Heart

EDV



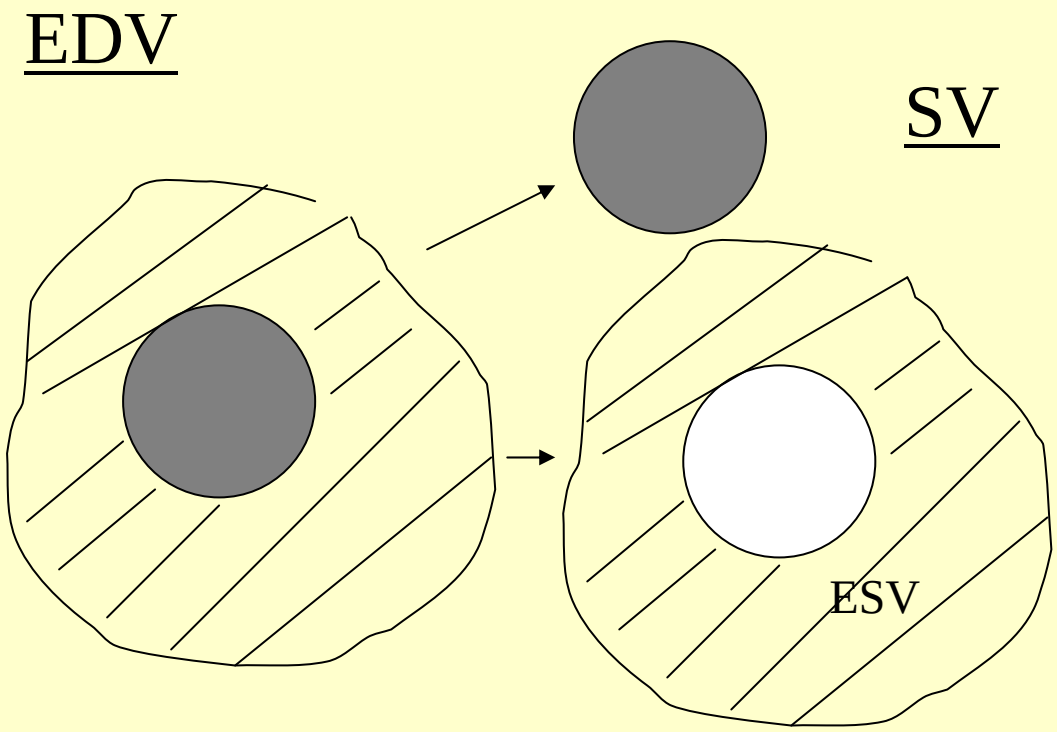
SV (stroke volume): EDV – ESV

ESV (endsystolic volume)

EF (ejection fraction) = SV/EDV minim. ~ 50 %

# Ejection fraction (EF)

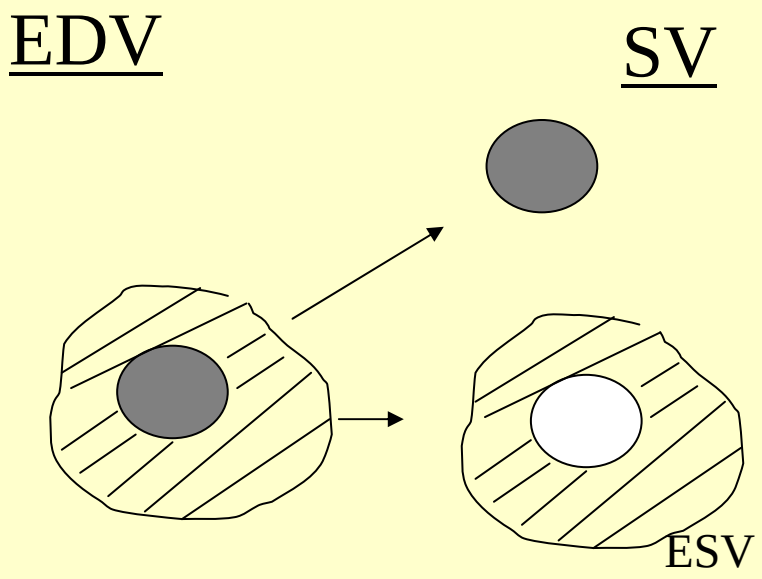
## Systolic heart failure = decrease of EF




- SV does not change
- Increased preload
- Increased enddiastolic pressure
- Dilatation of heart
- Increase end diastolic volume

# Ejection fraction (EF)

## Diastolic heart failure = EF does not change

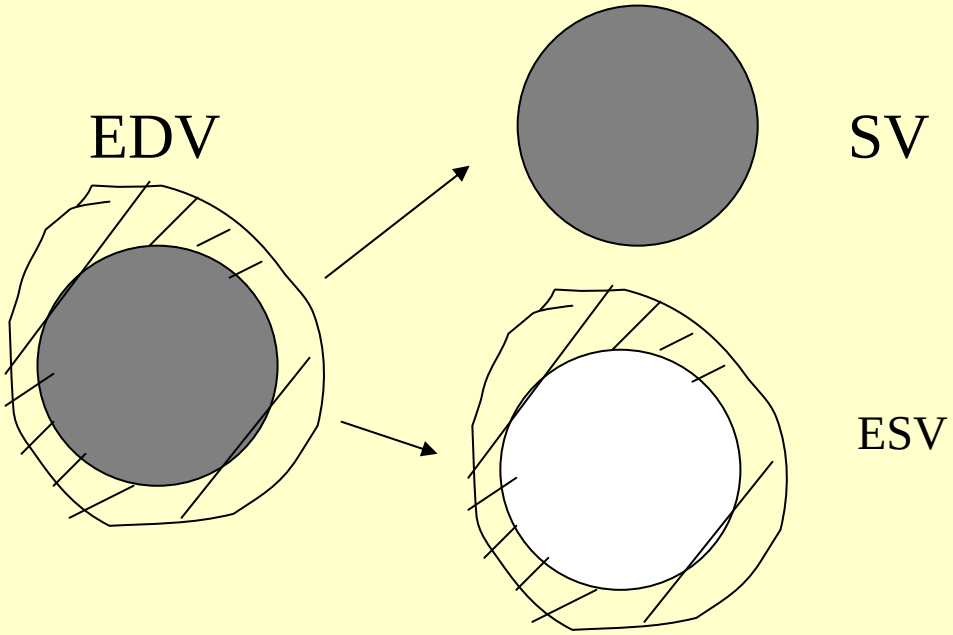



- Decreased LV diastolic compliance associated with increased LV diastolic pressure
- Decreased end-diastolic volume (preload-dependent)
- Depressed myocardial contractile function
- EF does not change or increase
- SV decline

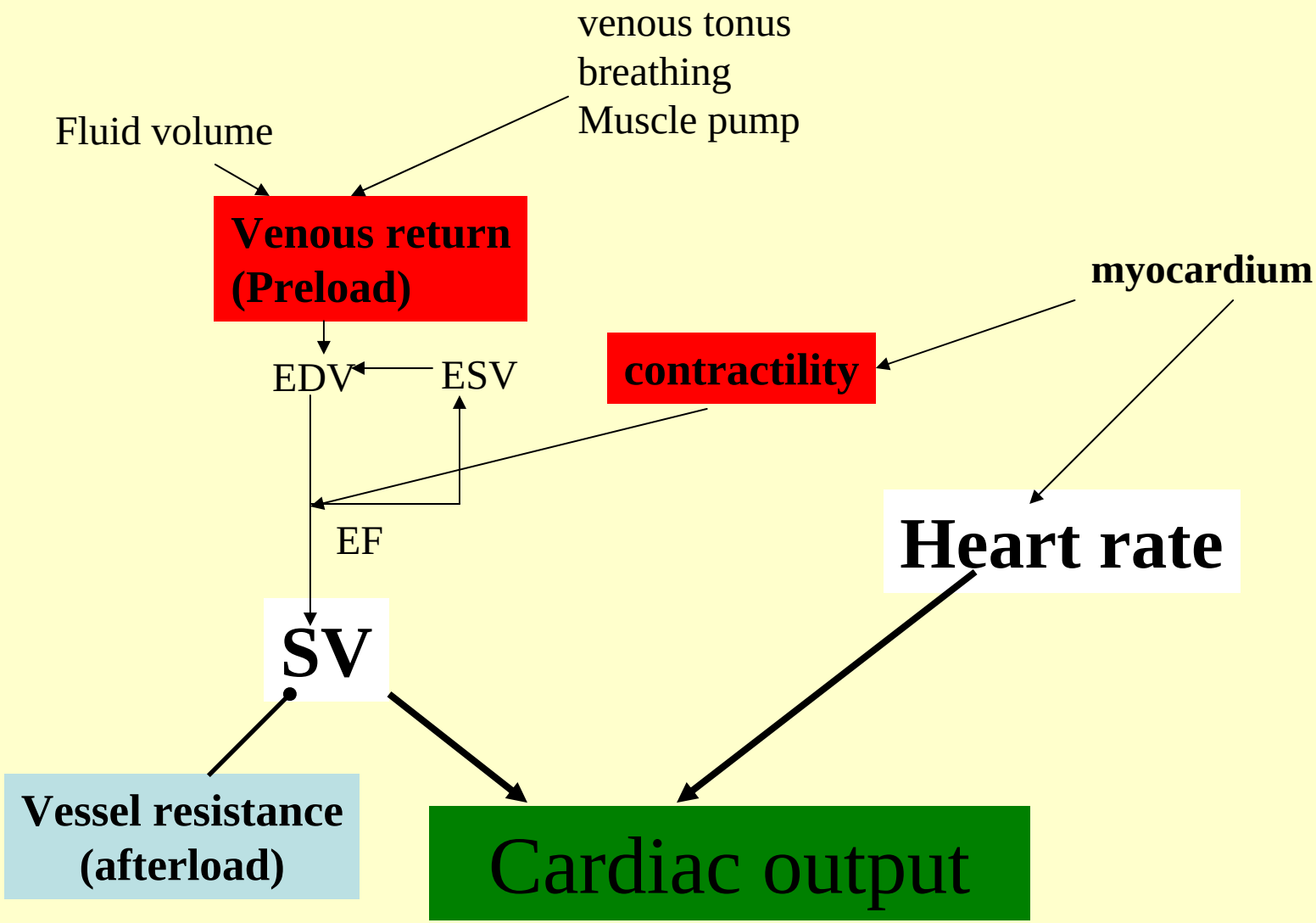
# Ejection fraction (EF)

Heart stimulated by sympathetic nerves (e.g. in shock)

EF increases, e.g. up to 80 %



# Cardiac output



# Cardiac output

Is determined by heart rate (HR) and stroke volume (SV)

$$CO = HR \times SV$$

- Normal values: 4–7 L/min

Measurment:

- Thermodilution (standard) method – Swan-Ganz catheter
- Fick Principle - C

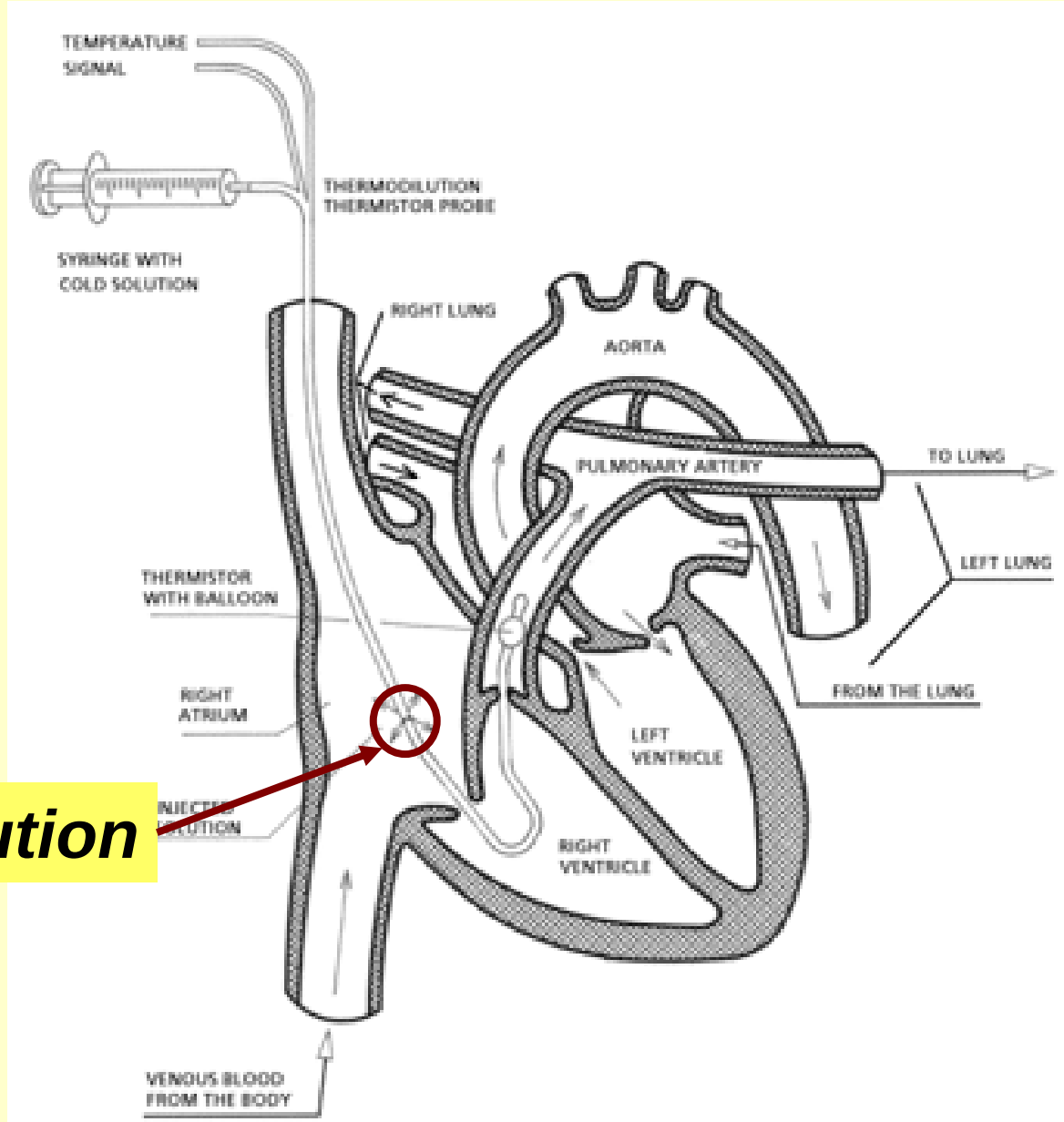
$$\text{Cardiac Output} = \frac{\text{oxygen consumption}}{\text{arteriovenous oxygen difference}} \times 100$$

- Noninvasive methods (Ultrasound with Doppler)

# Thermodilution method

- Indicator dilution principle (temperature change)
- A known amount of solution at given (low) temperature is injected rapidly into the right atrial lumen

# Thermodilution method




***Injected cooler solution***

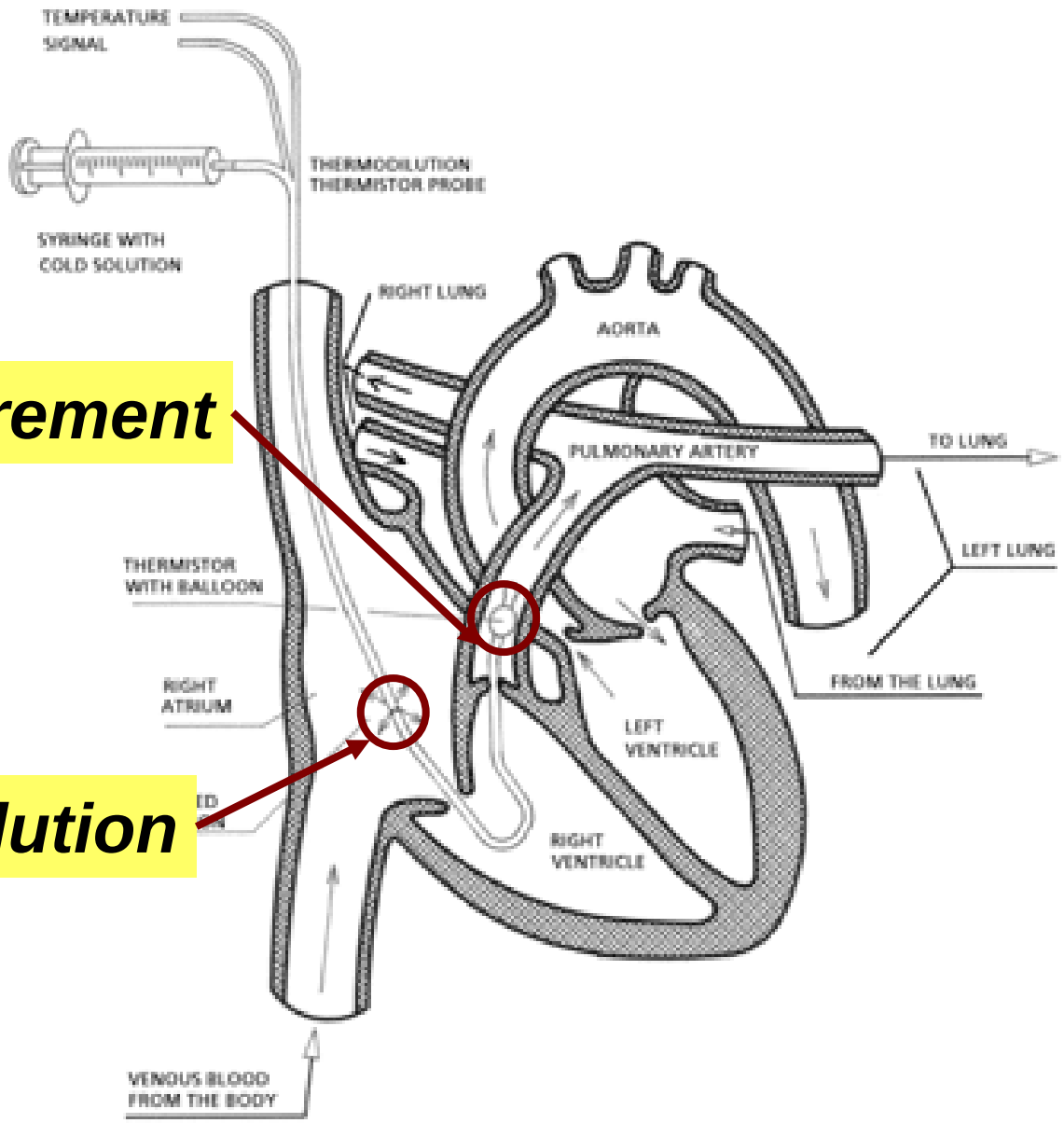
# Thermodilution method

- Indicator dilution principle (temperature change)
- A known amount of solution at a known temperature is injected rapidly into the right atrial lumen
- This cooler solution cools the surrounding blood, and the temperature is measured downstream in the pulmonary artery by a thermistor embedded in the catheter

# Thermodilution method

***Temperature measurement***

***Injected cooler solution***



# Thermodilution method

- Indicator dilution principle (temperature change)
- A known amount of solution at a known temperature is injected rapidly into the right atrial lumen
- This cooler solution mixes with and cools the surrounding blood, and the temperature is measured downstream in the pulmonary artery by a thermistor embedded in the catheter
- The resultant change in the temperature is then plotted on a time-temperature curve

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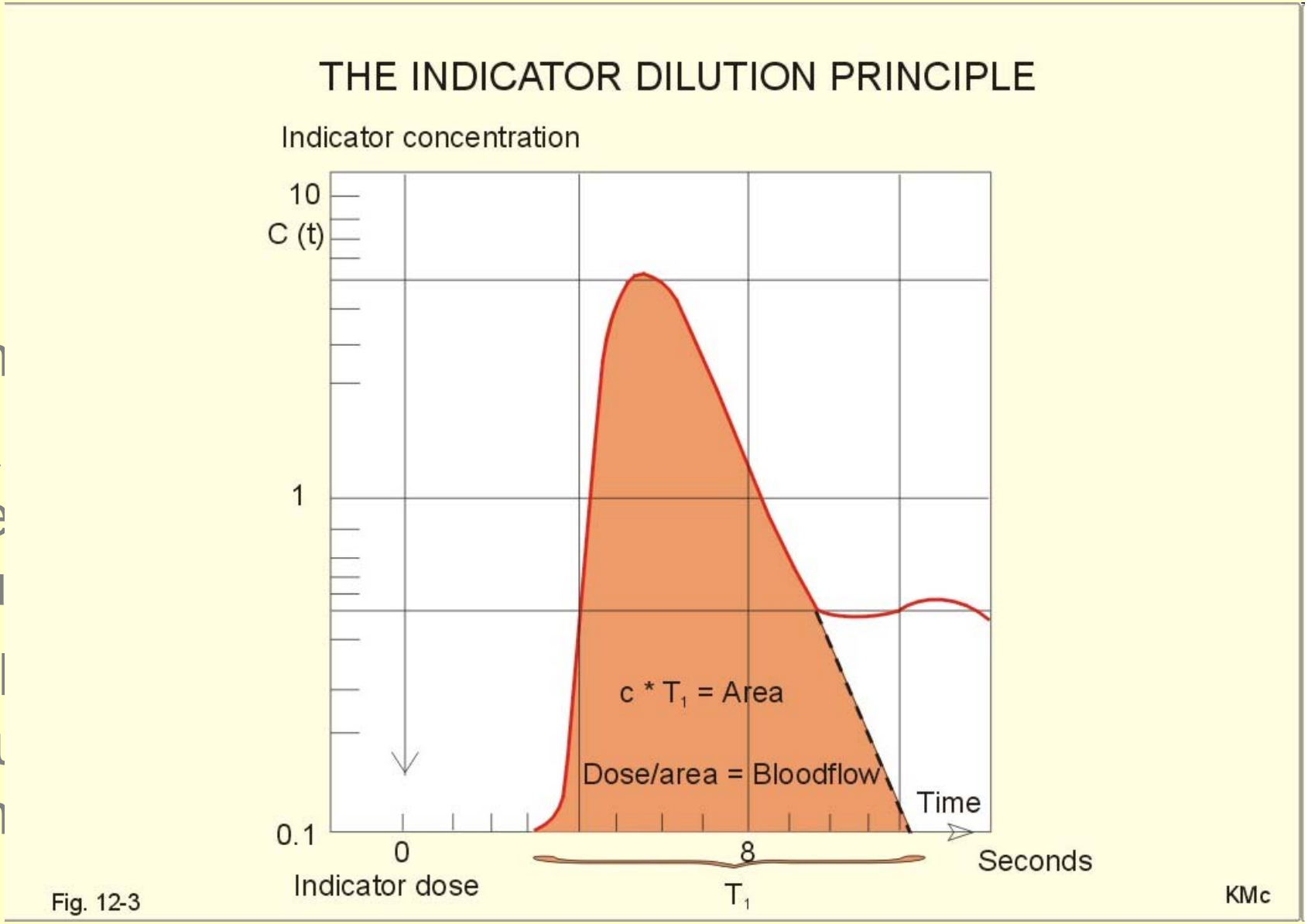


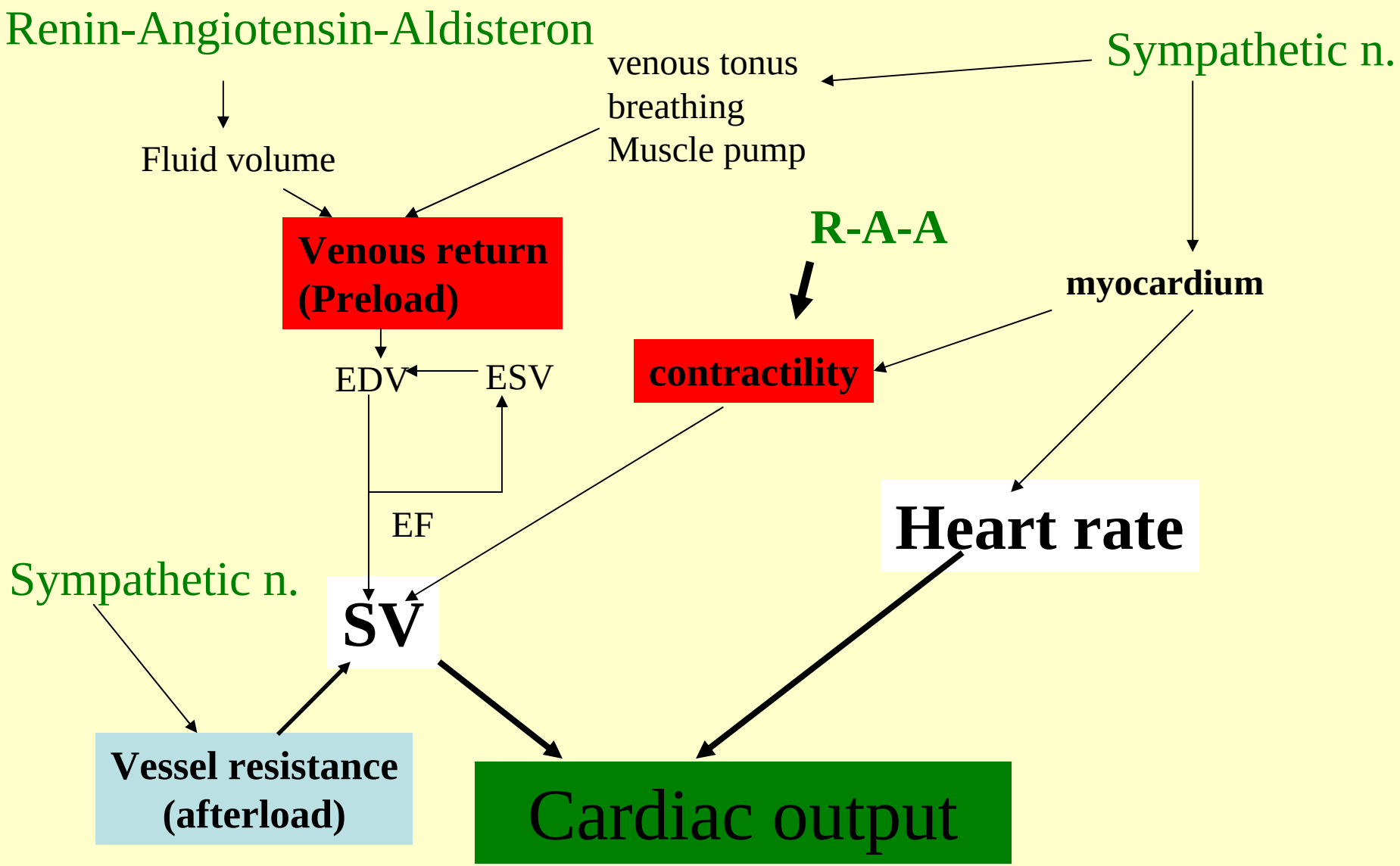

- The resultant change in the temperature is then plotted on a time-temperature curve

# Cardiac index

**CI** = CO / body surface area

- Normal values: 2.8 – 4.2 L/min/$m^2$

# Cardiac output



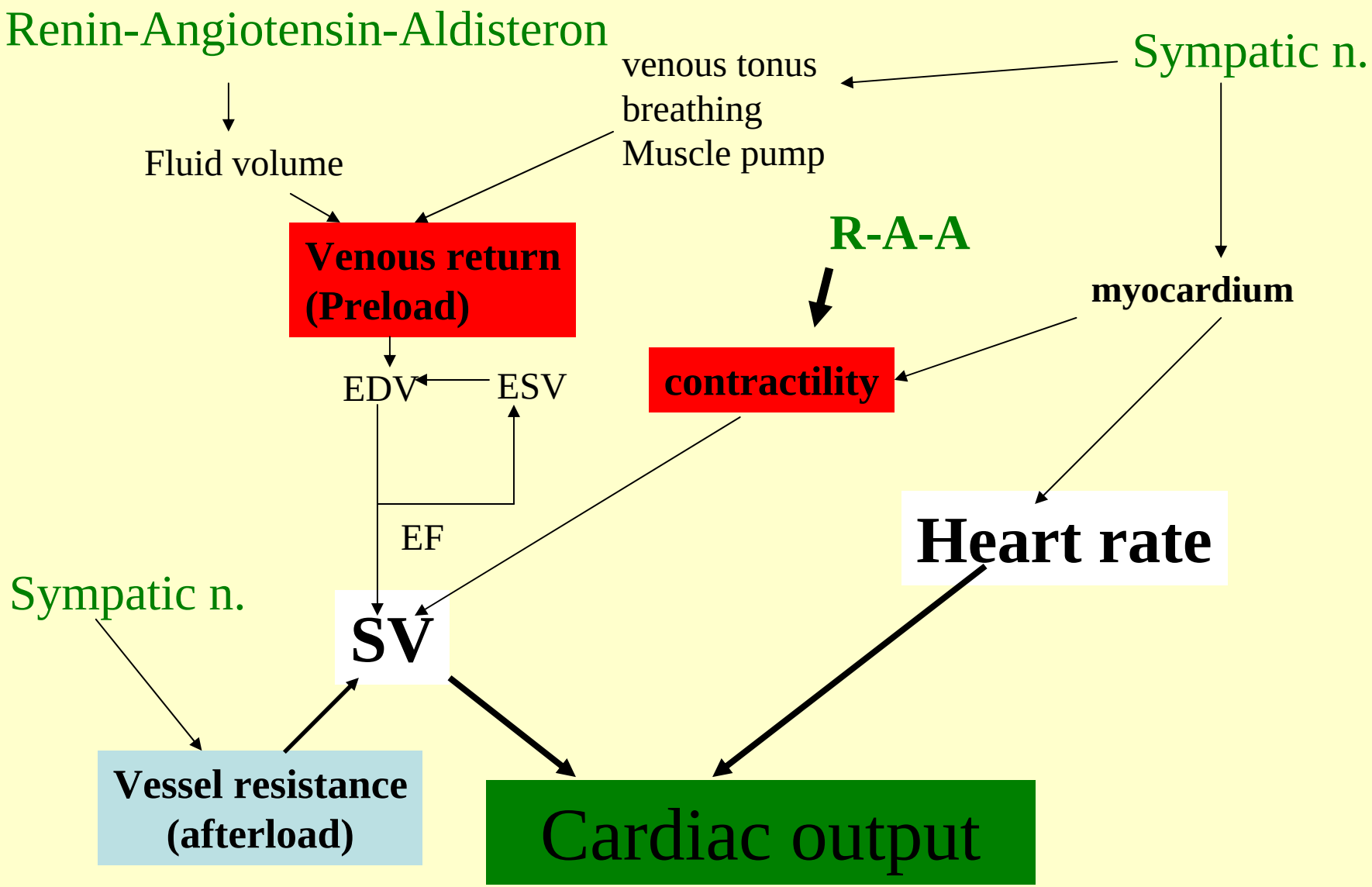
# Heart failure

Clinical syndrome associated with decreased cardiac output

Diagnostic criteria

- Symptoms of heart failure
- Signs of fluid retention
- Objective evidence of a structural or functional abnormality of the heart at rest

# Systolic Function of Heart

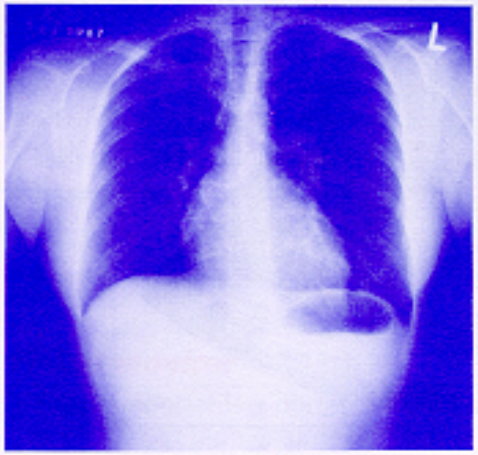


# CASE – Heart failure

# Imaging methods

- Ultrasound – Echo
- Chest X-ray
- Angiography - Coronarography
- MRI – Magnetic resonance imaging
- CT – computer tomography
- PET (positrone emission tomography – evaluation of heart metabolism
- Radioisotope methods

# Chest X-ray



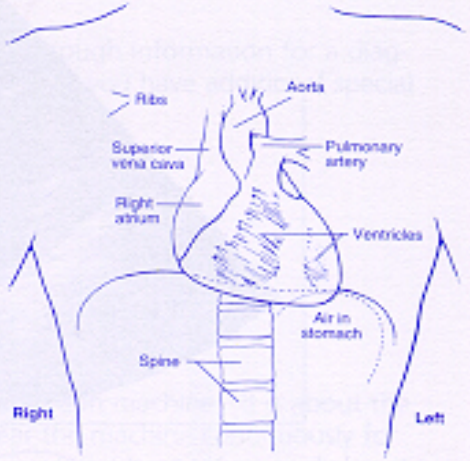



*Figure 2.a Chest X-ray*
Notice the position of the heart and its major vessels in the normal x-ray shown above.

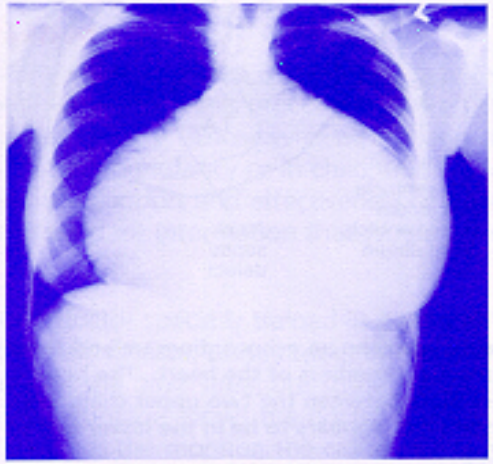
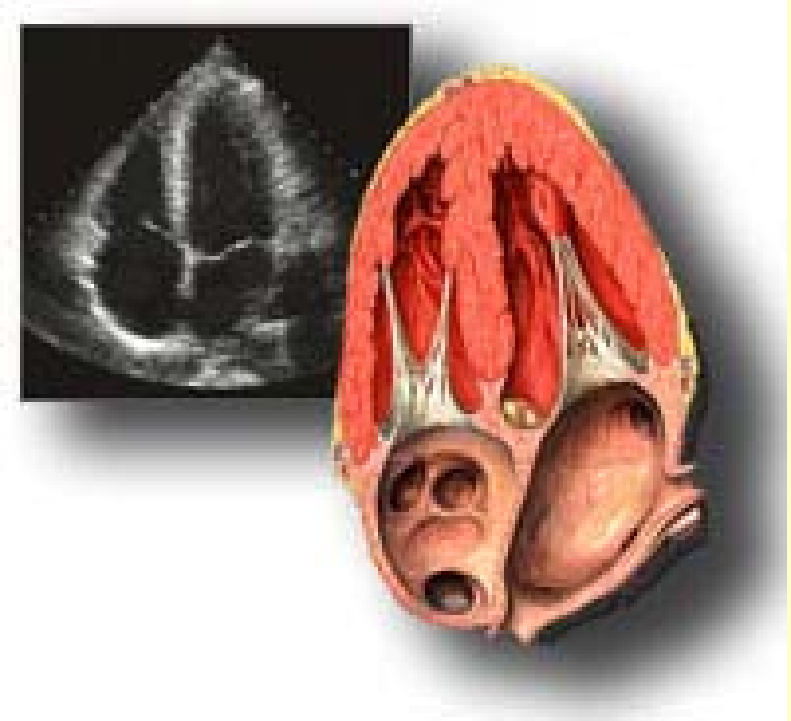


*Figure 2.b*
Compare this x-ray of an abnormally enlarged heart with the one above.

# Echokardiography (cardiac ultrasound) (2D, 3D)

- Size and mobility of the heart and its parts
  - myocardium thickness, mobility of the myocardium, valve shape and mobility, papillary muscles, size of myocardial cavities, pericardium
- Mechanical manifestations of ischemia
  - segmental kinetic defects of myocardium
    - segments corresponds to areas supplied with certain branches of coronary arteries
    - hypokinesis, akinesis, dyskinesis

# Ultrasound – Echo



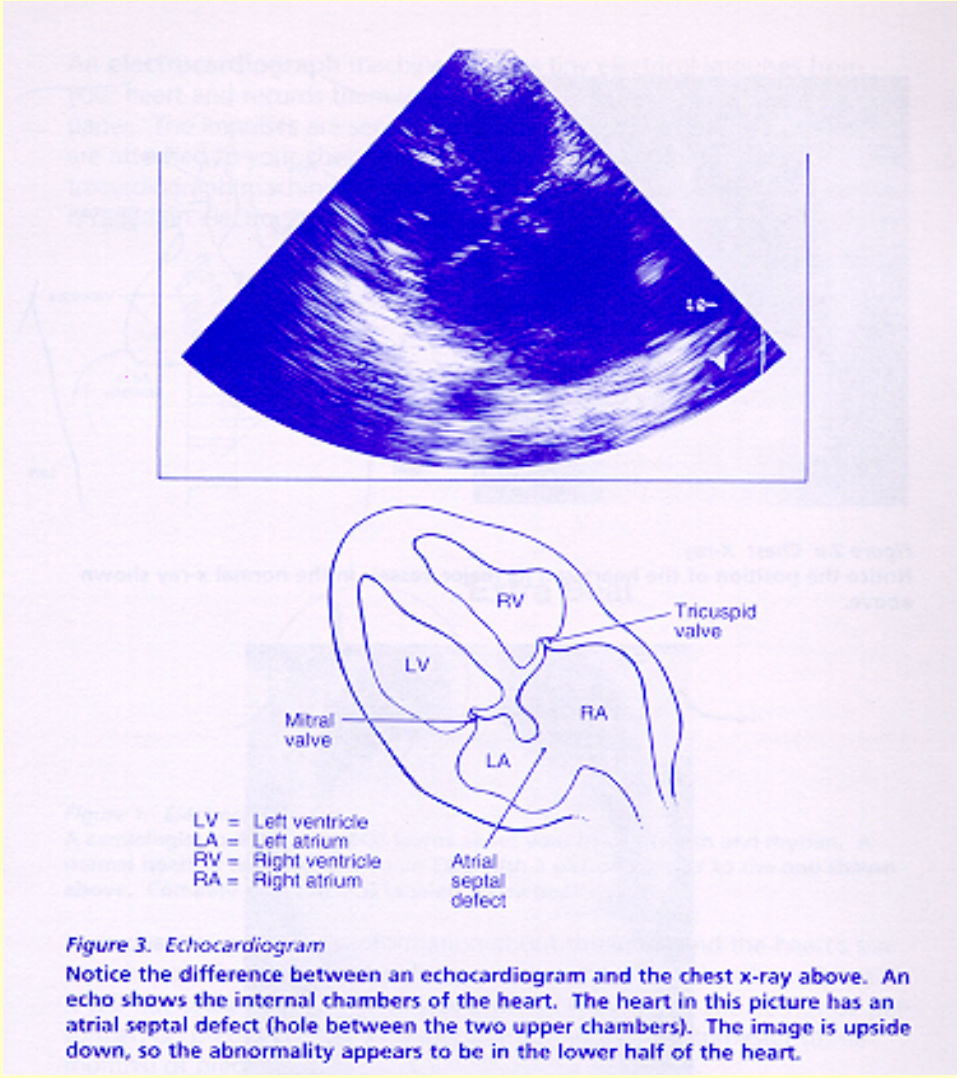
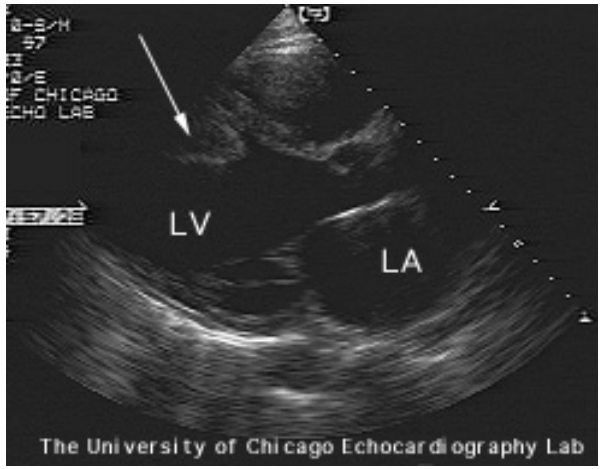
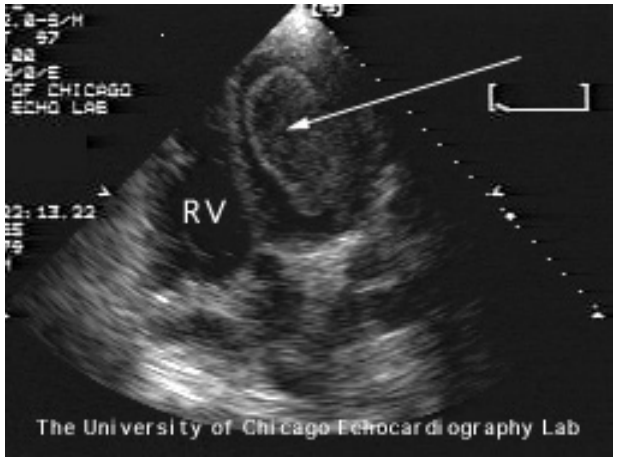


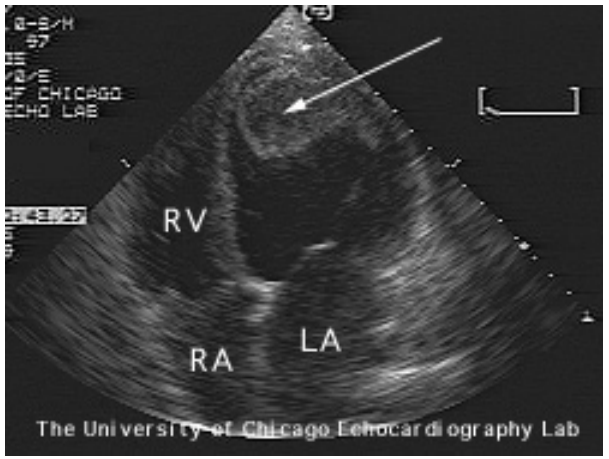

*Figure 3. Echocardiogram*

**Notice the difference between an echocardiogram and the chest x-ray above. An echo shows the internal chambers of the heart. The heart in this picture has an atrial septal defect (hole between the two upper chambers). The image is upside down, so the abnormality appears to be in the lower half of the heart.**

# Thrombus in Left Ventricle (Echokardigrafy)



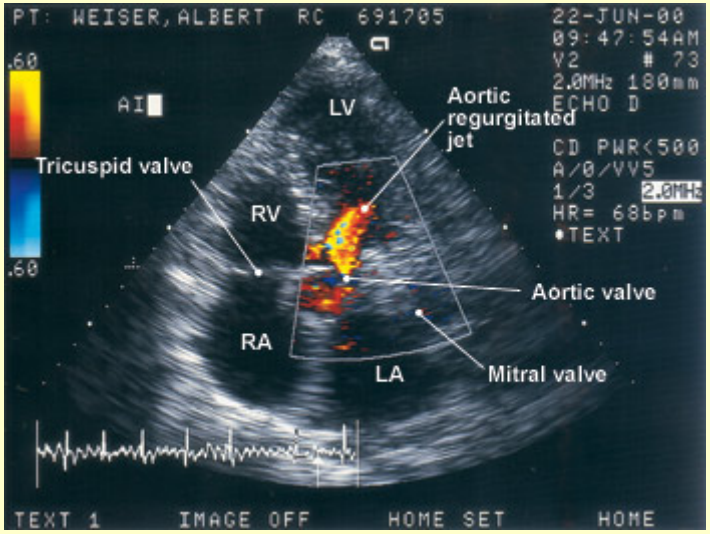

occupies a substantial portion of the LV apex

# Echocardiography with doppler

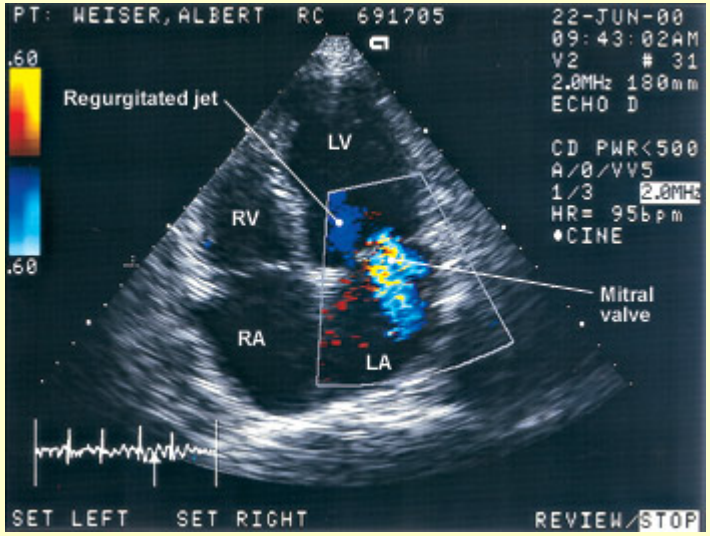
- blood flow in the heart
  - direction
  - velocity of the blood flow
  - type of blood flow (laminar or non-laminar)
  - pressure gradients
  - EF (ejection fraction)
  - CO (cardiac output)

# Ultrasound – Echo

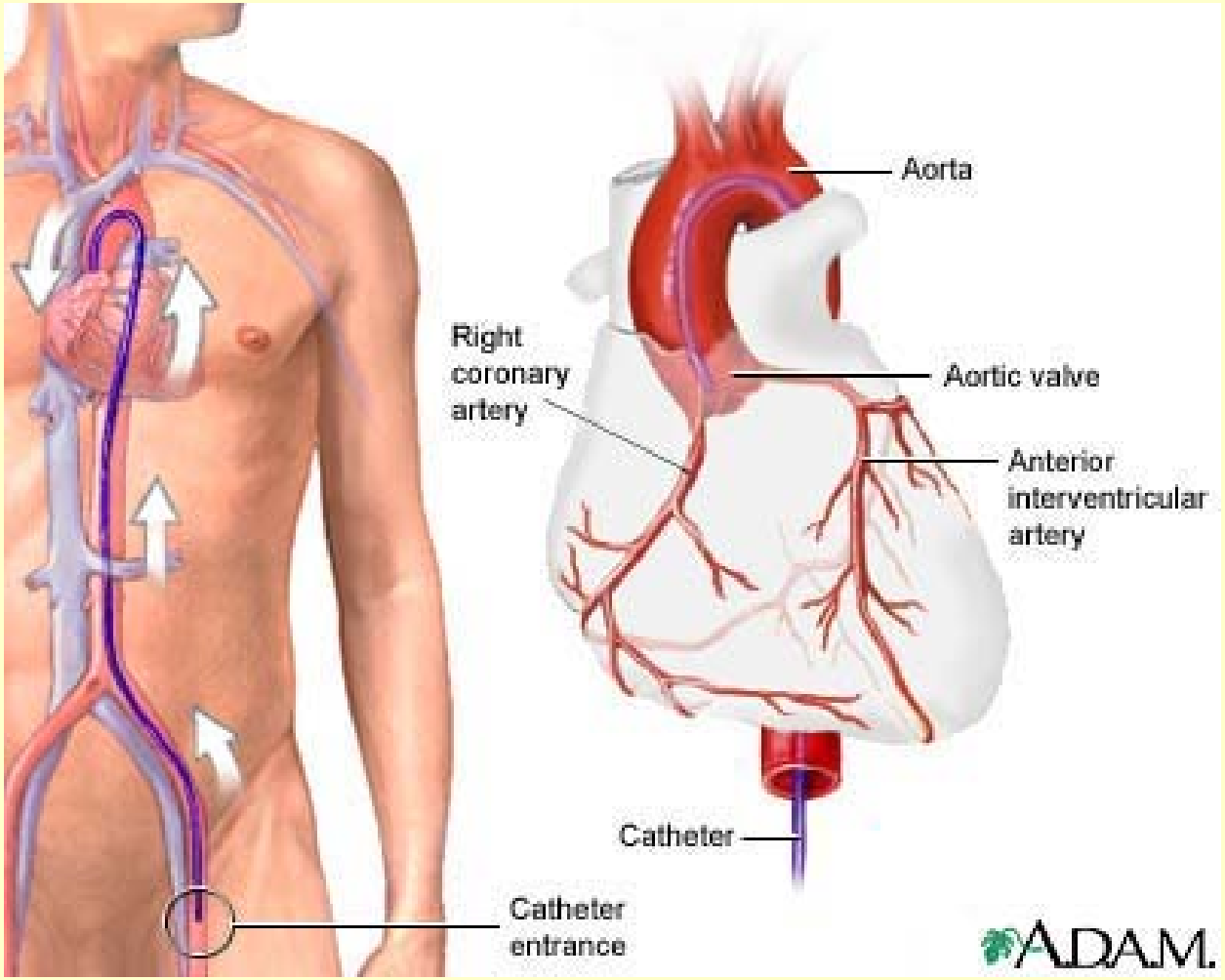
Aortal insuficiency (regurgitation)




Mitral insuficiency (regurgitation)

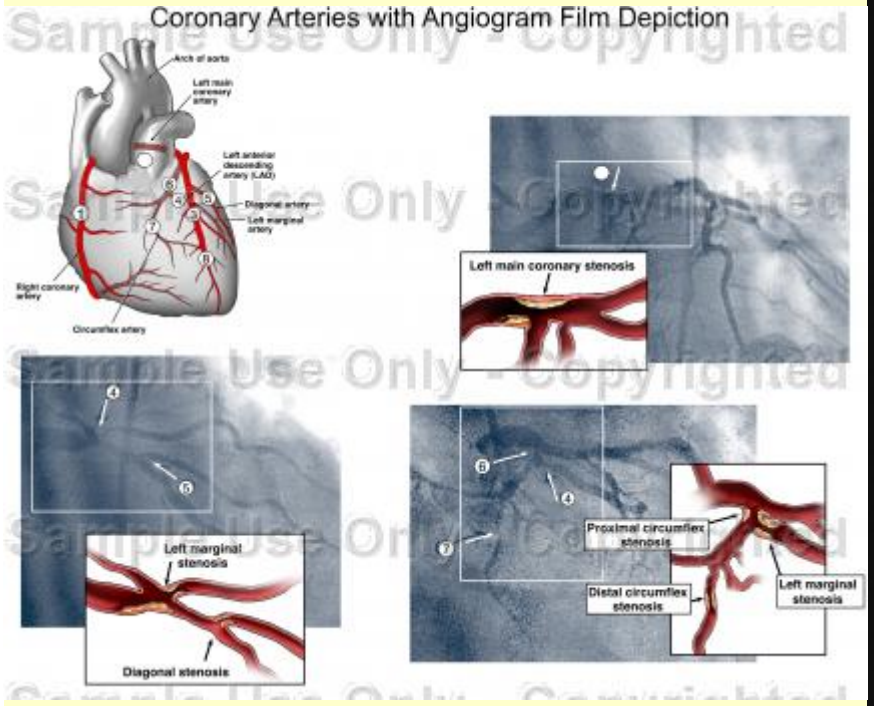


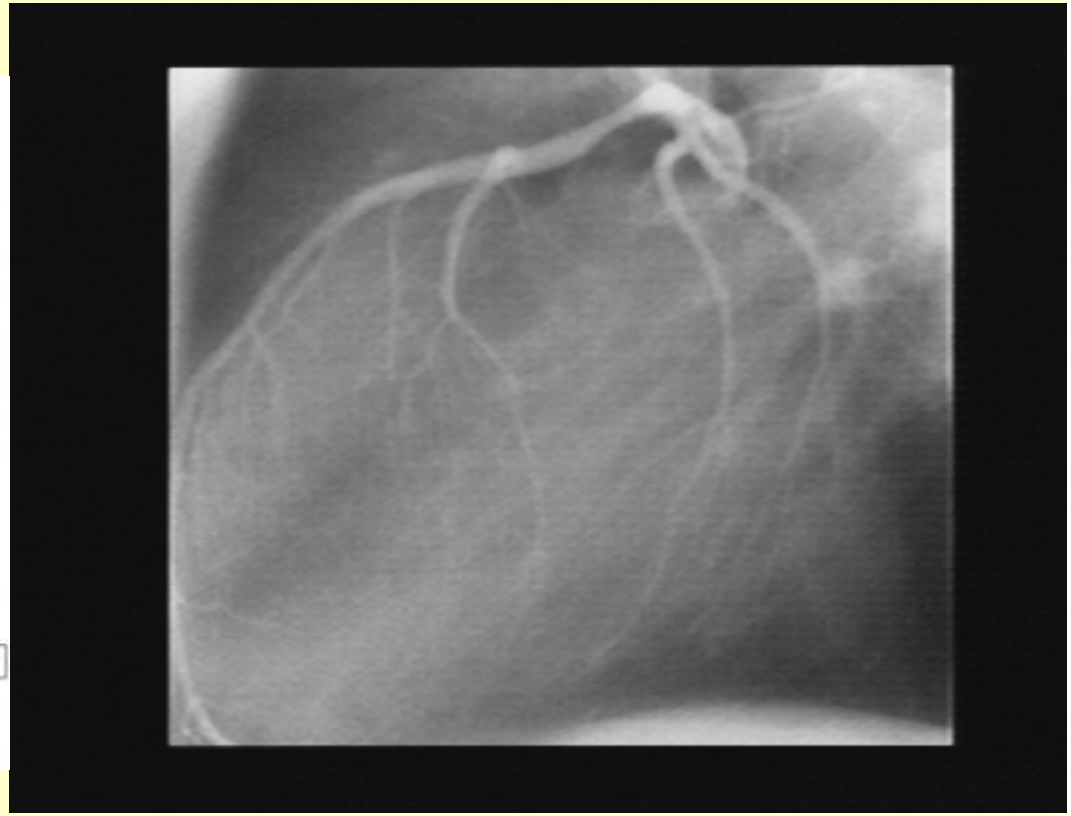
# Coronarography




about 3 to 5 complications for 1000 exams

# Coronarography



Radiological contrast product **is rapidly injected into the left and right coronary artery**

# Coronarography

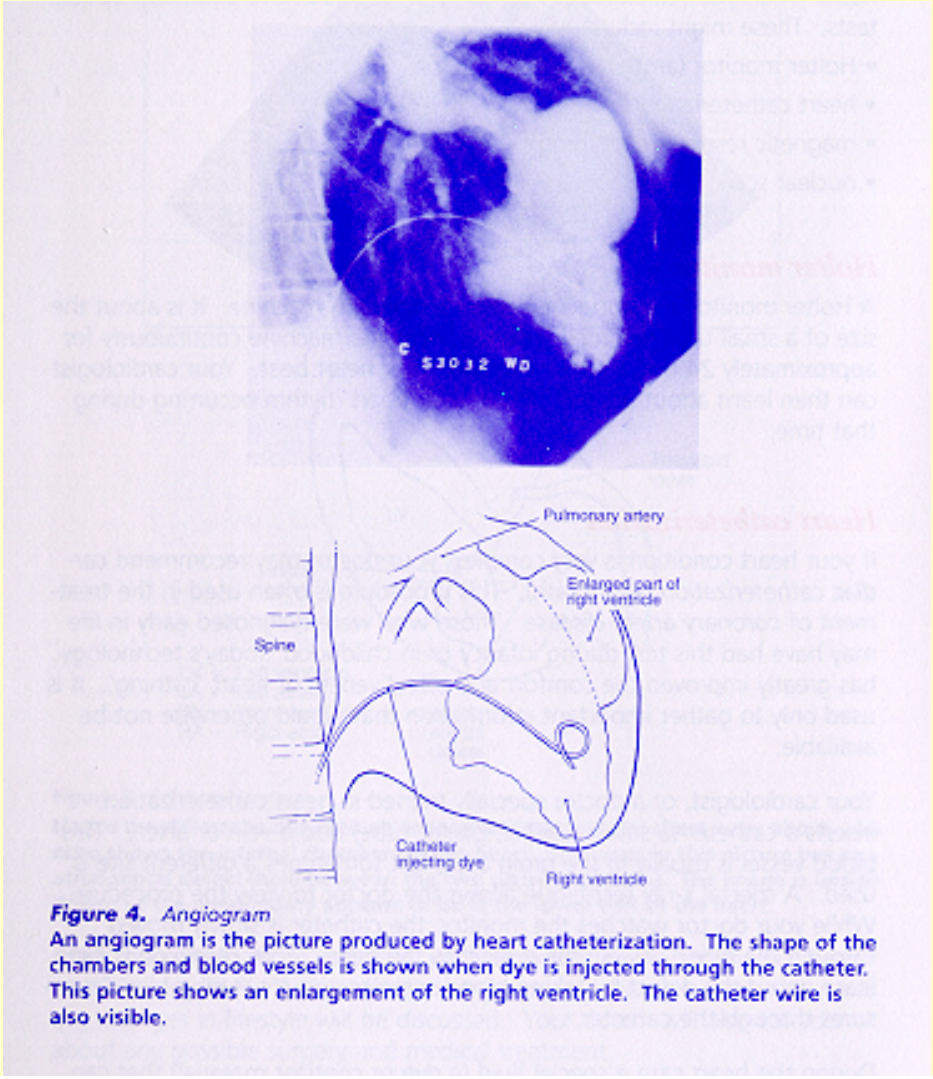



***Figure 4.*** *Angiogram*
An angiogram is the picture produced by heart catheterization. The shape of the chambers and blood vessels is shown when dye is injected through the catheter. This picture shows an enlargement of the right ventricle. The catheter wire is also visible.

# To remember

- Cardiac output
- Ejection fraction
- Manifestation of heart failure
- Brain natriuretic peptide (BNP)
- Echocardiography (with Doppler)
  - anatomic and functional evaluation of the heart

# II - Arrhythmias

Jan Živný
Department of Pathophysiology
jzivny@LF1.cuni.cz

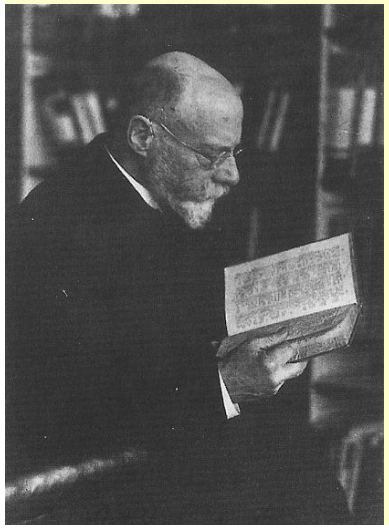
# Outline

- Introduction to ECG measurement
- ECG Interpretation
- Myocardial Infarction
- Arrhythmia
- Summary

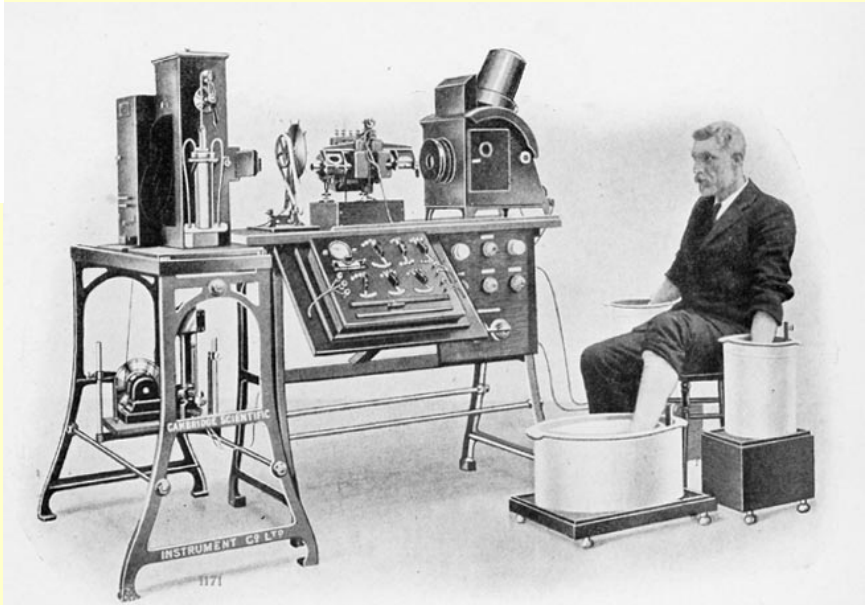
# Willem Einthoven (1860 – 1927)

- Physiologist from University of Leiden (Holland)
- Develped string galvanometer (~1903) and used it to measure electrical activity of the heart from limb leads (Einthoven triangel)

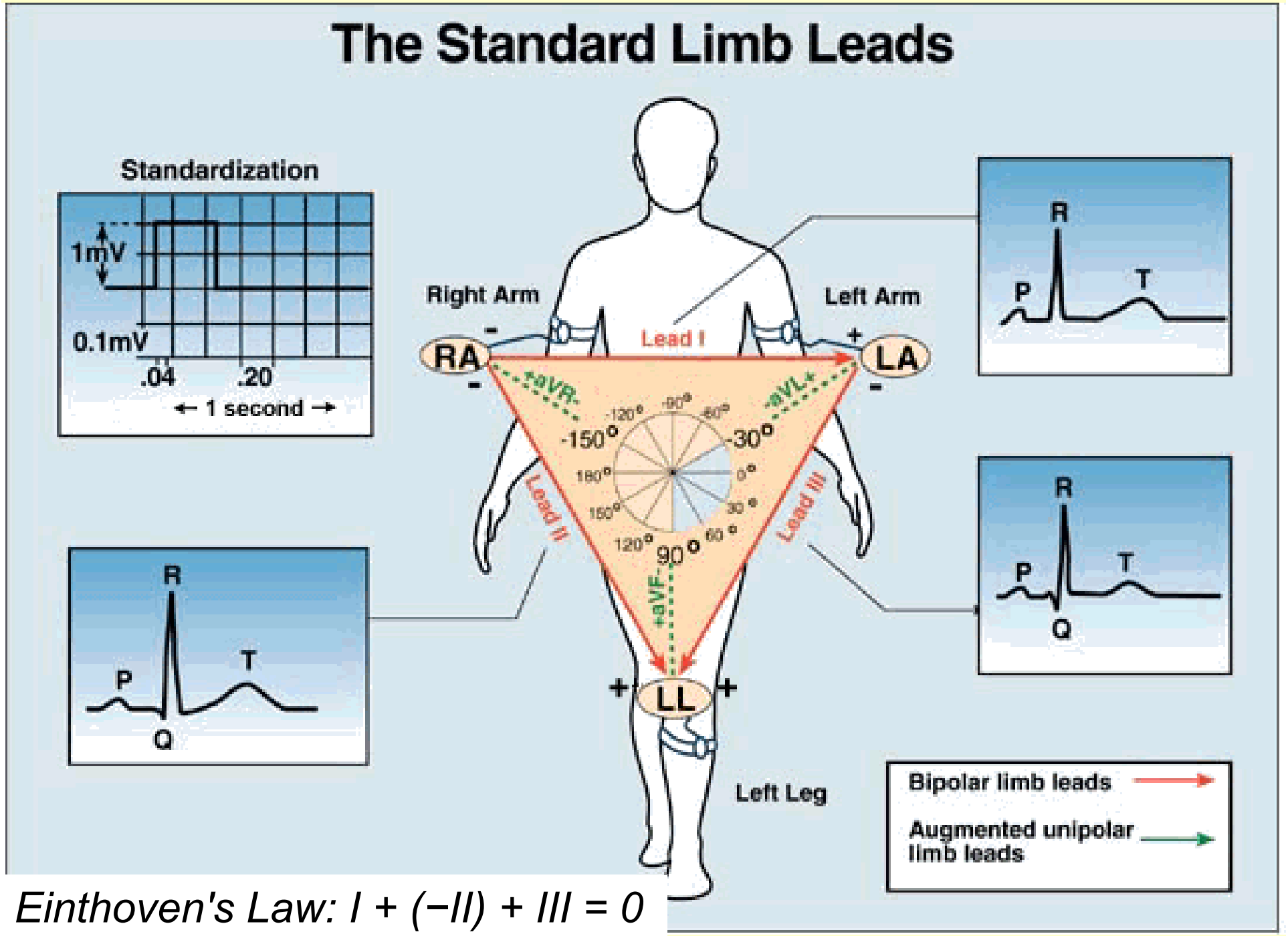
Introduced the designation of ECG deflections P Q R S T



In 1924 awarded Nobel price



Photograph of a Complete Electrocardiograph, Showing the Manner in which the Electrodes are Attached to the Patient, In this Case the Hands and One Foot Being Immersed in Jars of Salt Solution




*Einthoven's Law: I + (−II) + III = 0*

# Frank Wilson

- 1934
- By joining the wires from the right arm, left arm and left foot with 5000 Ohm resistors defined an 'indifferent electrode' = Wilson Central Terminal
  - acts as an earth and is attached to the negative terminal of the ECG
- Wilson defined the unipolar limb leads VR, VL and VF
  - electrode attached to the positive terminal of the ECG

# Emanuel Goldberger

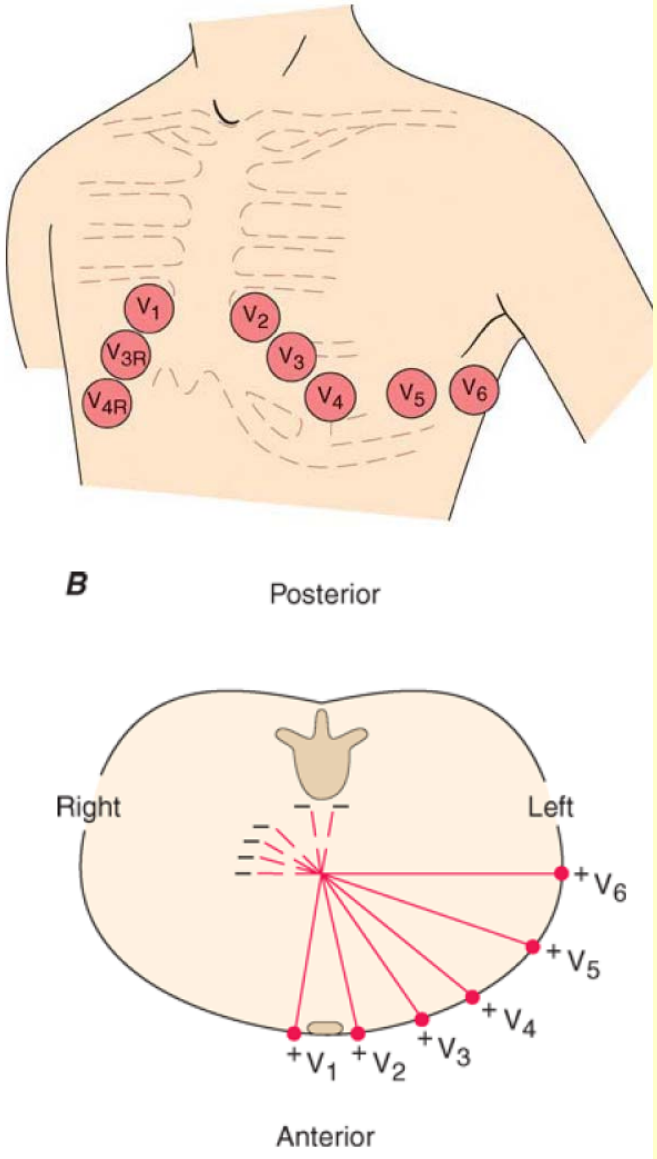
- 1942 increases the voltage of Wilson's unipolar leads by >50% and creates the augmented limb leads aVR, aVL and aVF

- Lead augmented vector right (aVR)
  - positive electrode on the right arm
  - negative electrode is a combination of the left arm and the left leg electrodes
- Lead augmented vector left (aVL)
  - positive electrode on the left arm
  - negative electrode is a combination of the right arm and the left leg electrodes
- Lead augmented vector foot (aVF)
  - positive electrode on the left leg
  - negative electrode is a combination of the left arm and the right arm electrodes

# Charles Wolferth and Francis Wood

- 1932 described the clinical use of chest leads
  - Wolferth CC, Wood FC. The ectrocardiographic diagnosis of coronary occlusion by the use of chest leads. Am J Med Sci 1932;183:30-35
- 1938 The American Heart Association and the Cardiac Society of Great Britain define the standard positions and wiring, of the chest leads V1 - V6.
  - Barnes AR, Pardee HEB, White PD. et al. Standardization of precordial leads. Am Heart J 1938;15:235-239

# Location of standard chest leads (4th and 5th intercostal area)




V1: right from sternum 4th intercostal area

V2: left from sternum 4th intercostal area

V3: between V2 and V4

V4: left in mid-clavicular line in 5th intercostal area

V5: horizontally left from V4 in anterior axillary line

V6: horizontally left from V5 in mid-axillary line

# ECG Interpretation

Before each analysis check standardization (calibration) and technical features (including lead placement and artifacts)

# ECG Interpretation

1. Rhythm analysis
2. Measurements (usually made in frontal plane leads)
3. Conduction analysis
4. Waveform description
5. ECG interpretation and summary
6. Comparison with Previous ECG (if any)

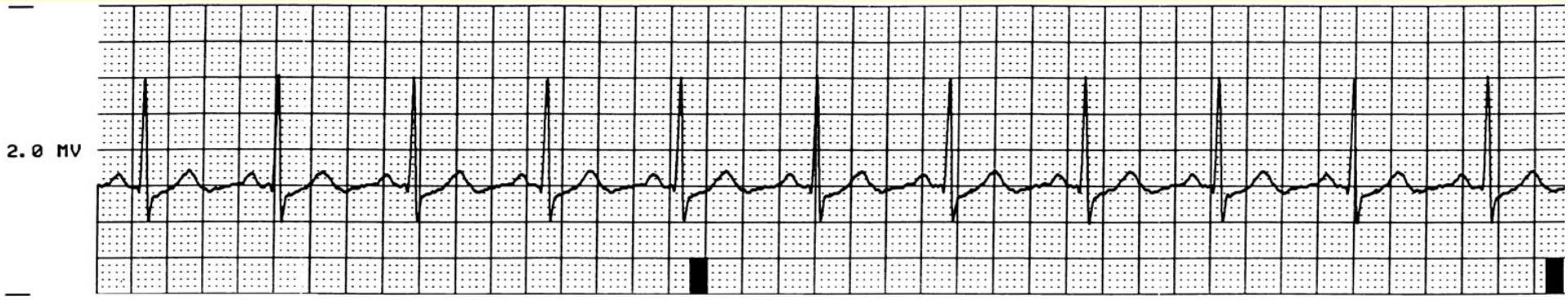
# 1. Rhythm Analysis

- Basic rhythm
  - **"normal sinus rhythm“**
  - **other “abnormal” rhythms** (e.g. sinus tachycardia, atrial fibrillation, etc.)
- Identify additional rhythm events if present
  - premature ventricular complexes (PVC's)
  - premature atrial complexes (PAC's), etc

# Sinus rhythm

- P wave is present
- P wave have constant configuration
- PQ interval is between 120 - 210 ms
- QRS complexes of normal width (60 – 120 ms)
- Intervals between QRS komplexes are constant
- HR is between 60 and 100 bpm

- The P waves in leads I and II must be upright (positive) if the rhythm is coming from the sinus node and each P wave is followed by QRS



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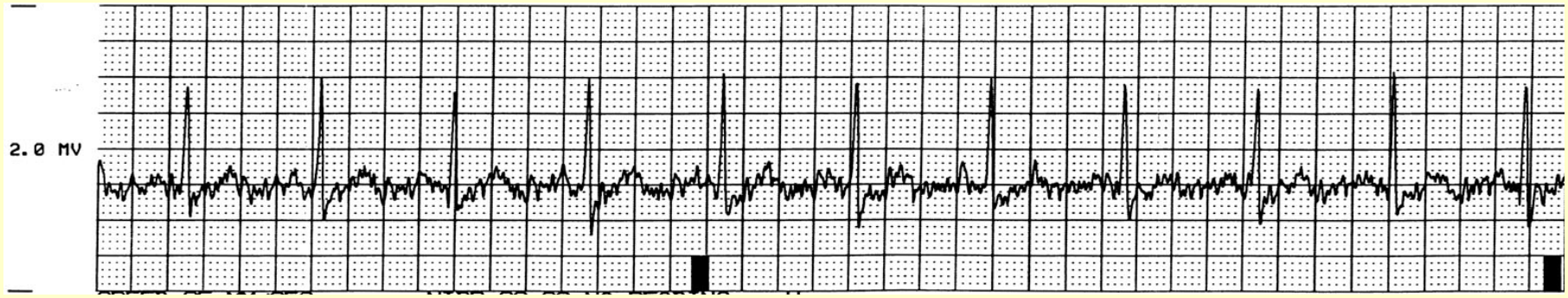
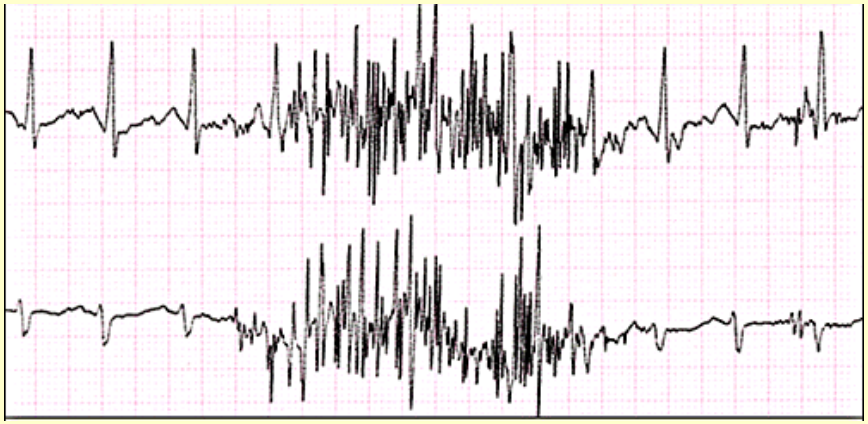


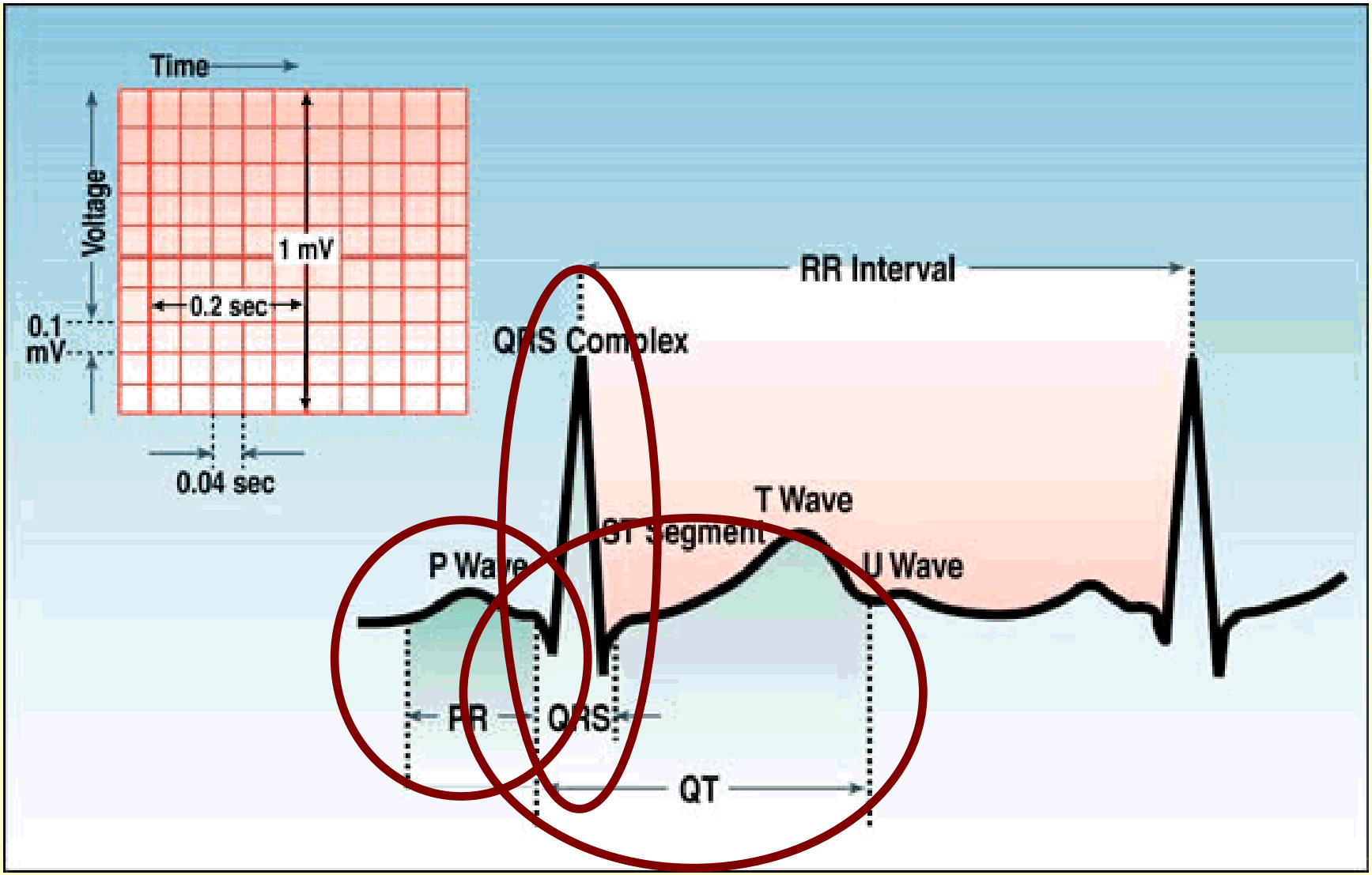



Artifact – muscle tremor

- Sinus rhythm is masked by irregular electric activity of skeletal muscles

# 2. Measurements

- Heart rate
  - state atrial and ventricular, if different
- PR (PQ) interval / AV conduction
  - from beginning of P to beginning of QRS (120 – 200 ms)
- QRS duration / intraventricular conduction
  - width of most representative QRS (60-120 ms)
- QT interval
  - from beginning of QRS to end of T (varies with HR)
- QRS axis in frontal plane

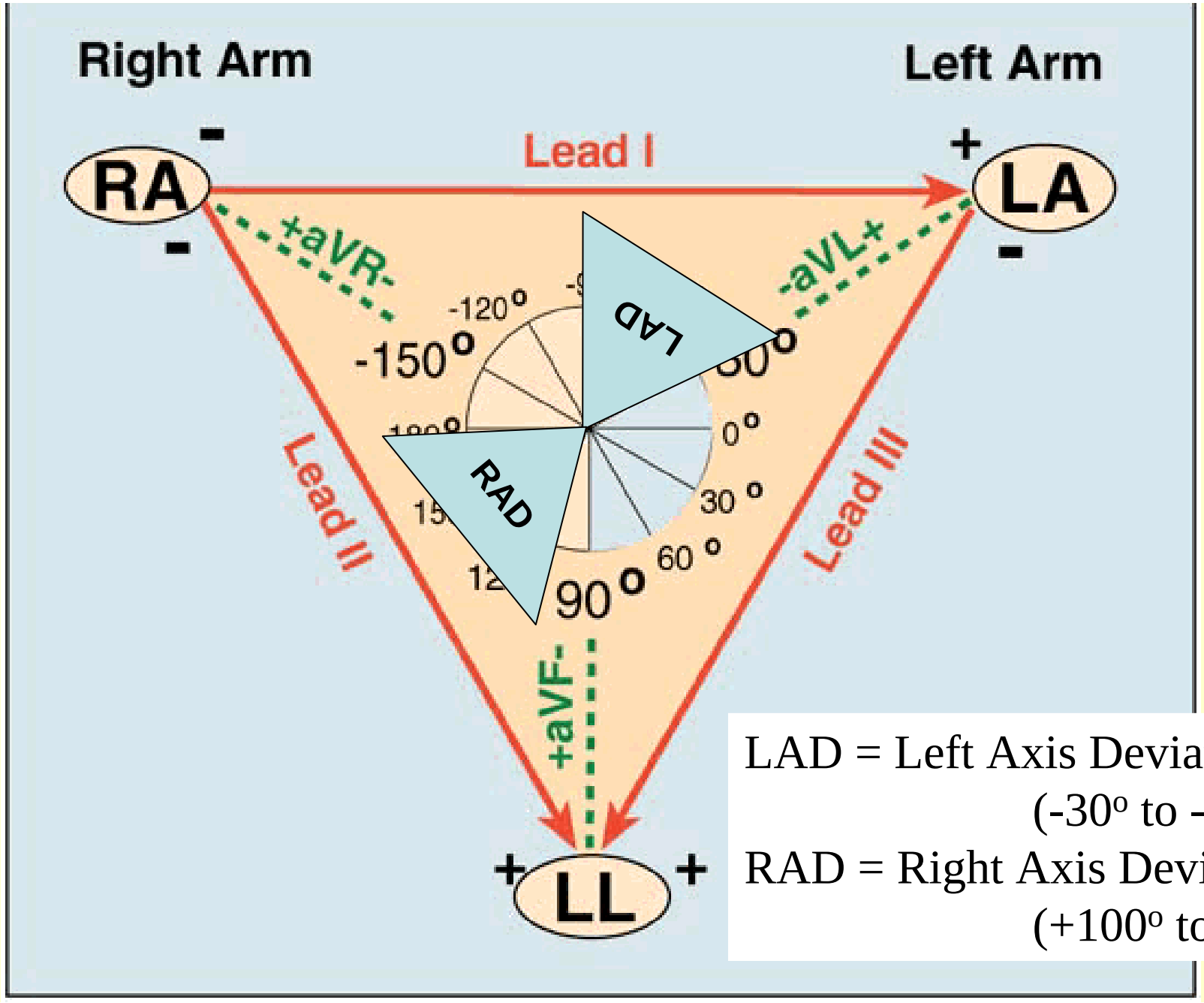



- PR (PQ) interval: 120 – 200 ms
- QRS complex: 60 – 120 ms
- QT interval: varies with heart rate

# QRS axis

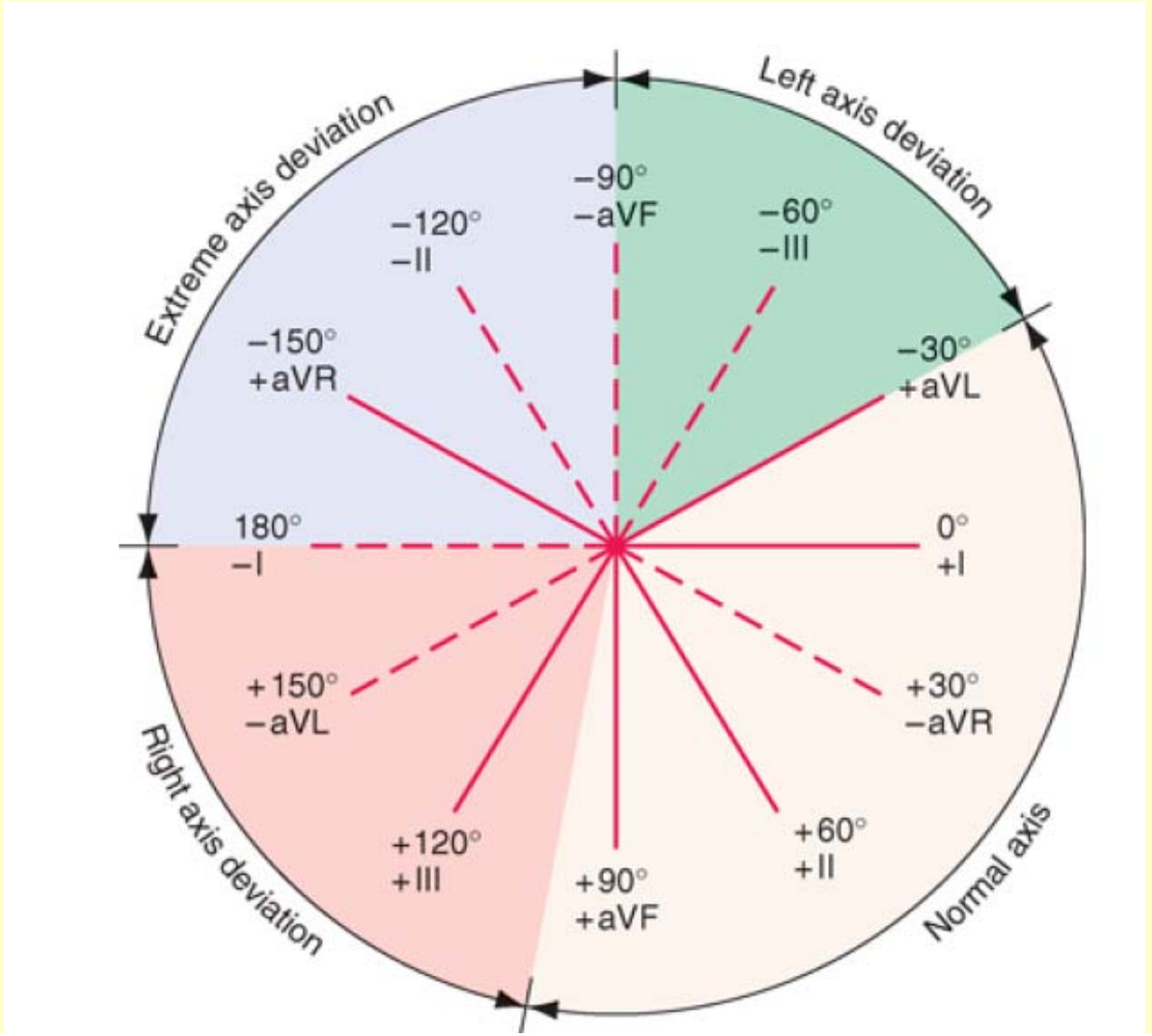
- The QRS axis represents the average direction of ventricular activation in the frontal plane
- Can inform about changes in the sequence of ventricular activation
  - conduction defects (e.g. left anterior fascicular block)
  - indicator of myocardial damage (e.g. myocardial infarction).

Normal axis: -30° to +100°. Voltage in leads I and II is positive




LAD = Left Axis Deviation
(-30° to -90°)

RAD = Right Axis Deviation
(+100° to +180° )


Extreme axis deviation
Left axis deviation
Right axis deviation
Normal axis
−90°
−aVF
−120°
−II
−60°
−III
−150°
+aVR
−30°
+aVL
180°
−I
0°
+I
+150°
−aVL
+30°
−aVR
+120°
+III
+90°
+aVF
+60°
+II

# Determination of QRS axis

A. One isoelectric lead is present
   - The lead with equal forces in the positive and negative direction
   - The QRS axis is perpendicular to that lead's orientation (two directions)
   - chose the perpendicular that best fits the direction of the other ECG leads

B. No isoelectric lead
   - Usually two leads that are nearly isoelectric (always $30^{o}$ apart)
   - Find the perpendiculars for each lead and chose an approximate QRS axis within the 30o range

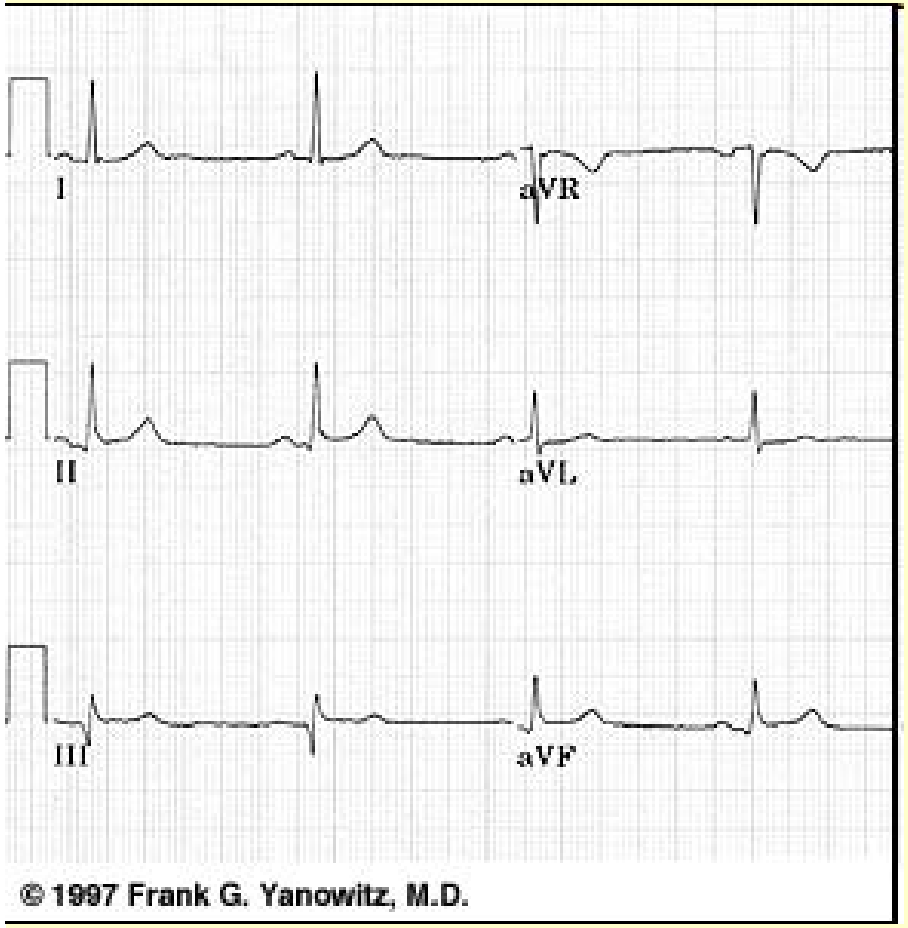
C. Each of the 6 frontal plane leads is small and/or isoelectric
   - The axis cannot be determined (indeterminate axis)
   - normal variant

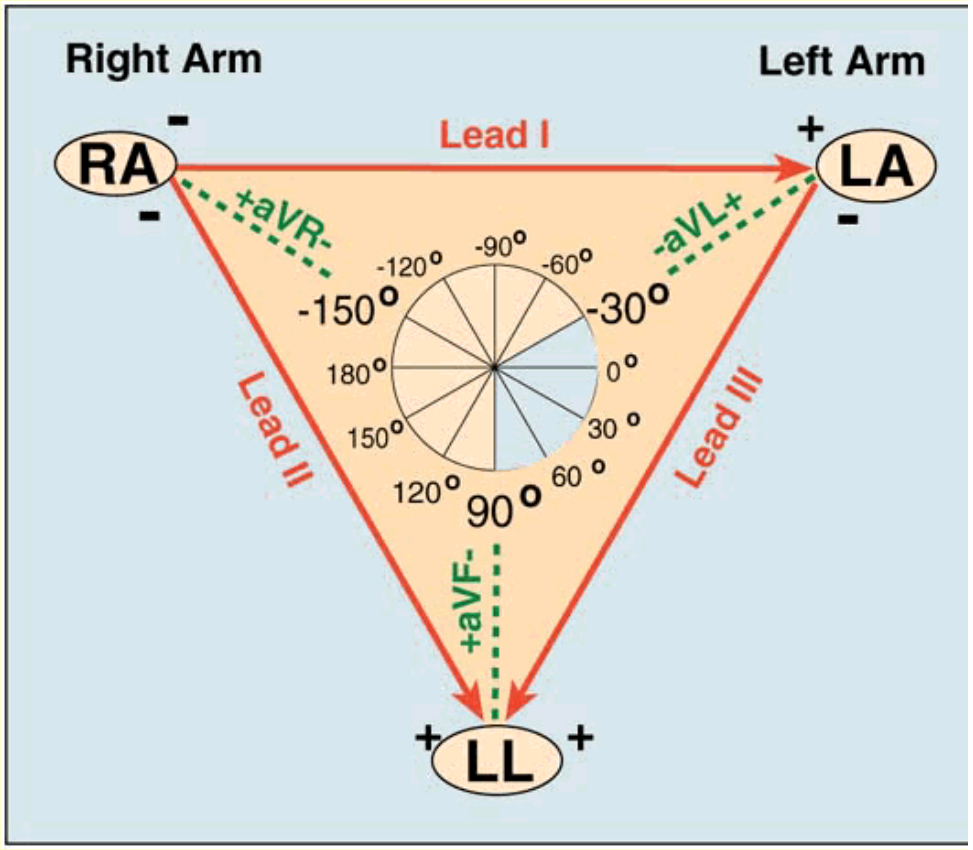
A. +90 degrees

B. +150 degrees

C. +30 degrees

D. - 45 degrees




Lead III is isoelectric

- Average direction of ventricular activation is perpendicular to lead III. (i.e. +30$^{o}$ or -150$^{o}$)

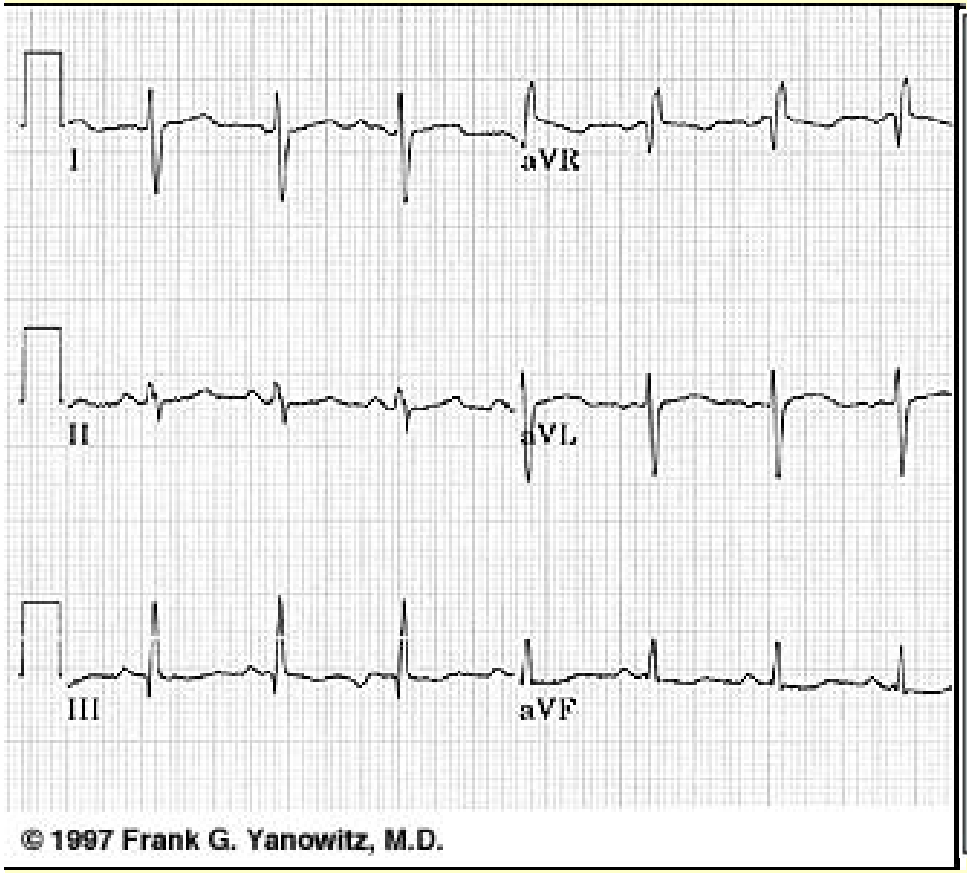
Lead I is positive and Lead III. Is positive = Physiological QRS axis (~ + 30°)

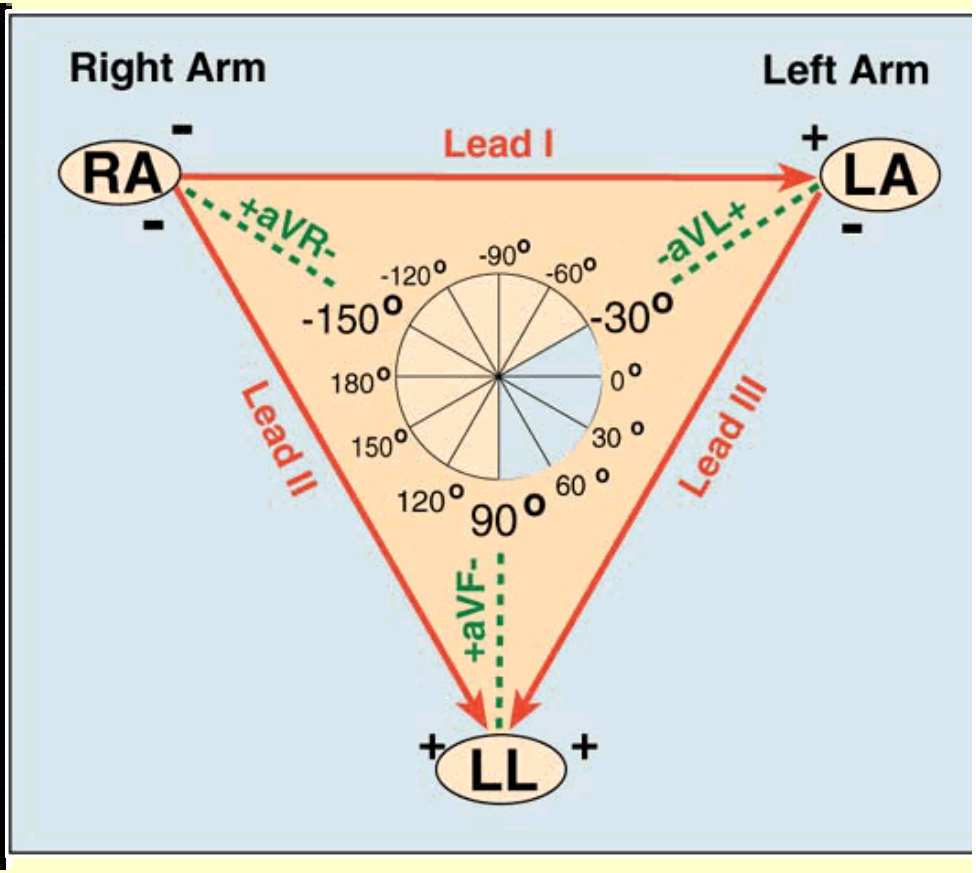
A. +90 degrees

B. -30 degrees

C. -45 degrees

D. +150 degrees



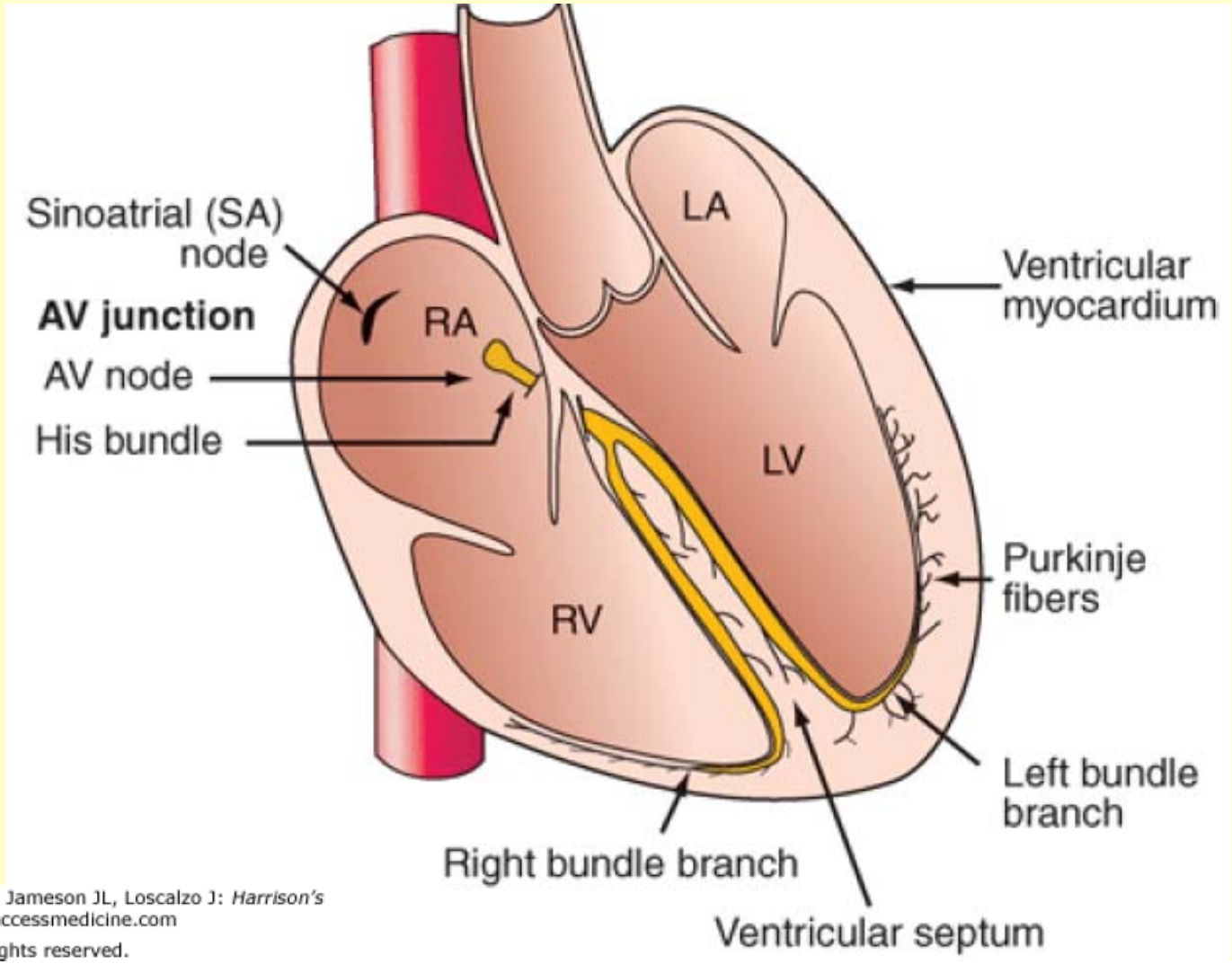

Lead II is isoelectric

- Average direction of ventricular activation is perpendicular to lead II. (i.e. -30$^{o}$ or +150$^{o}$)

Lead I is negative and Lead III. Is positive = Right Axis Deviation (RAD ~ + 150°)

# 3. Conduction Analysis

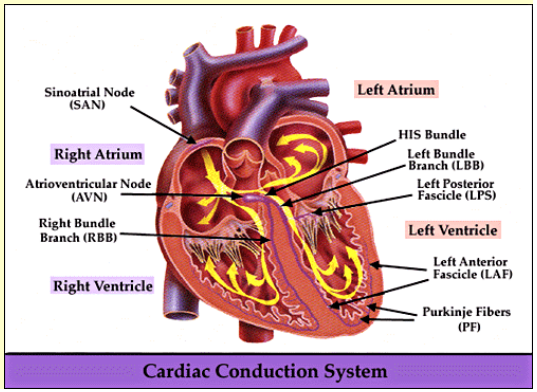
- "Normal" conduction
  - sino-atrial (SA)
  - atrio-ventricular (AV)
  - intraventricular (IV) conduction




Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine, 18th Edition*: www.accessmedicine.com

# 3. Conduction Analysis

- Conduction abnormalities
  - SA block (exit blocks):
    - 2nd degree (type I vs. type II)
  - AV block:
    - 1st, 2nd (type I vs. type II), and 3rd degree
  - IV blocks:
    - bundle branch, fascicular, and nonspecific blocks



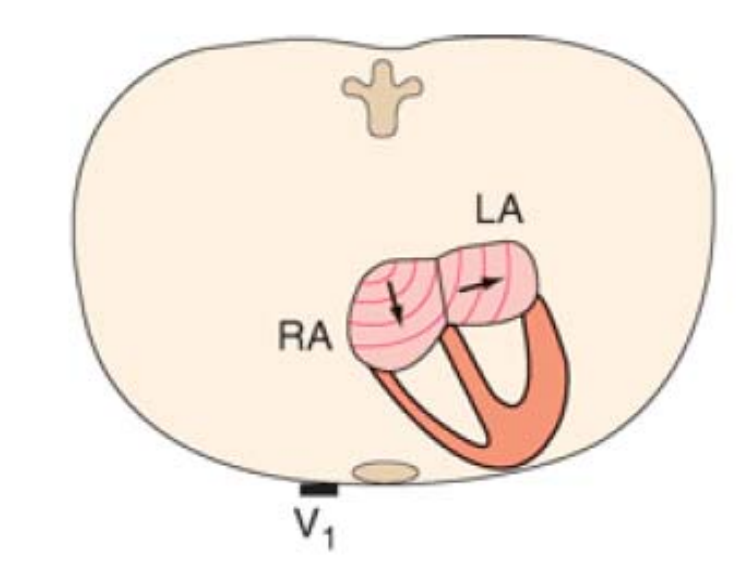
# 4. Waveform Description

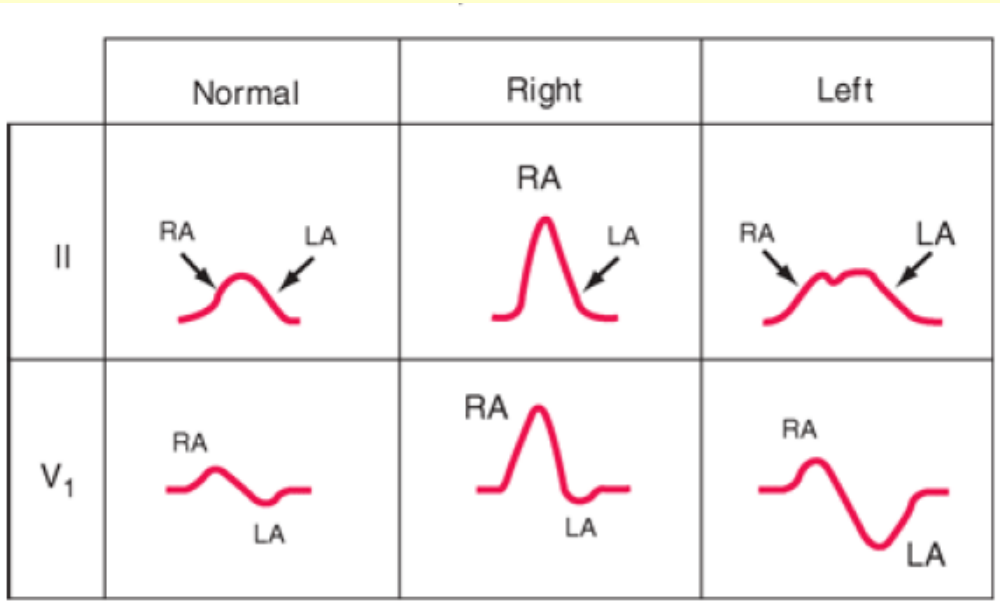
- Analyze the 12-lead ECG for abnormalities in each of the waveforms
  - ST segments:
    - abnormal ST elevation and/or depression
  - T waves:
    - abnormally inverted T waves
  - U waves
    - prominent or inverted U waves.

# P waves:

- are they too wide, too tall, look “funny” (i.e., are they ectopic), etc.?

- Right atrial (RA) overload: tall, peaked P waves in the limb or precordial leads
- Left atrial (LA) abnormality: broad, often notched P waves in the limb leads and a biphasic P wave in lead V1 with a prominent negative component (delayed depolarization of the LA)

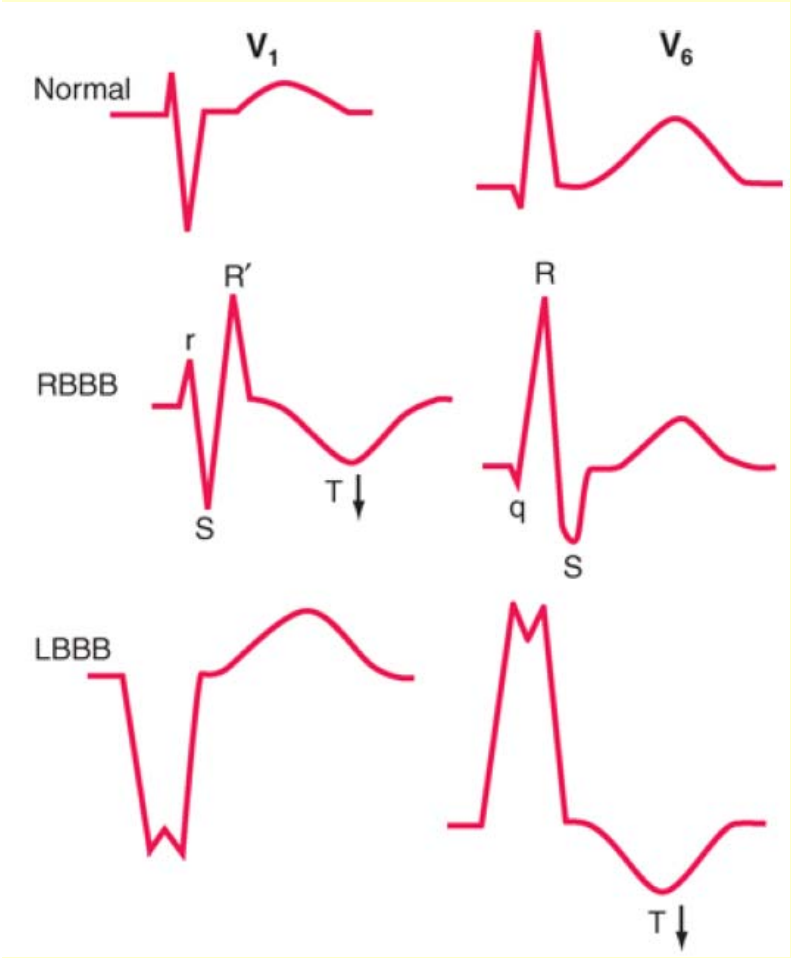


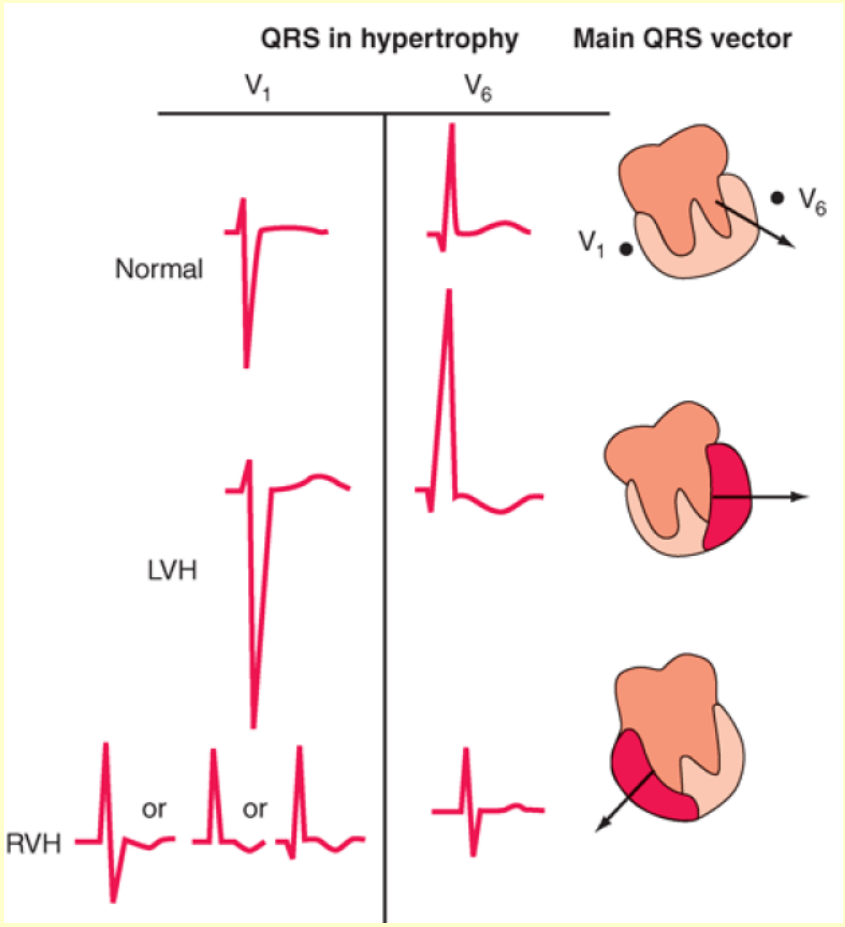




*MK Park, WG Guntheroth: How to Read Pediatric ECGs, 4th ed. St. Louis, Mosby/Elsevier, 2006.*

# QRS complexes:

- atypical QRS pattern, abnormal voltage, pathologic Q waves, etc.



# 5. ECG Interpretation

- Conclusion
  - Normal  X Abnormal  X  Borderline
- Abnormal ECG e.g.:
  - MI (location, acute, old)
  - Rhythm abnormalities
  - Blocks
    - Left anterior fascicular block (LAFB)
    - Left ventricular hypertrophy (LVH)
  - Nonspecific ST-T wave abnormalities

# Diagnostic approaches in cardiology

# III - Myocardial ischemia
# II - Arrhythmias

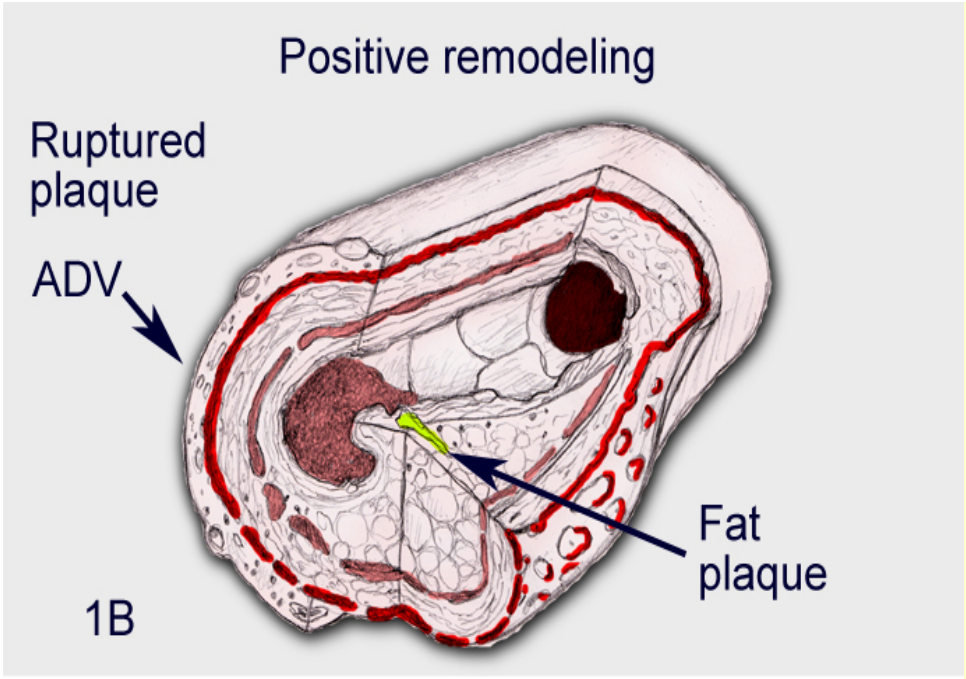
Jan Živný
Department of Pathophysiology
jzivny@LF1.cuni.cz

# Coronary artery disease

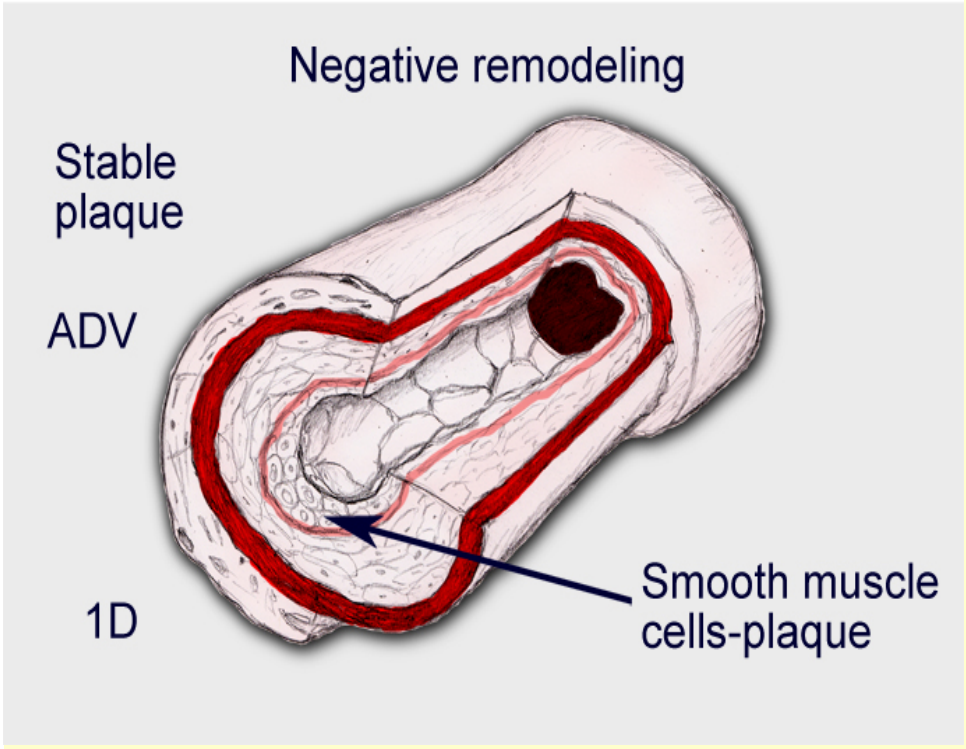
- Caused by aterosclerosis of large and medium-sized muscular arteries
- Is characterized by:
  - Endothelial dysfunction
  - Vascular inflammation
  - Buildup of lipids, cholesterol, calcium, and cellular debris within the intima of the vessel wall

# Atherosclerotic buildup results in:

- Plaque formation
- Vascular remodeling
- Acute and chronic luminal obstruction
- Abnormalities of blood flow




- Arterial wall bulges outward and the lumen remains uncompromised
- More prone to plaque rupture and ACS than to stable angina
- May eventually progress to the negative remodeling stage




- The atheroma steadily grows inward, causing gradual luminal narrowing
- Usually lead to the development of stable angina or plaque rupture and thrombosis.

# Acute coronary syndrome (ACS)

- Representing ongoing myocardial ischemia or injury caused by rupture of an atherosclerotic plaque and partial or complete thrombosis of the infarct-related artery.
- A spectrum of clinical presentations
  - Unstable angina
  - Non–ST-segment elevation myocardial infarction (NSTEMI)
  - ST-segment elevation myocardial infarction (STEMI)

# Myocardial infarction

- Ischemic injury to myocardium
  - occurs when the blood supply is insufficient to meet the tissue demand for metabolism
- Most myocardial infarctions occur in lesions that are less than 70% severe
- Caused by rupture of coronary atherosclerotic plaques with superimposed coronary thrombosis (> 90% MIs)

# Manifestation of MI

- Crushing chest pressure
- Diaphoresis
- Malignant ventricular arrhythmias
- Heart failure
- Cardiac shock
- Sudden cardiac death (w/o necrosis - takes time to develop)

- Clinically silent in as many as 25% of elderly patients

# STEMI

- ST elevations on the ECG reflect active and ongoing transmural myocardial injury
- Most persons with STEMI develop Q waves (without reperfusion therapy)
- Q waves reflecting a dead zone of myocardium (irreversible damage)

Decission:

Thrombolysis or with primary percutaneous coronary intervention (PCI

# Unstable angina/ NSTEMI

- ECG without ST elevations
- May have other ECG changes ST-segment depression or T-wave morphological changes
- presence of cardiac enzymes
- recommend that in patients with suspected myocardial infarction, cardiac biomarkers should be measured at presentation

# Diagnosis of MI

- *Laboratory studies*
  - Cardiac biomarkers/enzymes:
- *Electrocardiography*
  - confirmatory of the diagnosis in approximately 80% of cases
- *Cardiac imaging*
  - To definitively diagnose or rule out coronary artery disease

# Diagnosis of MI - Laboratory studies

Cardiac biomarkers

- Troponin:
  - contractile protein released when myocardial necrosis occurs
- Creatine kinase (CK):
  - CK-MB increase within 3-12 hours of the onset of chest pain (peak values within 24 hours, and return to baseline after 48-72 hours)
- Myoglobin:
  - release more rapidly than troponin

Other

- Complete blood count
- Chemistry profile
- Lipid profile
- C-reactive protein and other inflammation markers

# Diagnosis of MI - ECG

- The finding depend on the localization and the size of the affected area
  - Q type MI
  - non-Q type MI (2/3 of MI, usually depression of ST segment or inversion of T wave)
- MI caused by complete occlusion of coronary artery usually result in homogenous transmural tissue defect and Q type MI
- MI caused by subtotal occlusion
  - heterogeneous tissue defect with non Q type MI

# Cause and consequences of MI

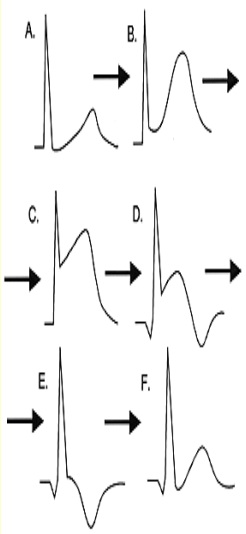
- Most frequent mechanism of MI
  - rupture of atherosclerotic plaque followed by thrombosis of coronary artery

- Pathological changes of myocardium
  - subendocardial or transmural ischemia
  - necrosis
  - fibrosis (scar)

# Development of STEMI on ECG to Q type MI

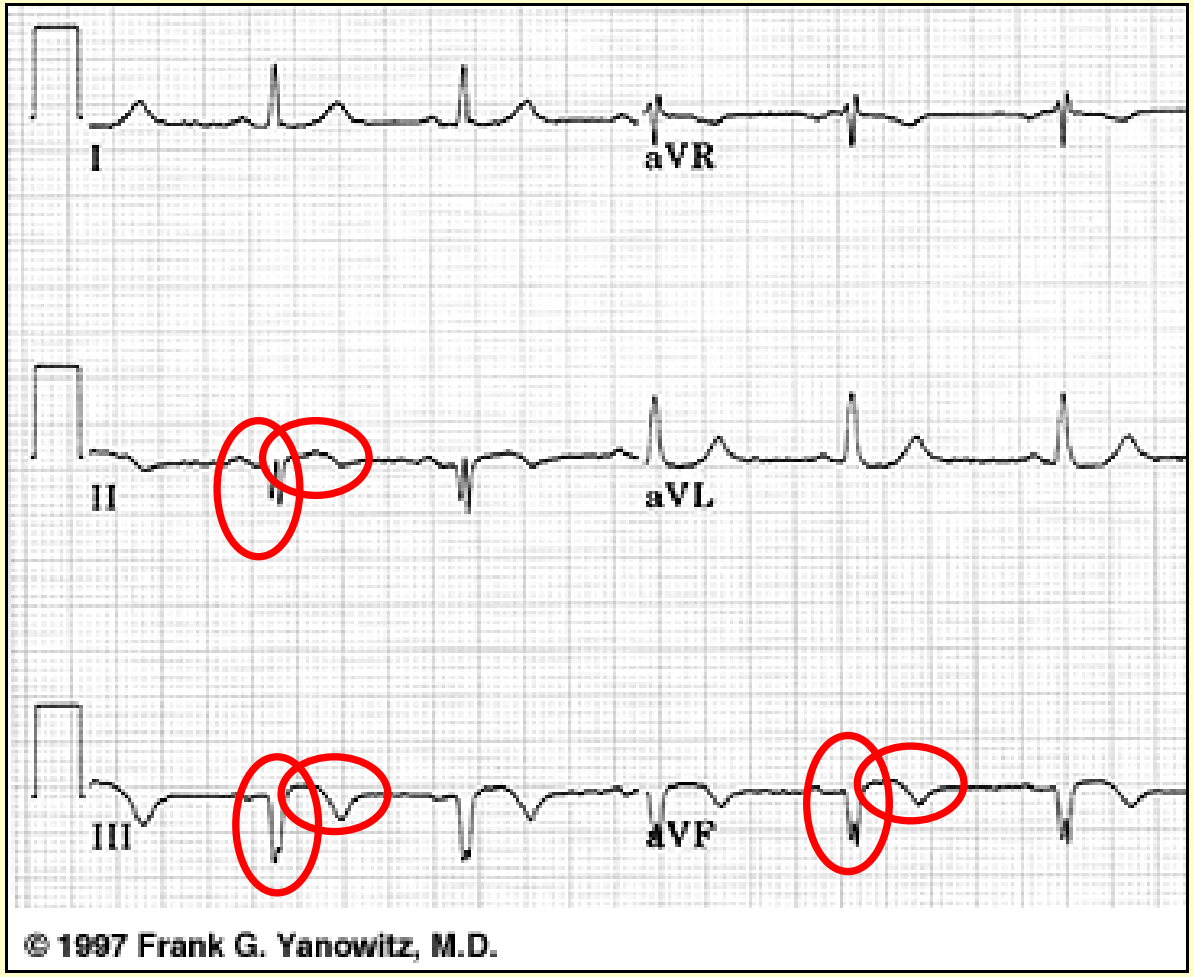
- Increased T wave amplitude and width (may also see ST elevation) – **minutes to hours**
- Marked ST elevation with hyperacute T wave changes (transmural injury) - **hours**
- Pathologic Q wave, less ST elevation, terminal T wave inversion (necrosis) – **hours to days**
- Pathologic Q waves, T wave inversion (necrosis and fibrosis) **- days**
- Pathologic Q waves, upright T waves (fibrosis) **days to weeks**

Pathologic Q wave: duration >0.04 s and/or >25% of R-wave



Evolution of Acute MI

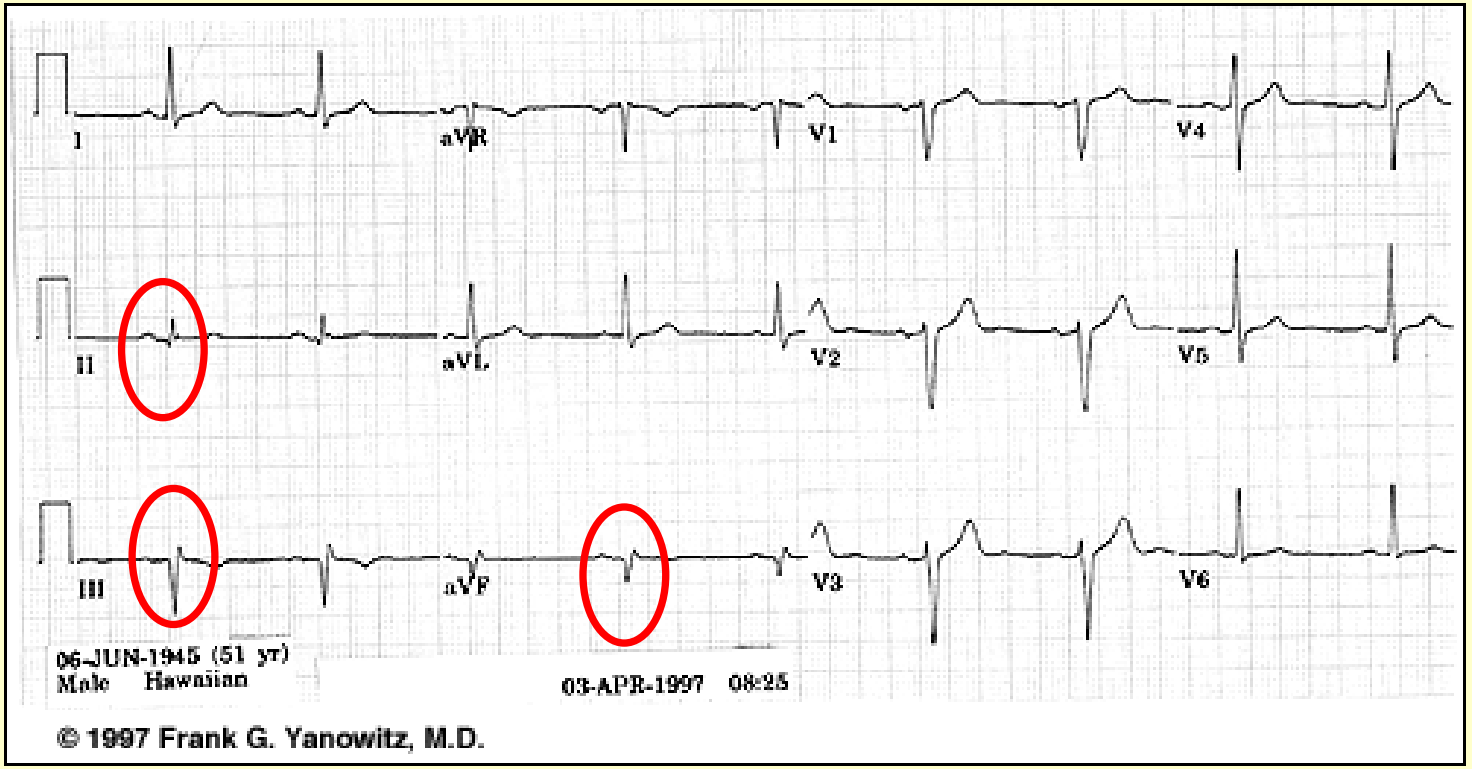
# Inferior MI




Fully developed inferior MI:

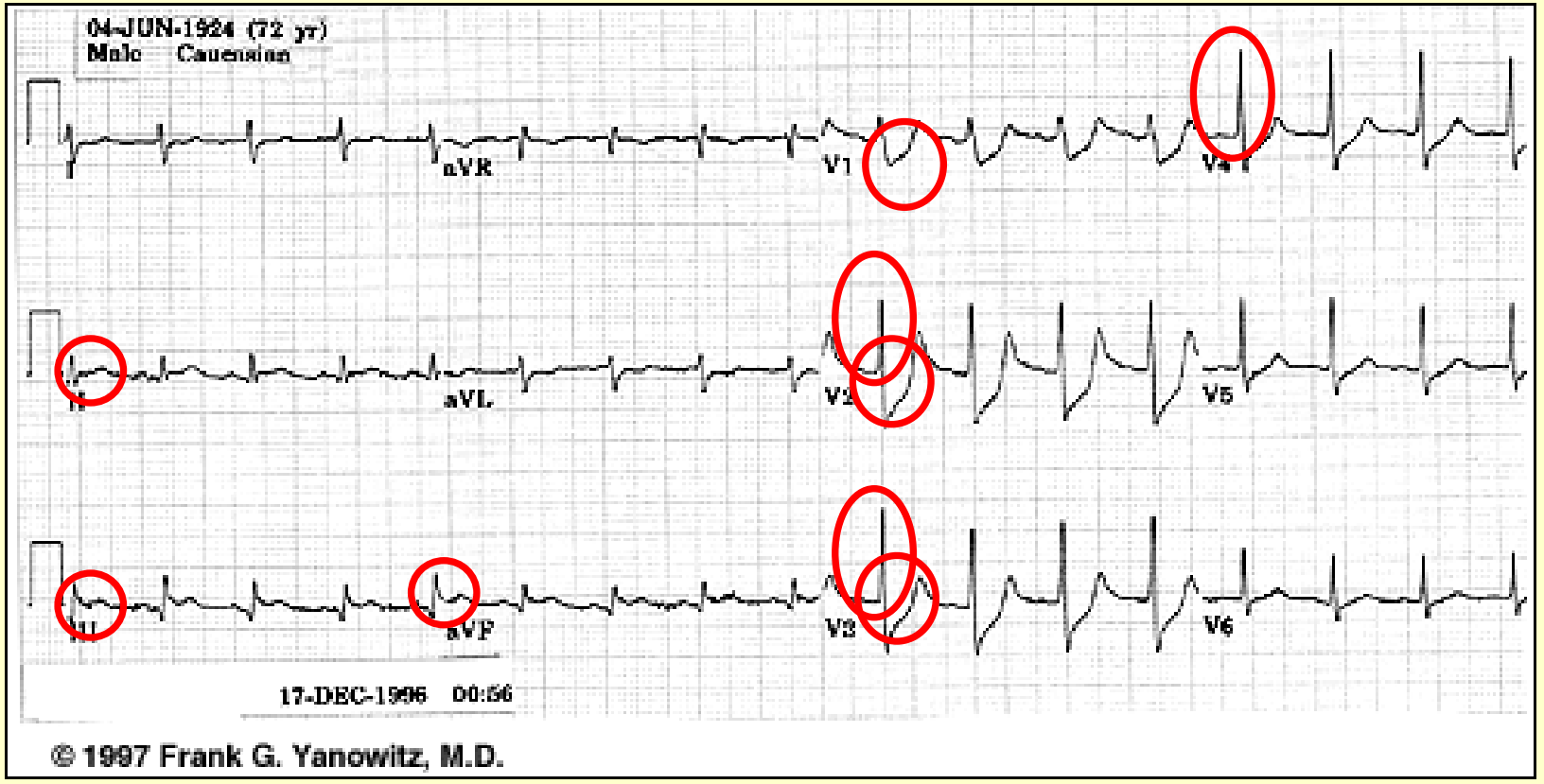
- ve II, III, aVF
  - Q-
  - ST elevation
  - T inversion
- Q is deepest in lead III (> aVF > II)

# Older inferior MI



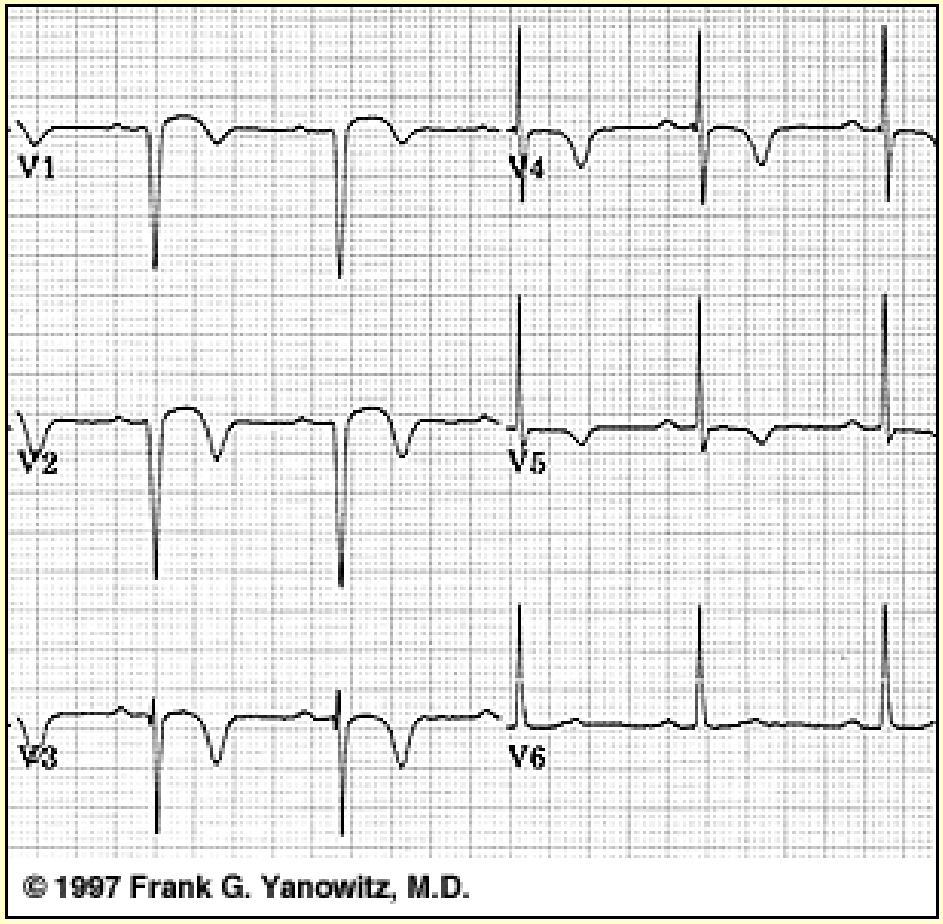

- Starší spodní IM
- Q ve svodech III, aVF a II

# Acute inferior and posterior MI




- note tall R waves V2-4, marked ST depression V 1-3, ST elevation in II, III, aVF)

# Fully evolved anteroseptal MI



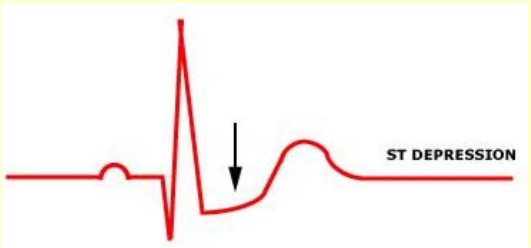

- QS waves in V1-2,
- qrS complex in V3,
- ST-T wave changes

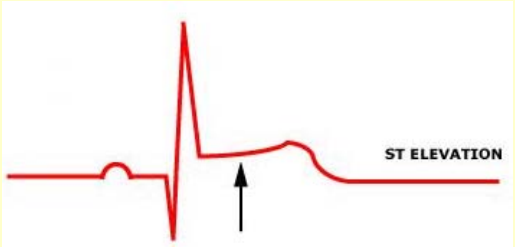
# Non-Q MI

- Usually MI caused by subtotal occlusion and heterogeneous tissue defect

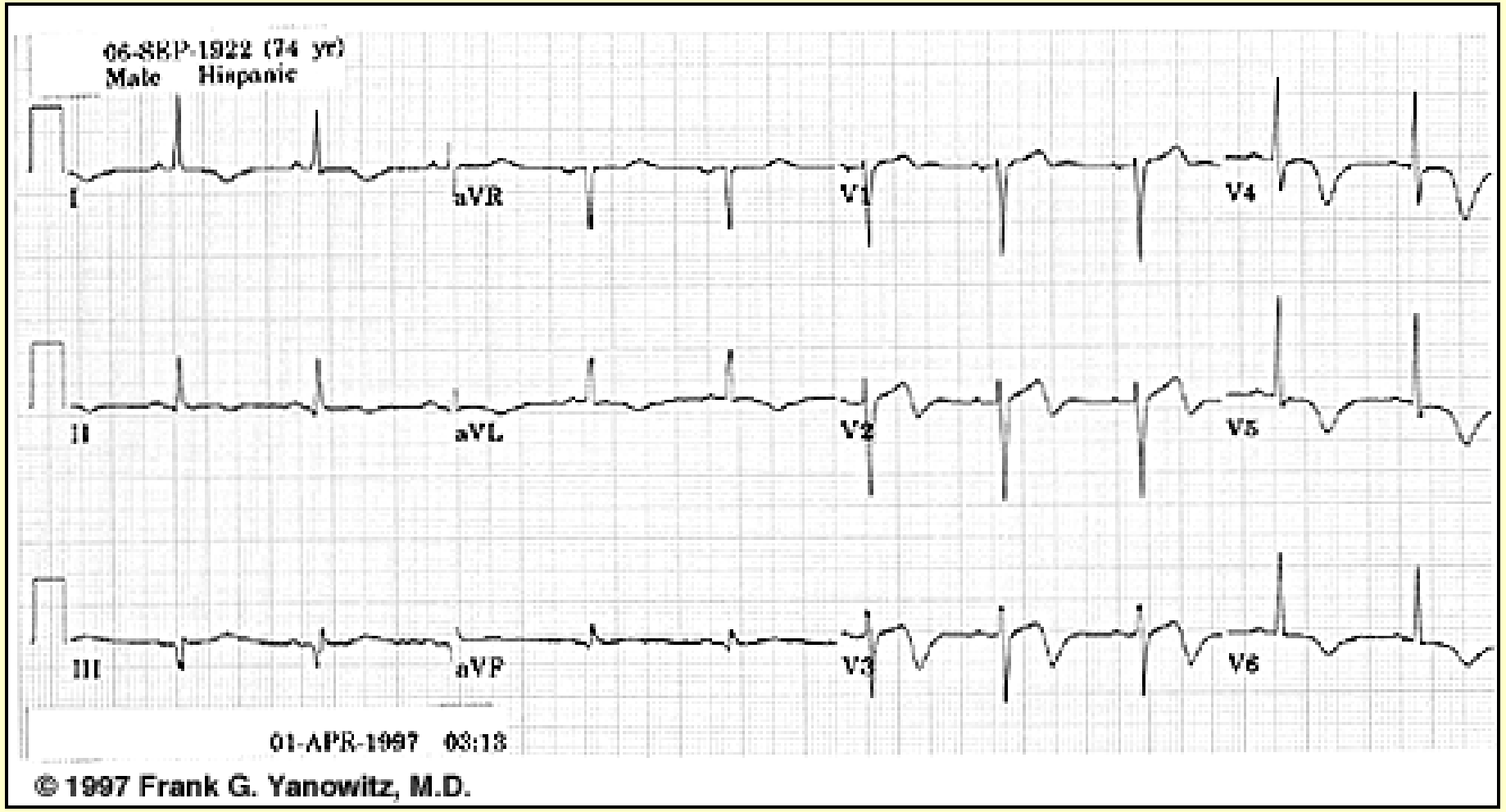
# Non-Q MI

- Gradual changes in ST segment and T wave (in patients with typical chest pain and “heart“ enzyme elevation
- ST-T changes:
  - Depresion of ST segment (often)
  - Elevation of ST segment (less often)
  - Symetrical inversion of T wave (often)
  - Combination of ST-T changes



# Non-Q Wave MI

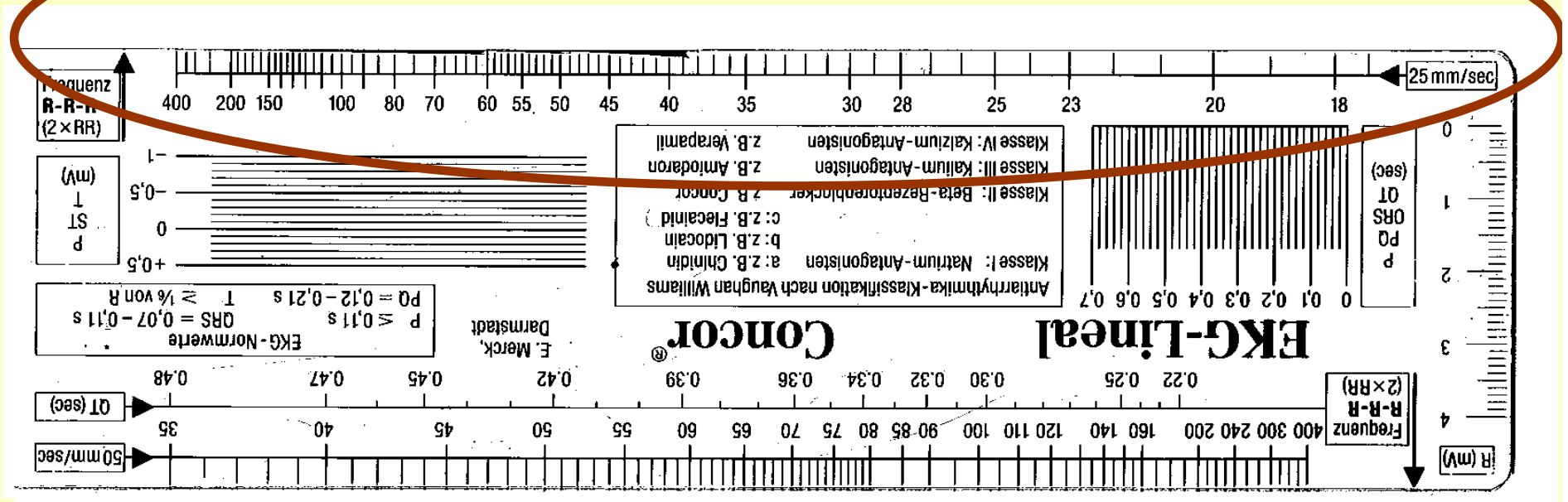

06-SEP-1922 (74 yr)
Male Hispanic
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
01-APR-1997 03:13
© 1997 Frank G. Yanowitz, M.D.

# ECG ruler

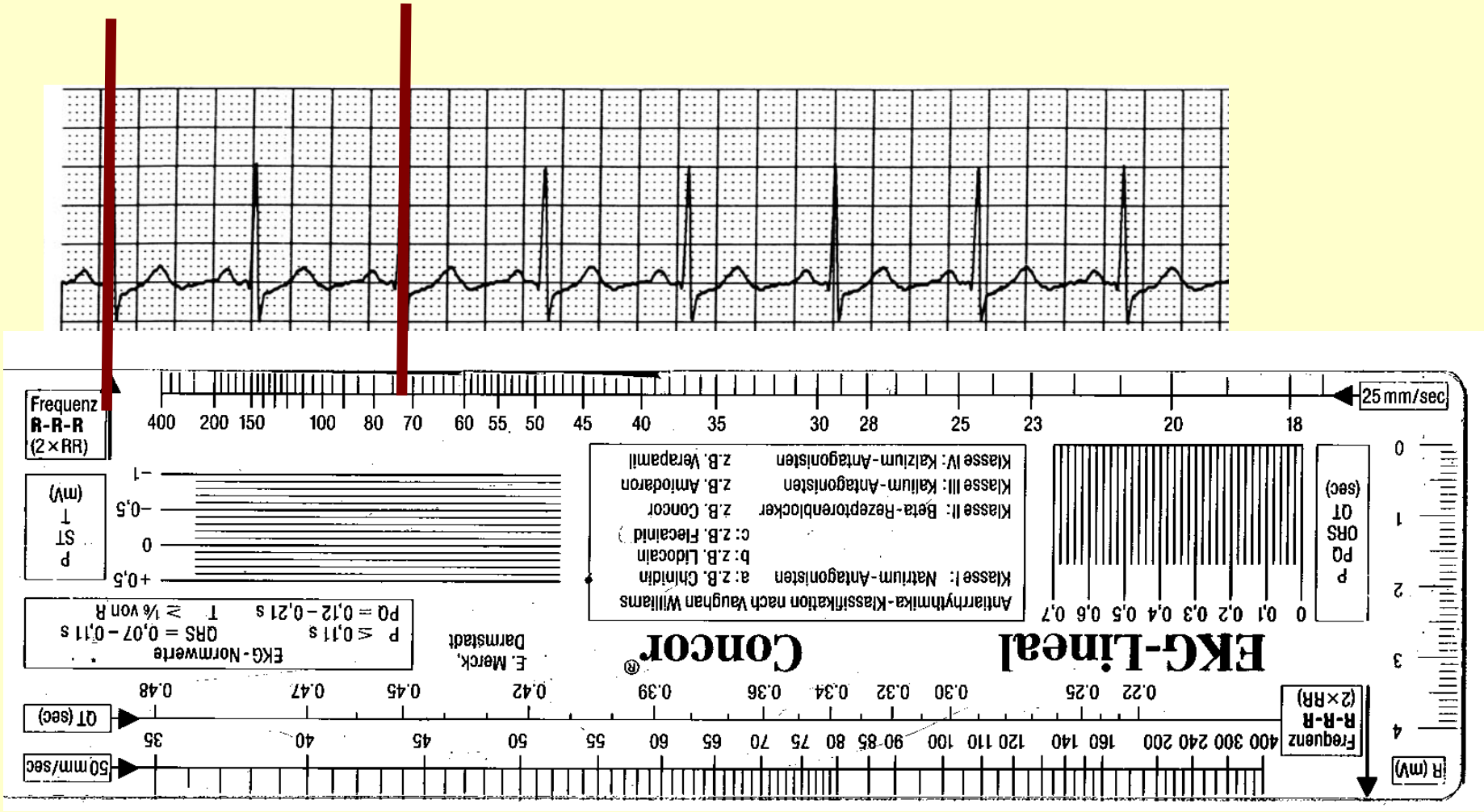
- Measurement of HR and intervals on ECG record

# Measurement of heart rate

- Atrial HR
- Ventricular HR
- Recording speed 50 mm/s or 25 mm/s

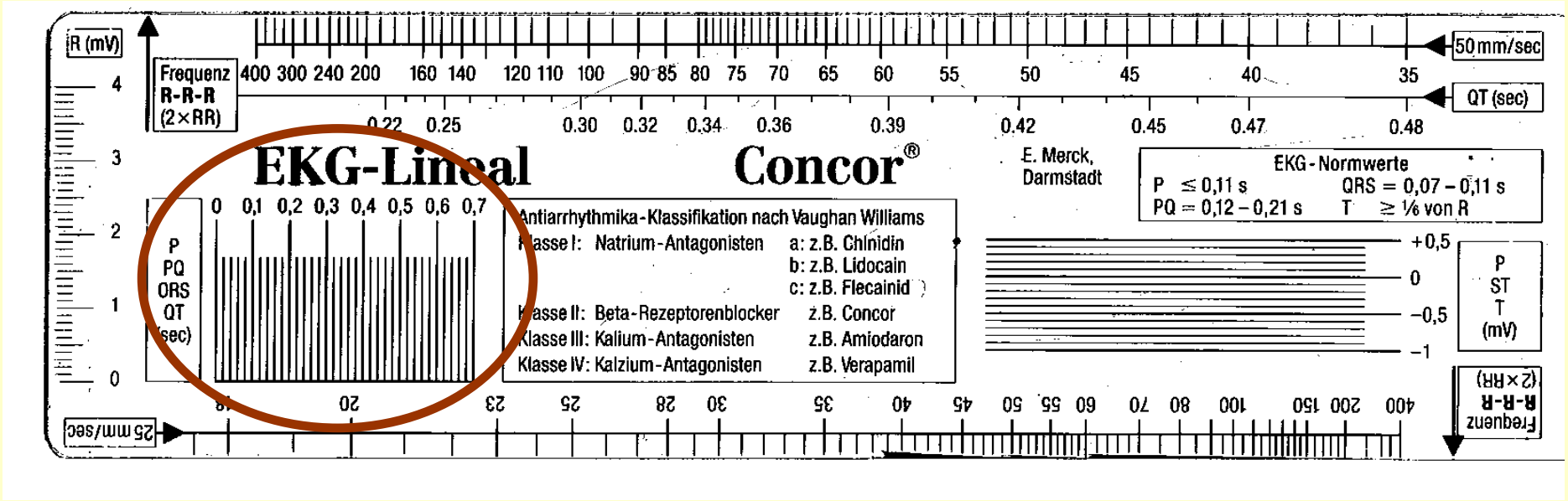


# ECG ruler


Frequenz
R-R-R
(2×RR)
400 200 150 100 80 70 60 55 50 45 40 35 30 28 25 23 20 18
25 mm/sec
P
ST
T
(mV)
+0,5
0
−0,5
−1
Antiarrhythmika-Klassifikation nach Vaughan Williams
Klasse I: Natrium-Antagonisten a: z.B. Chinidin
b: z.B. Lidocain
c: z.B. Flecainid
Klasse II: Beta-Rezeptorenblocker z.B. Concor
Klasse III: Kalium-Antagonisten z.B. Amiodaron
Klasse IV: Kalzium-Antagonisten z.B. Verapamil
0 0,1 0,2 0,3 0,4 0,5 0,6 0,7
P
PQ
QRS
QT
(sec)
0 1 2 3 4
R (mV)
EKG-Normwerte
P ≤ 0,11 s QRS = 0,07 – 0,11 s
PQ = 0,12 – 0,21 s T ≥ ¼ von R
E. Merck, Darmstadt
Concor®
EKG-Lineal
Frequenz
R-R-R
(2×RR)
400 300 240 200 160 140 120 110 100 90 85 80 75 70 65 60 55 50 45 40 35
50 mm/sec
0.22 0.25 0.30 0.32 0.34 0.36 0.39 0.42 0.45 0.47 0.48
QT (sec)

# Analysis of conduction

- Interval measurements (speed 50 mm/s)
  - PR interval: 0.12 and 0.20 s
    - AV conduction
  - QRS interval: 0.07 - 0.11 s
    - ventricular conduction




For speed 25 mm/s the value need to be multiplied by 2x

# Examinations in Cardiology
# Arrhythmia

Jan Živný
Department of Pathophysiology
jzivny@LF1.cuni.cz

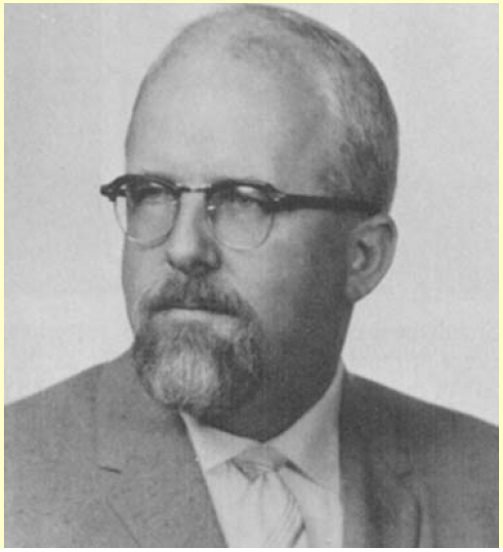
# Outline

- methods to diagnose arrhythmia
- classification of arrhythmias
- ECG changes in Arrhythmias
- Summary

# Arrhythmia diagnosis

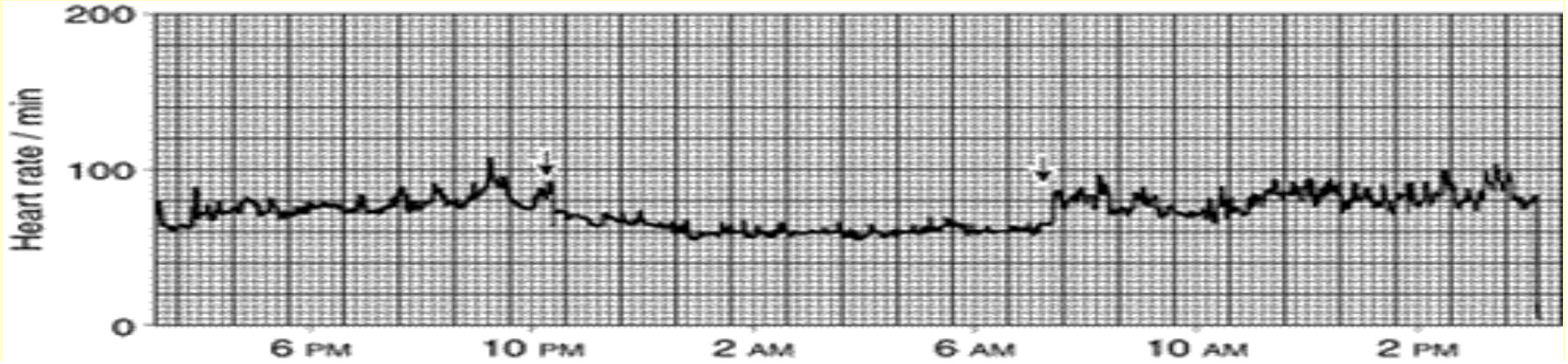
- ECG recording
- Holter 24 h ECG monitoring
- Computer analysis of ECG recordings

# 24-h ambulatory ECG (Holter) monitor



**Norman “Jeff” Holter**

*The original Holter biotelemetry apparatus in 1947 weighing 85 lb (38 kg)*
Corday et al. Detection of phantom arrhythmias and evanescent electrocardiographic Abnormalities. JAMA 1965



# Classification of arrhythmias Based of localization of the defect

- Supraventricular
  - Sinus node
  - Atrial
  - Junction
- Ventricular

# Classification of arrhythmias Pathophysiological

- Pacemaker defect (Abnormal electrical impulse formation) e.g.:
  - Sick sinus
  - Ectopic Focus
- Conduction defect e.g.:
  - Accelerated AV conduction
  - AV blocks
- Combined e.g.:
  - 3rd degree blocks

# ECG changes in Arrhythmias

# Atrial and atrioventricular conduction abnormalities

# Short PR: < 120 ms (Preexcitation syndromes)

- WPW (Wolff-Parkinson-White) Syndrome:
  - An accessory pathway (called the "Kent" bundle)
    - connects the right atrium to the right ventricle or the left atrium to the left ventricle
    - early activation of the ventricles (delta wave) and a short PR interval

# Short PR: < 120 ms (Preexcitation syndromes)

- LGL (Lown-Ganong-Levine):
  - An AV nodal bypass track into the His bundle exists
  - this permits early activation of the ventricles without a delta-wave

# Short PR: < 120 ms (Preexcitation syndromes)

- AV Junctional Rhythms with retrograde atrial activation
  - inverted P waves in II, III, aVF:
  - Retrograde P waves may occur
    - before the QRS complex (short PR interval),
    - in the QRS complex (hidden from view)
    - after the QRS complex ( in the ST segment)

# Short PR: < 120 ms (Preexcitation syndromes)

- Ectopic atrial rhythms originating near the AV node
  - the P wave morphology is different from the sinus P

# Prolonged PR: > 200 ms

- First degree AV block: PR interval usually constant > 200 ms
  - Intra-atrial conduction delay (uncommon)
  - Slowed conduction in AV node (most common site)
  - Slowed conduction in His bundle (rare)
  - Slowed conduction in bundle branch

# Prolonged PR: > 200 ms

- Second degree AV block
  - PR interval may be normal or prolonged; some P waves do not conduct
  - Type I (Wenckebach): Increasing PR until nonconducted P wave occurs
  - Type II (Mobitz): Fixed PR intervals plus nonconducted P waves
- [Third degree AV block
  - AV dissociation: Some PR's may appear prolonged, but the P waves and QRS complexes are dissociated]

# Intraventricular conduction abnormalities

# Prolonged QRS (>100 ms)

- QRS duration 100 – 120 ms
  - Incomplete right or left bundle branch block
  - Nonspecific intraventricular conduction delay (IVCD)
  - Some cases of left anterior or posterior fascicular block

# Prolonged QRS (>100 ms)

- QRS duration > 120 ms
  - Complete RBBB or LBBB
  - Nonspecific IVCD (intraventricular conduction defect)
  - Ectopic rhythms originating in the ventricles
    - ventricular tachycardia
    - pacemaker rhythm

# QT Interval

- Heart rate dependent
  - corrected QT = QTc = measured QT x sq-root RR in seconds
  - upper limit for QTc = 0.44 sec

# QT Interval

- Long QT Syndrome – (LQTS)
  - QTc > 0.47 sec for males and > 0.48 sec
  - Increased vulnerability to malignant ventricular arrhythmias:
    - syncope
    - sudden death
    - Torsade-de-pointes
      - a polymorphic ventricular tachycardia characterized by varying QRS morphology and amplitude around the isoelectric baseline.

Thank you !

# ECG Interpretation

# ECG Interpretation

1. Measurements (HR, intervals, QRS axis)
   - usually made in frontal plane leads
2. Rhythm analysis
3. Conduction analysis
4. Waveform description
5. ECG interpretation and summary
6. Comparison with Previous ECG (if any)

# ECG ruler

- Measurement of HR and intervals on ECG record

# Measurement of heart rate

- Atrial HR
- Ventricular HR
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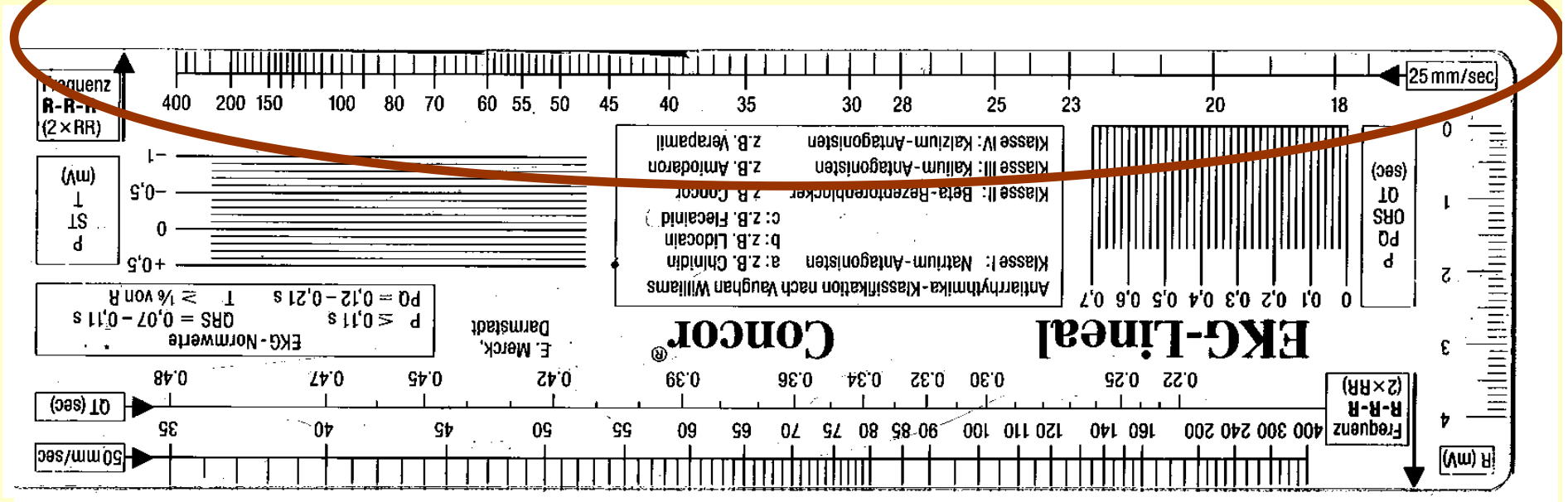

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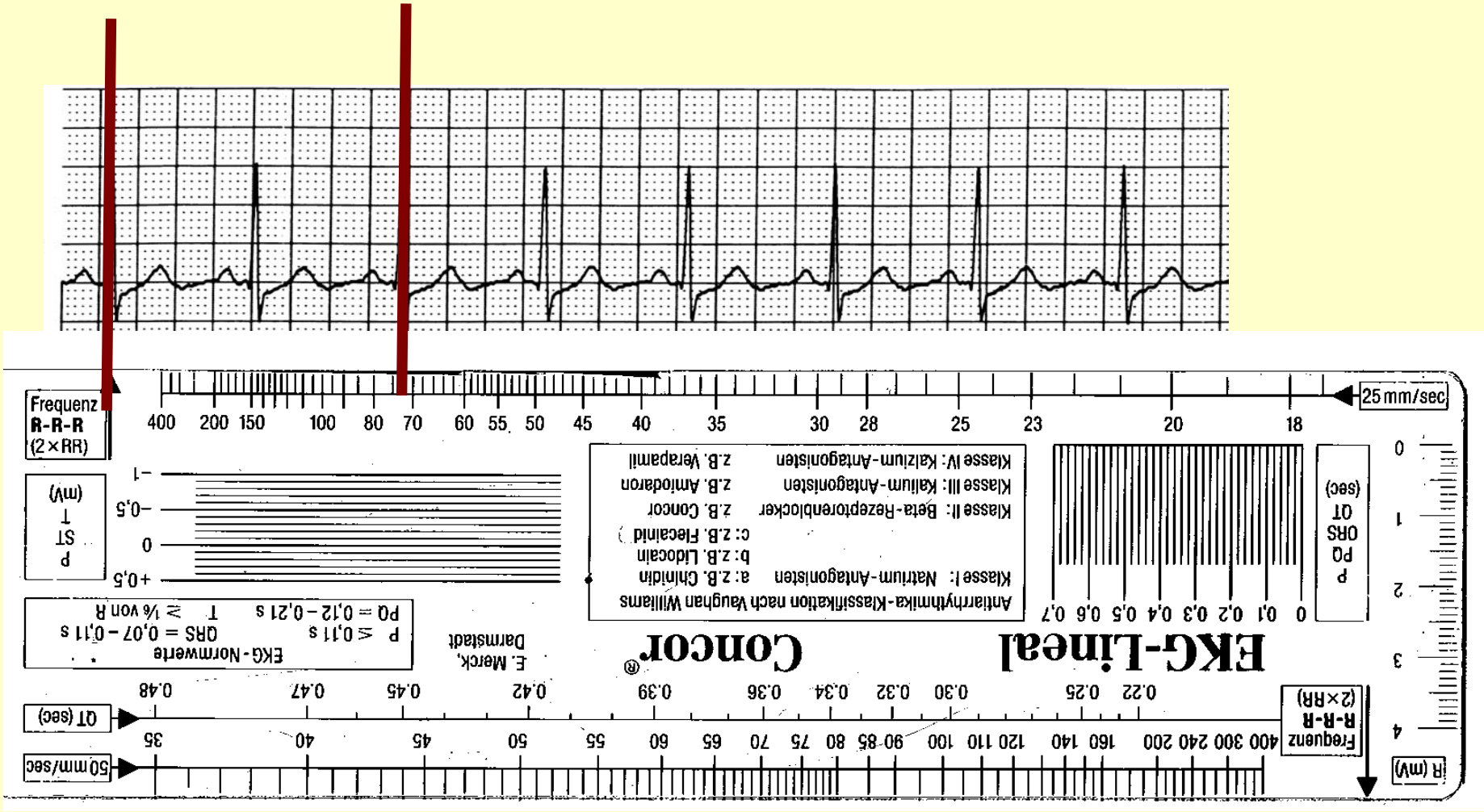
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P
PQ
QRS
QT
(sec)
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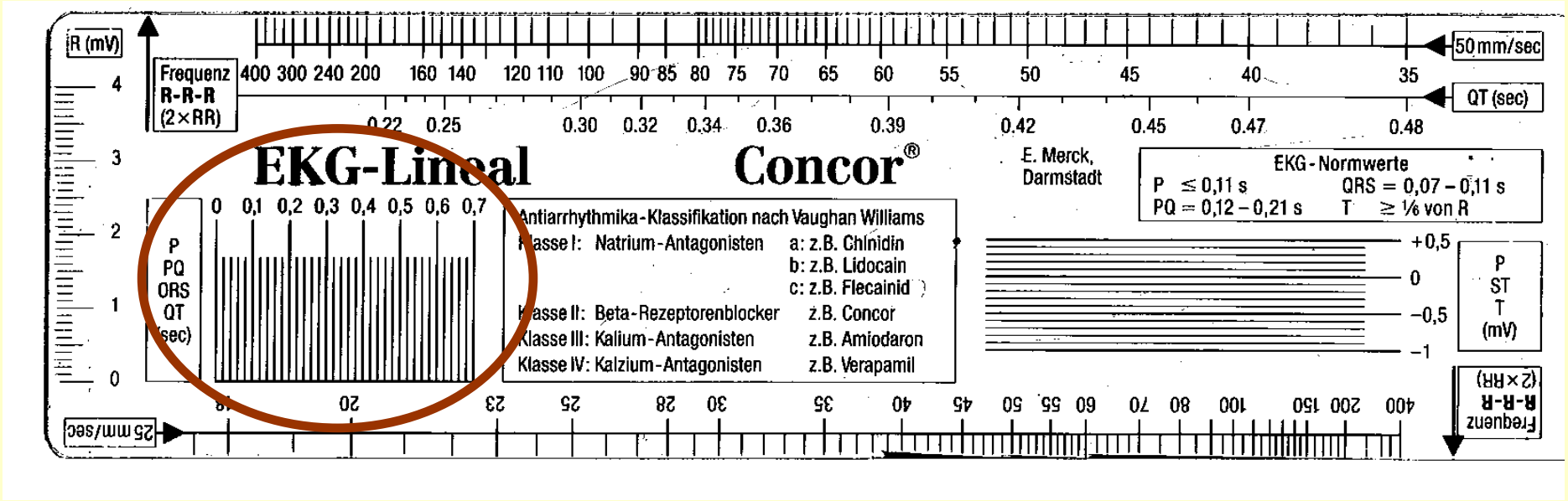
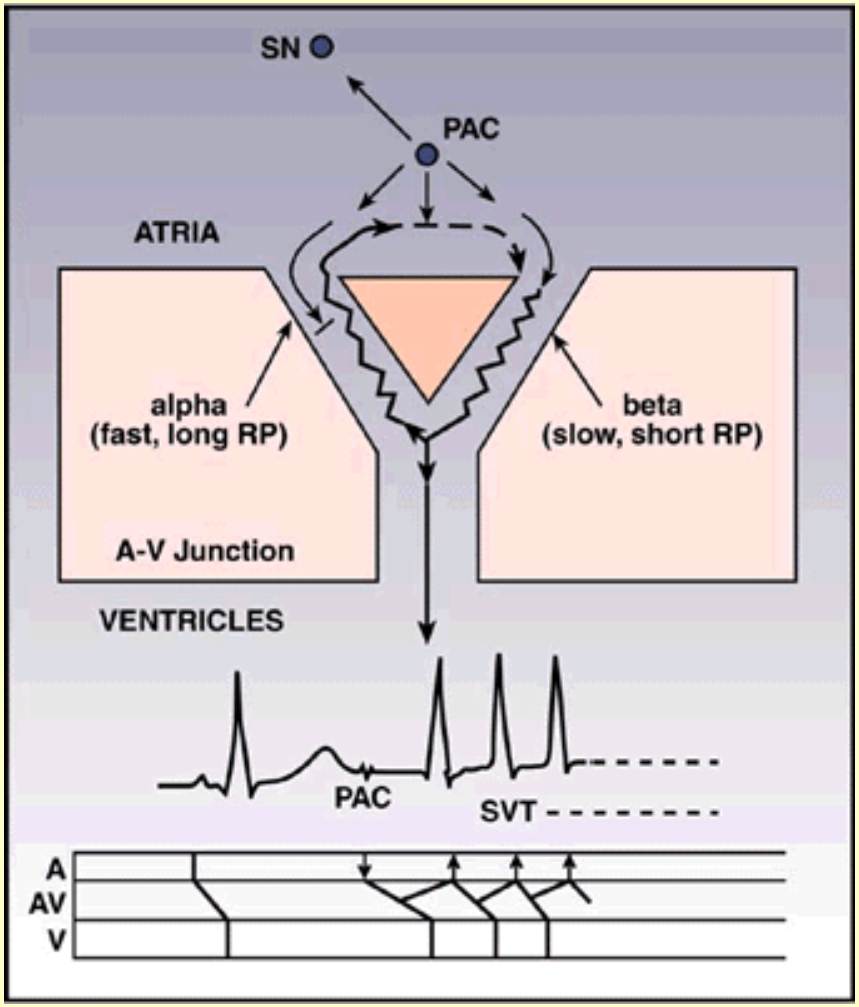



For speed 25 mm/s the value need to be multiplied by 2x

# Re-entry




## Example

- Dual AV nodal pathways with different electrical properties.
  - alpha is a fast AV nodal pathway with a long refractory period (RP)
  - beta is the slow pathway with a short RP
- During sinus rhythm alpha is always used because it conducts faster
  - PAC finds alpha still refractory and must use the slower beta pathway
- By the time it traverses beta alpha has recovered allowing retrograde conduction back to the atria
  - The retrograde P wave can reenter the AV junction because of beta's short refractory period

Thank you !