



Signs and symptoms in cardiovascular problems in children

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Agenda

1. **History taking** in cardiac problems in children
2. Examination of the **heart**
 - Heart sounds
 - Heart murmurs
3. **Cyanosis**
4. **Clubbing**
5. **Heart failure-** signs & symptoms



History taking in cardiac problems

- **Prenatal history**
 - previous prenatal US/scans,
 - prenatal echocardiography
- **Neonatal period- eating problems (breastfeeding)?**
- **Excessive sweating** of the child (big effort)
- **Cyanosis**
- **SOB**
- **Physical activity-** compare to peers
- **Condition of teeth !**
- **Fainting? / Syncope?**- anytime? In what conditions?
- **Family history of**
 - congenital heart defects,
 - cardiac arrest
 - sudden deaths
 - arrhythmia?



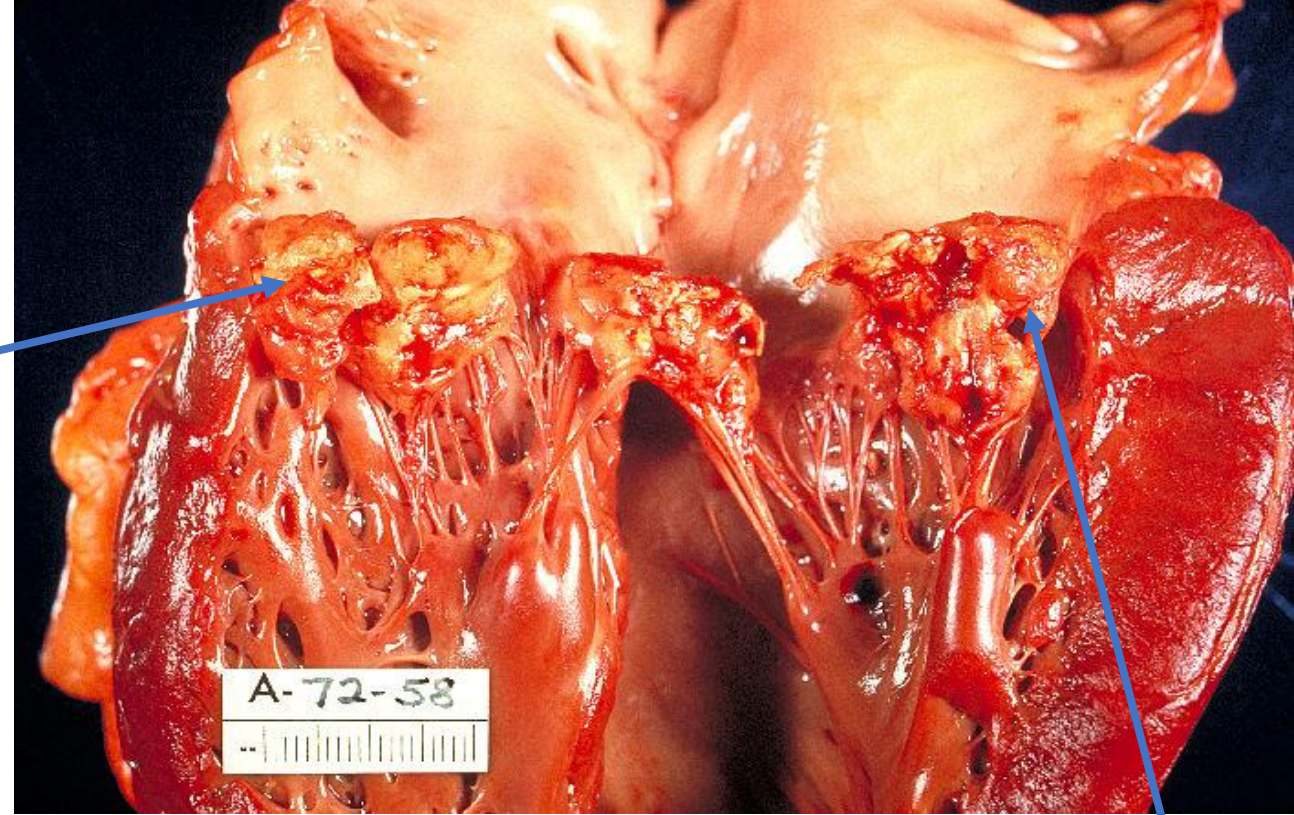
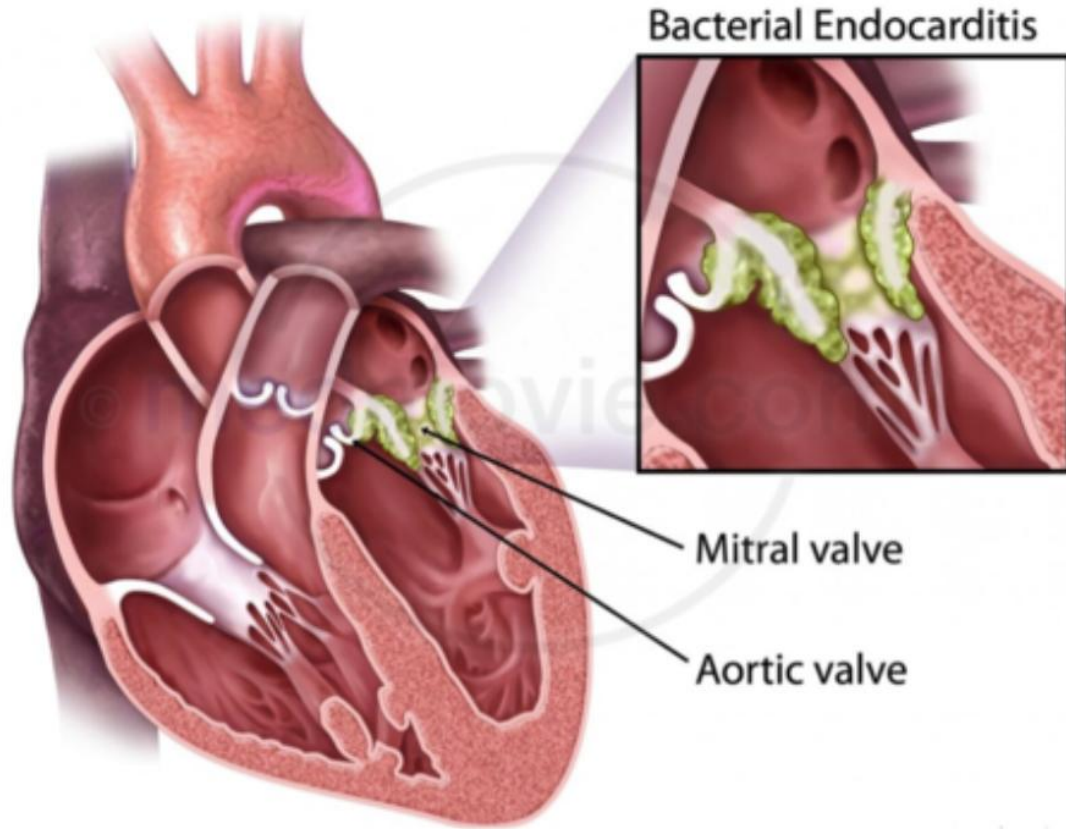
History taking in cardiac problems

- **Prenatal history**
 - previous prenatal US/scans,
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- **Neonatal period- echocardiography**
- **Eating problems (breastfeeding?)**- neonatal period, infancy
- **Excessive sweating** of the child
- **Physical activity**- compare to peers
- **Fainting? / Syncope?**- anytime? In what conditions?
- **Condition of teeth !**
- **Family history of**
 - congenital heart defects,
 - cardiac arrest,
 - sudden deaths
 - arrhythmia?



Infective endocarditis

bacterial vegetations on mitral valve



- **Staphylococci** (*St. aureus*)
- **Streptococci** (*S. viridans* including *S. oralis*, *S. mitis*, *S. sanguis*, *S. gordonii* and *S. parasanguis*- the primary habitats for these organisms are the oral cavity and upper respiratory tract)
- **Enterococci**
- **HACEK** (are a normal part of the human microbiota, living in the oro-pharyngeal region: *Haemophilus*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella*, *Kingella*)

History taking in cardiac problems

- **Prenatal history**
 - previous prenatal US/scans,
 - prenatal echocardiography
- **Neonatal period- echocardiography**
- **Eating problems (breastfeeding?)- neonatal period, infancy**
- **Excessive sweating of the child**
- **Physical activity- compare to peers, NYHA scale**
- **Condition of teeth !**
- **Family history of**
 - congenital heart defects,
 - cardiac arrest,
 - sudden deaths
 - arrhythmia? (eg. LQTS, Brugada syndrome)
- **Syncope?- anytime? In what conditions? Prodrome symptoms?**



EXAMINATION OF THE HEART

SOUNDS & MURMURS

PHYSICAL EXAMINATION OF THE HEART

1. Inspection
2. Palpation
3. Percussion
4. Auscultation



Funnel chest (pectus excavatum)



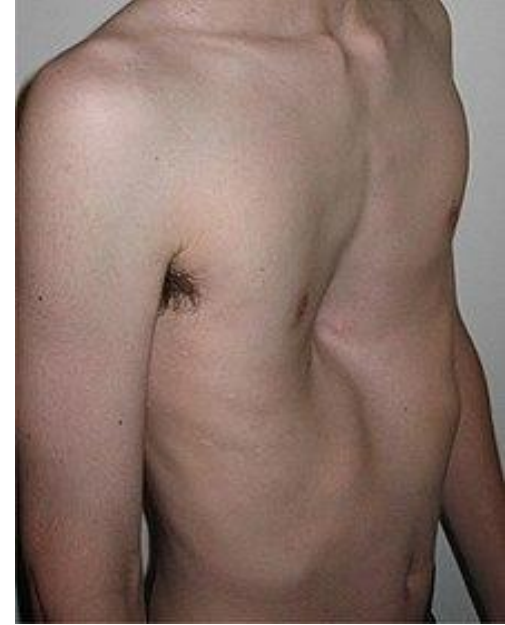
PHYSICAL EXAMINATION OF THE HEART

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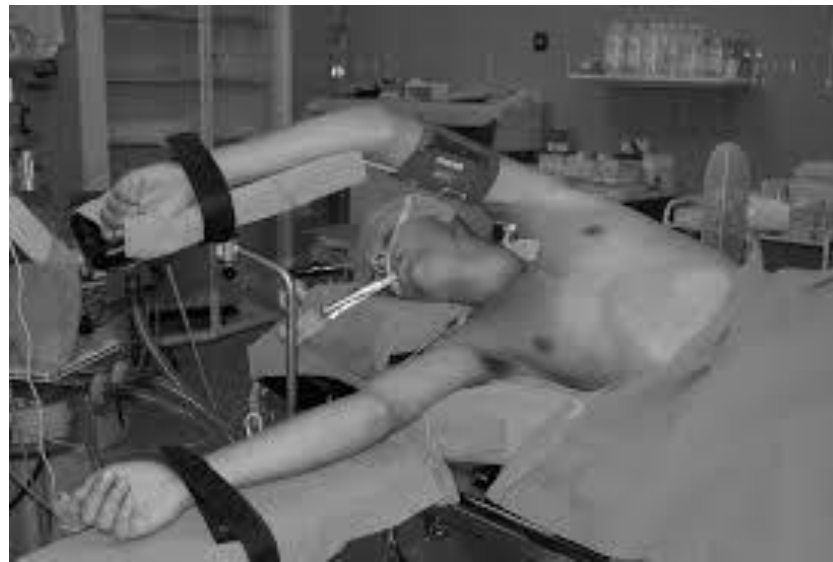
4. Auscultation



Funnel chest (pectus excavatum)

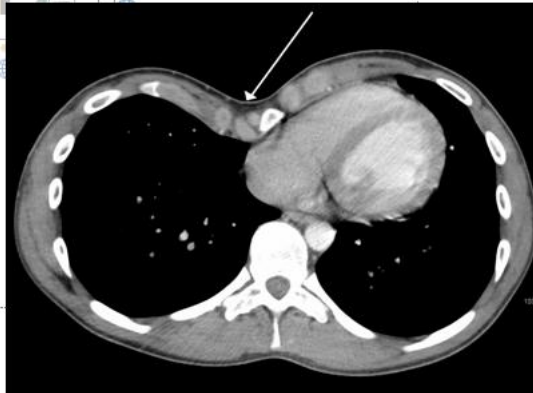


Scars- after cardiothoracic surgeries/ cardiovascular interventions

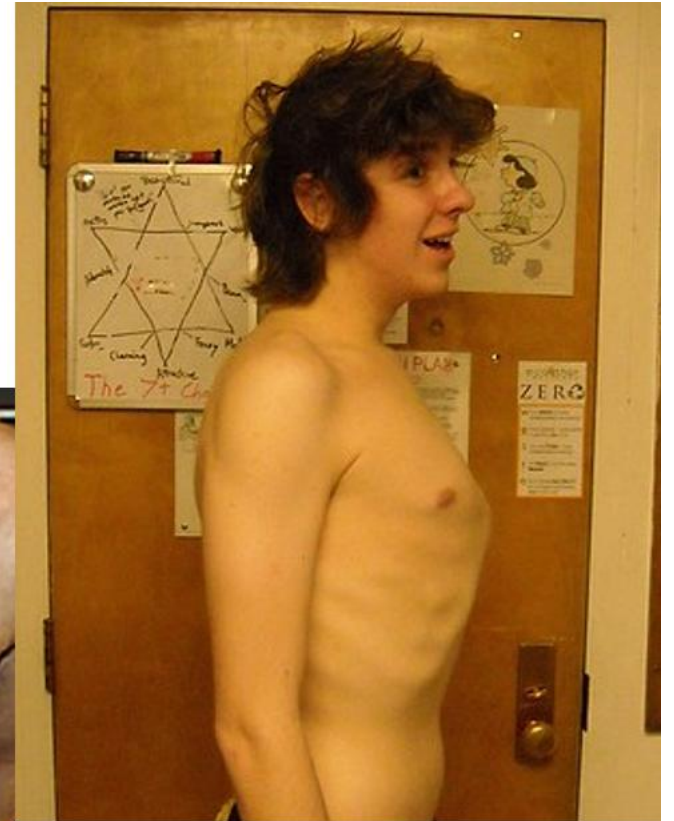


Chest shape

**Pectus
excavatum**



**Pectus
carinatum**



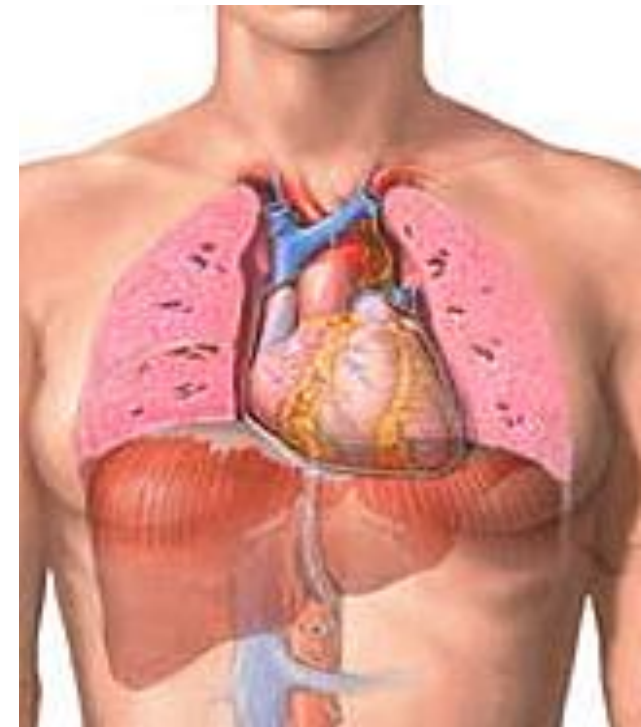
PHYSICAL EXAMINATION OF THE HEART

1. Inspection
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Palpation

- **Apex beat (apical impulse)**-vibration resulting from the heart striking against the chest wall during systole
- **Chest (precordial)thrill** - vibratory sensation felt on the skin overlying an area of turbulence and indicates a loud heart murmur



PHYSICAL EXAMINATION OF THE HEART

1. Inspection

2. Palpation

3. Percussion

4. Auscultation



Funnel chest (pectus excavatum)





DIAPHRAGM

- transmits higher frequency sounds
- suppress low frequency sounds

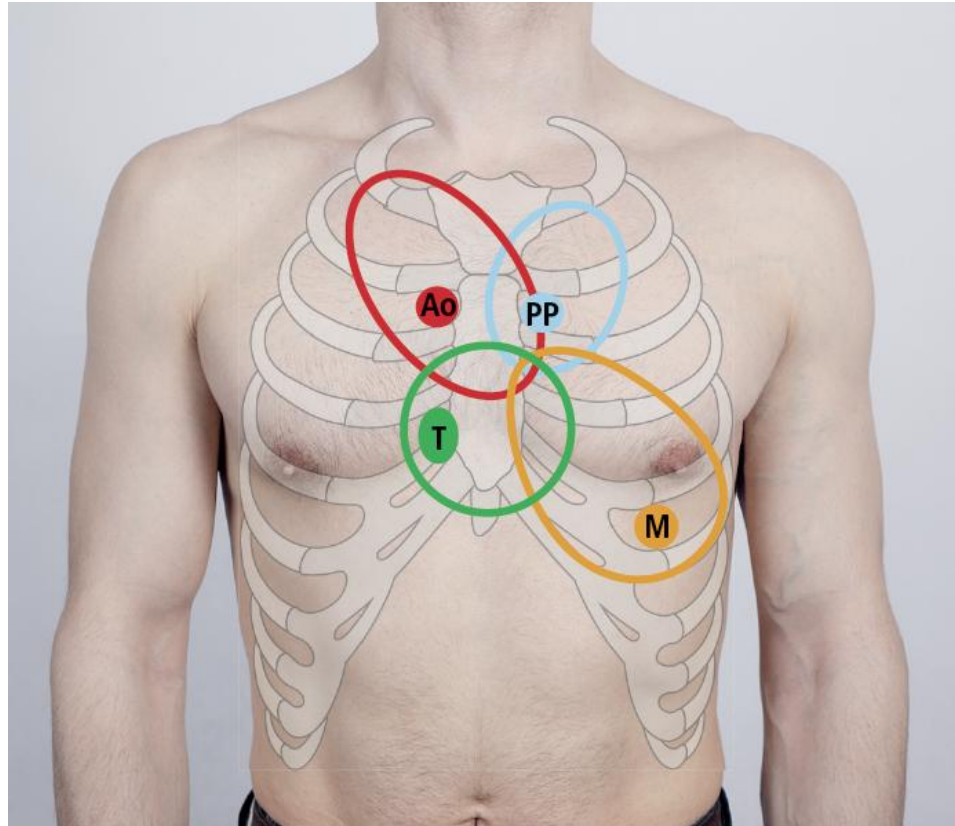
BELL

- transmits low frequency and quiet sounds,
- Suppresses high frequency sounds

Auscultation of the heart valves

2. AORTIC VALVE- 2nd right intercostal space

4. TRICUSPID VALVE- right 4th intercostal space , lateral to the sternal border

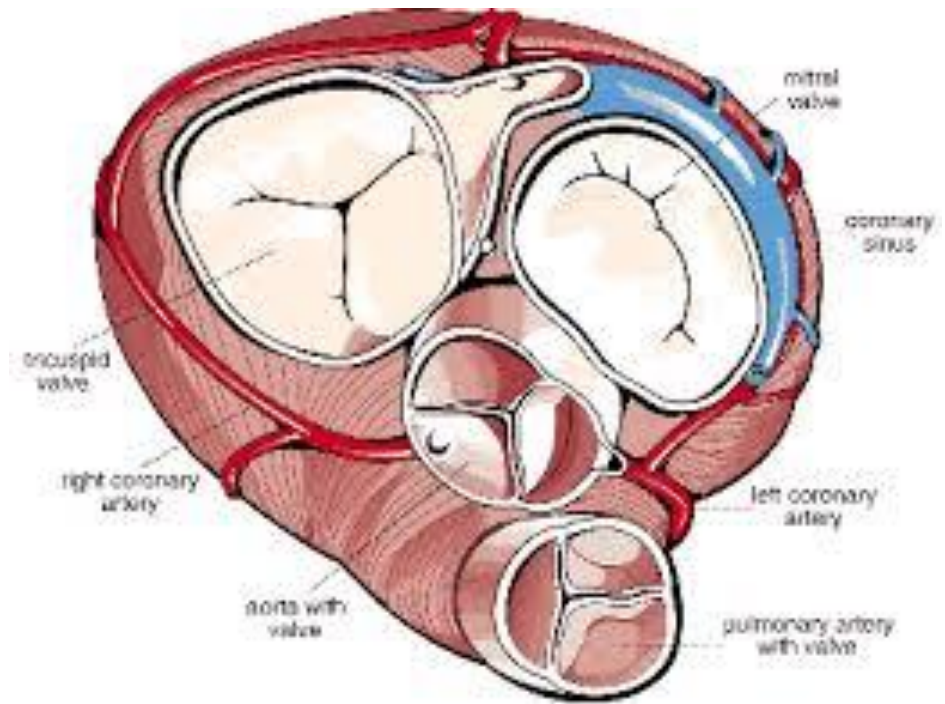


3. PULMONARY VALVE- 2nd left intercostal space

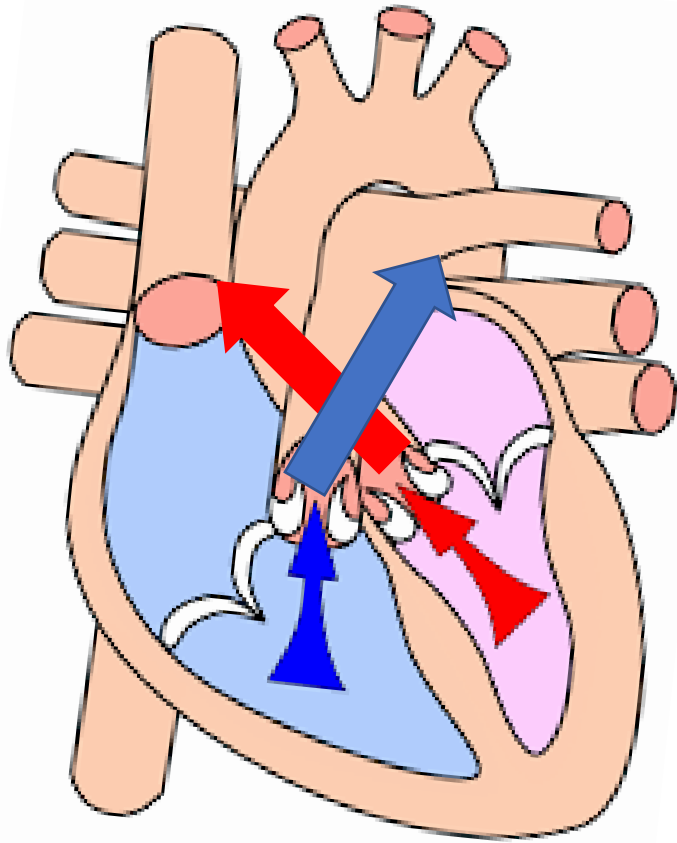
ERBS POINT- left 4th intercostal space , lateral to the sternal border

1. MITRAL VALVE- apical beat

back



front



systole

Heart rate (PALS)

General Vital Signs and Guidelines

Age	Heart Rate (beats/min)	Blood Pressure (mmHg)	Respiratory Rate (breaths/min)
Premature	110-170	SBP 55-75 DBP 35-45	40-70
0-3 months	110-160	SBP 65-85 DBP 45-55	35-55
3-6 months	110-160	SBP 70-90 DBP 50-65	30-45
6-12 months	90-160	SBP 80-100 DBP 55-65	22-38
1-3 years	80-150	SBP 90-105 DBP 55-70	22-30
3-6 years	70-120	SBP 95-110 DBP 60-75	20-24
6-12 years	60-110	SBP 100-120 DBP 60-75	16-22
> 12 years	60-100	SBP 110-135 DBP 65-85	12-20

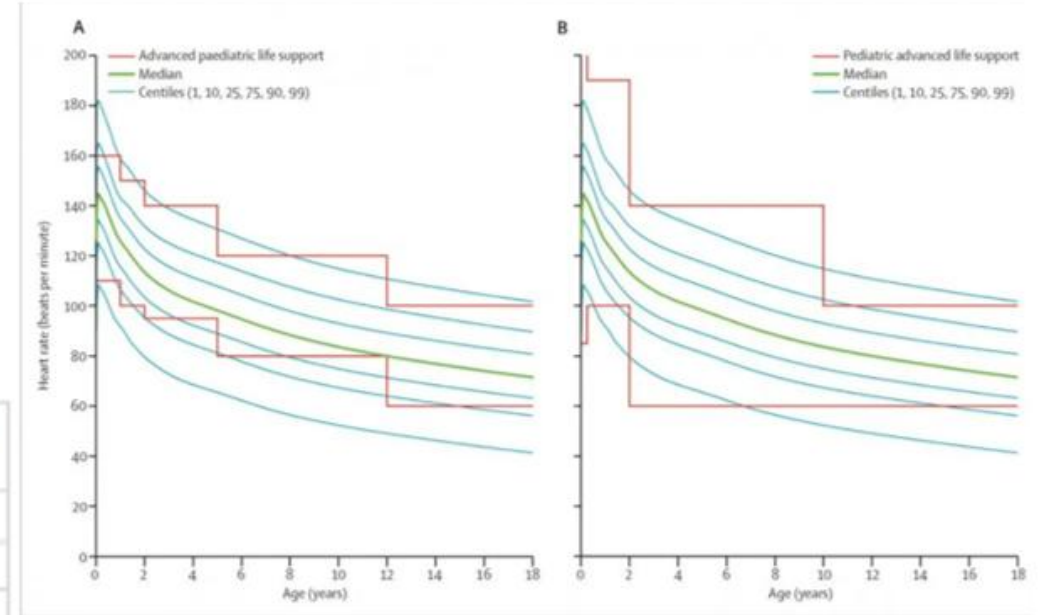
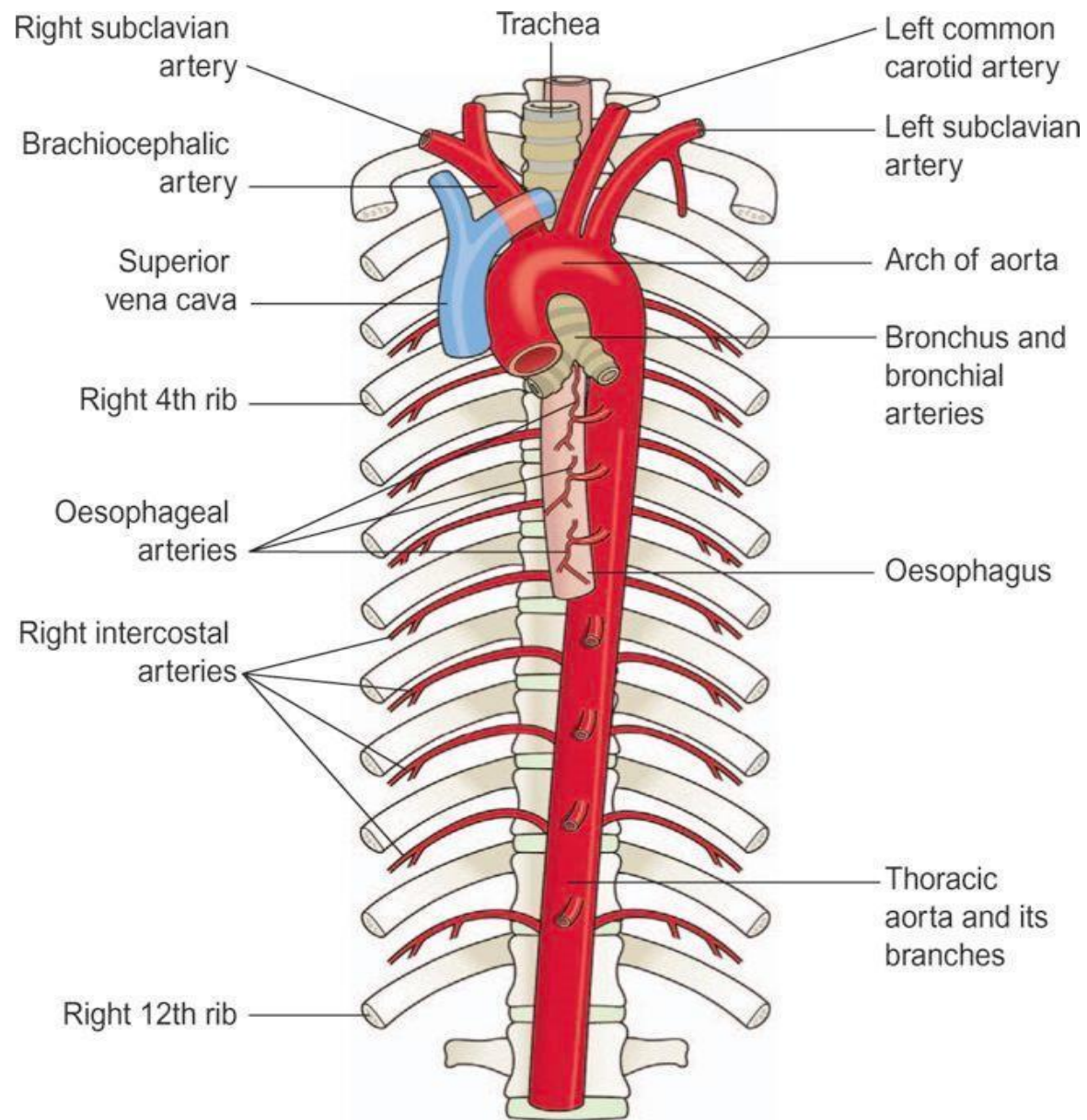
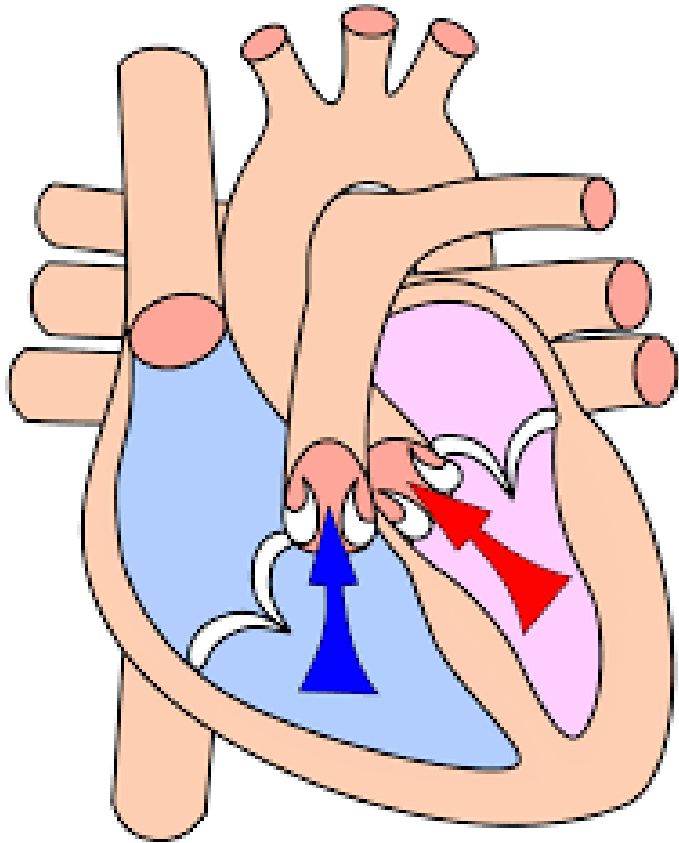


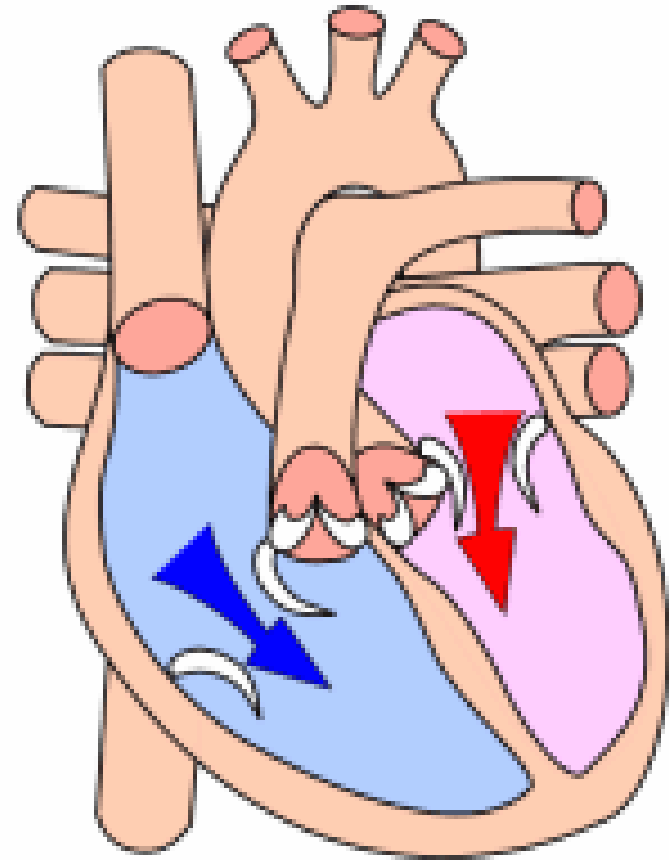
Figure 5: Comparison of heart rate centiles with paediatric reference ranges from the advanced paediatric life support (A) and pediatric advanced life support (B) guidelines



S1 THE CARDIAC CYCLE S2



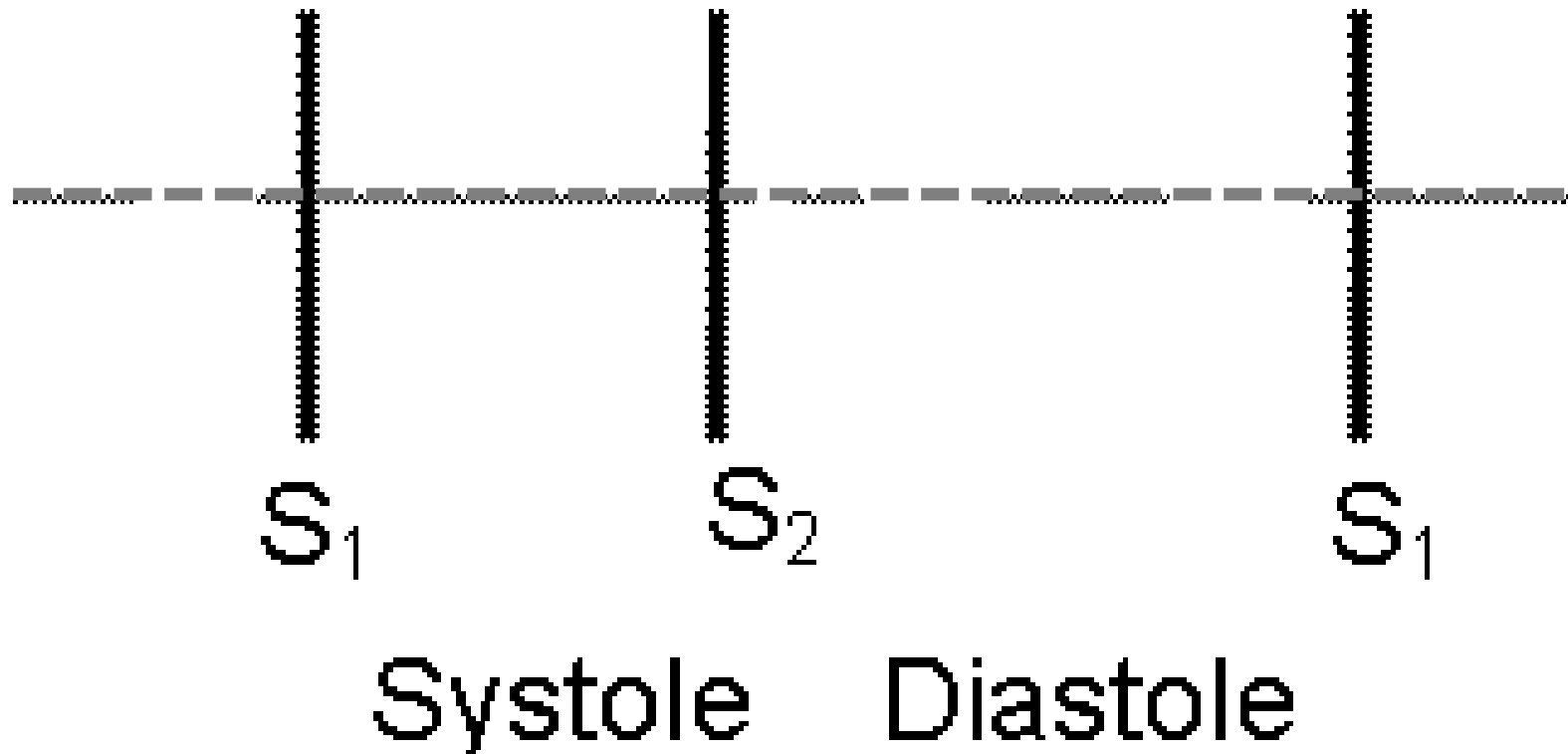
SYSTOLE



DIASTOLE

NORMAL HEART SOUNDS

=WHAT DO I HEAR?



Normal heart sound

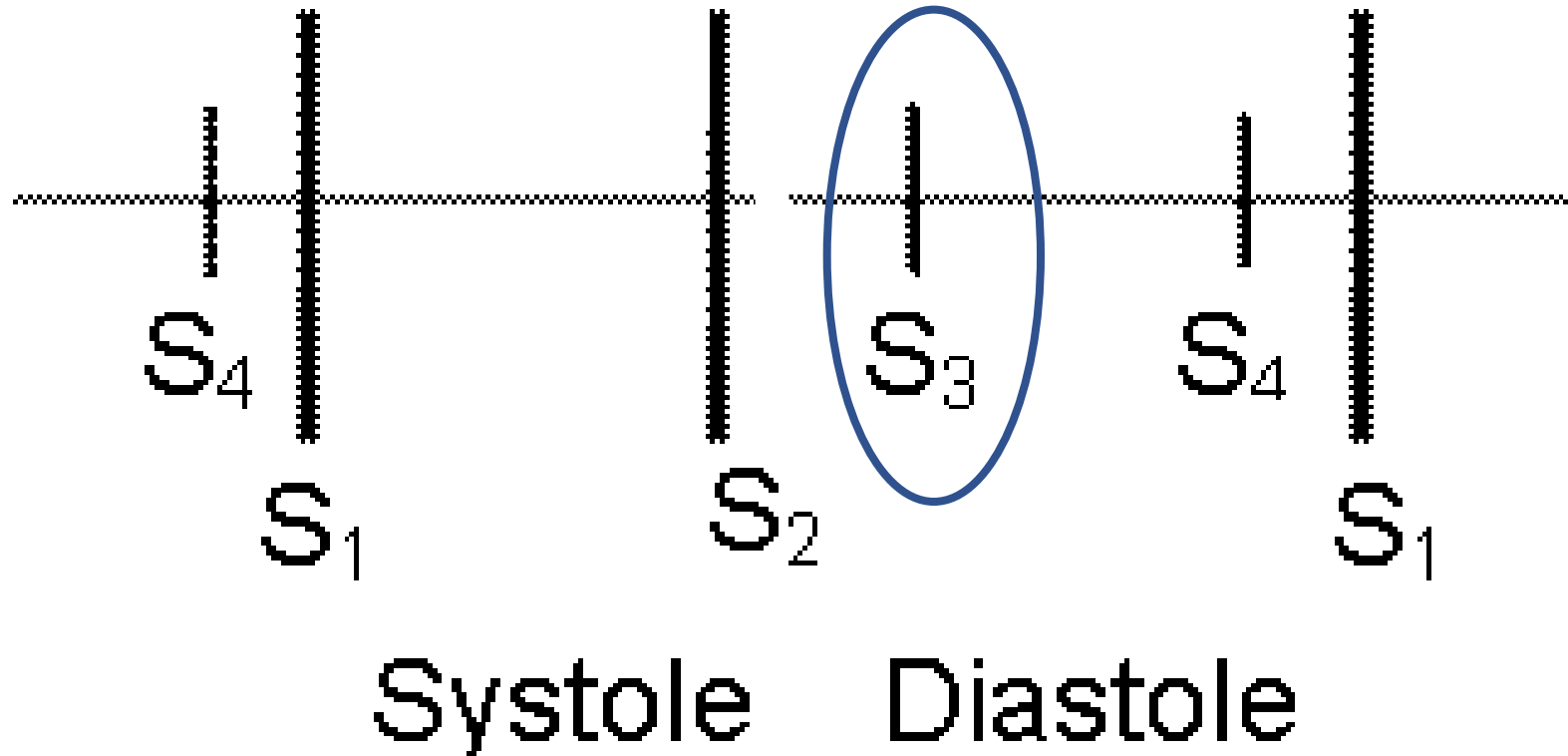
<https://www.youtube.com/watch?v=FtXNnmifbhE>

ABNORMAL HEART SOUNDS & MURMURS

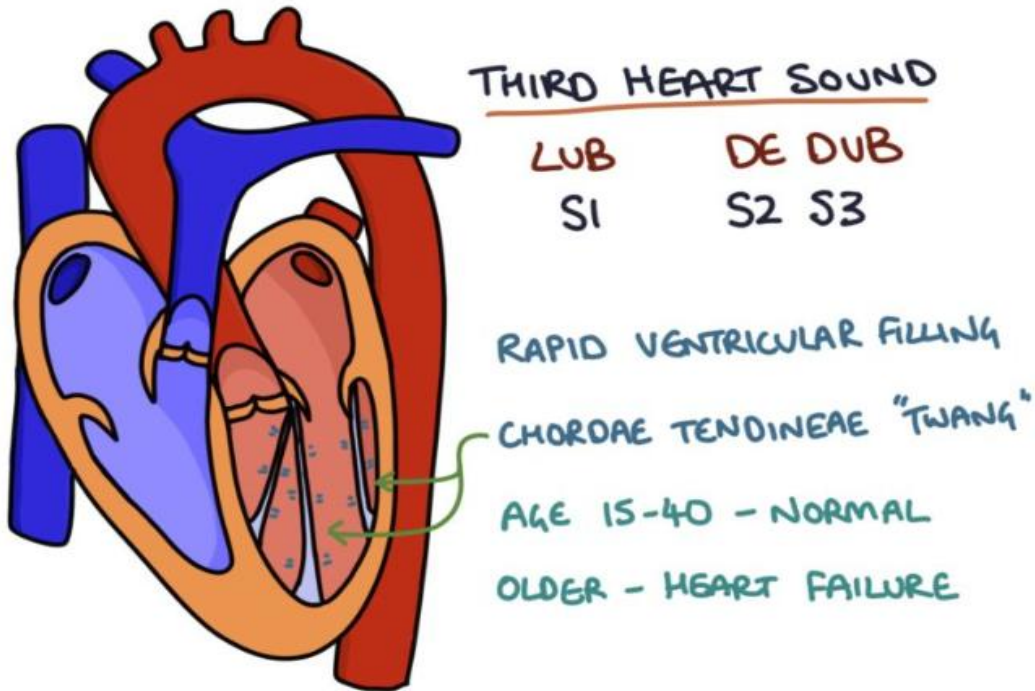
1. Extra heart sounds (S3, S4)
2. Splitting of S1 & S2
3. Alteration in intensity
4. Additional heart sounds (extrasystoly)
5. Murmurs

NORMAL HEART SOUNDS

=WHAT DO I HEAR?



A third heart sound (S3)



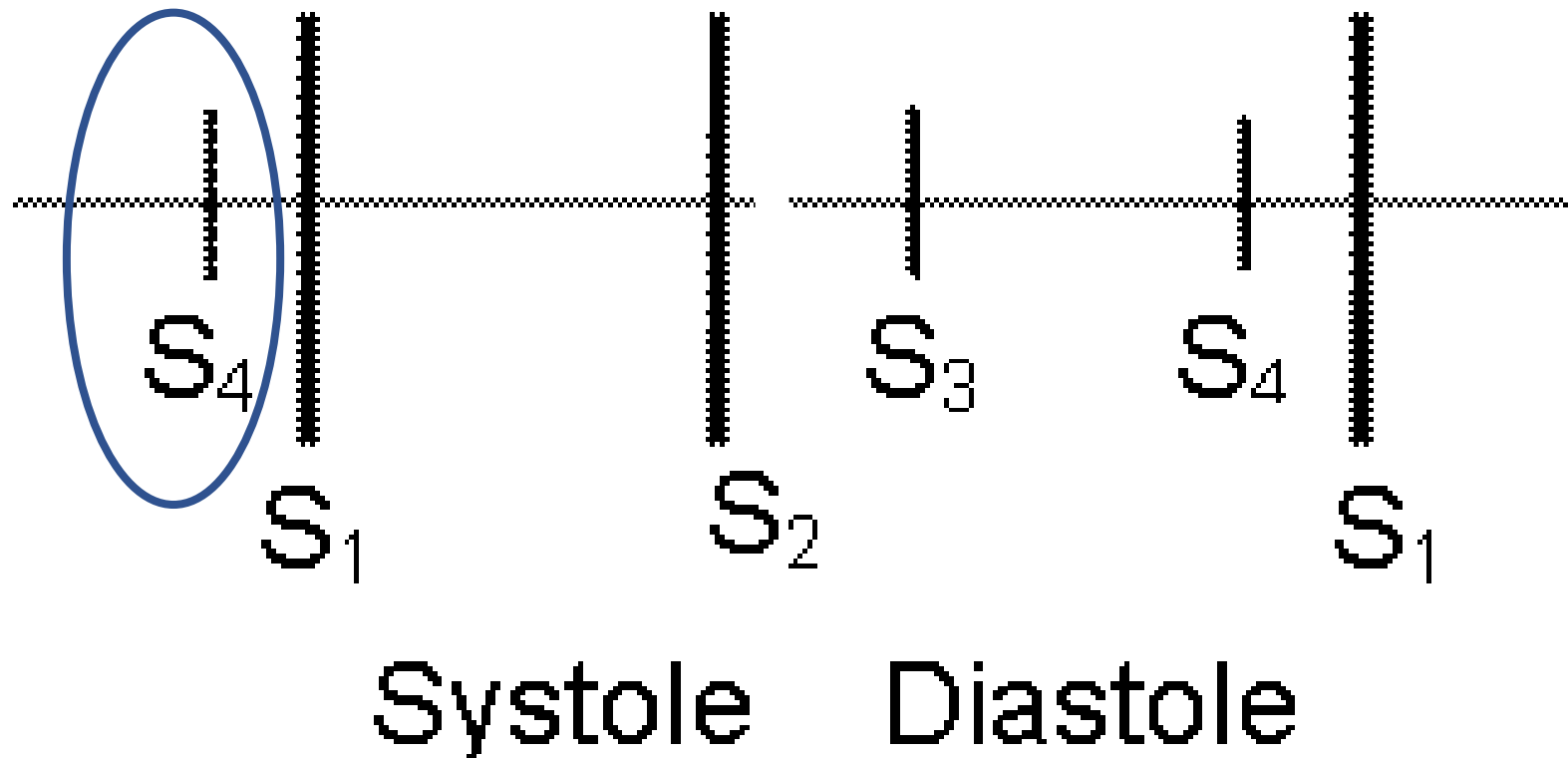
- is heard roughly 0.1 seconds after the second heart sound.
- **Due to rapid ventricular filling** causing the ***chordae tendineae*** to pull to their full length &
- „twang like a guitar string”
- **can be normal in children, teenagers and young healthy adults (15-40 years) - because the heart functions so well that the ventricles easily allow rapid filling**
- In older patients it can indicate heart failure, as the ***ventricles*** and ***chordae*** are stiff and weak so they reach their limit much faster than normal

S3 słuchamy

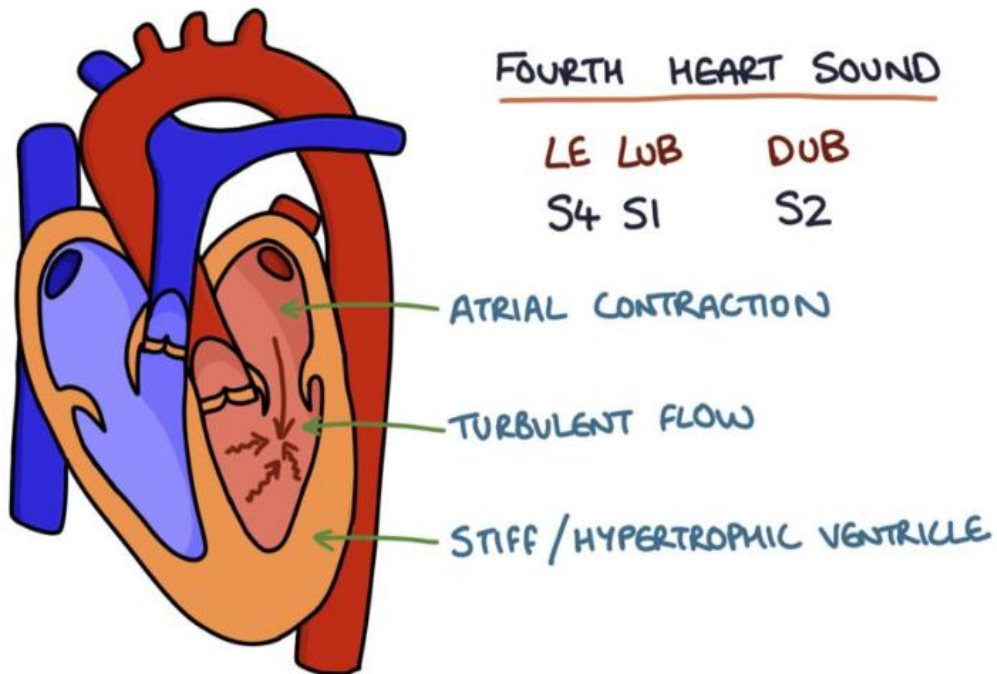
https://www.youtube.com/watch?v=_i2D1KZkN1w

NORMAL HEART SOUNDS

=WHAT DO I HEAR?



A fourth heart sound (S4)



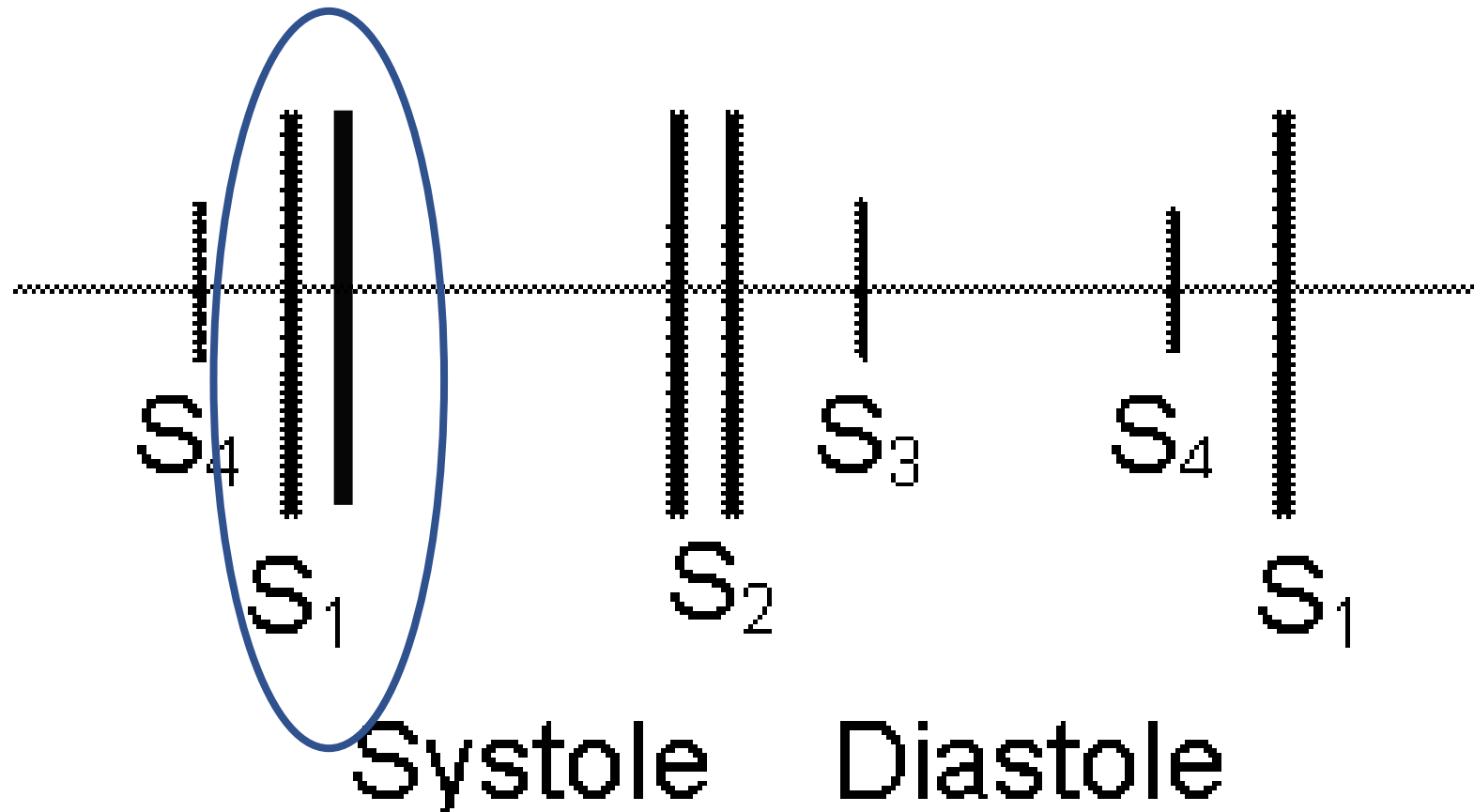
- is heard directly before **S1**.
- is always abnormal
- relatively rare to hear
- indicates a stiff or hypertrophic ventricle and is caused by **turbulent flow from an atria contracting against a non-compliant ventricle**
- eg. hypertrophic cardiomyopathy, fibrosis after MI
- „gallop of the heart”

S4- słuchamy

<https://www.youtube.com/watch?v=KcMF8rJDTIk>

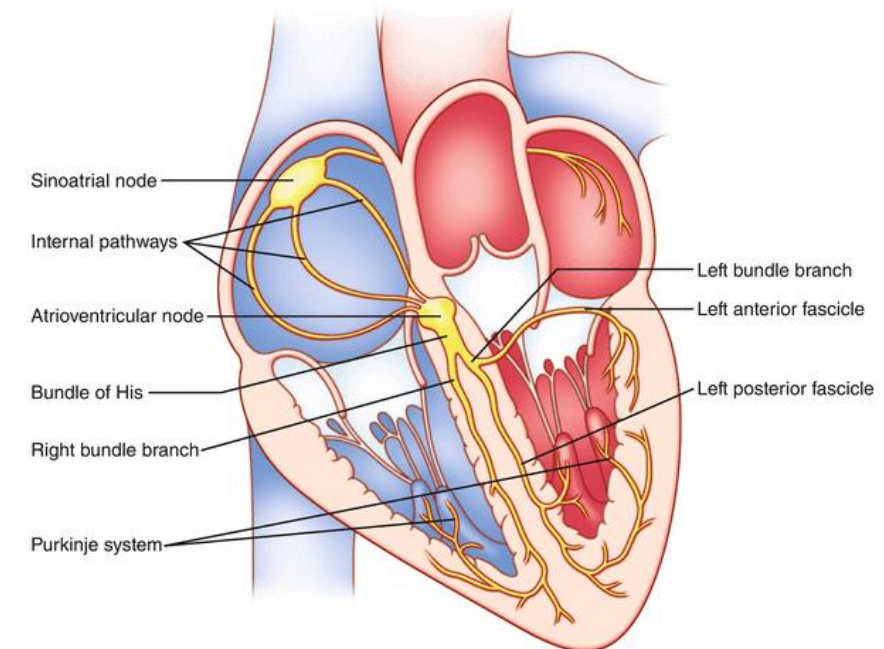
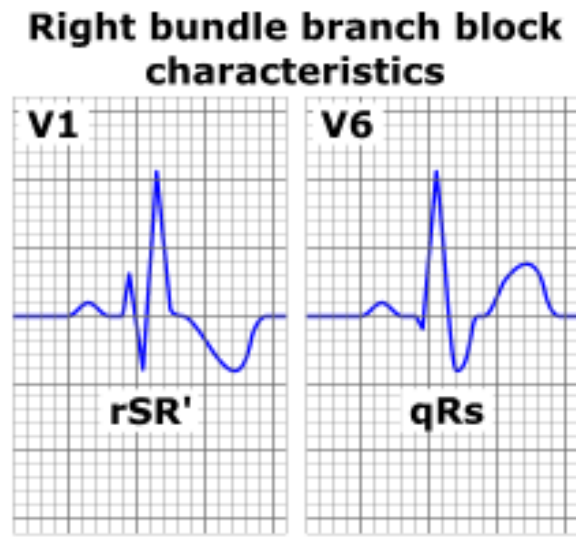
NORMAL HEART SOUNDS

=WHAT DO I HEAR?



Split of S1

- You will listen to S1 split **when MV and TV do not close exactly at the same time**
- M1 usually closes first (M1), then the TV (T1)
- S1 split is **usually normal finding**
- **May be heard in RBBB**
- **Differentiate from S4**

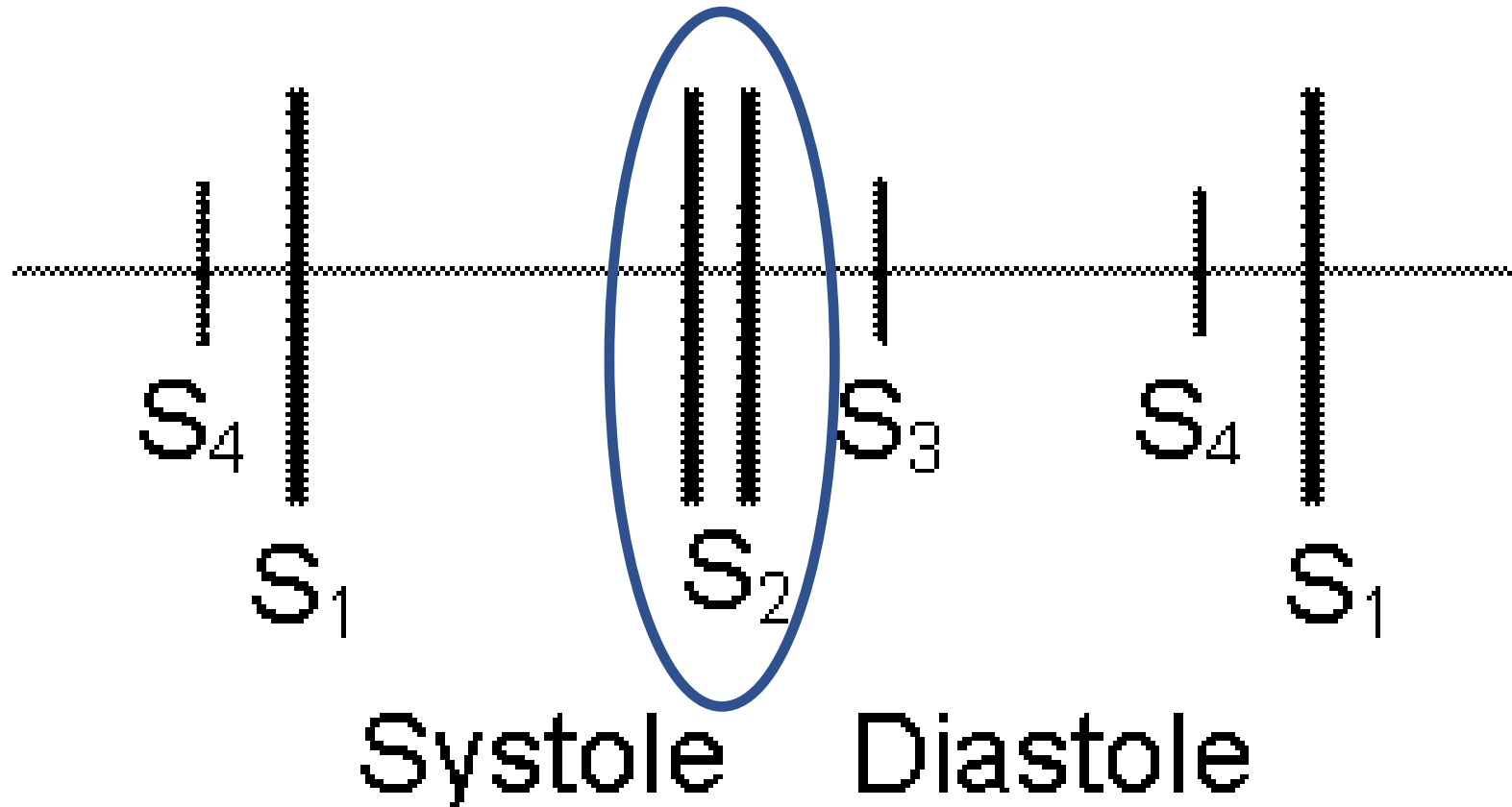


Split S1-słuchamy

<https://www.youtube.com/watch?v=kvQ2IU3ILRo>

NORMAL HEART SOUNDS

=WHAT DO I HEAR?



Split S2

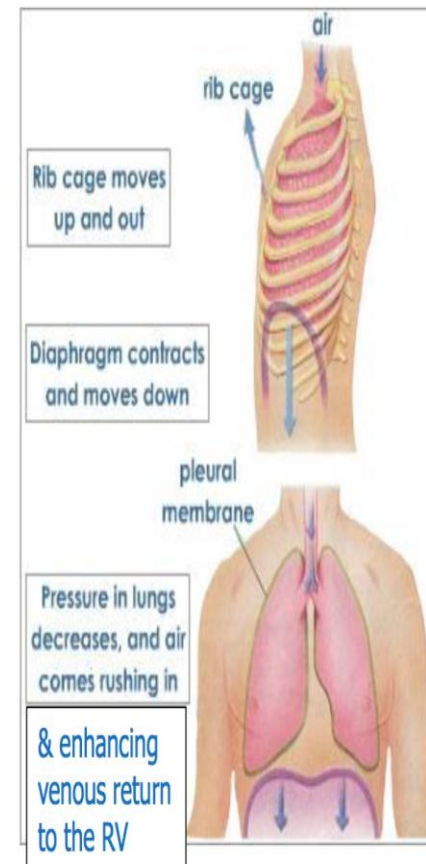
- Split of S2 can be normally heard **on deep inspiration**
- because **when a person takes a deep breath, a decrease of intrathoracic pressure causes an increase of venous blood (VCS & VCI)...**
- This **rapid venous return** on the right side of the heart **may cause a slight delay RV emptying and a slight delay of PV closure** (in comparison to Ao valve)

I. Ao II. PV → split of S2

- Physiological- become quiet in expiration, pathological S2 split is heard on inspiration & expiration
- Pathological- children with ASD & PS

During the isovolumetric relaxation, the aortic valve closes before the pulmonary valve

Inspiration causes splitting of S₂ (A₂, P₂), delaying closure of the pulmonic valve.



Intrathoracic pressure decreases
↓
Increases venous return to the RV
↓
Increases RV preload (RVEDV)
↓
Results in increased stroke volume and prolongs RV ejection time
↓
Delays closure of P₂

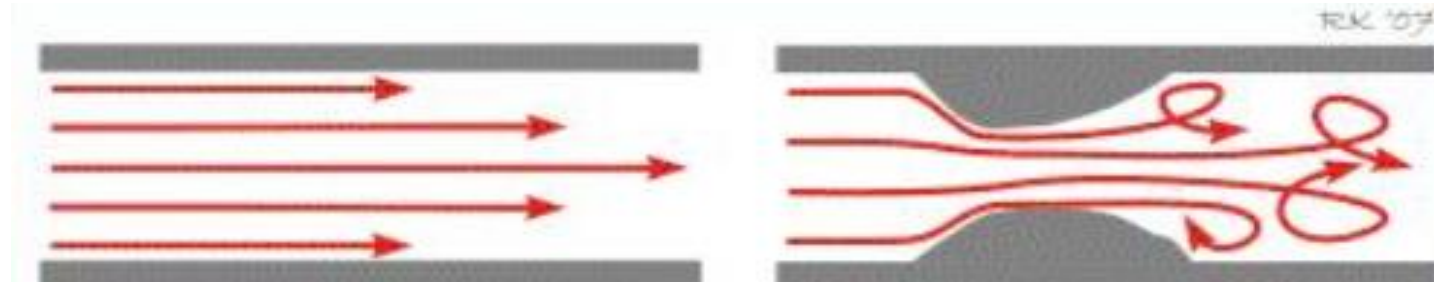
Split S2 – słuchamy

<https://www.youtube.com/watch?v=98HM1fr3cq4>

HEART MURMURS



HEART MURMURS



Present when:

1. **Turbulent flow** (eg. Stenotic valve, constricted vessel, hole in atrial or ventricular septum- VSD, ASD)
2. **High flow through normal orifice** (eg. hyperkinetic circulation)
3. **Backward or regurgitant flow** through the valve

Assessing a Murmur (SCRIPT mnemonic)

S – Site: where is the murmur loudest?

C – Character: soft / blowing / crescendo (getting louder) / decrescendo (getting quieter) / crescendo-decrescendo (louder then quieter)

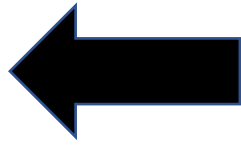
R – Radiation: can you hear the murmur over the carotids (AS) or left axilla (MR)?

I – Intensity: what grade is the murmur?

P – Pitch: is it high pitched or low and grumbling? Pitch indicates velocity.

T – Timing: is it systolic or diastolic?

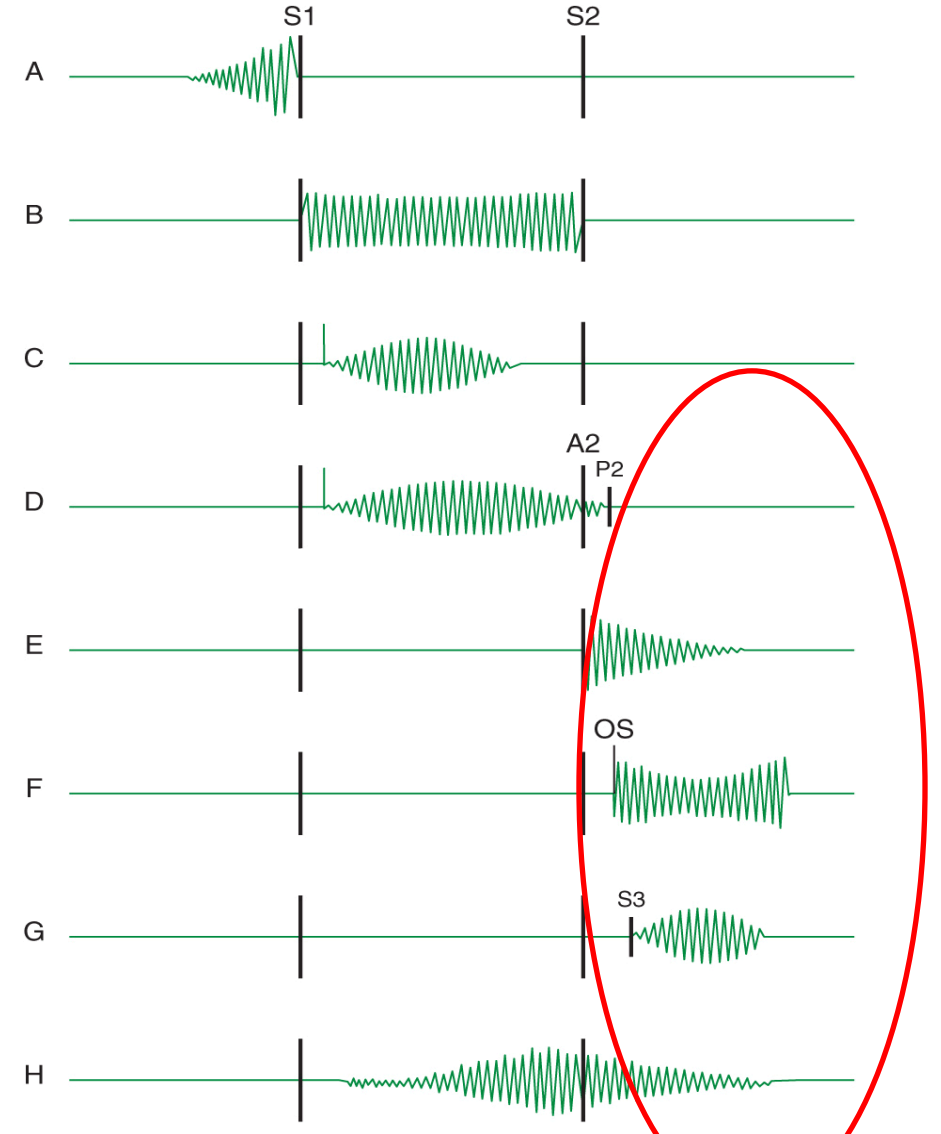
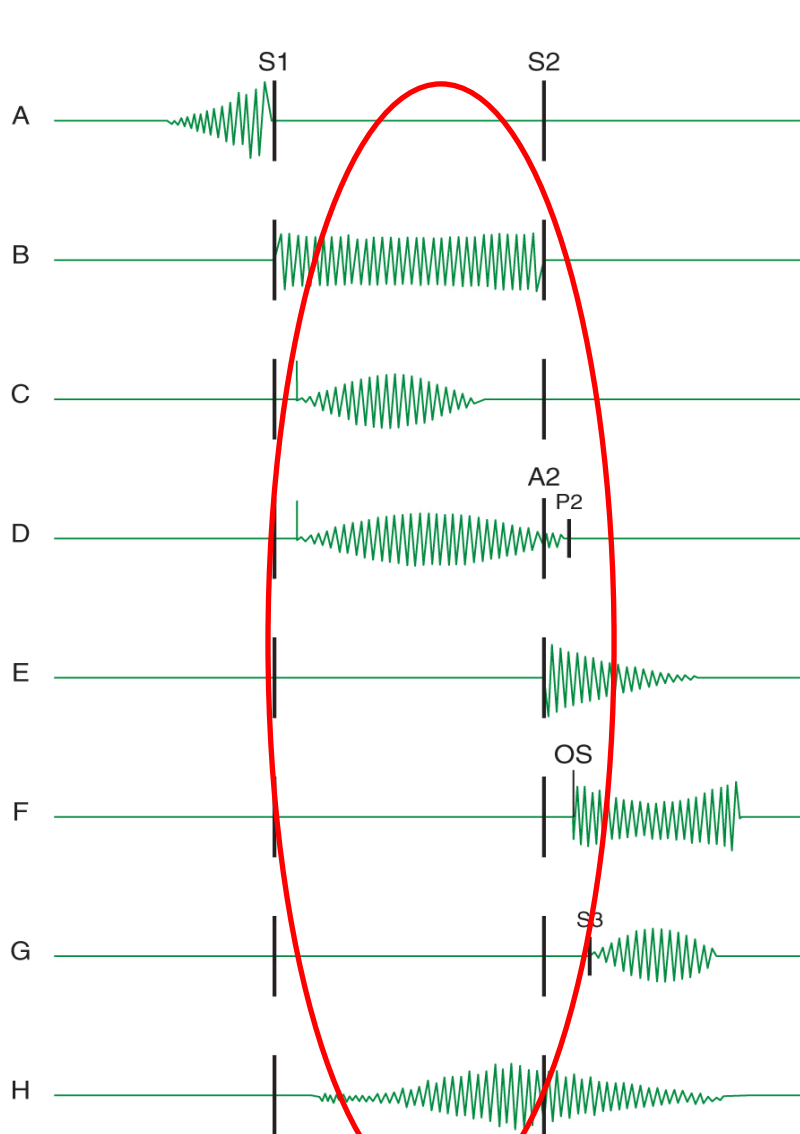
SYSTOLIC



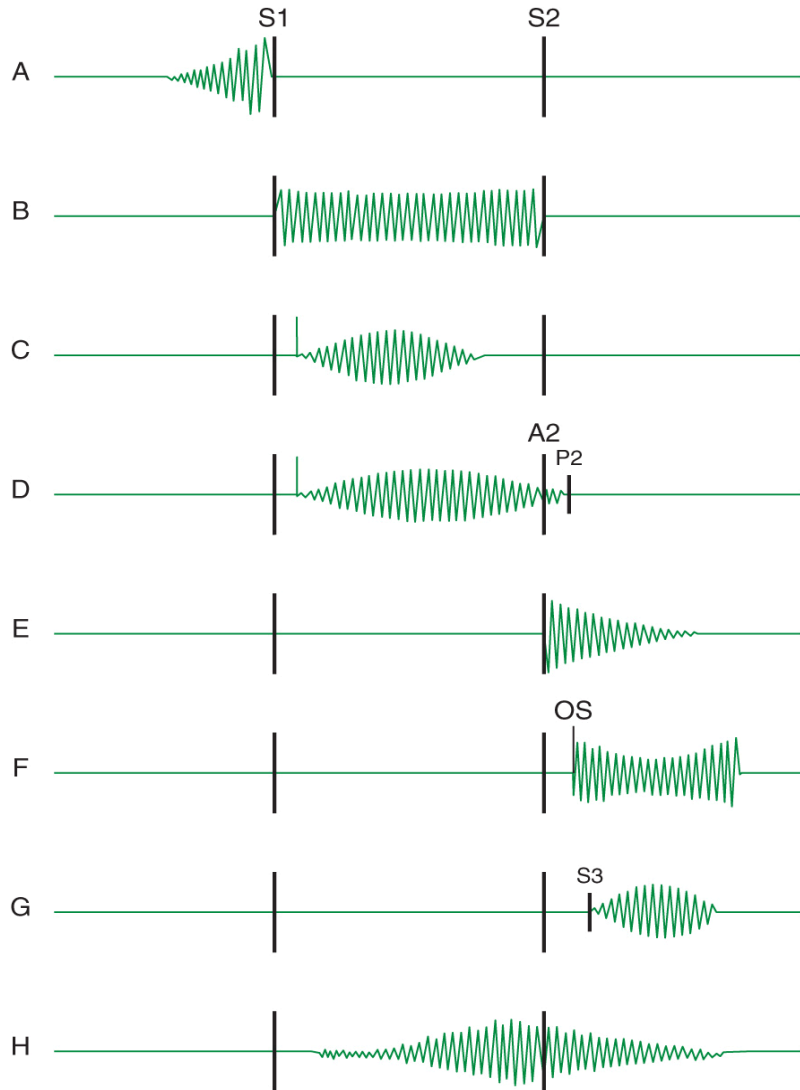
MURMURS



DIASTOLIC



MURMURS



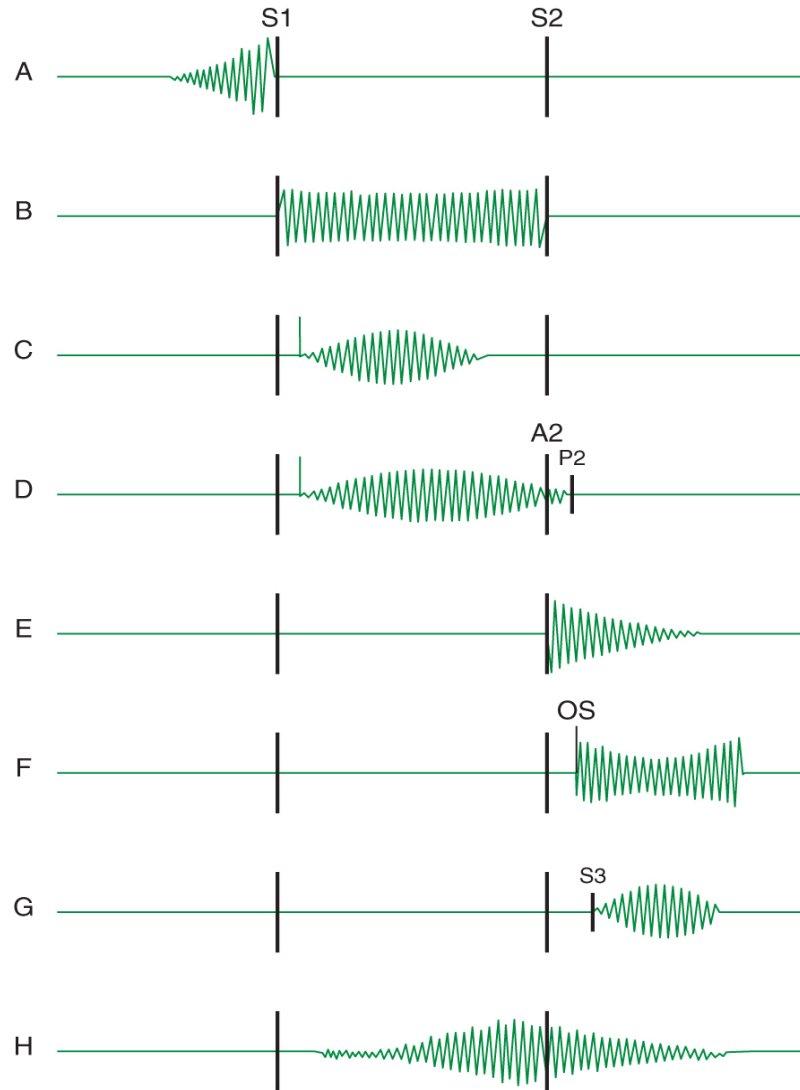
- **Holo-** systolic= **Pansystolic**

- **Early-** systolic

- **Mid-** systolic

- **End-** systolic

MURMURS



- **End-diastolic**

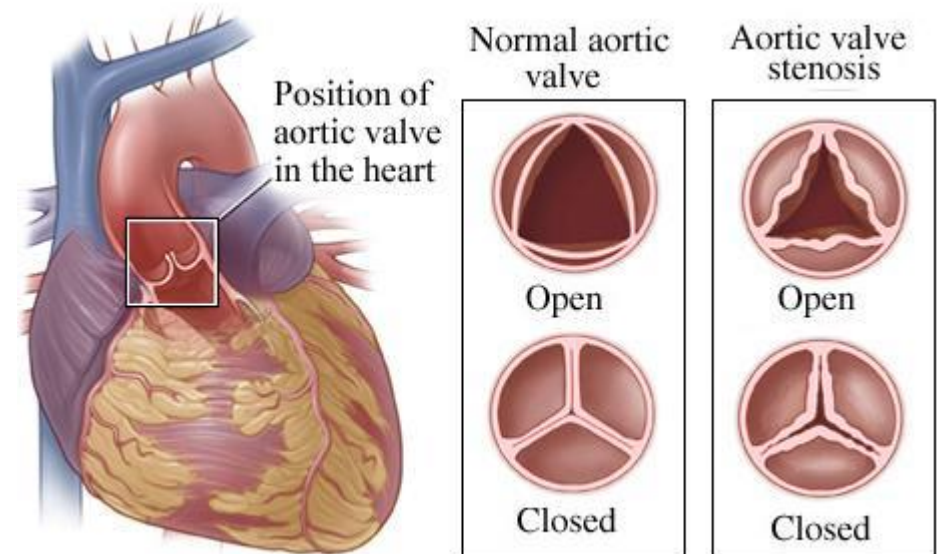
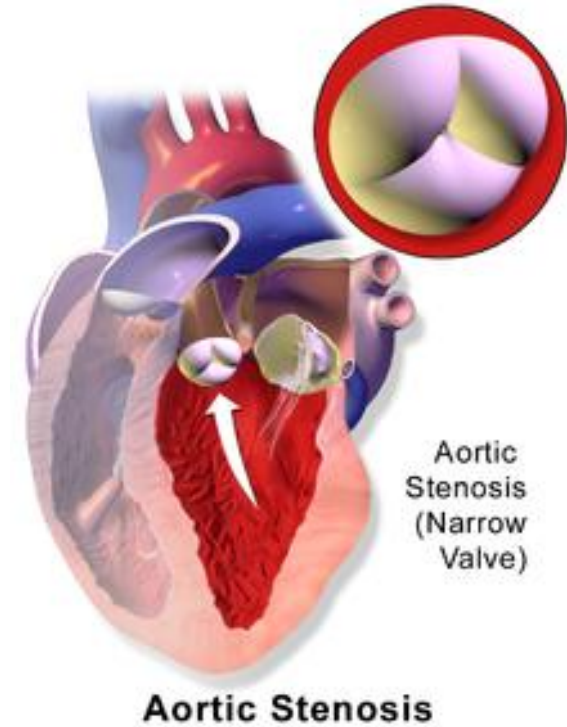
- **Early-** diastolic

- **Holo-** diastolic

- **Mid-** diastolic

Aortic stenosis (AoS)

- In a child with AoS, because the AoV is very narrow, **the pressure in LV is much higher** than normal and the **heart must work harder** to pump blood out into the body arteries
- Over time this CHD can cause **hypertrophy and damage to the overworked heart muscle**- should be treated (balloon or surgery)
- On examination- **systolic ejection murmur** due to **turbulent flow through the AoV**

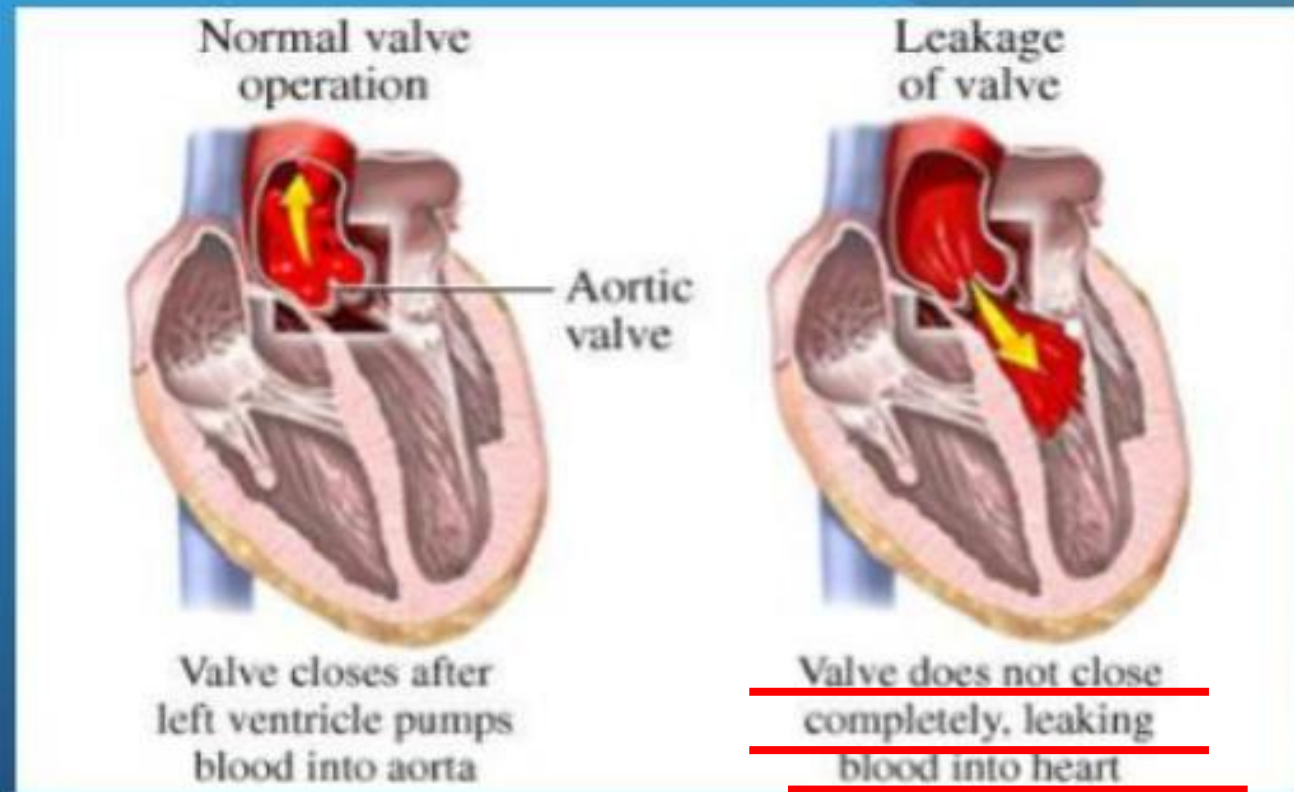


Aortic stenosis- słuchamy

<https://www.youtube.com/watch?v=pgDWz1JybzE&t=15s>

Pathology: Aortic Valve Regurgitation

- Incompetent aortic valve
- Maybe in conjunction with aortic stenosis
- Maybe associated with dilated aorta root
- Heard on diastole

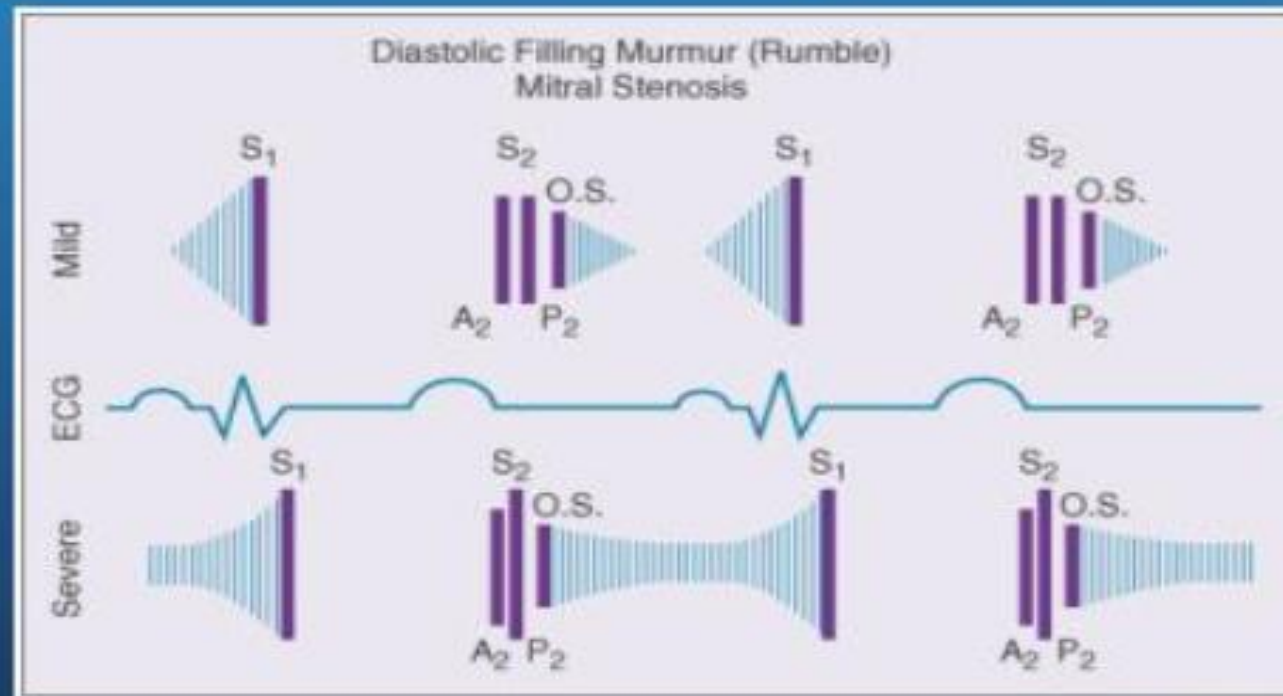


Ao regurgitation- słuchamy

<https://www.youtube.com/watch?v=uZysrKXHJMM>

Pathology: Mitral

- Mitral stenosis
 - Mid-diastolic and pre-systolic low frequency “rumble”
 - Murmur after mitral opening snap
 - Opening snap: depend on calcification \propto 1/valve mobility
 - Often difficult to hear as soft and low frequency
 - Aerobic exercise and left lateral decubitus position accentuate

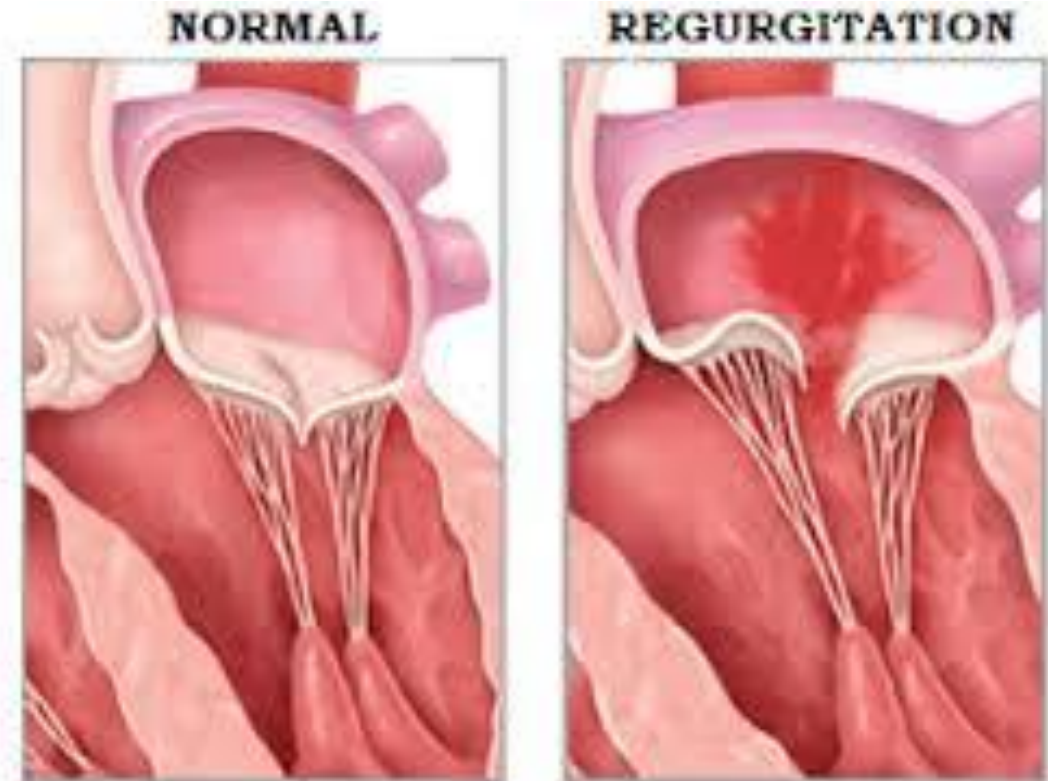
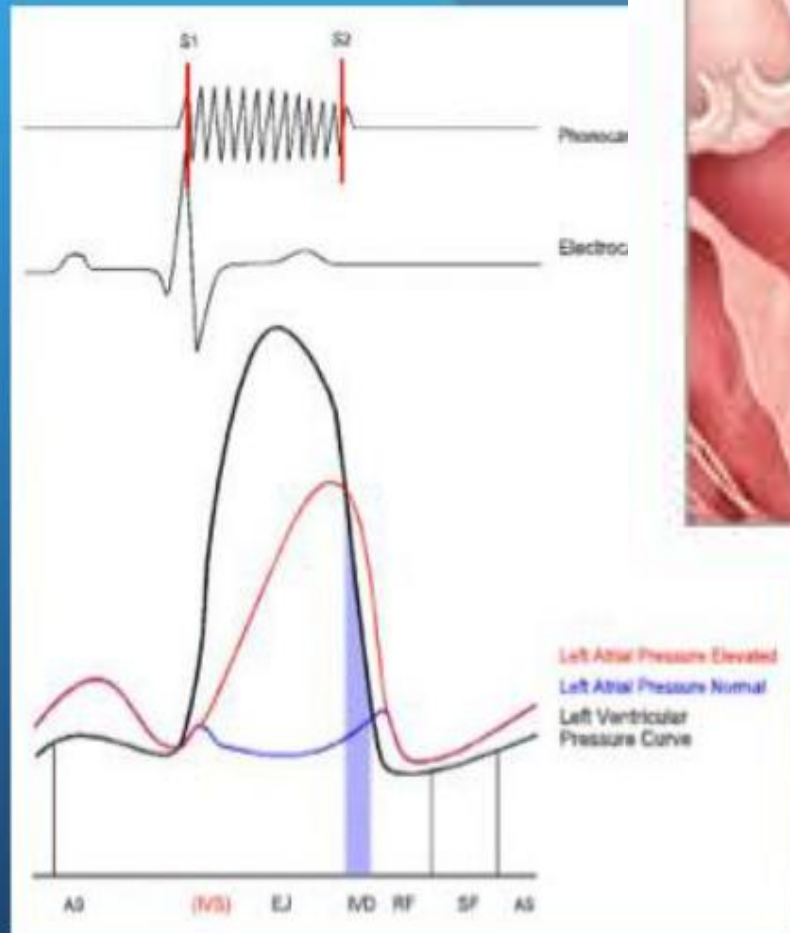


Mitral stenosis- słuchamy

<https://www.youtube.com/watch?v=5oCPtZo4pUY>

Pathology: Mitral

- Mitral regurgitation
 - Holosystolic constant high-frequency
- Maneuvers:
 - Typical MR and Rheumatic MR:
 - Increase with increase afterload
 - Not effected much by respiration
- Mitral valve prolapse:
 - Significant changes with respiration

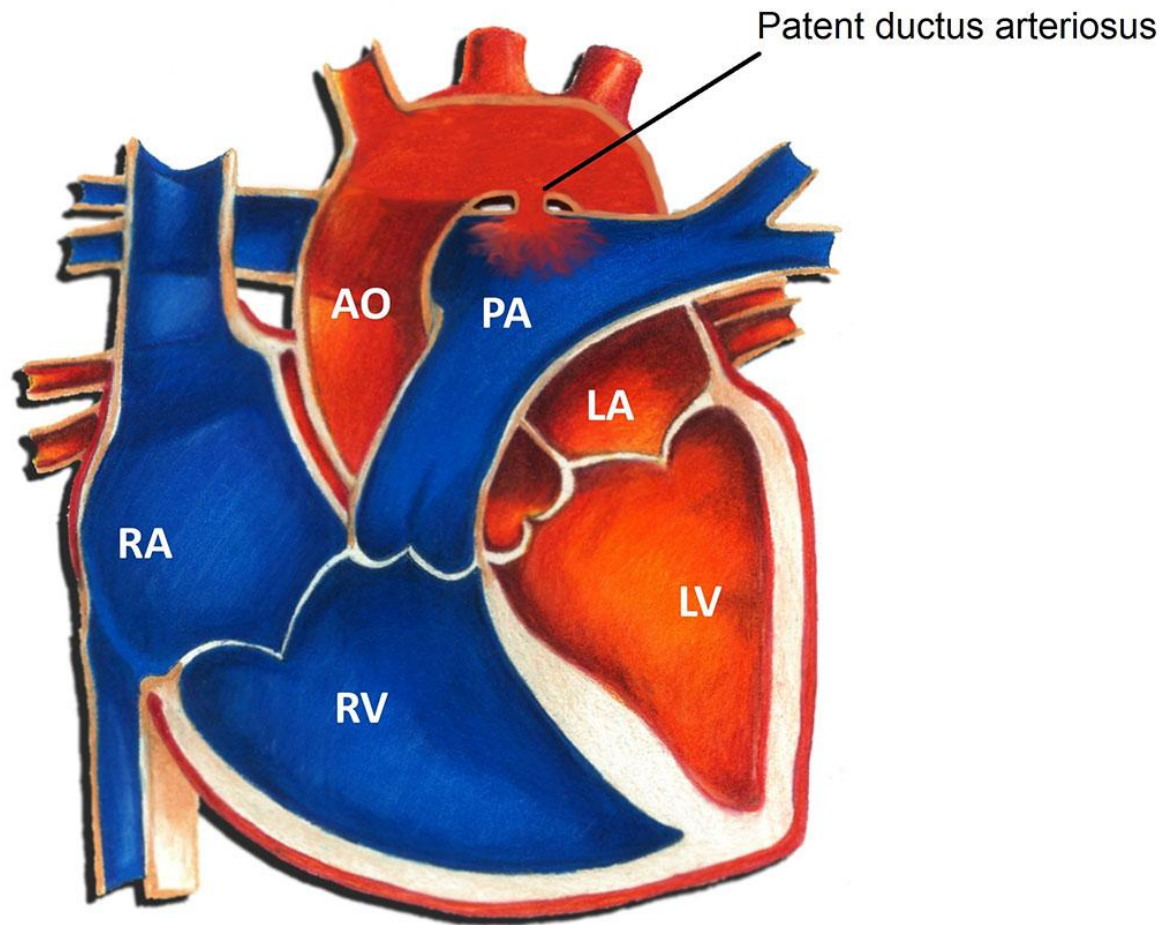


→ MV cannot close completely causing leakage of the blood during systole of the heart cycle

Mitral- Tricuspid Valve Regurgitation Holosystolic Murmur

<https://www.youtube.com/watch?v=MzORJbyHTT0>

Continuous machinery murmur- PDA (Gibson's murmur)



- In PDA, abnormal blood flow occurs between Ao and pulmonary trunk
- Typical machinery systolic-diastolic murmur

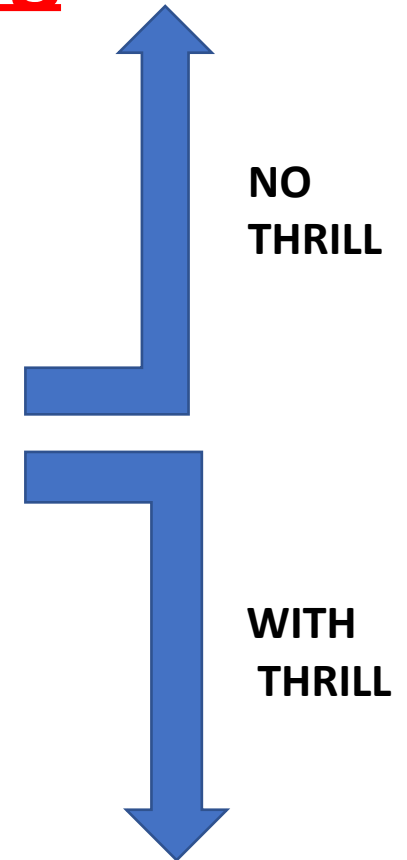
Continuous murmur- PDA

<https://www.youtube.com/watch?v=LduljbtF7kA>

Murmur Grade- Levine's grading

1. Difficult to hear
2. Quiet
3. Easy to hear with stethoscope, but no palpable thrill
4. Easy to hear with a palpable thrill
5. Can hear with stethoscope barely touching chest
6. Can hear with stethoscope off the chest

- Grading a murmur is quite subjective
- If in doubt it is probably grade 2 or 3



Assessing a Murmur (SCRIPT mnemonic)

S – Site: where is the murmur loudest?

C – Character: soft / blowing / crescendo (getting louder) / decrescendo (getting quieter) / crescendo-decrescendo (louder then quieter)

R – Radiation: can you hear the murmur over the carotids (AS) or left axilla (MR)?

I – Intensity: what grade is the murmur?

P – Pitch: is it high pitched or low and grumbling? Pitch indicates velocity.

T – Timing: is it systolic or diastolic?

Special manoeuvres/ positions

can be used to emphasise certain murmurs:

- Patient on their left side → ***mitral stenosis, galop of the heart***
- Patient sat up, leaning forward and holding exhalation → ***aortic regurgitation***

difficult in children- poor cooperation

Physiology: Maneuvers

- Position

- Supine:

- Increase preload:
increased blood volume
in heart

- Rapid standing:

- Decrease preload:
decreased blood volume
in heart

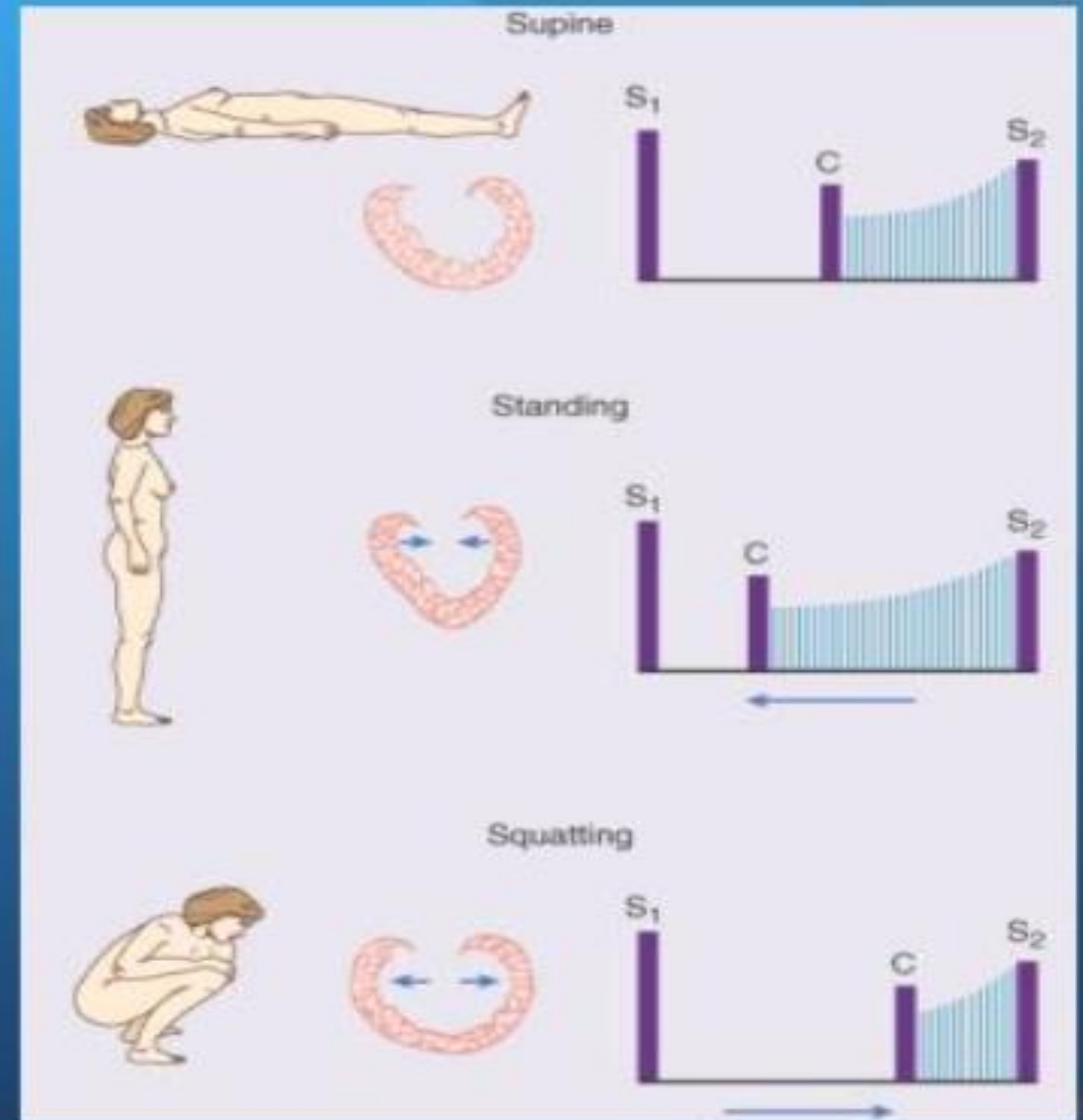
- Valsalva (bear down)

- Decrease venous return
(preload)

Most murmurs become quiet, except
from hypertrophic cardiomyopathy

- Breathing

- Deep breath in: Increased
preload → Murmurs from right heart
 - Deep breath out: Decrease
preload



But....

... not every murmur is a pathology



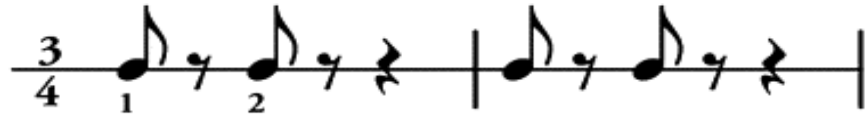
Innocent murmurs



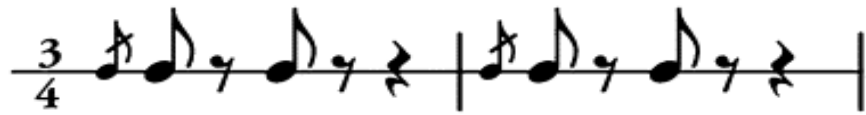
- In healthy children
- Caused by turbulent blood flow through **anatomically healthy heart**
- **Asymptomatic**
- Most of them **systolic (1 exception)**
- **Postural**- volume varies with sitting/ standing
- Intensifies with **increased cardiac output** (eg. fever, emotions, exercise)
- **Short** duration
- **Soft** and **quite** in quality < 3 grade
- **No radiation**
- Otherwise **normal physical examination**- no palpable thrill, no SOB, no FTT, normal BP, HR, SaO₂

Heart sounds

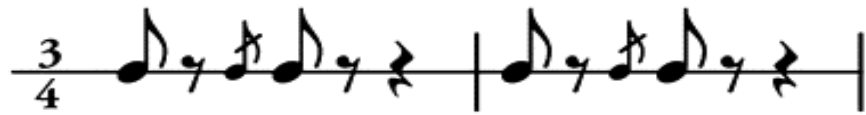
Normal 1st and 2nd heart sounds



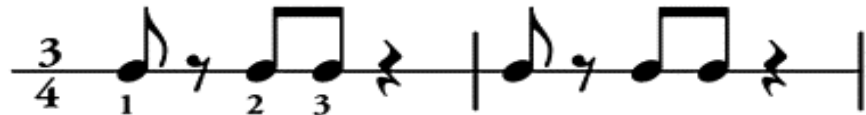
Split 1st heart sound



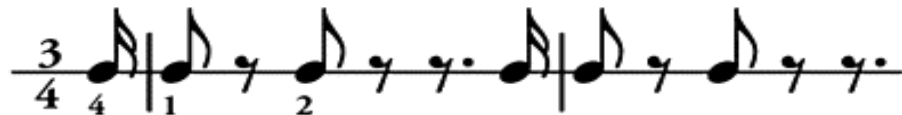
Split 2nd heart sound



Added 3rd heart sound



Added 4th heart sound

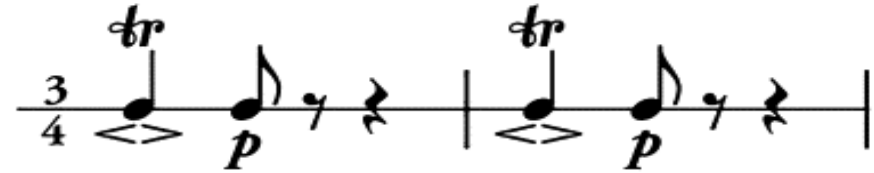


Summation gallop

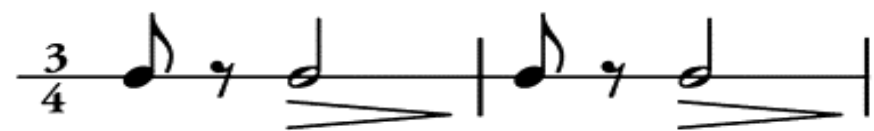


Left-sided murmurs

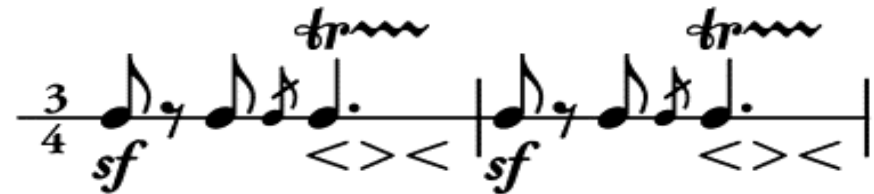
Aortic stenosis



Aortic regurgitation



Mitral stenosis



Mitral regurgitation



CYANOSIS



CYANOSIS

- the bluish or purplish **discoloration** of the **skin or/ and mucous membranes** due to **low oxygen saturation** of the tissues near the skin surface
- Is a result of **deoxygenated haemoglobin** or **abnormal haemoglobin** in the blood
- Is apparent **when there is ≥ 5 g/dl of reduced haemoglobin or ≥ 0.5 g/dl of methaemoglobin**
- Anemic patients may not become cyanotic even in the presence of marked arterial desaturation
- In the light-skinned patients cyanosis is usually noted with arterial $\text{SaO}_2 < 85\%$, whereas:
- In the dark-skinned patients, the SaO_2 may be lower

CYANOSIS

CENTRAL

is due to a circulatory or ventilatory problem that leads to poor blood oxygenation in the lungs

- lips
- tongue

PERIFERAL

Is due to an inadequate or obstructed circulation

- only the extremities or fingers



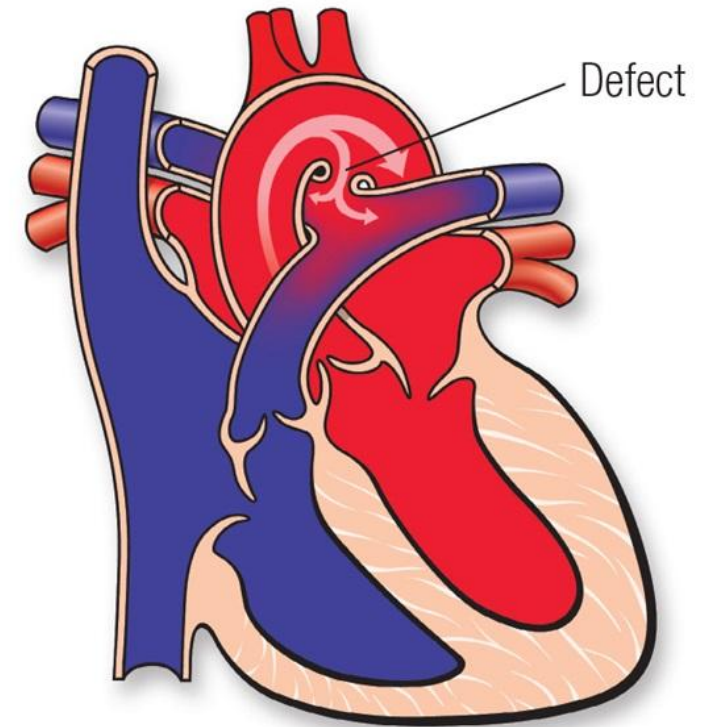


Causes of central cyanosis

1. Cardiovascular diseases:

- Congenital heart disease with R-L shunt
- Heart failure
- Valvular heart disease
- Myocardial infarction

Patent Ductus Arteriosus



Causes of central cyanosis

2. Respiratory system:

- Severe pneumonia
- Bronchiolitis
- Bronchospasm
- Pulmonary hypertension
- Pulmonary embolism
- Hypoventilation
- Chronic obstructive pulmonary disease
- Cystic fibrosis



Causes of central cyanosis

3. Central nervous system (impairing normal ventilation):

- Intracranial **hemorrhage**
- **Drug overdose** (e.g. heroin) → **apnea** or/ and **airway obstruction**
- **Tonic–clonic seizure** (e.g. grand mal seizure)



Causes of central cyanosis

4. Blood :

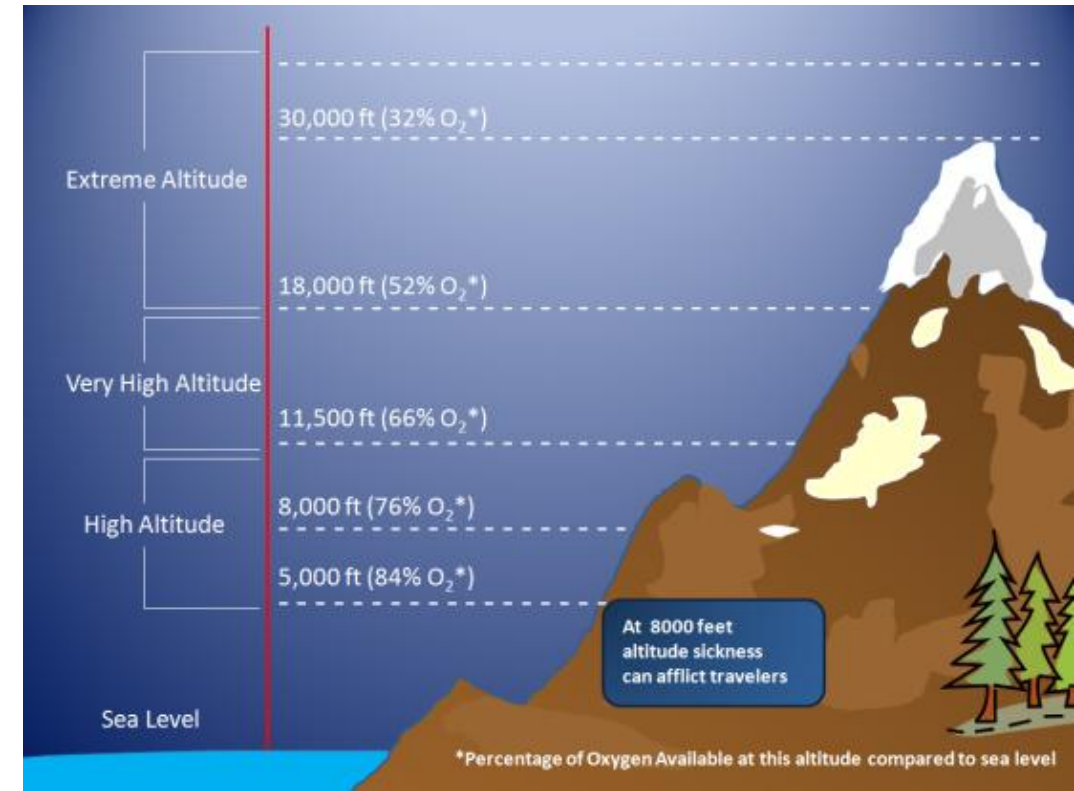
- **Congenital cyanosis** (HbM Boston) arises from a mutation in the α -codon which results in a change of primary sequence
- **Methemoglobinemia**- patient appears cyanosed even in the presence of a normal arterial oxygen level due to conversion of iron in hemoglobin from the ferrous $[\text{Fe}^{2+}]$ to the ferric $[\text{Fe}^{3+}]$ → acquired (drugs, chemicals & toxins eg. aniline dyes, chlorates, and bromates)



Causes of central cyanosis

5. Others:

- **High altitude**, cyanosis may develop in ascents to altitudes >2400 m.
- **Hypothermia**
- Severe obstructive sleep apnea (**apnea**)



Peripheral cyanosis

may be due to the following causes:

- All common causes of central cyanosis
- Reduced cardiac output (e.g. heart failure or hypovolaemia)
- Cold exposure
- Chronic obstructive pulmonary disease (COPD)
- Arterial obstruction (e.g. Raynaud phenomenon)
- Venous obstruction (e.g. deep vein thrombosis, DVT)



Is it cyanosis?



Is it cyanosis?



Argyria or argyrosis is a condition caused by excessive exposure to chemical compounds of the **element silver** or to **silver dust**

- skin turns purple or purple-grey
- **Generalised (with mucus membranes, eyes)**
- Argyria **worsens** and builds up as exposure to silver continues, and **does not resolve once exposure stops**

CLUBBING



NAIL CLUBBING



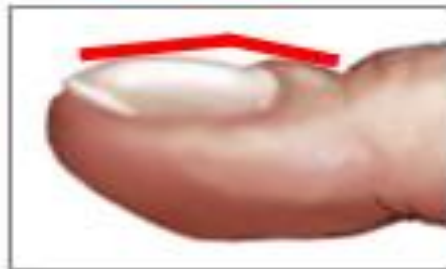
NAIL CLUBBING

- is a deformity of the **fingers** and/or **toes nails** associated with a number of diseases, mostly of the heart and lungs
- occurs when **the tips of the fingers enlarge and the nails curve around the fingertips,**
- usually over the course of years
- is often **the result of low oxygen** in the blood and could be a sign of various types of heart and/ or lung disease
- Pathomechanism- unknown

$< 180^\circ$ NAIL CLUBBING



Normal angle
of nail bed



Distorted angle
of nail bed

Clubbed fingers



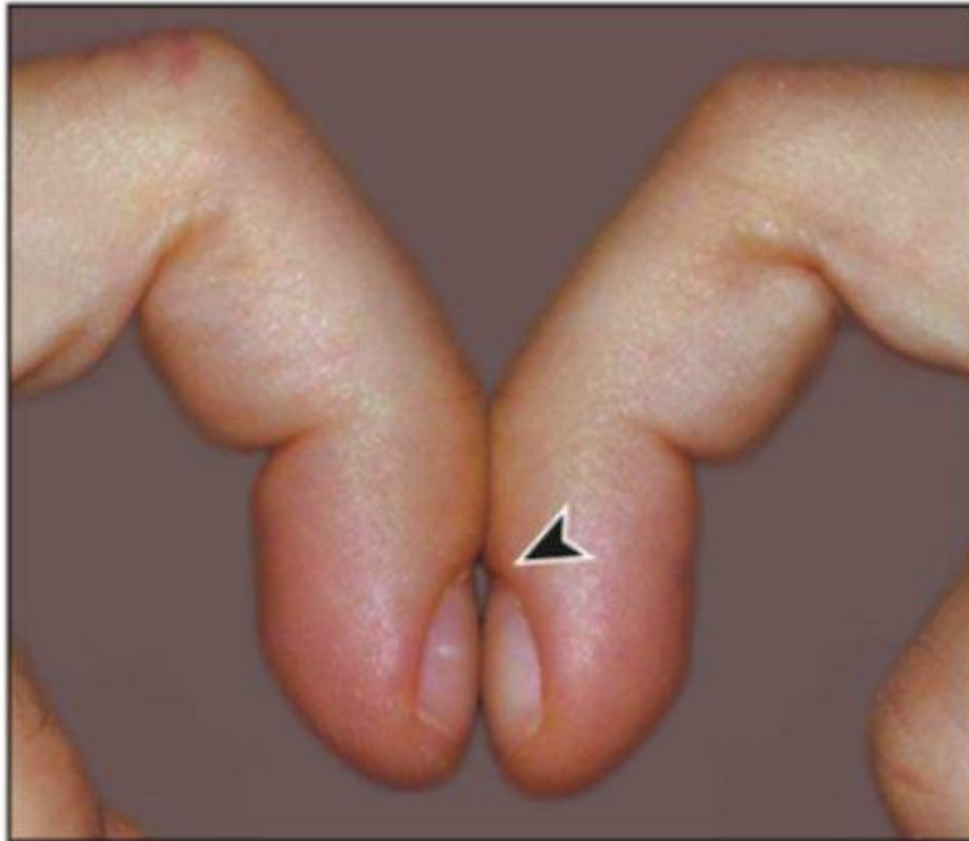
NAIL CLUBBING- TEST (Schamroth sign)



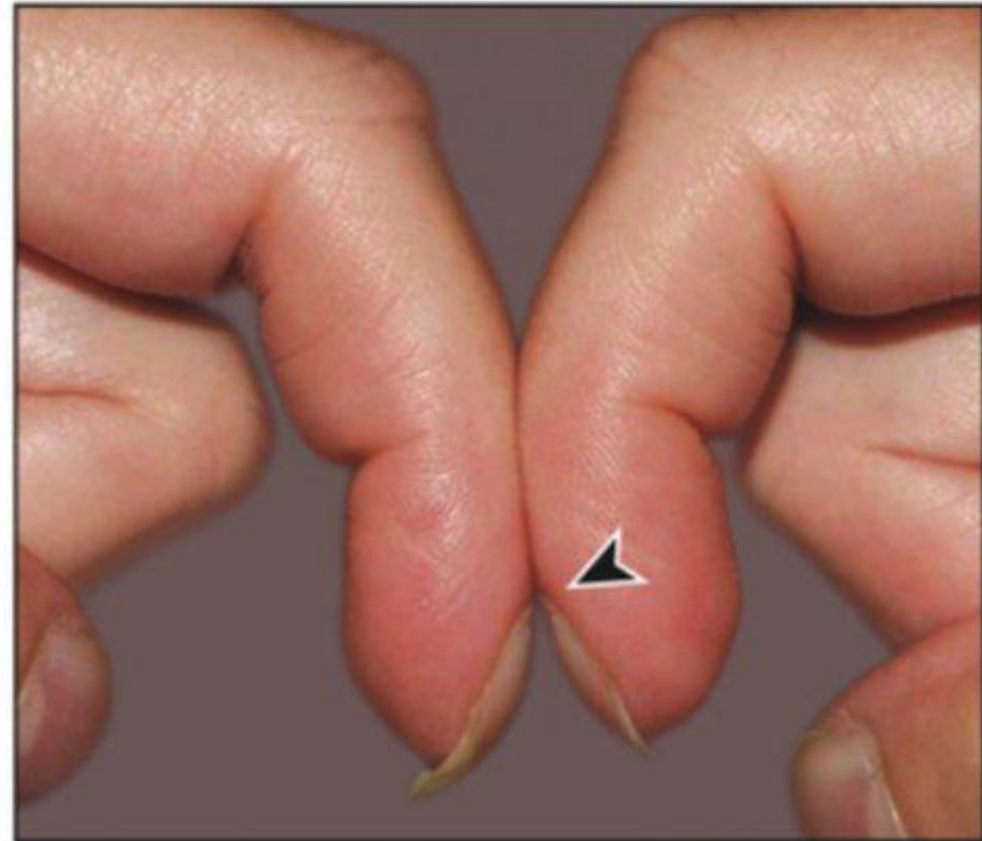
NAIL CLUBBING- TEST

Schamroth sign

Normal



Clubbed



NAIL CLUBBING





NAIL CLUBBING ↔ DRUMSTICKS



NAIL CLUBBING



NAIL CLUBBING



NAIL CLUBBING



NAIL CLUBBING- COMMON CAUSE



1. **Congenital cyanotic heart defects**
2. Any heart disease leading to **chronic hypoxia**
3. **Infective endocarditis**
4. **Pulmonary conditions** (eg CF, pulmonary hypertension)
 - **Lung cancer** mainly non-small-cell (54% of all cases), not seen frequently in small-cell lung cancer (< 5% of cases)
 - **Interstitial lung disease** most commonly idiopathic pulmonary fibrosis
 - **Complicated tuberculosis**
 - Suppurative lung disease: lung abscess, empyema, bronchiectasis, **cystic fibrosis**
 - **Mesothelioma** of the pleura
 - **Arteriovenous fistula** or malformation
 - **Sarcoidosis**

NAIL CLUBBING- COMMON CAUSE

5. **Hereditary**

6. **GI disease** (Crohn's disease, ulcerative colitis, cirrhosis, especially in primary biliary cirrhosis)

7. **Idiopathic**

FINAL TEST! REMEMBER!



HEART FAILURE

HEART FAILURE IN ADULTS

New York Heart Association (NYHA) Functional Classification

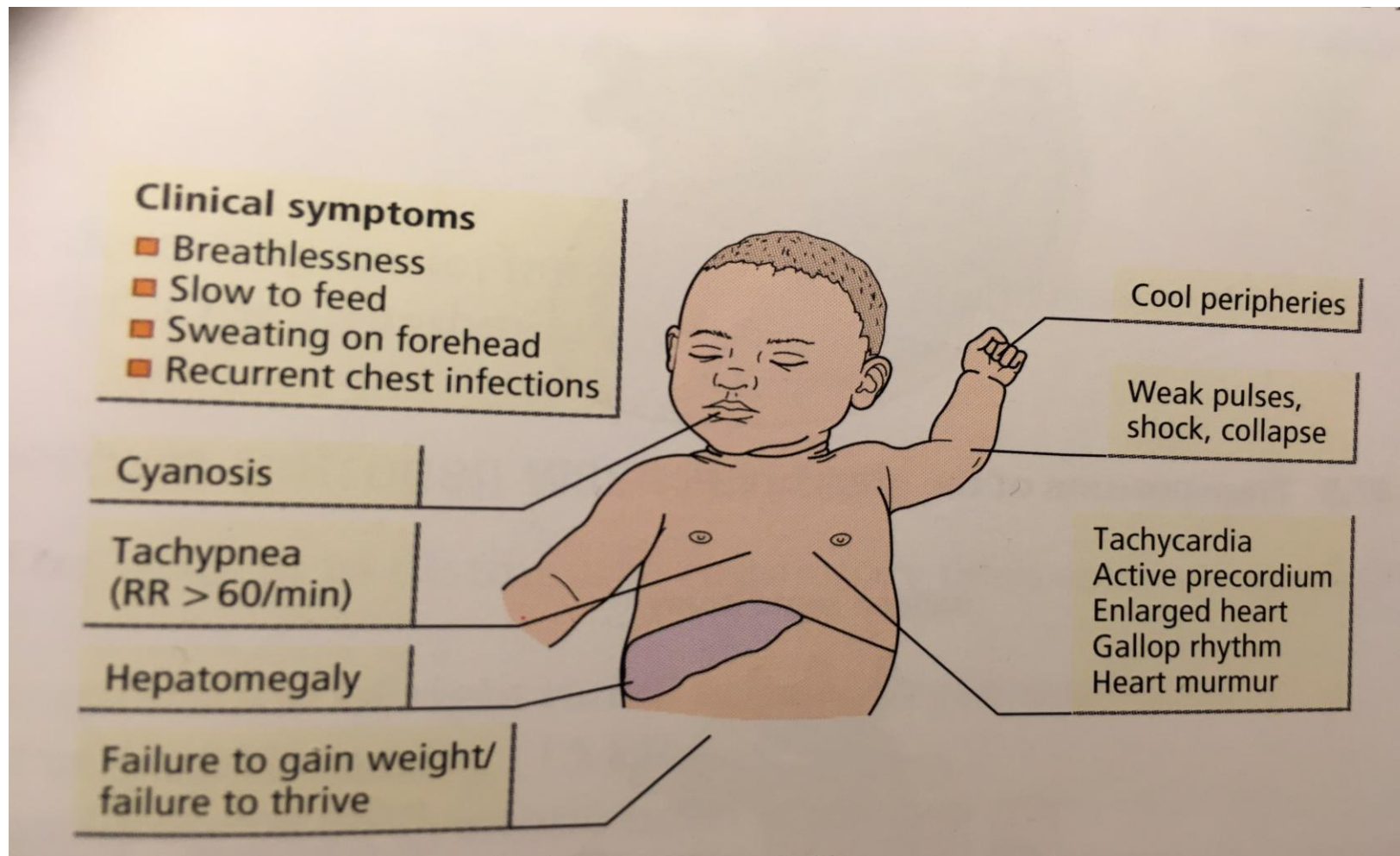
Classifying the extent of heart failure

NYHA Class	Symptoms
I	Cardiac disease, but no symptoms and no limitation in ordinary physical activity, e.g. no shortness of breath when walking, climbing stairs etc.
II	Mild symptoms (mild shortness of breath and/or angina) and slight limitation during ordinary activity.
III	Marked limitation in activity due to symptoms, even during less-than-ordinary activity, e.g. walking short distances (20–100 m). Comfortable only at rest.
IV	Severe limitations. Experiences symptoms even while <i>at rest</i>

Heart failure in children

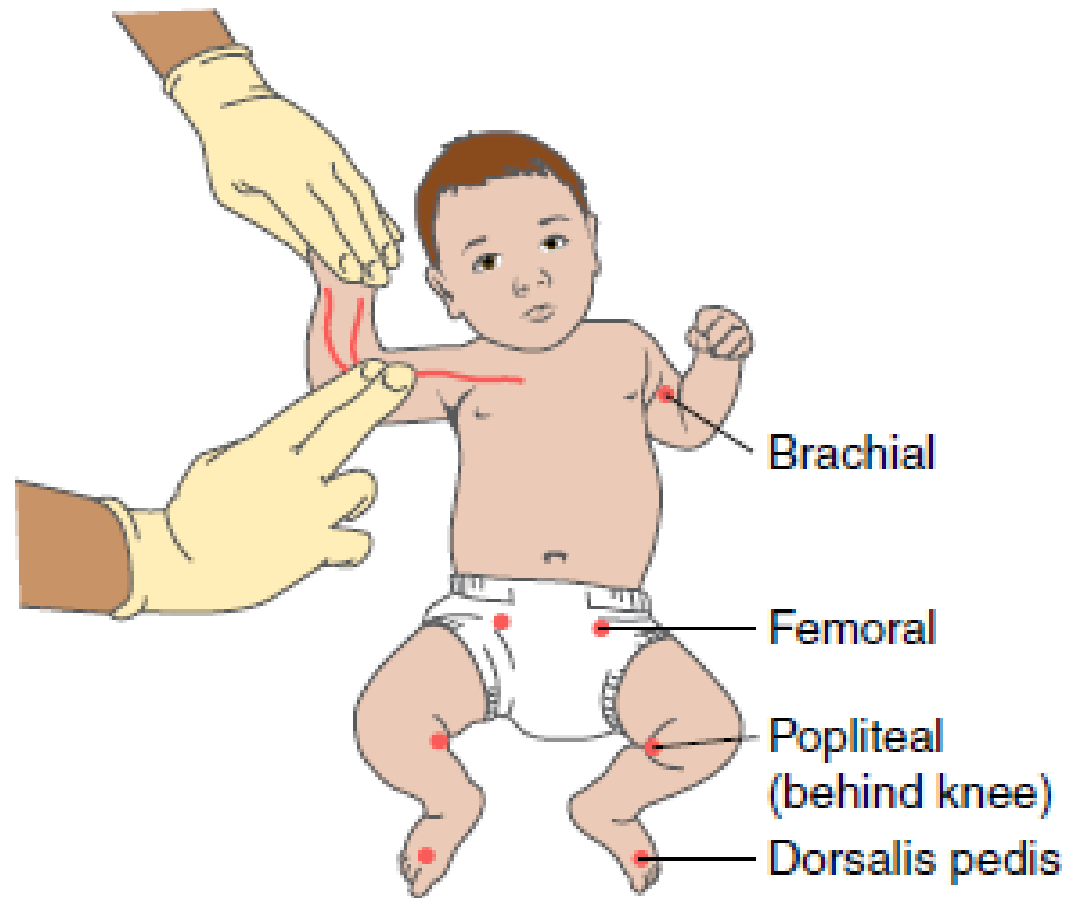
- May be manifested by symptoms of **poor tissue perfusion alone** (eg. fatigue, poor exercise tolerance *infants !, confusion) or/and
- by symptoms of **congestion of circulation** (eg. SOB, pleural effusion, pulmonary/peripheral oedema, hepatomegaly) without evoking compensatory mechanisms
- **Underlying pathophysiology mechanisms** leading to HF include
 - increased afterload (pressure work) eg. valves stenosis
 - increased preload (volume work) eg. shunts
 - myocardial abnormalities (eg. Cardiomyopathies)
 - tachyarrhythmias

HEART FAILURE IN CHILDREN





Checking the pulse in children

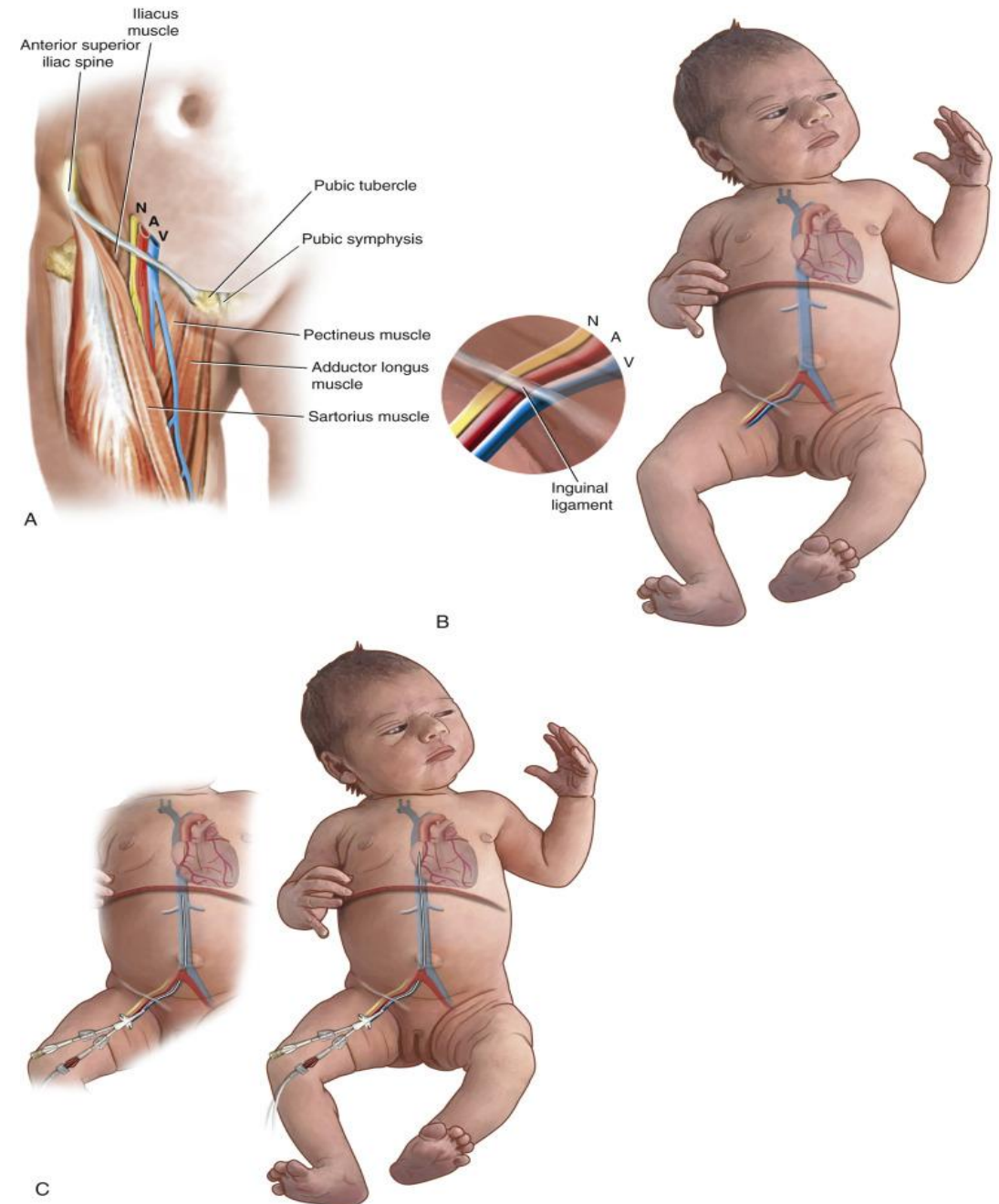


Pulse - Brachial

- Used for infants and small children
- Place fingertips of first 2 or middle 3 fingers over the brachial pulse area
 - Inside of the elbow
- Lightly press your fingertip on the pulse area



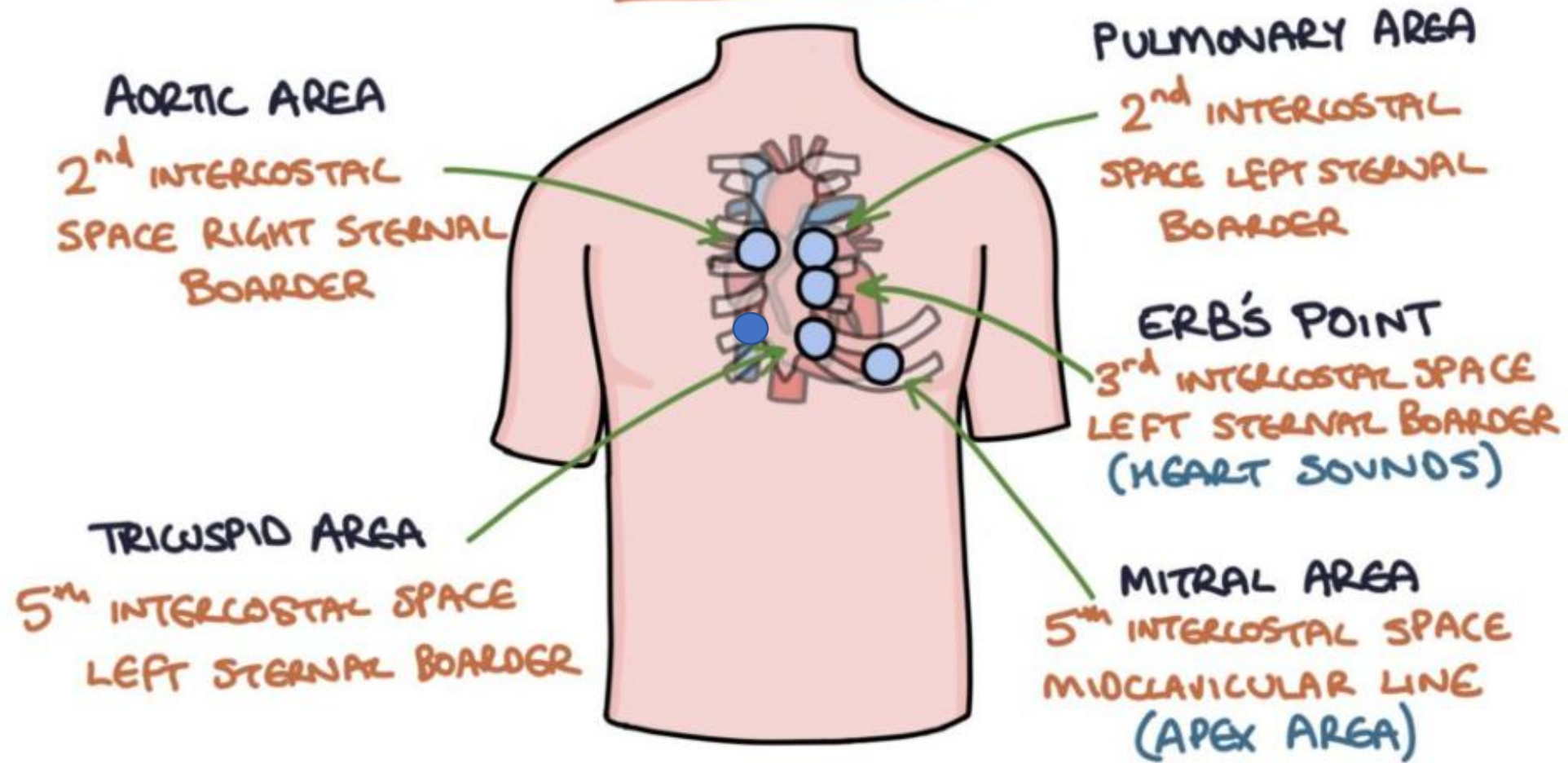
Checking the pulse in neonates



Thank you for your attention!

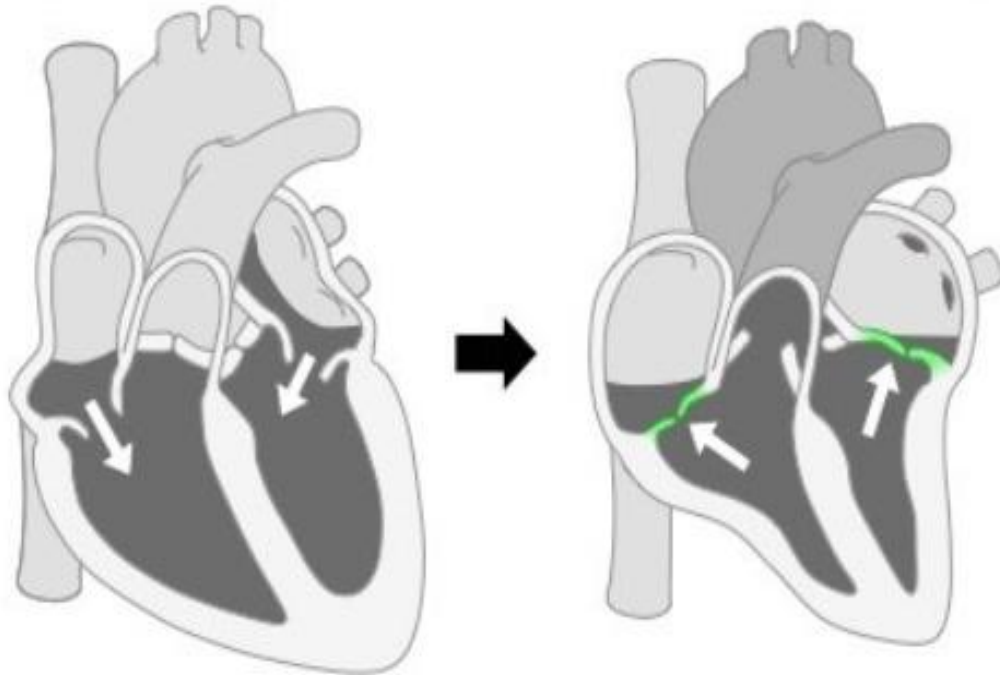


AUSCULTATION



Heart Valves and Heart Sounds

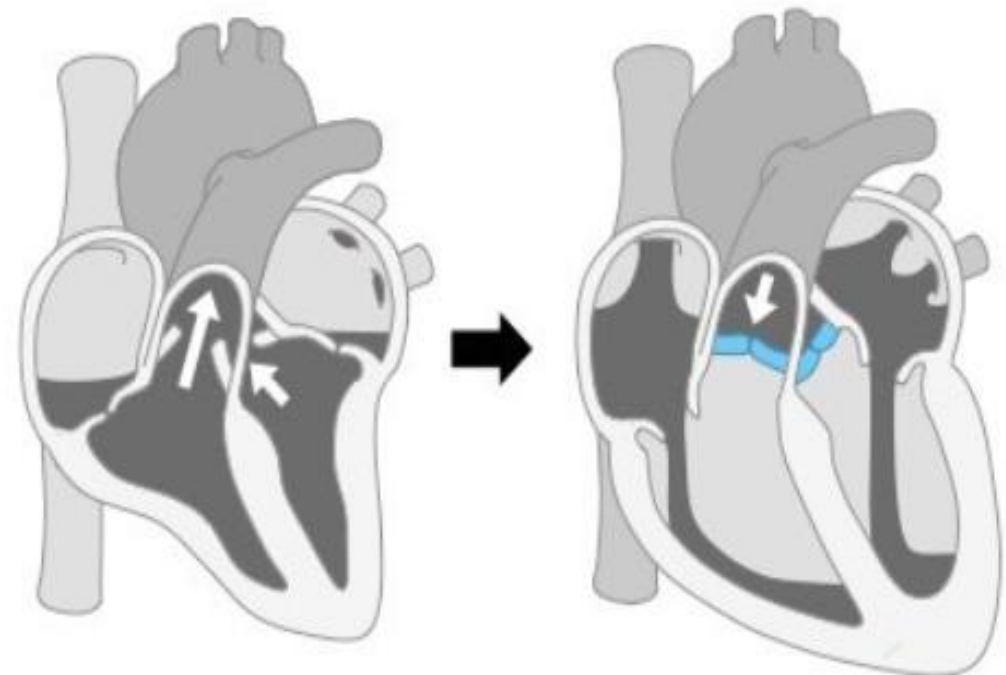
First Heart Sound ('Lubb')
Closure of the *atrioventricular valves*



Atrial Systole

Ventricular Systole

Second Heart Sound ('Dupp')
Closure of the *semilunar valves*



Ventricular Systole

Ventricular Diastole

Systolic Murmurs

Early systolic

Mitral

Acute MR

VSD

Muscular

Nonrestrictive with pulmonary hypertension

Tricuspid

TR with normal pulmonary artery pressure

Mid-systolic

Aortic

Obstructive

Supravalvular—supravalvular aortic stenosis, coarctation of the aorta

Valvular—AS and aortic sclerosis

Subvalvular—discrete, tunnel or HOCM

Increased flow, hyperkinetic states, AR, complete heart block

Dilation of ascending aorta, atheroma, aortitis

Pulmonary

Obstructive

Supravalvular—pulmonary artery stenosis

Valvular—pulmonic valve stenosis

Subvalvular—infundibular stenosis (dynamic)

Increased flow, hyperkinetic states, left-to-right shunt (e.g., ASD)

Dilation of pulmonary artery

Late systolic

Mitral

MVP, acute myocardial ischemia

Tricuspid

TVP

Holosystolic

Atrioventricular valve regurgitation (MR, TR)

Left-to-right shunt at ventricular level (VSD)

Early Diastolic Murmurs

Aortic regurgitation

Valvular: congenital (bicuspid valve), rheumatic deformity, endocarditis, prolapse, trauma, post-valvulotomy

Dilation of valve ring: aortic dissection, annulo-aortic ectasia, cystic medial degeneration, hypertension, ankylosing spondylitis

Widening of commissures: syphilis

Pulmonic regurgitation

Valvular: post-valvulotomy, endocarditis, rheumatic fever, carcinoid

Dilation of valve ring: pulmonary hypertension; Marfan syndrome

Congenital: isolated or associated with tetralogy of Fallot, VSD, pulmonic stenosis

Mid-Diastolic Murmurs

Mitral

Mitral stenosis

Carey-Coombs murmur (mid-diastolic apical murmur in acute rheumatic fever)

Increased flow across nonstenotic mitral valve (e.g., MR, VSD, PDA, high-output states, and complete heart block)

Tricuspid

Tricuspid stenosis

Increased flow across nonstenotic tricuspid valve (e.g., TR, ASD, and anomalous pulmonary venous return)

Left and right atrial tumors (myxoma)

Severe AR (Austin Flint murmur)

Continuous Murmurs

Patent ductus arteriosus

Coronary AV fistula

Ruptured sinus of Valsalva aneurysm

Aortic septal defect

Cervical venous hum

Anomalous left coronary artery

Proximal coronary artery stenosis

Mammary souffle of pregnancy

Pulmonary artery branch stenosis

Bronchial collateral circulation

Small (restrictive) ASD with MS

Intercostal AV fistula

Abbreviations: AR, aortic regurgitation; AS, aortic stenosis; ASD, atrial septal defect; AV, arteriovenous; HOCM, hypertrophic obstructive cardiomyopathy; MR, mitral regurgitation; MS, mitral stenosis; MVP, mitral valve prolapse; PDA, patent ductus arteriosus; TR, tricuspid regurgitation; TVP, tricuspid valve prolapse; VSD, ventricular septal defect.

Source: E Braunwald, JK Perloff, in D Zipes et al (eds): *Braunwald's Heart Disease*, 7th ed. Philadelphia, Elsevier, 2005; PJ Norton, RA O'Rourke, in E Braunwald, L Goldman (eds): *Primary Cardiology*, 2nd ed. Philadelphia, Elsevier, 2003.

Practical approach to physical examination of circulation

- **Airway**

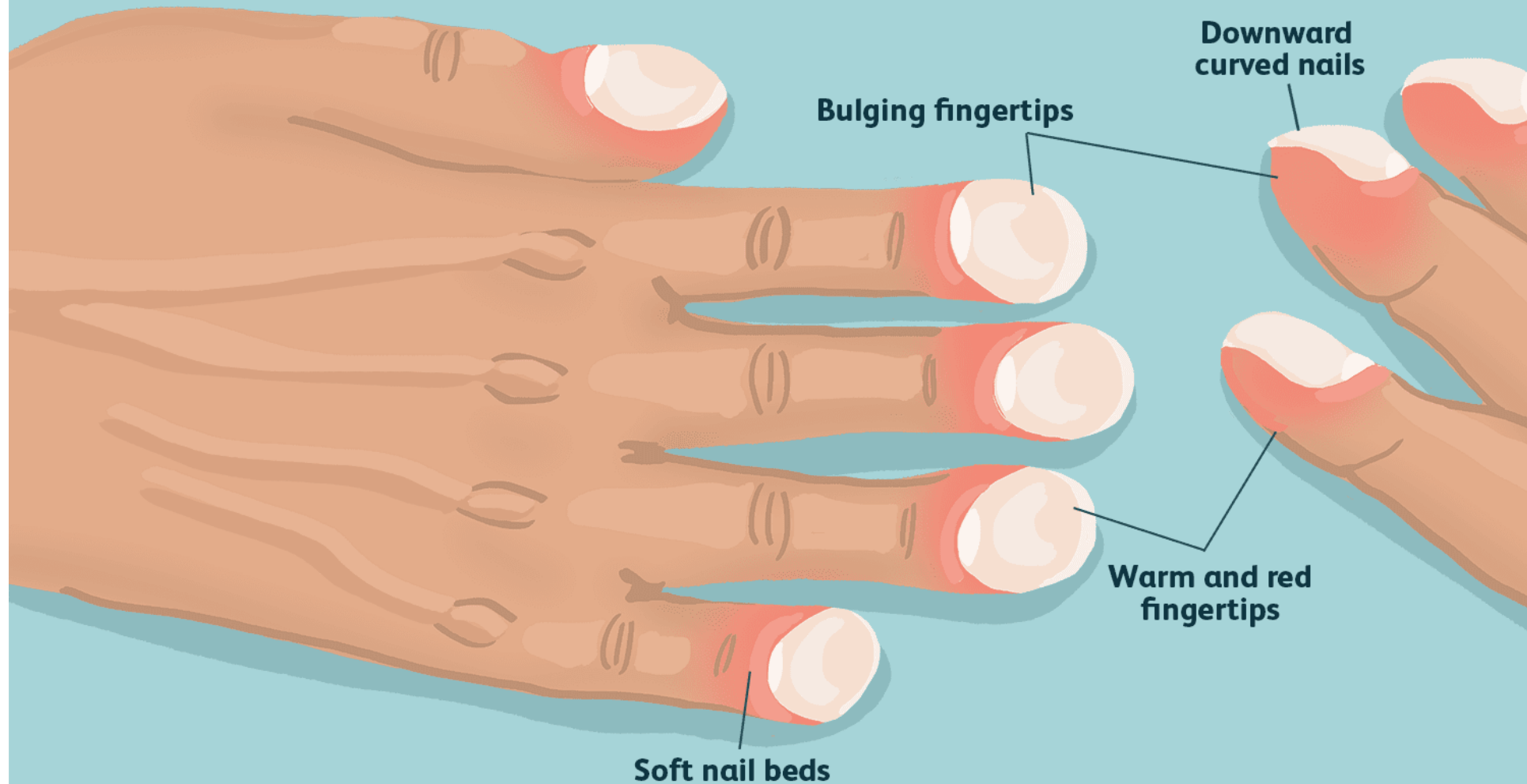
- **Breathing**

- Respiratory Rate
- Tidal Volume
- Work of extra muscles
- Oxygenation

- **Circulation**

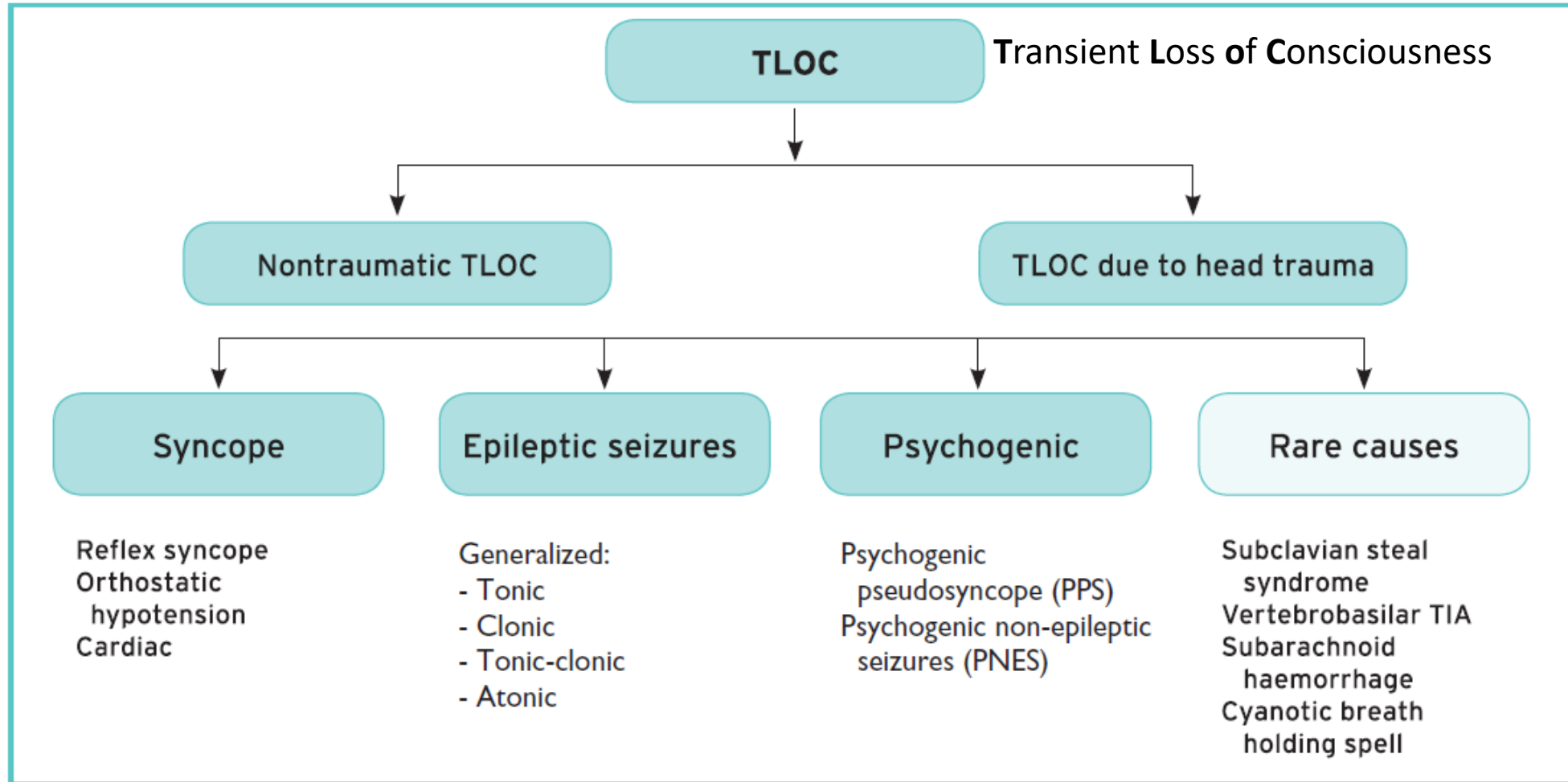
- HR
- BP
- PULSE (PRESENT? AMPLITUDE?)
- PERFUSION (CRT, SKIN- COLOUR- CYANOSIS?, WARM? / COLD?, SWEATY?)
- PRELOAD (JUGULAR VEINS, HEPATOMEGALY, CRACKLES IN LUNGS)
- DIURESIS (RENAL PERFUSION)

Clubbing: Common Symptoms



2018 ESC Guidelines for the diagnosis and management of syncope

Syncope- history taking



Syncope- types

1. Reflex

Vasovagal:

- orthostatic : standing, emotional, pain

2. Syncope due to orthostatic hypotension & vasodepression

- a) **Drug-induced** OH (most common cause of OH): e.g. vasodilators, diuretics, phenothiazine, antidepressants
- b) **Volume** depletion: haemorrhage, diarrhoea, vomiting, etc.
- c) **Primary** autonomic failure (neurogenic): pure autonomic failure, Parkinson's disease, dementia with Lewy bodies
- d) **Secondary autonomic failure** (neurogenic): diabetes, amyloidosis, spinal cord injuries

3. Cardiac

Arrhythmia as primary cause:

- a) Bradycardia:
 - sinus node dysfunction (including bradycardia/tachycardia syndrome)
 - atrioventricular conduction system disease
 - b) Tachycardia: supraventricular or ventricular
- Inherited familial : LQTS, Brugada syndrome

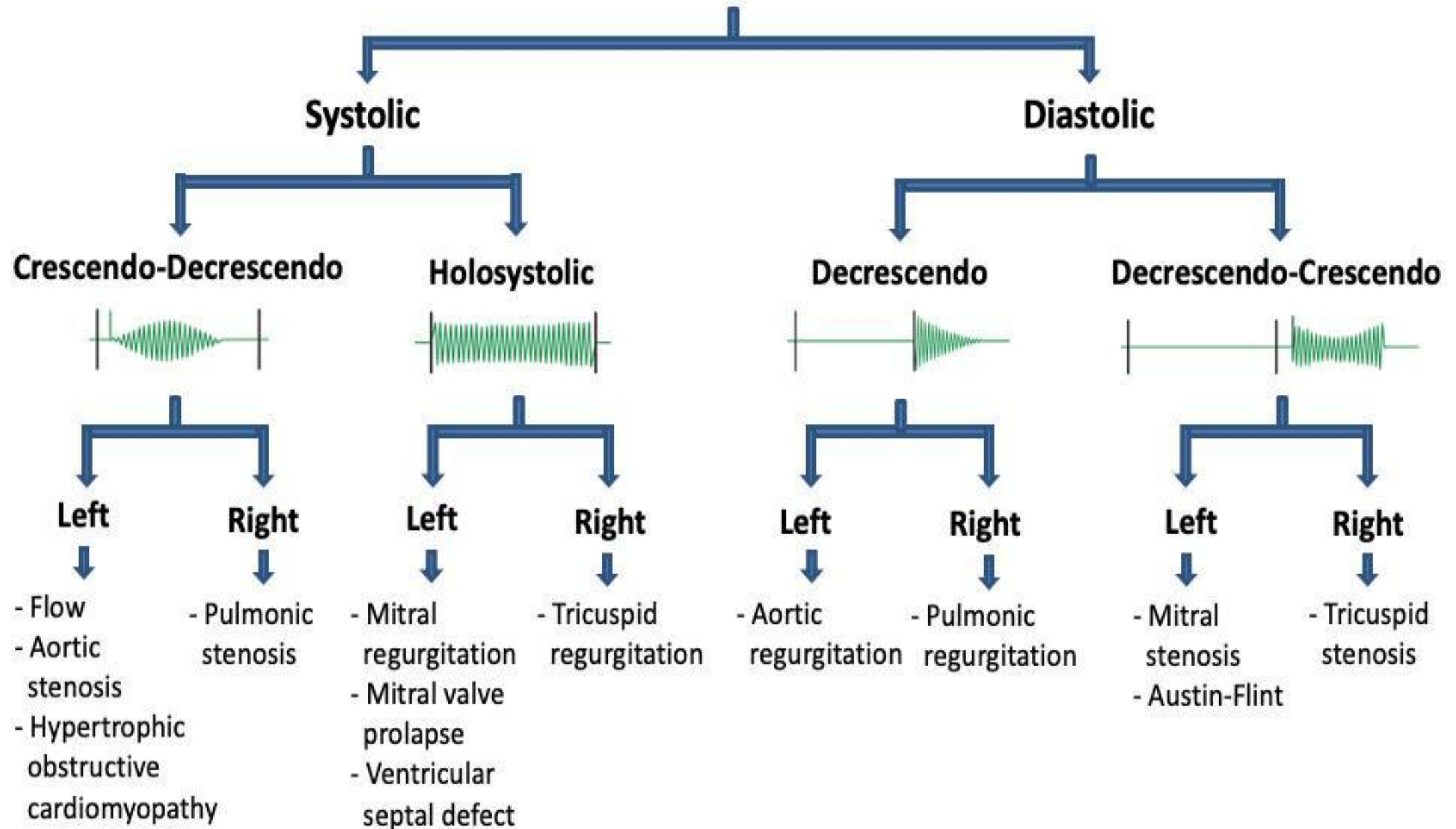
Structural cardiac defects:

Aortic stenosis, acute myocardial infarction/ischaemia, hypertrophic cardiomyopathy, cardiac masses (atrial myxoma, tumours, etc.), pericardial disease/tamponade, congenital anomalies of coronary arteries, prosthetic valve dysfunction

Cardiopulmonary and great vessels: pulmonary embolus, acute aortic dissection, pulmonary hypertension



Heart Murmurs Adults





ESC

European Society
of Cardiology

European Heart Journal (2018) 39, 1883–1948

doi:10.1093/eurheartj/ehy037

ESC GUIDELINES

Definition

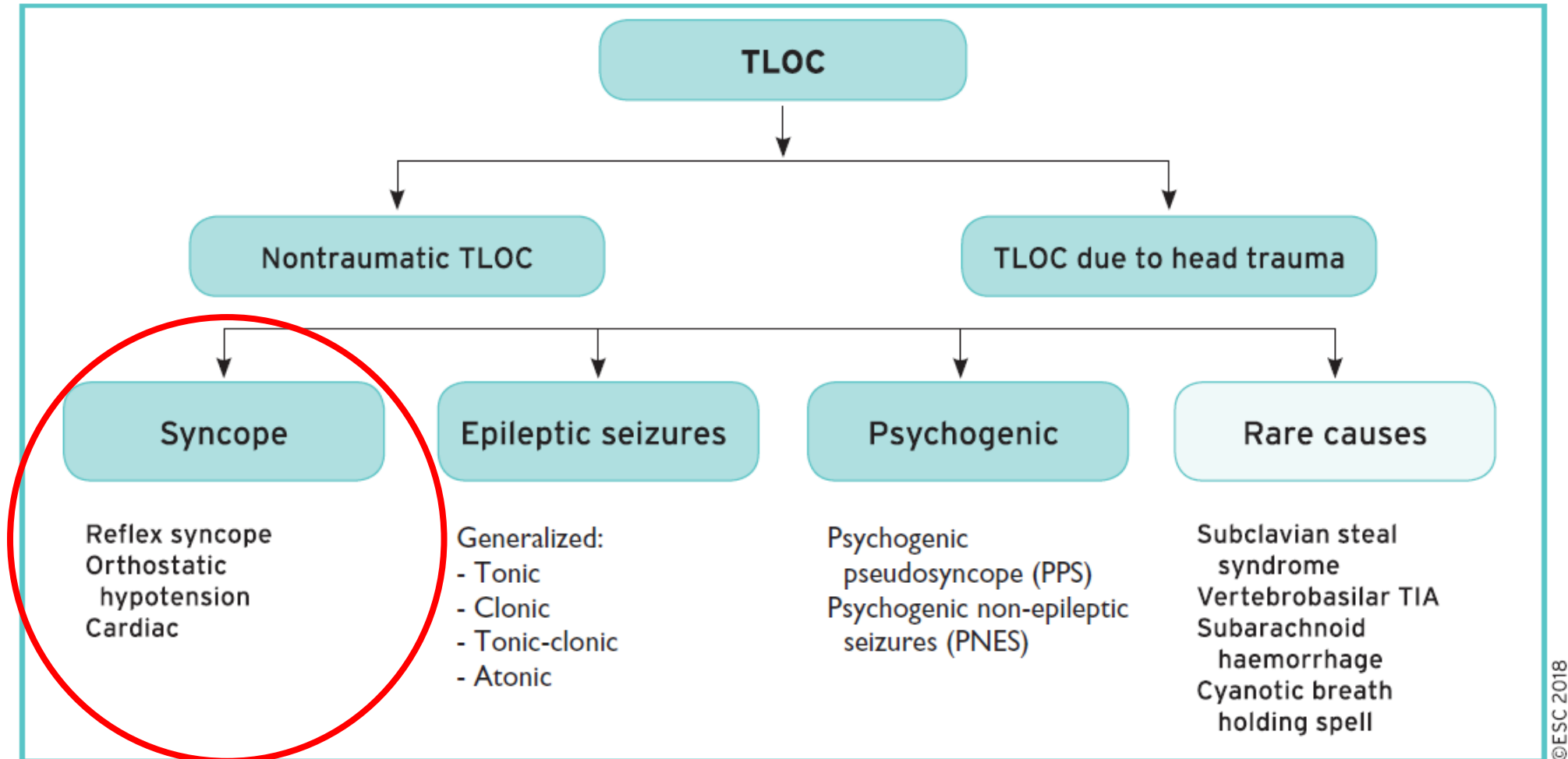
2018 ESC Guidelines for the diagnosis and management of syncope

Syncope

- is defined as transient loss of consciousness (TLOS)
due to cerebral hypoperfusion,
characterized by a rapid onset, short duration, and spontaneous & complete recovery
- Benign or life-threatening condition (depending on a reason)
- **Taking history** + examination + additional tests

2018 ESC Guidelines for the diagnosis and management of syncope

Syncope- taking history



Syncope- symptoms

Vasovagal

- There are typical symptoms (prodromal symptoms) before the loss of consciousness such as: **lightheadedness, sweating, pale skin, blurred vision, nausea, vomiting or feeling warm**
- Benign
- Most common

Cardiac

- No prodromal symptoms
- During or after physical activity
- (+) Family history of sudden deaths
- Chest pain
- Life-threatening- send to paediatric cardiologist