Practical 2 – Cardiovascular Physiology

2.1. The influence of ions and mediators on heart. Effect of vagal stimulation on heart activity and vagal escape.
2.2. Cardiac cycle.
2.3. Peripheral manifestations of cardiac activity: Heart sounds. The phonocardiogram recording and analysis.

2.1. **The influence of ions and mediators on the** heart. Effect of vagal stimulation on heart activity and vagal escape

High **Calcium** ions (hypercalcemia)

- low concentration → ↑ inotropism, ↑ chronotropism
- high concentration → heart stops in systole (rigor calcis)
- ECG: rounded T wave, PR interval longer, QT interval shorter

Hypocalcemia

- leads to ↓ inotropism, ↓ chronotropism
- ECG: longer ST, tall and sharp T wave

High **Potassium** ions (hyperpotassemia)

- low concentration → ↓ inotropism, ↓ chronotropism
- high concentration → heart stops in diastole
- ECG: tall and sharp T wave, large QRS complex (branch block aspect), Ventricular fibrillation

Hypopotassemia

- ST segment depression, longer QT, flat or inverted T wave, U wave

Hydrogen (**lower pH**)

- high lactic acid
- same action as hyperpotassemia
- large concentrations make irreversible damage to the heart

Parasympathetic and sympathetic nervous systems innervate the heart. Sympathetic fibers are distributed both to atriums and ventricles, while parasympathetic fibers are supplying the atriums, SA node and AV node.

Stimulation of the sympathetic nervous system (adrenaline, noradrenaline release) increases the rate and force of contraction of the heart. Stimulation of the parasympathetic nervous system (vagal nerves, through acetylcholine) decreases the depolarization rhythm of the sinoatrial node and slows transmission of excitation through the atrio-ventricular node.

Adrenalin - sympathetic system mediator acting on α and β receptors

- ↑ inotropism, ↑ chronotropism

Acetylcholine - parasympathetic system mediator acting on muscarinic receptors

- Distribution through vagus nerve: right branch – sino-atrial node, left branch – AV node
- Sino-atrial node - ↓ heart rate, AV node - ↓ conductibility

If vagal stimulation is excessive, the heart stops beating. After a short time (few seconds), the ventricles begin to beat again, at a lower frequency. This phenomenon is called ***vagal escape*** and may be the result of an idioventricular rhythm, acetylcholine depletion or sympathetic stimulation.

Vagal stimulation effect on the frog heart and vagal escape can be observed on a Marey cardiograph, by stimulating the nerve with a DC current using an electrode.

In clinics, parasympathetic stimulation and decrease in pulse rate is obtained through the different vagal maneuvers:

- *Valsalva maneuver* - moderately forceful attempted exhalation against a closed airway/ glottis. It leads to increased air pressure in the lungs that stimulate the parasympathetic receptors; may be used to stop episodes of supraventricular tachycardia.
- *Oculo-cardiac/oculo-depressor reflex* (Dagnini–Aschner reflex) - grip applied to extraocular muscles and/or compression of the eyeball through the eyelids for 15-20 seconds can decrease the heart rate. This reflex is mediated by nerve connections between the ophthalmic branch of the trigeminal cranial nerve via the ciliary ganglion, and the vagus nerve. Used in the clinic to stop the paroxistic atrial tachycardia episodes or for differential diagnostics of arrhythmias.
- *Carotic sinus massage* (Czermak-Hering test) is performed at the patient's bedside by applying moderate pressure with the fingers, repeatedly massaging the left or the right carotid arteries, to determine bradycardia and hypotension, to diagnose carotid sinus syncope and to differentiate supraventricular tachycardia from ventricular tachycardia.

- *Viscero–vagal reflex* (Goltz reflex) - a mechanical stimulation of the vagus receptors from the gut / internal organs, determines the decrease of heart rate or even heart stop.

2.2. Cardiac cycle

Cardiac cycle is the succession of one ventricular systole and diastole. Each time the pace-maker of the heart produces an action potential, a wave of electrical depolarization spreads throughout the myocardium, initiating contraction (**systole**) followed by a series of mechanical events that causes blood to flow in and out of the heart and ending with relaxation (**diastole**). In the same time frame (duration of one cardiac cycle), the atria also complete a contraction (atrial systole) and relaxation (atrial diastole) cycle. Note that the atria and ventricles do not contract and relax simultaneously.

The **duration of a cardiac cycle** is dependent on heart rate (beats/min). The duration of a cardiac cycle = 60 seconds/heart rate.
For a heart rate of 75 beats/minute the duration of a cardiac cycle is 0.8s (0.3s for the ventricular systole and 0.5s for the ventricular diastole; 0.1s for the atrial systole and 0.7s for the atrial diastole).

Phases of the cardiac cycle - relative to the ventricular activity: ventricular systole/contraction and ventricular diastole/relaxation.
For practical reasons the cardiac cycle is divided into four phases:

1) The ventricular filling / inflow phase: ventricles relaxed, atrioventricular valves open, semilunar valves closed
   - Rapid ventricular filling
   - Decreased ventricular filling. Diastasis
   - Atrial contraction- supplementary ventricular filling
2) The isovolumetric contraction: ventricles contracting, both types of valves are closed, intraventricular pressure increases
3) The ejection / outflow phase: ventricles contracting, semilunar valves open, then close, atrioventricular valves remain closed
   - Rapid ejection (fast muscle shortening)
   - Decreased ventricular ejection (slow muscle shortening)
4) The isovolumetric relaxation: ventricles relaxed, both types valves closed

All the valvular events (opening /closing) are passive events brought on by pressure gradients. There are clinical ways to distinguish the systole and diastole from heart sounds.

Heart period	Diastole	Systole			Diastole	Systole			Diastole	Systole			Diastole
Phase of cardiac cycle	1	2	3	4	1	2	3	4	1	2	3	4	1
AV valves	Open	Closed			Open	Closed			Open	Closed			Open
Semilunar valves	Closed	Open			Closed	Open			Closed	Open			Closed

Phase of cardiac cycle:

1 = VFP (ventricular filling)

2 = ICP (ventricular volume unchanged)

3 = VEP (ventricular ejection)

4 = IRP (ventricular volume unchanged)

2.3. Peripheral manifestations of cardiac activity: Heart sounds. The phonocardiogram recording and analysis.

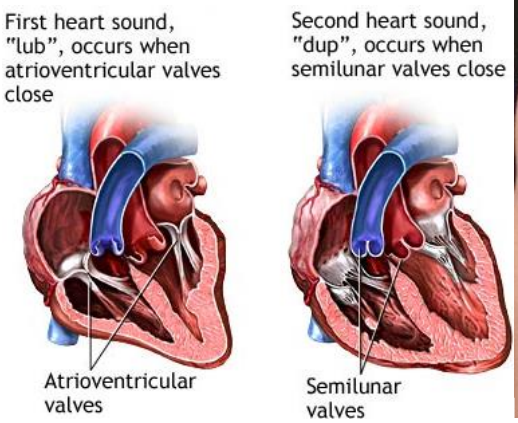
The **heart sounds** are produced by a summed series of mechanical events, as follows:

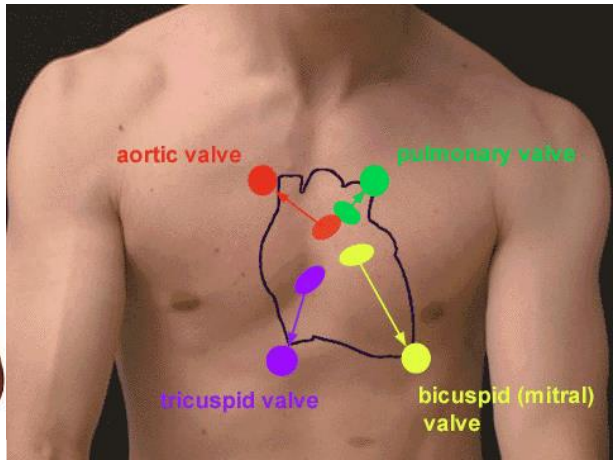
a. Valvular events = vibrations caused mostly by the closing of the heart valves; opening of the valves produces a vibration of lesser intensity.
b. Muscular events = vibrations of the myocardium during contraction.
c. Vascular events = vibration produced by the sudden distension of the arterial walls during ejection.
d. Vibrations caused by the acceleration/deceleration of the blood flow.

N.B. Not every sound is composed of all these components, the most prominent component being the closure of the valves.

4 areas of auscultation (where the sounds coming from a specific valve are best transmitted).

a. The primary aortic area: second right interspace, adjacent to the sternum; there is a secondary aortic auscultation area in the third left interspace, also adjacent to the sternum (known as the Erb's point).
b. The pulmonary area: second left interspace
c. The tricuspid area: the fourth and fifth interspaces, adjacent to the left sternal border or on the xyfoid process.
d. The mitral area at the cardiac apex in the fifth left interspace, on the medio-clavicular line.





The phonocardiogram is recorded using microphones placed on the thorax in the main auscultatory areas; these microphones filtrate the sounds in between 25 and 200 Hz. It is usually recorded together with the ECG.
Note that in normal ausculation only sound 1 and 2 should be heard, but on phonocardiogram we can record all four sounds. Sounds 3 and 4 can sometimes be palpated.

There are two types of sounds: high frequency sounds associated with closing and opening of the valves and low frequency sounds related to early and late ventricular filling events.
High-frequency sounds - better heard with the diaphragm of the stethoscope
Related to closing and opening of the **AV valves**
a. Mitral (M1) and tricuspid (T1) closing sounds (**S1**)
b. Opening snaps-mitral and tricuspid (diastolic sounds)
c. Nonejection sounds –click caused by mitral valve prolapse (mid-late systolic sound)
Related to closing and opening of the **semilunar valves**
d. Aortic (A2) and pulmonic (P2) closure sounds (**S2**)
e. Early valvular ejection sounds or clicks- aortic and pulmonic (systolic sounds)
Low-frequency sounds- better heard with the bell of the stethoscope
f. Physiologic third heart sound (**S3**), or in pathological conditions protodiastolic gallop
g. Fourth heart sound (**S4**)

The first heart sound (S1) – systolic sound:
- appears at 0.02 – 0.04s after the QRS complex, and lasts 0.12-0.15s
- the "lub", frequency of 30-40Hz
- produced, in this order, by: closing of the mitral valve, closing of the tricuspid valve, opening of the pulmonary valve, opening of the aortic valve.
- Best heard in the mitral and tricuspid auscultatory areas
- Loud S1 in:
  - Mitral stenosis (calcified but mobile valve)
  - Tachycardia –short diastole
  - Short PR interval –short interval between atrial and ventricular contraction
  - Complete AV block "canon sound" when the atria contract against a closed atrioventricular valve
- Reduced intensity of S1 in:
  - Thick chest wall, pericardial effusion, reduced contractility of the heart (all sounds have reduced intensity)
  - Long PR interval
  - Mitral regurgitation-imperfect closure
  - Mitral stenosis (immobile valve)
- Wide Splitting of S1- right bundle branch block

The second heart sound (S2) – diastolic sound
- appears in the terminal period of the T wave, lasts 0.08 – 0.12s
- the "dub", frequency of 50-70 Hz
- produced, in this order, by: closing of the aortic valve, closing of the pulmonic valve, opening of the tricuspid valve, opening of the mitral valve.
- Best heard in the 2nd left interspace near the sternal border

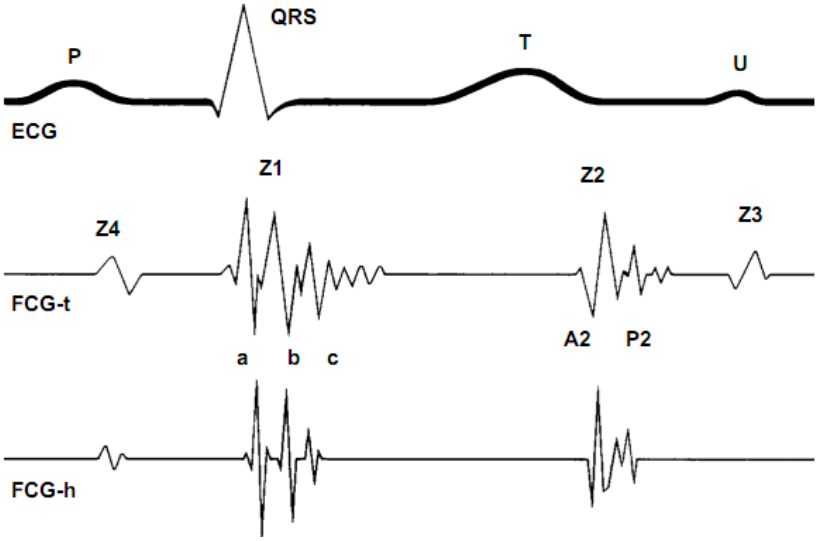
- Physiologic splitting that varies with respiration (wider splitting with inspiration).
- Pathological splitting:
  - Large variable splitting (A2 P2) with right bundle branch block (late systole of the right ventricle)
  - Fixed splitting- atrial septal deffect (ASD)
  - Paradoxical splitting (P2 A2)- heard in expiration and disappears in inspiration- severe aortic stenosis (long left ventricle ejection); left bundle branch block (late left ventricular systole); severe pulmonary hypertension (early closure of pulmonary valve)
- Loud S2:
  - Systemic hypertension
  - Fibrous aortic valves
  - Pulmonary hypertension (louder P2)
- Reduced intensity of S2:
  - Aortic stenosis
  - Pulmonary stenosis

The third heart sound (S3) - ***protodiastolic sound*****:**

- early diastolic, lasts 0.02-0.04 s, low frequency
- it is normal in children and individuals with a thin thoracic wall
- it is produced by the blood flow that hits the ventricular wall during the rapid filling phase

The fourth heart sound (S4) – pre-systolic sound:

- late diastolic-just before S1
- lasts 0.04-0.10s, low frequency
- It is caused by the blood flow that hits the ventricular wall during the atrial systole, causing it to vibrate.
- It is physiological only in small children, if heard otherwise it is a sign of reduced ventricular compliance.
- Is heard as a *presystolic (atrial) gallop*



Heart murmurs are sounds that last longer than 150 ms, in contrast to heart sounds that are shorter. They are caused by turbulent blood flow through the heart or great vessels.

Types of heart murmurs:

- Systolic murmurs
  - Ejection murmurs- midsystolic
    - Aortic stenosis
    - Pulmonic stenosis
  - Regurgitation murmur- holosystolic
    - Mitral regurgitation
    - Tricuspid regurgitation
  - Other systolic murmurs
    - Vascular murmurs
    - Ventricular septal defect
- Diastolic murmurs
    - Mitral stenosis
    - Tricuspid stenosis
    - Aortic regurgitation
    - Pulmonary regurgitation
- Continuous murmurs –patent ductus arteriosus