Physical examination of the heart

3rd Medical Department
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2012, 3rd year
- Inspection
- Palpation
- percussion
- auscultation!
- (echocardiography!!)

The heart is a generator of vibrations from "0" to about 2000 Hz
Inspection of the face:

„For by his face straight shall you know his heart”

Shakespeare, King Richard III
Act III, scene IV-53
Inspection of the precordium observe from the foot, patients head and trunk elevated 30° chest abnormalities:
Inspection of the chest

chest abnormalities:
barrel shaped – emphysema
pectus excavatum-”caved-in”sternum
carinatum- bulging of the sternum
excavatum, funnel
(a Latin term meaning hollowed chest)

10/15/2012

carinatum, pigeon
a protrusion of the sternum and ribs
Precordial pulsations due to the heartbeat

Areas of inspection and palpation

- The sternoclavicular area
- The aortic area
- The pulmonic area
- The right ventricular (left parasternal) area
- The apical (left ventricular area)
- The epigastric area
- Ectopic (variable-location) areas
Palpation

* Movements felt on the chest surface, frequency below 15-20 Hz
  
  - **Cardiac apex impulse** - 5th interspace, midclav
  
  - **Antihorral rotation of the heart in early systole** and tapping of the apex on the chest wall
Characteristics of the impulse:

- amplitude,
- duration(sustained),
- direction(e.g. outward during systole abnormal),
- time,
- quickness,
- location,
- distribution
Normal findings (figure)

Apex impulse (left ventricular thrust) 
the point of maximal outward movement

- minimal amplitude, brief in duration, outward in early systole, midclav.line 5th interspace, small area: 2-3 cm diameter
- **Parasternal retraction**: the lower parasternal area moves inward during systole, due to volume change during ejection (sustained)
left ventricular impulse
This is from an old book
**Abnormal findings**

- *Left ventricular hypertrophy*: exaggeration of LV thrust-amplitude, duration, dysplacemnt
- **pressure load**: aortic stenosis
- **volume load**: aortic insuff-amplitude+, sustained, mid and late syst. retraction
Abnormal findings

- Right ventricular hypertrophy: sustained systolic lift lower parasternal region ("V2,"), most often mitral stenosis
Anterior wall of the right ventricle
Ischaemic heart disease:
outward paradox bulge of LV during systole, sustained, wide area,
♦ infarction and angina-dyskinesis, akinesis
valvular heart disease

- mitral stenosis: parasternal lift due to elevated pulmonary pressure
- mitral incompetence: the thrust increased in amplitude occasionally second systolic peak - LV volume overload
HOCM (IHSS) hypertrophic obstructive cardiomyopathy: double outward movement - a rocking sensation

- Palpable heart sounds: hyperdynamic state - accentuated S1
- Thrills: vibrations from loud murmurs - low frequency (a cot’s purr)

fremissement cattaire

кошачье мурлыканье
Percussion of the heart

is of limited value, on the surface of the chest from resonance to dullness.

In old textbooks you find:

relative and absolute dullness.

left border within the left midclavicular line compare with the impulse location
Auenbrugger, Leopold

„Inventum novum ex percussione thoracis …“ 1761

He found that with light percussion of the chest, he could make a guess on the character and position of the organs in the chest.

He learned in his father’s wine cellar that by percussion of the barrels one can determine the level of wine.

He applied this to the heart!
Percussion of the heart

Long tradition, by now obsolete

Anemburger (1761)

Corvisart (1808)

Diary (1828)

plessimeter vs. a heater
absolute dullness relative dullness

Auenbugger 1761
Corvisart 1808
„direct” percussion

Piory 1828
„indirect”
use of plessimeter

relative dullness absolute dullness
The outer limits of cardiac dullness

1. to the left when the diaphragm is higher
2. within the midclav. line in concentric hypertrophy
3. displacement to the right in left sided PTX or right sided atelectasis (collapse of the lung)
23. ábra. A relatív ( argued ) és abszolút ( shaded ) szívtompulat a) fiziológián, b) balra irányuló szívmegnagyobbodás, c) jobbra irányuló szívmegnagyobbodás és d) pericardialis fluidum esetén
4. situs inversus

5. historic significance: valvular diseases before the invention of X-ray
Auscultation of the heart

- First discussed in HARVEY's De motu cordis 1628
- Fetal heart sounds: Marsac 1680 - ridiculed
- Ancient Egypt ??
- Auscultation:
  - Corvisart~1810 "immediate" auscultation
  - Laennec 1816 "mediate" auscultation
  - A rolled quire of paper "stethoscope"

a young female patient
The human ear is most sensitive to vibrations between 1000-4000 Hz

heart sounds: 30-1000 Hz
diaphragm for high frequency
bell for low freq

- Quiet room,
- comfortable temperature,
- exposure of the patient to the waist
Fig. 18-2. Graph indicating the pressure threshold of audibility.
Fenyvesi

The stethoscope

- earpiece
- rubber tubing: short, thick
- bell
- diaphragm

Which is the most important component?

Rapaport-Sprague
Littmann

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Binaural stethoscope
Bartlett
1880
Fig. 18-2. The stethoscope collection of Dr. E. Grey Dimond. Sixteen monaural stethoscopes, beginning with Laennec's (1, 2), a thought-provoking collapsible model (3), versions turned out of wood (4 through 9), others of metal (11, 13 through 16)—some of which can be regulated for pocket convenience (4, 8, 13, 14)—and two unusual models (10, 12). Instrument 10 has a solid wood terminus to aid resonance, and 12 shows perhaps the earliest incorporation of a diaphragm. Stethoscopes 1, 3, 8, 10, 11, 12, 14, and 16 are from the collection of Paul Dudley White, M.D. (Courtesy of Dr. E. Grey Dimond)
Fig. 18-10. Everything is wrong!! How not to employ the technique of auscultation.
Phonocardiography:

graphic (analogue) record of the heart sounds and murmurs, synchronized with ECG.
Significance in timing and teaching.
rarely used?
Aortic stenosis
Areas of auscultation

1. aortic: primary 2nd right interspace secondary 3rd left interspace adjacent to the sternum
2. pulmonary: 2nd left interspace
3. tricuspid: 4th-5th interspace left sternal border
4. mitral: cardiac apex

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Areas of auscultation

This is orientative-systematic approach, so called "inching" is of great use. In each area listen to sounds and murmurs.
Heart sounds:

brief auditory vibrations characterized by

• intensity (loudness)
• frequency (pitch)
• quality (timbre)
Heart murmurs:
auditory vibrations more prolonged than a sound, characterized by
- timing in the cardiac cycle
- intensity
- frequency
- configuration (shape)
- duration
- direction of radiation
How to learn and practice auscultation
In each area listen to **sounds** and **murmurs**

- S1, S2: intensity, constancy, splitting
- Extra sounds: in systole, in diastole

- **Opening and closing** of the valves: high frequency
- **Filling sounds**: low frequency
The FIRST HEART SOUND, S1

- main components M1 and T1 closure
  ◆ coincident with the coaptation, but not clapping together—rather halting of the blood
  ---vibration of the "cardiohemic" system
- splitting of S1 (not audible)
- **RBBB** → M1T1
- **RV pacing** → T1M1 "reversed splitting"
- or **ectopics**
Intensity of S1:

1. integrity of the valve closure
2. mobility of the valve
3. velocity of closure: LV pressure rise in early syst
Intensity of S1 depends on the mitral component (M1) with shorter PR at the beginning of LVP rise the mitral leaflets are wide apart, M1 is delayed and coincides with a higher velocity of LV pressure rise: velocity of closure long PR-----mild soft S1 short PR---- loud S1
Hemodynamic correlates of the S1: the first high frequency component of the S1 is delayed from LV-LA pressure crossover by 30 ms (inertial flow)
mitral leaflet motion
complete AV block

Long PR interval

Short PR interval

soft S1

lauud S1
Intensity of S1

4. status of ventricular contractility
5. transmission characteristics of the thorax
S1 in pathologic conditions in mitral stenosis: loud, late M1 increased left atrial pressure

1. It delays A-V pressure crossover, it occurs at a higher dP/dt
2. Prevents "preclosure" of the valves
decreased intensity of M1
LBBB
LV dysfunction
acute aortic regurgitation-
early "preclosure" of the
mitral valve
Go to video

(ICI ausč1)
Ejection sounds

**Aortic valvular**: nonstenotic congen. bicuspid stenosis p.m. at the apex, timing: anacrotic notch of the upstroke of the aortic pressure pm.aorta

**Aortic vascular**: sclerotic aortic root

**Pulmonary valvular**: decreases with inspiration

**Pulmonary vascular**: dilatation
Aortic ejection sound
Pulmonic ejeciton sound
Accentuated pulm2
Opening snap
NONEJECTION SOUNDS

systolic click: prolapse of the mitral valve
pm.apex, **timing**: increased LV volume---

shift to S2(squatting, supine, vasopressor)

**decreased LV volume**

shift to S1(standing, Valsalva, amyl nitrite)
The second heart sound, S2

- "the key to auscultation of the heart" Leatham
- A2-aortic pressure incisure
- P2-pulmonic pressure incisure
- A2 and P2 coincide with the complete closure of aortic and pulmonic valve leaflets. Not "clapping'',
  but deceleration of the blood column
- Delay from the ventricular pressure drop to the incisure :"hangout" pulmonary longer than aortic
Splitting of S2

- Normal "physiological" splitting: expiration 30ms, inspiration 50ms--"P2 moves away", prolongation of RV systole. pm.: 2nd left interspace
- Wide physiological splitting
  1. delayed pulmonic closure
     electric: RBBB, LV ectopic, LV pace
     mechanical: pulm hypertension, stenosis
     ASD: increased RV stroke volume
  2. Early aortic closure
     shortened "LVET": MI, VSD

The respiratory variation now attributed to impedance variation, but increased RV filling and protracted ejection is also possible
Splitting of S2

- **Reversed splitting**: always pathology, P2 precedes A2, in inspiration P2 moves to A2, so the splitting narrows

  - **Causes**
    - 1. *delayed aortic closure*
      - electric: LBBB, RV ectopic, RV pace
      - mechanical: LBBB distal type, LV outflow obstruction, hypertension, IHD
    - decreased impedance to outflow: increase of "hangout"-post-srenotic dilatation of Ao
    - 2. *early pulmonic closure*
      - early electrical activation
Splitting of S2

- **Fixed splitting**
  inability of RV to delay in inspiration: "common” atrial chamber, ASD*
- **Narrow splitting**
  pulmonary hypertension--decrease of "hangout"
- **Single S2**
  all causes of reversed splitting inaudibility of S2
  old age
  emphysema
Opening snaps

- Opening of the normal AV valves is silent
- MS in thickened deformed leaflets
  a sudden stopping
  of the opening: crisp, sharp sound
  p.m. left sternal border to apex
- intensity: mobility of the valve,
  calcification: „silent MS” or deafness?

A2-OS : 0.03-0.15s

- TS difficult to detect, rarely a single pathology
  p.m. left sternal border
- Tumor plop of atrial myxoma
Pulmonic ejection sound
Accentuated pulm2
Opening snap
third heart sound, S3

- **Low frequency sound** "protodiastolic gallop" rapid filling of the LV, A2-S3 = 120-200 ms
- Physiologic in children and young adults
- Pathologic exaggeration of the normal S3
  - **causes:** LV dysfunction,
    - increased filling P, decreased LV compliance
    - (DCM, IHD, restrictive CM)
    - excessive early diastolic filling (anaemia, thyreotox., A-V valve incompetence)

- **theories of origine:**
  - 1. valvular: diastolic tensing of the A-V valves at the end of rapid filling **NO!**
2. **ventricular**: interplay between the force of delivery of blood and the ability of the LV to accept it. At the *elastic limits* of LV the blood column suddenly halts → vibration (mostly accepted)

3. **impact theory**: with simultaneous intracardiac and external recording
   - the external is lauder
   - not coincident
   - hence it is caused by the impact of the heart to the chest wall (new ?)
The fourth heart sound, S4

- *Precordial vibration from atrial contraction*
  - palpable and audible
- **Pathological!** ECG P-wave--S4→ 70ms
  - p.m. left lateral at the maximal impulse
- **Names:**
  - atrial diastolic gallop, presystolic gallop
  - "gallop" or "canter"? See JAMA
- **Causes:** see table
- S3+S4 "summation gallop" may occur
To the Editor. -In his 1894 textbook, Potain,' taught by Bouillard, described the bruit de galop, an abnormal heart sound causing a rhythm that he likened to the sound of galloping horses: "Fort analogue au bruit des chevaux que nous entendons chaque jour galoper dans les rues ou sur la promenade." Today, gallop is regularly used to mean triple or quadruple cardiac rhythms. This is incorrect. Among equestrians, gallop has a precise meaning: it denotes the faster, four-beat gait~ in which each hoof lands separately. When the gallop is slowed and shortened ("collected" in equestrian terminology), it becomes the canter, a three-beat gait in which a forefoot and the opposite hindfoot land simultaneously. This distinction is clear in the literature of both classical horsemanship and veterinary medicine.' Indeed, in dressage, the most ancient and advanced form of equitation, it is considered a serious fault to perform a four-beat gallop when one is asked for the three-beat canter.

Therefore, to be correct in communicating auscultatory findings, a three-beat rhythm could be called a canter. Gallop should be reserved for four-beat rhythms. Frederick W. Hund, MD

Glenwood Springs, Colo
■ Prosthetic valve sounds
  - Depend on the type of the prosthesis, and the position

■ External sounds
  - Pacemaker sounds: twitch contraction of an intercostal muscle
  - Pericardial friction rub
Heart murmurs

- Relatively prolonged series of auditory vibrations of varying: intensity (loudness), frequency (pitch)
- quality (musical, harsh), configuration (envelope), duration
- grading from 1 to 6
- thrills (palpable), usually associated with loud murmurs

"pressure head" → flow velocity
- small ASD → high velocity → loud murmur
- large ASD → low velocity → no murmur
Systolic murmurs

Ejection murmurs: forward flow across the LV or RV outflow tract (stenosed area)
- delay between the S1 and the beginning of murmur, as P in the ventricle must exceed the aortic or pulmonic P.
- crescendo-decrescendo character "diamond-shaped"
- ends before the closure of the corresponding semilunar valve
- intensity parallels changes in cardiac output
Types of systolic murmurs

- **Innocent murmurs** - systolic ejection, without structural abnormalities
  - in small children *Still's murmur* - LV outflow vibratory, left sternal border 3th-4th interspace
  - in older age *musical murmur* of sclerotic aorta
    mandatory echo to exclude AS !!,
    now we know it is Ao stenosis
  - supraclavicular arterial - exclude carotid disease

- **Functional ejection systolic murmurs** - produced by high cardiac output states:
  - thyrotoxicosis, pregnancy, anaemia, fever,
  - exercise peripheral a-v fistula
midsystolic murmur, from healthy children, the last of four in a febrile state (Still murmur)
LV outflow tract murmurs

- **Obstruction**: acquired or congenital
  - valvular, subvalvular or supravalvular
    - *intensity* depends on pressure-drop, crescendo-decrescendo
    - p.max: 2nd right interspace → neck,
    - in elderly patients → apex
- **Congenital valvular AS** + ejection sound
- in **severe obstruction** reversed splitting of S2
- **LV hypertrophy** → S4 (atrial sound)
RV outflow tract obstruction

- Mostly congenital: valvular, infundibular (associated with great VSD), proximal pulmonary artery
  - **valvular**: late peaking murmur + ejection sound
  - **infundibular**: tetralogy of Fallot - increasing sten. - shunting to left, less flow through RV outfl. tract
  - if severe, murmur envelops A2 and no P2

- large VSD
- severe pulm sten
- "overriding aorta"
- RV hypertrophy

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Systolic regurgitrant murmurs

- High V-A pressure difference  *holosystolic* or *pansystolic* regurgitation
  - murmur plateau-like

- **Mitral regurgitation**: starts at S1, goes beyond S2
  - S3 may be present as a sign of rapid early diastolic filling
  - P.max at the apex, radiates to the axilla
  - intensity depends on the gradient
Systolic regurgitrant murmurs

- High V-A pressure difference  
  *holosystolic* or  
  *pansystolic* regurgitation  
  
  - murmuru plateau-like  
  - starts at S1, goes beyond S2

- **Tricuspid regurgitation**
  - P.max left sternal border  
  - intensity increases with inspiration, Carvallo’s sign, prominent jugular v wave
Mid - and late systolic regurgitant murmurs

- papillary muscle dysfunction

- mitral prolapse syndrome: after the systolic click a regurgitation starts
- **Hypertrophic obstructive cardiomyopathy** (HOCM)
  - the massive septum and the "SAM" mid and late systolic outflow tract obstruction
  - ejection murmur : left sternal edge
  - distorted mitral apparatus regurgitation
  - + S3 and S4
- **Ventricular septal defect**
  - P. max off the sternal border, 4-5-6 interspace
  - Pansystolic, accompanied with a **thrill**
  - Intensity correlates poorly with the size
  - Small muscular defect (Roger) → high velocity flow → loud murmur
- Early systolic regurgitant murmur
  - acute mitral regurg. , normal atrium
  - organic tricuspid regurg
Respiration
Right-sided murmurs generally increase with inspiration. Left-sided murmurs usually are louder during expiration.

Valsalva maneuver
Most murmurs decrease in length and intensity. Two exceptions are the systolic murmur of HCM, which usually becomes much louder, and that of MVP, which becomes longer and often louder. After release of the Valsalva, right-sided murmurs tend to return to baseline intensity earlier than left-sided murmurs.
Exercise

Murmurs caused by blood flow across normal or obstructed valves (e.g., PS and MS) become louder with both isotonic and isometric (handgrip) exercise.

Murmurs of MR, VSD, and AR also increase with handgrip exercise.
Positional changes

With standing, most murmurs diminish, 2 exceptions: the murmur of HCM, which becomes louder, and that of MVP, which lengthens and often is intensified.

With brisk squatting, most murmurs become louder, but those of HCM and MVP usually soften and may disappear.

Passive leg raising usually produces the same results as brisk squatting.
Murmurs originating at normal or **stenotic** semilunar valves increase in intensity during the cardiac cycle **after a VPB** or in the beat after a long cycle length in AF. By contrast, systolic murmurs due to **atrioventricular valve regurgitation** do not change, **diminish** (papillary muscle dysfunction), or become **shorter** (MVP).
Pharmacological interventions

In the initial relative **hypotension after amyl nitrite inhalation**, murmurs of MR, VSD, and AR **decrease**, whereas murmurs of AS **increase** because of increased stroke volume.

During the later tachycardia phase, murmurs of MS and right-sided lesions also increase. This intervention may thus distinguish the murmur of the Austin-Flint phenomenon from that of MS. The response in MVP often is biphasic (softer then louder than control).

*If an electrocardiogram or chest X-ray has been obtained and is abnormal, echocardiography is indicated.*
THE VALSALVA MANEUVER

CONTROL

Mitral regurgitation
Aortic stenosis
Hypertrophic CM
Mitral prolapse

VALSALVA

$S_1$ $S_2$

$S_1$ $S_2$

DIMINISHED VENTRICULAR FILLING
<table>
<thead>
<tr>
<th>DIAGNOSIS</th>
<th>SYSTOLIC MURMUR</th>
<th>SECOND SOUND</th>
<th>EFFECT OF POSTURE</th>
<th>AMYL NITRITE</th>
<th>PHENYLEPHRINE</th>
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</thead>
<tbody>
<tr>
<td>1. Hypertrophic obstructive cardiomyopathy</td>
<td></td>
<td>Variable i.e. reversed partially reversed narrow or normal</td>
<td>Changes in intensity of systolic murmur</td>
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<td>2. Mitral regurgitation</td>
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<td>a. Pure severe</td>
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<td>b. Papillary muscle dysfunction</td>
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<td>c. Billowing posterior leaflet</td>
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<td>d. Rheumatic of moderate degree</td>
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<td>3. Valvular aortic stenosis</td>
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<td>a. Mild to moderate</td>
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<td>b. Marked</td>
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<td>4. Ventricular septal defect</td>
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<td>5. Innocent vibratory systolic murmur</td>
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No change from control
Degree of increase
Degree of decrease
Diastolic murmurs

- Early diastolic murmurs
  - Aortic regurgitation: high frequency, decrescendo, may be very faint
    ♦ P. max mid left sternal border, better if the patient leans forward
  - Pulmonary regurgitation
    ♦ pulm. hypertension incompetence
    ♦ in MS difficult to tell from AI: Graham Steell murmur
    ♦ in organic pulmonary incompetence without pulmonary hypertension the murmur is
    ♦ low or medium pitched
DIASTOLIC MURMURS

EARLY DIASTOLIC

LEFT SIDED
S₁  A₂

RIGHT SIDED
S₁  A₂̅P₁

MID DIASTOLIC
S₁  S₂

LATE DIASTOLIC (PRESYSTOLIC)

LEFT SIDED
S₁  S₂  S₁

RIGHT SIDED
S₁  S₂
Diastolic Filling Murmur (Rumble)
Mitral Stenosis

Mild

S\_1

\begin{align*}
\text{S\_2} &\quad \text{O.S.} \\
\text{A\_2} &\quad \text{P\_2} \\
\end{align*}

Severe

\begin{align*}
\text{S\_2} &\quad \text{O.S.} \\
\text{A\_2} &\quad \text{P\_2} \\
\end{align*}
Middiastolic murmurs

- Low pitched, rumbling murmur
- **Mitral stenosis (MS)**: at the site of LV impulse; patient turned on the left side. *Duration* depends on severity.
- **Tricuspid stenosis**: best heard at the xiphoid area, it is augmented on inspiration.
- **VSD**: may cause an increased flow in the mitral area.
- **S3+S4**: in tachycardia they coincide causing a middiastolic murmur: e.g. rheumatic fever + valvulitis: **Carey-Coombs** murmur.
Late diastolic murmurs

- In **MS** and **TS** atrial systole → acceleration of flow through the narrow orifice
- The **Austin Flint** murmur
  - late diastolic with mid-diastolic extension, it is introduced by S3
  - caused by *aortic regurg* → *increased LVEDP*
  - premature closure of the mitral valve or
  - the *aortic regurgitant jet* causes a shudder of the *anterior mitral leaflet*
Continuous murmurs

- begins in systole and extends through S2 into part or all diastole
- High to low pressure shunts
  - PDA left infraclavicular area and 2.interspace
  - Sinus of Valsalva rupture into the right atrium
    - lower sternal border-diastolic accentuation
distinguish from to-and-fro murmurs like aortic stenosis and regurgitation, coarctation of the aorta, branch pulmonary stenosis
Continuous Murmur vs. To-Fro Murmur

Continuous Murmur

To-Fro Murmur
Echocardiography
Aortic ejection sound in expiration