Physical examination of the heart

3rd Medical Department
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2016, 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)

10/20/2016

carinatum, pigeon
a protrusion of the sternum and the 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
- *Antihoral rotation of the heart in early systole and tapping of the apex on the chest wall*
LV Segmentation and Mechanics in HCM: Twisting the Rubik’s Cube Into Perfection!
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)
This is from an old book
left ventricular impulse
Abnormal findings

- *Left ventricular hypertrophy*: exaggeration of LV thrust-amplitude, duration, dysplacement
- **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, akinesia
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

Auenbrugger (1761)
Corvisart (1808)

Diorg (1828)

plethysmometer vs. a naked eye
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)
Percussion of the heart

23. ábra. A relatív (andbox) és abszolút (andbox) szívompulat a) fiziológiásan,
b) balra irányuló szívmeagnagyobbodás, c) jobbra irányuló szívmeagnagyobbodás és
d) pericardialis fluidum esetén
4. situs inversus
5. historic significance: valvular diseases before the invention of X-ray
Percussion
in Luisada: Cardiology 1959
5½ pages

In Braunwald 9th ed 2012
the term
not even mentioned in the index
Fig. 3-24. Schematic relationship between anatomical projection of the heart and vessels on the anterior chest wall and the absolute and relative areas of dullness: 1, Part of the heart which cannot be recognized through percussion, being too deep. 2, Area of relative dullness. 3, Area of absolute dullness.
Fig. 3-27. Left ventricular enlargement. A. Initial stage. B. Final stage.
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. 10.3. Graph indicating the curves on the threshold of audibility.
Fenyvesi

The stethoscope

Which is the most important component?

earpiece
rubber
tubing: short, thick
bell
diaphragm

Rapaport-Sprague
Littmann
10/20/2016
Binaural stethoscope
Fig. 18-2. The stethoscope collection of Dr. E. Grey Dimond. Sixteen masonic 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, 6, 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, 19, 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
How to learn and practice auscultation
The robust Lumify app is available on compatible Android smart devices via the Google Play Store. 2015!!, only in the US
Beware, these are not advertisements of two products, but just a teaching aid
Carefully performed clinical studies have shown that diagnostic ultrasonography can be superior to the physical exam. In one study, first-year medical students using point-of-care ultrasound outperformed board-certified cardiologists using bedside cardiovascular physical examination in identifying cardiac abnormalities, identifying 75% of conditions, as compared with 49% identified by the cardiologists.

NEJM 2014;370:1083
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
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 BASIC HEART SOUNDS

A

S₁  S₂  S₃  S₄

THE HEART SOUNDS
DESCRIPTIVE TERMINOLOGY

B

ES  MS  LS  ED  MD  LD

S₁  S₂

S₄

S₁  S₂

C
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

ECG

T P R
complete AV block

Long PR interval

Short PR interval

soft S1

laud 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 ausc1)
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 ejection 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, amylNitrite)
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
Figure 14-22
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-sclerotic 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

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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

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Pulmonic ejeciton 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, NO! ?)
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
The fourth heart sound is a low-pitched sound coincident with late diastolic filling of the ventricle due to atrial contraction. It thus occurs shortly before the first heart sound. Although it is also called the atrial sound, and its production requires an effective atrial contraction, the fourth heart sound is the result of vibrations generated within the ventricle. Commonly, its presence indicates increased resistance to filling of the left or right ventricle because of a reduction in ventricular wall compliance, and it is accompanied by a disproportionate rise in ventricular end-diastolic pressure. In patients with a fourth heart sound, its palpable correlate is often present: a concomitant brief presystolic outward movement of the chest wall.
Dressage for Cardiologists: Gallop or Canter?

To the Editor. —In his 1894 textbook, Potain,' taught by Bouillard, described the bruit de galop, an abnormal heartsound 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

JAMA, April 14, 1989—Vol 261, No. 14
- **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
ON CARDIAC MURMURS

AUSTIN FLINT, M.D.

Professor of the Principles and Practice of Medicine in the Bellevue Hospital Medical College, N.Y., and in the Long Island College Hospital

The American Journal of Medical Sciences 1862; 44:29-54

Yes 1862 !!!
I am going to use many quotations of this very educational paper.
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
    murmurs, 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
SYSTOLIC MURMURS

MIDSYSTOLIC

LEFT SIDED

S_1  A_2

RIGHT SIDED

S_1  A_2 P_2

HOLOSYSTOLIC

LEFT SIDED

S_1  A_2

RIGHT SIDED

S_1  A_2 P_2

EARLY SYSTOLIC

S_1  S_2

LATE SYSTOLIC

S_1  S_2
Types of systolic murmurs

- **Innocent murmurs** - systolic ejection, without structural abnormalities
  - in small children *Still's murmur* - LV outflow vibratory, medium-to-long with a musical quality left sternal border 3th-4th

In older age *musical murmur* of sclerotic aorta *mandatory echo to exclude AS*

!! now we know it is Ao stenosis
Types of systolic murmurs

- 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 on the phono) (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
    1. large VSD
    2. severe pulm sten
    3. "overriding aorta"
    4. RV hypertrophy

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Systolic regurgitrant murmurs

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

- Mitral regurgitataion:
  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
  
murmur 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

In his paper, Barlow described the features of mitral-valve prolapse in 90 patients with nonejection clicks, late systolic murmurs, or a combination of the two.
– **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

Roger’s murmur of ventricular septal defects is holosystolic and heard best at the left upper sternal border. The murmur is loud, and its sound has been compared with that of a rushing waterfall.
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.
Postventricular premature beat and atrial fibrillation

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).
THE VALSALVA MANEUVER

CONTROL

S₁
Mitral regurgitation

S₂
Aortic stenosis

Hypertrophic CM

Mitral prolapse

VALSALVA

S₁

S₂

C

DIMINISHED VENTRICULAR FILLING

C

<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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<tbody>
<tr>
<td>1. Hypertrophic obstructive cardiomyopathy</td>
<td></td>
<td>Variable ie - 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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- 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

The Key–Hodgkin murmur is a diastolic murmur of aortic regurgitation; it has a raspy quality, similar to the sound of a saw cutting through wood.
Diastolic murmurs

- **Pulmonary regurgitation**
  - pulm.hypertension  ➔  incompetence
  - in MS difficult to tell from AI:  *Graham Steell* murmur

He posited that the pulmonary regurgitation was usually the result of chronically elevated blood pressure in the pulmonary artery, resulting from mitral stenosis

- in organic pulmonary incompetence without pulmonary hypertension the murmur is low or medium pitched
DIASTOLIC MURMURS

EARLY DIASTOLIC

LEFT SIDED

RIGHT SIDED

MIDDIASTOLIC

LATE DIASTOLIC (PRESYSTOLIC)

LEFT SIDED

RIGHT SIDED
Diastolic Filling Murmур (Rumble)  
Mitral Stenosis

Mild

Severe

ECG

S1  S2  O.S.  S1  S2  O.S.  S1  S2  O.S.
A2   P2  A2   P2  A2   P2  A2   P2
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
The Carey Coombs murmur is a short mid-diastolic murmur caused by active rheumatic carditis with mitral-valve inflammation. The murmur is soft and low pitched, heard best at the apex.1
Late diastolic murmurs

- In MS and TS atrial systole → acceleration of flow through the narrow orifice
- The **Austin Flint** murmur
  - late diastolic with middiastolic 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*
The Austin Flint murmur is a mid-diastolic rumbling sound present in selected cases of nonrheumatic aortic regurgitation. The sound is indistinguishable from mitral stenosis. Flint postulated that the murmur was due to regurgitant flow onto the mitral valve that pushed back the mitral leaflets, decreasing the size of the mitral orifice and impairing flow from the left atrium to the left ventricle.
Continuous murmurs

- begins in systole and extends through S2 into part or all diastole

**Gibson murmur** is continuous, beginning after the first heart sound and extending through the second heart sound, which is distinctly audible over the unbroken rushing of the murmur

**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