CLINICAL EXAMINATION OF PRECORDIUM ALONG WITH RELEVANT GENERAL PHYSICAL EXAMINATION

**Scheme of Examination**

- Inspect the patient
- Feel the pulses, rate and rhythm
- Measure the BP
- Inspect the neck veins
- Palpate the precordium
- Auscultate the precordium
- Palpate the peripheral pulses

**Examination – Face and Neck**

- Jaundice
- Xanthelasmata
- Corneal arcus
- Malar flush (mitral stenosis)
- High arched palate (Marfan’s syndrome)
- Dental caries (infective endocarditis)
- Central cyanosis
- Carotid pulse character
  - Slow rising (AS)
  - Bisferiens (AS + AR)
  - Collapsing (AR)
  - Alternans (LVF)
  - Jerky (HOCM)
- Carotid bruit

**Arcus senilis (juvenilis)**

Arcus juvenilis. This ring is associated with premature atherosclerosis

**ARTERIAL PULSE**

Character of pulse is assessed by stroke volume & compliances and is best to assess by palpating a major
artery carotid artery and brachial artery

Examination - Pulse

- Radial artery
- Rate (normal = 60-100)
  - Bradycardia (<60)
  - Tachycardia (>100)
- Rhythm
  - Regular
  - Irregular
- Character of Pulse
- Radio emoral delay

THE ARTERIAL PULSE

- The anacrotic and dicrotic notches normally not be felt but are easily recorded
- Normally the arterial pulse is not palpable during diastole
- Normal pulse contour is altered with increasing distance from aortic valve characterized by a greater amplitude and velocity and a lower dicrotic notch. These changes become progressively more pronounced distally.
- A missing or extremely weak pulse may have major significance—atherosclerosis embolic occlusion, dissection, vascular compression or a congenital anomaly.

Valvular regurgitation anaemia, sepsis other causes of a large stroke typically produce a bounding with a high amplitude and wide pulse pressure.
Valvular stenosis impedes ventricular flow and may cause a slow rising weak pulse
Sinus rhythm produces a pulse that is regular in time and arrhythmias may cause irregularity in time and character.
**Difference b/w arterial and venous pulsation**

- Venous has two peak in each cardiac cycle.
- The height of the venous pulse varies with respiration.
- Abdominal compression causes the venous pulse to rise.
- Venous pulse is not palpable and can be occluded by light pressure.

**Jugular Venous Pressure**

- Patient at 45 degrees
- Good lighting
- Internal jugular vein
- Reflects right atrial pressure
- Zero point = sternal angle
- Visible but not palpable
- Complex wave form (a, c, v waves)
- Decreases on inspiration
- Hepatojugular reflux

**Jugular Venous Pressure**

- Abnormal if >3 cm above zero point:
  - RV failure
  - RV infarct
  - Tricuspid stenosis
  - Tricuspid regurgitation
  - Pericardial effusion
- SVC obstruction
- Fluid overload

**Examination - Blood Pressure**

- With Sphygmomanometer
- Systolic/diastolic pressure
- Normal <140/90 mmHg (lower in diabetes)
- Korotkoff sounds
- Use larger cuff width for large arms
- Difference between arms of <10 mmHg
- Rubber tube should cover 2/3 of the Four Arm

**Look For**
- Pulsus paradoxus = exaggerated reduction in BP with inspiration (>10 mmHg)
- Postural hypotension
- Hills Sign

**INSPECTION:**
- Chest observation gives clues to cardiopulmonary disease
- Can be insensitive

**INSPECTION (cont.):**
- Asymmetry can indicate RVE
- Increased A-P chest diameter indicates chronic air trapping/hyperinflation
- Pectus deformities--usually no significant cardiopulmonary consequences
- Kyphoscoliosis--can have cardiopulmonary effect
  - LV impulse outward movement like a ping pong
  - ball protruding between the ribs
    - Apex moves outward for the first third of systole
    - and falls away rapidly
      - Lasts for no more than 2/3 of systole
      - Sustained apex-hangs out to S2
  - correlates with pressure overload
  - (> 2/3 systole-hangs out to S2)
  - AS, LVH or LV systolic dysfunction

**PRECORDIAL PALPATION**
— Palpation of the precordium

— Place fingertips over apex to assesss for the position and character, place heel of the hand over left sternal edge for a parasternal heave or lift. Assess for presence of thrills in all areas, including aortic and pulmonary.

**Precordium - Palpation**

— Apex beat
  — Location
  — Character
    • Heaving
    • Thrusting
    • Double
    • Tapping

— Left parasternal heave

— Thrills (palpable murmurs)
  — Systolic
  — Diastolic

— Palpable P2 (pulmonary hypertension)

— Pacemaker box

**PALPATION:**

❤ Sometimes overlooked and not always helpful
❤ Use the most sensitive portion of the hand
❤ Lay the heel of R hand at left sternal border with fingertips pointing to left axilla.

**Palpation - Apex**

— Apex:
  • Best felt with fingertips or finger pads

— Normal Location:
  • No more than 10 cm from mid-sternal line in the supine position
  • Left decubitus position

— Normal Size:
- No larger than 3 cm

**LV/apical impulse (PMI):**

- Found with the fingertips
- Note interspace location, relation to the midclavicular/anterior axillary line, amplitude compared to RV impulse

**Common abnormalities**

- Volume overload displaced forcefully as in MR / AR
- Pressure overload discrete, thrusting such as AS / HTN
- Dyskinetic displaced incoordinate such as LV aneurysm

**Other abnormalities**

- Palpable S1 tapping at the mitral stenosis
- Palpable P2 in severe PH
- Left parasternal heave or lift felt by heel of hand
- Palpable thrill as in AS

**Apical Impulse (abnormal):**

- Hyperdynamic impulse in normal location: think increased cardiac output or LVH
- Hyperdynamic and downward/leftwardly displaced: think LVE
- Indistinct impulse associated with RVH
- Precordial heave is seen with RVE

**LV/apical impulse (abnormal):**

- Strong impulse is due to increased cardiac output or LVH
- Downward/leftward displacement—LVE (with or without LVH)

**RV impulse:**

- Felt at the LSB—usually slight
- RVH (without RVE)—parasternal tap (sharply localized, quickly rising)
- RVE (with or without RVH)—parasternal lift (diffuse, gradually rising)

**Thrills:**

- Palpation of a loud murmur
- Found in the precordial, suprasternal, or carotid artery area
- If low intensity murmur, probably just a pulsation and NOT a thrill

**AUSCULTATION:**

**Where to listen:**

- Apex/5LICS (mitral area)
- Left lower sternal border/4LICS (tricuspid and secondary aortic area)
Right middle sternal border/2RICS (aortic area)
Left middle sternal border/2LICS (pulmonary area)

Where to listen (cont.):
Left and right infraclavicular areas
Left anterior axillary line
R and L interscapular areas of back (for pulmonary/aortic collaterals)

How to listen:
Have a system, e.g. method of inching
Listen systematically: S1, S2, systolic sounds, systolic murmurs, diastolic sounds, diastolic murmurs

Normal heart sounds
AUSCULTATION

Radiation of systolic murmurs.
- Towards the base of the neck in aortic stenosis.
- Toward the axilla in mitral incompetence.
- Towards the left shoulder below left clavical in Pulmonary stenosis
- Murmurs can be differentiated by feeling the carotid pulse e.g. systolic murmurs are with the pulse

S1:
- phenomena From closure of the A-V valves
- Best heard at the apex and LLSB
- Often sounds single unless slow heart rate

S1 (cont.):
- If split heard better at the apex, may actually be S4 or ejection click
- Tends to be more low-pitched and long as compared to S2
- Differentiate S1 from S2 by palpating carotid pulse:

- S1 comes before and S2 comes after carotid upstroke

Abnormalities of S1:
- Slowed ventricular ejection rate/volume
- First degree AV block
- Mitral insufficiency
- Increased chest wall thickness
- Pericardial effusion
- Hypothyroidism
Increased S1:
  - Increased A-V valve flow velocity (acquired mitral stenosis, but not congenital MS)

Wide splitting of S1:
  - RBBB (at tricuspid area)
  - PVC’s
  - VT

S1:
- Produced due to closure vibrations of aortic and pulmonary valves

Have two components A2 and P2 (aortic and pulmonary closure sounds)
- Best heard at LMSB/2LICS
- Higher pitched than S1—better heard with diaphragm

S2:
- Normally split due to different impedance of systemic and pulmonary vascular beds
- Audible split with > 20 msec difference
- Split in 2/3 of newborns by 16 hrs. of age, 80% by 48 hours
- Harder to discern in heart rates > 100 bpm

S2 splitting (normal, cont.):
- Respiratory variation causes ↑ splitting on inspiration; ↓ pulmonary vascular resistance
- When supine, slight splitting can occur in expiration
- When upright, S2 usually becomes single with expiration

S2 splitting (abnormal):
- Persistent expiratory splitting
  - ASD
  - RBBB
  - Mild valvar PS
  - Idiopathic dilation of the PA
WPW

Single S2:

- Single S2 occurs with greater impedance to pulmonary flow, P2 closer to A2
- Single and loud (A2): TGA, extreme ToF, truncus arteriosus
- Single and loud (P2): pulmonary HTN!!
- Single and soft: typical ToF
- Loud (not single) A2: CoA or AI

Extra heart sounds

S3 (gallop):

- Usually physiologic
- Low pitched sound, occurs with rapid filling of ventricles in early diastole
- Due to sudden intrinsic limitation of longitudinal expansion of ventricular wall
- Makes Ken-tuck-y rhythm on auscultation

S3 (cont.):

- Best heard with patient supine or in left lateral decubitus
- Increased by exercise, abdominal pressure, or lifting legs
- LV S3 heard at apex and RV S3 heard at LLSB

S3 (abnormal):

- Low frequency sound in early diastole
- Seen with Kawasaki’s disease--disappears after treatment
- If prolonged/high pitched/louder:
  - May be due to diastolic flow rumble indicating increased flow volume from atrium to ventricle

S4 (gallop):

- Nearly always pathologic
- Can be normal in elderly or athletes
Low pitched sound in late diastole
Due to elevated LVEDP (poor compliance) causing vibrations in stiff ventricular myocardium as it fills
Makes “Ten-nes-see” rhythm

S4 Associations:
CHF!!!
HCM
severe systemic HTN
pulmonary HTN
Ebstein’s anomaly
myocarditis

Click:
Usually pathologic
Snappy, high pitched sound usually in early systole
Due to vibrations in the artery distal to a stenotic valve

Can be associated with:
Valvar aortic stenosis or pulmonary stenosis
Truncus arteriosus
Pulmonary atresia/VSD
Bicuspid aortic valve
Mitral valve prolapse (mid-systolic click)
Ebstein’s anomaly (can have multiple clicks)

Friction rub:
Creaking sound heard with pericardial inflammation
Classically has 3 components; can have fewer than 3 components
Changes with position, louder with inspiration

Murmur:
What is a murmur?
• A sound/vibration made by blood flowing through a normal valve or an abnormal valve.
• A sound made by blood flowing backwards through a leaking valve
Forward flow through an incompetent valve, septal defect, or PDA
Murmurs may be functional or pathologic

**Detection & Identification**

- Listen with the patient in both supine & sitting position/expiration & inspiration
  - Position in the cardiac cycle
  - Graded intensity & duration
  - PMI
  - Radiation of murmur
  - Loudness, pitch, & quality
  - Effect of respiration & interventions
  - Whether or not S2 is normally split
  - Accompanying thrill
  - Accompanying change in heart sound

**Position in the Cardiac Cycle**

- Systolic Murmurs
  - Between S₁ & S₂
  - Classified as early, mid, late, holosystolic

- Diastolic Murmurs
  - Between S₂ & S₁
  - Classified as early, mid, late

- Continuous Murmurs
  - Begins with S₁ and continues through S₂ into diastole

**Pitch & Loudness (Intensity) of Murmurs**

- The pressure differential across an orifice for blood flow that determines the velocity of that flow for a given orifice
When the pressure differential is high, so is the velocity of flow.

As the velocity increases, so does the turbulence, loudness, & pitch of the heart murmur

Loudness is determined by the rate of blood flow & the turbulence produced through a given orifice

### Grading Intensity of Murmurs

- **Grade I**
  - faint audible only with concentration & by experienced person
- **Grade II**
  - Faint, need to focus to hear
- **Grade III**
  - Not loud, but somewhat louder than grade II; of intermediate intensity

### Grading Intensity of Murmurs

- **Grade IV**
  - intermediate intensity; generally associated with a palpable vibration or thrill;
- **Grade V** - Loud, easily audible
- **VI** - audible with the stethoscope partially off the chest

### Character of murmurs

- **Waxing and Waning**
  - **Crescendo Murmur**
    - Begins faintly and increases in loudness
  - **Decrescendo Murmur**
    - Loud at first & then fades away
  - **Crescendo-Decrescendo Murmur**
• Which is diamond-shaped-first increasing in loudness & then fading away

**Heart Murmurs**

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**Systolic**

- Pansystolic
  - Mitral regurgitation
  - Tricuspid regurgitation
  - Ventricular septal defect

- Ejection systolic
  - Aortic stenosis
  - Pulmonary stenosis
  - HOCM
  - Atrial septal defect

- Late systolic
  - Mitral valve prolapse

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**Diastolic**

- Early diastolic
  - Aortic regurgitation
  - Pulmonary regurgitation

- Mid-diastolic
  - Mitral stenosis
  - Tricuspid stenosis
  - Atrial myxoma

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**Continuous**

- Patent ductus arteriosus
- Arteriovenous fistula

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**Pericardial friction rub**

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**Functional Murmurs**

*Common in Asymptomatic Adults*
• Characterized by
  – Grade I–II @ LSB
  – Systolic ejection pattern - no ↑ with Valsalva
  – Normal precordium, apex, S1
  – Normal intensity & splitting of second sound (S2)
  – No other abnormal sounds or murmurs

Summary

A. Presystolic murmur
  — Mitral/Tricuspid stenosis
B. Mitral/Tricuspid regurg.
C. Aortic ejection murmur
D. Pulmonic stenosis (spilling through S2)
E. Aortic/Pulm. diastolic murmur
F. Mitral stenosis w/ Opening snap
G. Mid-diastolic inflow murmur
H. Continuous murmur of PDA

HANDS/ARMS

• HANDS
  — Clubbing
  — Splinter Haemorrhages
  — Cyanosis / cool peripheries
  — Tendon Xanthoma
  — Tar Staining

• ARMS
  — Pulse
  — Rate / Rhythm
  — +/- Delay

Detection of clubbing

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