

Guidelines for Examination of Cardiovascular System

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INSPECTION

1. FACIES

Apprehensive facies produced by pain, anxiety and respiratory distress (MI, angina, PE, P edema, arrhythmias as VT, fast AF)

A. Skin Color and Texture:

- Malar flush: long-standing MS, rash across the nose and nose in SLE.
- Brick red color of polycythemia (may cause HTN, thrombosis, MI)
- Bronze skin in hemochromatosis(Cardiomypathy)
- Brown + buccal pigmentation in Addison's disease (hypotension)
- Flushing & telangiectasia in carcinoid syndrome (tricuspid & pulmonary valve disease)
- Moon face in cushing's disease (HTN)
- Coarseness & dryness in myxedema (bradycardia, heart failure, PE)
- Central cyanosis (right to left intracardiac shunt or lung disease)

B. Eyes and Lids

- Xanthelasma (hypercholesterolemia, DM)
- Lid edema (myxedema, nephrotic syndrome, SVC syndrome...)

- Exophthalmos, lid retraction in thyrotoxicosis (A.F, high output failure)
- Corneal arcus in young people indicates severe hypercholesterolemia
- Blue sclera in marfan syndrome, Ehlers-Danlos syndrome (associated with AR, MVP, ASD)
- Lenses (subluxation in Marfan syndrome superior, homocystenuria inferior)
- Pupils (Argyll Robertson sign) react to accommodation not to the light seen in neurosyphilis (AR, calcification in the ascending aorta)

C. **Bony Developmental Abnormality**

- Large head (Paget's disease): High-out failure
- Acromegaly (HTN, CHF)
- Marfan syndrome (with long narrow face, lens subluxation, long arm, arachnodactyly)(AR, aortic dissection, MVP)
- Williams syndrome (small elf-like forehead, turned up nose, egg shaped teeth, low set ears) associated with supravalvular aortic stenosis
- Noonan's syndrome (widely set eyes, web neck) associated with PS

D. **Hands**

- Tremor may indicate thyrotoxicosis (AF, CHF)
- Clubbing of the fingers (cardiac cause: congenital heart disease, bacterial endocarditis)

- Capillary pulsation (AR, thyrotoxicosis, pregnancy)
- Splinter hemorrhage (bacterial endocarditis, acute glomerulonephritis)
- Osler's nodes (0.5-1 cm painful reddish-brown subcutaneous papules occur on the tip of the fingers or toes, palm of the hand, plantar aspect of the feet (bacterial endocarditis)
- Arachnodactyly (long slender hand and fingers) marfan syndrome

BREATHING PATTERNS

1. Is the patient using accessory muscles of respiration? (P edema, asthma, COPD, fulminant pneumonia)
2. Breathlessness + wheezing (asthma, COPD, LV failure)
3. Stridor (indicating upper airway obstruction) life-threatening situation
4. Cheyne-stokes respiration (CHF, strokes, oversedation, uremia)

CYANOSIS

1. Is not apparent till Hb < 5g/dl (central)
2. In CHD cyanosis is observed if R to L shunt is > 25% of CO and not improved by 100% of O₂
3. Good examination of tongue, lips, ear lobes, fingers, toes is recommended

FOUR TYPE OF CYANOSIS

- **Central cyanosis** blue tongue, lips, and extremities with warm peripheries (CHD, lung disease as emphysema, pneumonia, ARDS, chronic bronchitis, sometimes CHF)
- **Peripheral cyanosis** (result from sluggish circulation in the peripheries) reduction in oxygenated Hb occur in capillaries (extremities are blue & cold) etiologies: low CO, hypovolemic shock)
- **Differential cyanosis** (lower limb cyanosed, upper limb pink) in CHD: PDA with reversed shunt due to PHTN
- **Reversed differential cyanosis.** The cyanosis of the fingers exceeds that of the toes; seen in transposition of the great vessels (blood from RV ejected into the AO reaches the upper limbs and head, blood from LV ejected into PA reaches the lower limb via PDA)

PALPATION

1. PULSE: determine

- ✓ Rate
- ✓ Rhythm
- ✓ Character
- ✓ Symmetry

Examine both radial, carotid, femoral, tibial, and dorsalis pedis pulses

- **Rate** at rest > 100/min (tachycardia) seen in anxiety, pain, CHF, PE, hyperthyroidism, anemia, fever, medications
- **Rate** < 60/min (bradycardia) due to (medications, MI, hypothyroidism, hypothermia, SSS,...)
- **Rhythm** (regular or irregular indicating AF, frequent PAC's, PVC's, ...etc.)

➤ **Character**

1. **Collapsing pulse** (water hammer pulse) jerky pulse with full expansion followed by sudden collapse (AR, PDA, A-V fistulas, pregnancy, paget's disease, thyrotoxicosis, anemia)
2. **Alternating pulse** pulses alternans (regular rate, amplitude varies from beat to beat) seen in LVF
3. **Pulsus bisferiens** (two strong systolic peaks separated by a midsystolic dip) seen in HOCM, AS/AI
4. **Anacrotic pulse** slow rising pulse in A.S. (Parvus et tardus)
5. **Dicrotic pulse**, two systolic and diastolic peaks (sepsis, hypovolemic, cardiogenic shock)
6. **Paradoxical pulse** (amplitude decreases with inspiration and increases during expiration) seen in cardiac tamponade, COPD, massive P.E.

PERIPHERAL VEINS

- **JVP:**

Differentiation of the jugular and carotid pulse wave

	Jugular	Carotid
Character	3 positive waves	1 wave
Effect of respiration	↓ on the inspiration ↑ on expiration	No effect
Venous compression	Easily eliminate pulse wave	No effect
Effect of changing position	More prominante when recumbent, less prominante when sitting	No effect
Abdominal pressure	JV is more visible	No effect

Normal JVP:

- H Period of slow filling of atria before atrial contraction
- a Atrial systole

- X Atrial relaxation
- C Bulging of TV into RA during V systole
- X' RA pressure falls because of pulling of RA floor during V systole. Some authors refer to this as X wave
- V filling of RA while TV is closed
- Y Decline in RA pressure when TV opens

- X' descent: systolic
- Y descent: diastolic

Abnormalities of Venous Wave

1. Giant "A" wave seen in RA contraction against an obstructed TV (TS, atresia, myxoma) high resistance to RA emptying (RVH, PHTN, PS, PE, ...)
2. Cannon "A" wave: (RA contracts against closed TV) seen in CHB.
3. Prominent "V" wave (V wave caused by RA filling against TV closure coincide with S2 and T wave on the ECG) seen in significant TR, VSD, ASD causing diastolic RA overloading
4. Kussmaul's sign: paradoxical rise with inspiration (constrictive pericarditis, severe RHF)

PALPATION

1. **APEX BEAT:**

Patient should be examined in the supine, sitting, and left lateral decubitus position. Normal apical impulse occurs during early systole with an outward motion imparted to the chest wall. Normal apex beat is palpable as brief outward impulse (intersection of left mid clavicular line and 5th intercostal space by the fingers.

Apex beat > 2cm indicate LV enlargement.

Double apical impulse caused by LVH and forceful LA contraction.

2. **LEFT PARASTERNAL LIFT**

Best appreciated by the distal palm or with the finger tip.

Palpable anterior systolic movement sustained up to S2 indicate RVH.

Giant presystolic lift seen in HCM.

3. **ABDOMEN**

Abdominal aorta (aneurysm)

Liver (hepatomegaly, pulsatile liver)

Ascitis

AUSCULTATION

AREAS TO AUSCULTATES

1. Apex (mitral area) murmur originated from the MV are best heard
2. Lower sternal edge (tricuspid area)
3. Lower left parasternal (4th intercostal space) murmur of AR is best heard
4. Upper left parasternal (pulmonary area, 2nd left intercostal space)
5. Upper right parasternal (aortic area, 2nd right intercostal space murmurs arising from aortic valve area best heard)
6. Below the left clavicle: continuous murmur of PDA is best heard
7. Posterior chest for bruits caused by bronchial collaterals in case of coarctation of the aorta
8. Other areas: (abdominal aorta, renal arteries, carotide, femoral arteries)

FIRST HEART SOUND (S₁)

- Produced by M&T valve closure
- Best heard at the apex
- Occurs just before the palpable upstroke of the carotid pulse

S₁ is loudest when onset of ventricular systole finds mitral leaflets maximally recessed into LV cavity as in short PR, A.F. with short cycle length.

Factors influencing intensity of S₁

1. PR interval :
 - short PR-loud S₁
 - long PR-soft S₁

 2. Mitral Valve Disease: MS typically causes loud S₁. However, later in the course of the disease, when the valve becomes calcified and immobile, S₁ intensity decreases. Soft S₁ occur also in AR due to premature closure of MV.
- Intensity of S₁ increase in (MS, TS, myxoma, short PR)
 - Intensity of S₁ decrease in (fibrosis or calcification of MV, prolong PR, heart failure, MR, AR)
 - Variable intensity of S₁ in A.F., CHF, VT

SECOND HEART SOUND (S2)

- Closure of semilunar valves AV & PV (other theory is that deceleration of a column of blood in the root of aorta & PA at termination of systole leads to sound producing vibration audible as S2).
- Normally AV closes before PV. Inspiration splitting of S2 is due to delay in P2 due to:
 1. Increase capacitance of pulmonary vascular bed
 2. Increase-RV volume

Normally intensity of A2 exceeds that of P2.

- A) Loudness of A2 or P2 is proportional to the respective pressures in Ao or PA at onset of diastole, i.e., higher pressure – louder A₂ or P₂
 - A2 is louder with HTN, dilated aorta.
 - P2 is louder with pulmonary HTN, dilated PA
- B) Decrease intensity of A2 or P2 is due to decrease pressure beyond the valve, stiff semilunar valves (A.S, P.S.), chest wall or lung deformity (emphysema)

Wide but not fixed splitting of S2:

1. Delayed electrical activation of RV (RBBB, PVC from LV)
2. Prolonged RV mechanical systole (massive PE, Pulmonary HTN, P.S.)
3. Early aortic closure (MR)

Reversed Splitting:

1. Delay electrical activation of LV (LBBB, PVC from RV)
2. Prolonged LV mechanical systole (HTN, A.S., severe LV dysfunction)
3. Early pulmonic closure (TR), early electrical activation of RV (WPW)

Fixed Splitting: A.S.D., severe RV failure

THIRD HEART SOUND (S3)

1. Normal findings in younger patients
2. Due to passive diastolic filling of the ventricle (occurs 0.14 to 0.22 s after S2)
3. Best heard at the apex patient on the LLP
4. Causes of S3 include
 - Abnormal ventricular relaxation
 - Ventricular failure
 - Dilated and hypertrophic cardiomyopathy
 - Severe TR, MR
 - LV dyskinesia or aneurysm
 - Hyperdynamic states (AV fistula, thyrotoxicosis)

FOURTH HEART SOUND (S4)

1. Due to vigorous atrial contraction to propel blood into a stiff ventricle (absent in AF)
2. Best heard at the apex, patient on LL decubitus
3. S4 is heard in:
 - LVH
 - Acute MI
 - HOCM, DCM
 - Severe AS, PS
4. S4 occurs just before the S1 (# diagnosis with split S1: firm pressure by the diaphragm eliminates S4 but not split S1)

EJECTION CLICKS:

Three mechanisms:

1. Intrinsic abnormality of AV or PV (congenital bicuspid AV, congenital P.S.)
2. Pulsatile distension of dilated great artery (HTN, PHTN, dilated PA, aortic aneurysm)
3. Increase flow states (ASD → pulmonic EC) (truncus arteriosus → aortic EC)

Mid to Late Systolic Clicks:

1. Commonly heard in MVP
2. Sharp high pitched sound
3. Maneuvers that decrease LV volume move the click earlier.
4. Maneuvers that increase LV volume move the click later.

OPENING SNAP

1. Caused by the opening of the stenotic but pliable MV or TV (disappears in severe calcified MS or TS). Higher LA pressure leads to short S₂-OS
2. Occurs 0.08 s after S₂
3. Best heard between apex and left sternal border
4. DD: Split S₂. However, having the patient stand help to differentiate the two. The S₂ – OS interval widens, while split S₂ does the change or narrow.

MURMURS

Grading of intensity of murmurs:

Grade:

Grade I - So faint and heard only with special effort

Grade II - Soft but readily detected

Grade III - Prominent but not loud

Grade IV - Loud usually with palpable thrill

Grade V - Very loud with thrill

Grade VI - Heard without stethoscope on the chest wall

High pitch sounds (use diaphragm of stethoscope)

- S₁, S₂, murmurs of valvular regurgitation
- OS, clicks, obstruction of semilunar valves (AV, PV)
- Pericardial knock

Low pitch sound (use of bell of stethoscope)

- S₃, S₄ obstruction of AV valve (MV, TV)

Classification of Murmurs: (Systolic, Diastolic and Continuous)

I. Systolic Murmurs (SM)

- Stenosis of semilunar valve causes delay in peaking of SM related to prolongation of ejection. The duration not the intensity is proportional to severity of obstruction.
- Commonly encountered clinic problem is the differentiation of AS Vs benign aortic sclerosis. With aortic sclerosis there should be no clinical, ECG, or radiological evidence of heart disease. The carotid upstroke is normal. The SM peaks early with normal S₂.

- MR murmur is usually pansystolic, It can be late systolic in timing (suspect MVP, papillary muscle dysfunction). It can also be early systolic in acute severe MR with markedly increased LA pressure reducing late systolic LV-LA gradient.
- The SM of TR is best heard at LLSB or over xyphisternum and associated with large V-wave.
- The murmur of VSD parallels the pressure difference between the two ventricles. The murmur is typically pansystolic and associated with thrill. With significant pulmonary HTN, the murmur duration shortens.
- SM heard in the back may be caused by coarctation, aortic dissection, peripheral PA stenosis or pulmonary AV fistula.

II. *Diastolic Murmurs*

- Early diastolic murmurs:

Aortic regurgitation: configuration of the murmur reflects volume and rate of regurgitation flow. Therefore, with chronic AR, the aortic diastolic pressure consistently exceeds the LV diastolic pressure and the murmur is heard throughout diastole. However, with acute AR, the LV diastolic pressure is very high (because the LV is not dilated and unprepared), so the murmur tend to short.

Pulmonary regurgitation murmurs secondary to pulmonary HTN (Graham-steel murmur) is high velocity blowing murmur that can last throughout diastole. While PR without elevation of PA pressure results in mid diastole murmur because the diastolic pressure exerted on PV is minimal in early diastole.

- Mid diastole murmurs
 - Majority originates across mitral or tricuspid valves.
 - Murmur is easily appreciated with increase flow through the valves.
 - Duration of the murmur correlates with severity.

- Mid diastolic murmurs occur with: (1) obstruction of AV valves (MS, TS, Austin flint) and (2) Increase flow across AV valves (functional obstruction), e.g., ASD, VSD.
- Late diastolic murmur (presystolic)
 - Represented by increased flow through obstructed mitral or tricuspid valves during atrial contraction
 - Can be heard (opposite to common belief) in MS or TS in patients with atrial contraction (AF)

III. Continuous Murmurs – due to

- (1) Aortopulmonary connection (PDA)
- (2) AV connection (AV fistula, anomalous origin of left coronary from PA)
- (3) Disturbance of flow in arteries or veins (mammary soufflé, cervical venous hum)

Common Valvular Lesions

MS: Malor flush, small volume pulse, \uparrow S₁, opening snap, mid diastolic murmur (MDM), and presystolic accentuation

Signs of Severity: 1) duration of murmurs (longer is severe)
2) S₁ — OS interval (shorter is severe)

MR: Soft S₁, wide splitting of S₂, S₃ present, systolic murmur (pansystolic, early, mid or late systolic)

Signs of Severity: Longer duration or confined to early systole

AS: Slow-rising pulse, S₁, (N or soft), S₂ (single, paradoxical splitting), S₄ present, ejection click if valve is mobile, ejection systolic murmur with late peaking.

Signs of Severity: 1. Single S₂ or paradoxical splitting
2. Longer murmur
3. Late peaking of murmur.

AR: Peripheral signs with chronic not acute AR. S₁ (soft), S₂ (absent, single), S₃ present.

Early diastolic murmur – short in acute AR

Ejection systolic murmur due to increase flow across AV

Austin flint – MDM due to narrowed mitral valve due to rising ventricular diastolic pressure

Signs of Severity: 1) Duration of murmur – longer is severe in chronic
2) Systolic ejection flow murmur
3) Austin flint
4) Reduced diastolic BP

T.S.: Prominent A-wave, OS, MDM at LSB with presystolic accentuation

Both the OS and the murmur are accentuated with inspiration, leg raising, squatting and manoeuvres that increase transtricuspid valve flow

Signs of Severity: 1) duration of murmurs (longer is severe)
3) S₁ — OS interval (shorter is severe)

TR: Prominent V-wave, pulsatile liver, soft S₁, S₃ present, pansystolic murmur at LSB increase in inspiration

Signs of Severity: Peripheral signs with TR, longer duration of the murmur

PS: Ejection click, S₁ (N)

S₂ (wide splitting), S₄ present

Ejection systolic murmur increase with inspiration

PR: S₁ (soft), S₂ wide splitting

S₃ present, S₄ present

Diastolic murmur increase in inspiration. If secondary to PHTN it begins in very early diastole. If secondary to organic valve lesion, it begins slightly later and ends in mid diastole.

Pulmonary HTN: Prominent A-wave

Early systolic click (sudden opening of P.V. into high pressure artery)

Prominent P₂ (due to increase force of pulmonary valve closure)

Left parasternal left (RVH)

Mid systolic ejection murmur (turbulent transvalvular pulmonary flow)

Signs of Severity:

1. Murmur of PR
2. Murmur and peripheral signs of TR
3. If advanced pulmonary HTN – look for signs of RVF

Manoeuvres to Differentiate Between Different Murmurs

Valsalva: During active strain phase most murmurs decrease in intensity except murmur of: 1) HOCM – typically get louder and 2) MVP – longer and louder

Respiration: Right sided sounds and murmurs get louder with inspiration (except for pulmonary ejection click)

Handgrip: By increasing BP (afterload), augments murmur of MR, AR but does not affect murmur of AS and tends to decrease murmur of HOCM

Prompt Squatting: Causes a rapid increase in venous return and increase in peripheral resistance. Because LV volume and peripheral resistance increase, the murmur of HOCM gets softer. The murmurs of MR and AR get louder because of increase resistance

Extrasystolic beats: Both HOCM and AS murmur get louder because of increased post-extrasystolic contractility, while MR murmur is unaffected.