CVS

STEPS OF EXAMINATION

(1) APPROACH THE PATIENT

- Read the instructions carefully for clues
- Approach the right hand side of the patient, shake hands, introduce yourself
- Ask permission to examine him "**I am just going to feel your pulse and listen to your heart; is that alright**?"
- Adjust the backrest so that the patient reclines at **45° to the mattress**
- Expose the top half completely

(2) GENERAL INSPECTION

STEPS	POSSIBLE FINDINGS
1. **Scan the patient**.	➢ Nutritional status: under/average built or overweight ➢ Abnormal facies: Marfanoid (AA, AR, MR), Down's (VSD), Turner's (coarctation, bicuspid AV, AS), Noonan's (PS), malar flush (MS) ➢ Dysponea (tachyponea + use of accessory muscles of respiration; the **s**calene and the **s**ternocleidomastoid) ➢ Earlobe creases → increased incidence of coronary artery disease (see theoretical notes)
2. Examine the **eyes**. Pull down the eyelid.	➢ Anaemia (pallor) in the conjunctivae at the guttering between the eyeball and the lower lid ➢ Cornea (arcus senilis) ➢ Pupil (Argyll-Robertson pupil) → consider syphilitic aortitis
3. Examine the **mouth**. Ask the patient to protrude his tongue. Teeth must be examined in all cases (infective endocarditis)	➢ Central cyanosis in the under-surface of the tongue (see theoretical notes for types of cyanosis)
4. Examine the **hands**: tell the patient "outstretch your hands like this (dorsum facing upwards)"… then "like this (palms facing upwards)"… demonstrate.	➢ Clubbing (congenital heart disease, infective endocarditis, atrial myxoma) ➢ Cyanosis (could be peripheral or central) ➢ Capillary pulsations (AR, PDA) ➢ Splinter haemorrhage, Osler's nodes and Janeway lesions (infective endocarditis)… see theoretical notes ➢ Palmer erythema (consider CO2 retention) ➢ Arachnodactyly (see theoretical notes) ➢ Xanthomas (dyslipidaemia) ➢ Cool peripheries (poor flow - hyperdynamic circulation)

(3) PULSE

STEPS	POSSIBLE FINDINGS
1. **Radial** pulse: ▪ In patients with AF, re-measure the rate by auscultation at cardiac apex, and calculate the pulse deficit ▪ If you suspect complete heart block, recount the pulse while standing (in complete heart block, HR does not increase on standing)	➢ Check for rhythm and rate.
2. Feel the **opposite radial** simultaneously.	➢ Check for any difference in pulse volume…see theoretical notes for causes of absent radial pulse
3. **Radio-femoral delay:** firmly apply the right thumb just below the mid-inguinal point while feeling the radial with your left fingers.	➢ In coarctation of aorta, femoral pulses are of low volume and delayed relative to radial pulse
4. Check for **collapsing pulse:** left up the arm and put the palmer aspect of the four fingers of your left hand on the patient's wrist just below where you can easily feel the radial pulse. Press gently with your palm, lift the patient's hand above his head and then place your right palm over the patient's axillary artery:	➢ If the pulse has a water-hammer character you will feel a flick (a sharp & tall up-stroke and an abrupt down-stroke) which will run across all four fingers and at the same time you may feel a flick of the axillary artery against your right palm ➢ If the pulse has a collapsing character but is not a frank water-hammer type then the flick runs across only two or three fingers
5. Glance at the **antecubital fossa** for catheter scars. Palpate the right brachial with your right thumb.	➢ Check for abnormal pulse volume or character
6. Glance at the carotid for Corrigan's sign (visible carotid pulsation in AR). Palpate the right **carotid** pulse with the tip of your left thumb (between the larynx and the mid point of the anterior border of the sternocleidomastoid) using gentle pressure backwards.	➢ Check for abnormal pulse volume or character (see theoretical notes for components of carotid pulse and abnormalities of the pulse volume and character)

(4) JVP: examine right JVP.

STEPS	POSSIBLE FINDINGS
1. The patient should be lying at 45 degrees and neck is fully supported by pillows so the sternomastoids are fully relaxed. The head should be turned slightly to the left side and a light shone obliquely across the neck to maximize the shadows of right venous pulsations	➢ Recognize the JVP and differentiate it from arterial pulsation (see theoretical notes) ➢ Identify the height in centimetres vertically above the sternal angle (normal 3-5 cm).
2. While observing the right JVP, palpate the left carotid with your right thumb to time the JVP waves in relation to the carotid pulse	➢ Check for abnormal waveforms (see theoretical notes for normal JVP and abnormalities of the JVP
3. If JVP is not visible, consider applying manoeuvres to check for low or very high levels (see theoretical notes)	

(5) <u>LOCAL INSPECTION</u>:

- Scars: median sternotomy scar**,** left lateral/inframammary thoracotomy scar
- Devices: pacemaker/AICD implant

(6) APEX BEAT

STEPS	POSSIBLE FINDINGS
1. Localize the apex beat first by inspection then by laying your fingers on the chest parallel to the intercostal spaces 2. If you cannot feel it, ask the patient to roll onto the left side). Then stand the index finger on it to localize the point of maximum impulse and assess the extent of its thrust	➢ Apex beat is defined as the most inferior and most lateral point of cardiac pulsation. ➢ A normal apical impulse briefly lifts the palpating fingers (just palpable) and is localized (in the 5th ICS medial to the left MCL)…see theoretical notes for abnormalities of the apex beat

(7) PALPATION

STEPS	POSSIBLE FINDINGS
1. **Mitral area**: place your hand from the lower left sternal edge to the apex	➢ Palpable S1 (tapping impulse of MS) ➢ Palpable S3 (prominent early diastolic rapid-filling wave), often accompanied by a third heart sound in patients with left ventricular failure or mitral valve regurgitation ➢ Palpable S4 (marked presystolic distension of the left ventricle), often accompanied by a fourth heart sound in patients with an excessive left ventricular pressure load or myocardial ischemia/infarction ➢ Systolic thrill of MR (acute MR is associated with thrill in one-half of cases) ➢ Diastolic thrill of MS (uncommon- best felt with the patient in the left lateral position)
2. **Left parasternal edge:** place the flat of your right palm (or the heel of your hand) parasternally over the left parasternal edge and apply sustained and gentle pressure. Ask the patient to hold his breathing in expiration.	➢ Left parasternal lift: starts in early systole and is synchronous with the LV apical impulse (See theoretical notes for causes of left parasternal lift). ➢ Systolic thrill of VSD or HCM ➢ Diastolic thrill of AR (uncommon- best felt along the left sternal border with the patient leaning forwards and holding his breath after expiration)
3. **Upper left sternal edge** using the flat or ulnar border of the hand**.** Check for**:**	➢ Palpable P2 in pulmonary hypertension ➢ Thrill of PS, PDA, or ruptured congenital sinus of Valsalva aneurysm ➢ Palpable pulmonary artery pulsations in pulmonary hypertension, increased pulmonary blood flow (ASD) or poststenotic pulmonary artery dilation.
4. Upper right sternal edge using the flat or ulnar border of the hand. Check for	➢ Systolic thrill of AS (may also be palpable at the apex, the lower sternum, or in the neck- best felt with the patient leaning forwards and holding his breath after expiration). N.B. thrill of subclavian artery stenosis may be heard over the subclavicular area.

(8) AUSCULTATION

STEPS	POSSIBLE FINDINGS
1. Listen at the **apex with the diaphragm** (time with the right carotid). If you hear systolic murmur (probably MR) → repeat on expiration, listen at the axilla and feel for thrill. 2. Listen at the **apex with the bell** (using light pressure). Repeat with patient in **left lateral position** and his breath held after expiration (If unsure about the presence of mid-diastolic murmur → you may ask the patient to touch her toes and then reclines 10 times). If you hear mid-diastolic murmur (probably MS) → time with the carotid and feel for thrill 3. Reposition the patient and listen with the diaphragm over the **lower left sternal edge**. If you hear systolic murmur (probably TR/VSD) time with the carotid, repeat on inspiration and feel for thrill. 4. Listen with the diaphragm over the **upper left sternal edge,** and the **upper right sternal edge**. If you hear systolic murmur (probably AS/PS) → time with the carotid and feel for thrill. 5. Auscultate **both carotids** (for bruits and radiated murmurs) 6. Ask the patient to **sit up and lean forwards** with his breath held after expiration. Listen over the right 2nd interspace and the left 3rd interspace. If you hear diastolic murmur (probably AR) → time with the carotid and feel for thrill. 7. Listen over the **lung bases** (for basal crackles and radiating murmurs) and check for sacral oedema	During auscultation at any area, identify and describe the following: ➢ **S1 & S2** (see theoretical notes for recognition and abnormalities of S1 & S2) S1 Just precedes the carotid pulsation, and S2 follows it)**:** ▪ Normally both are low pitched, best heard with the bell of the stethoscope ▪ See theoretical notes for ➢ Extra sound that may precede S1 (see theoretical notes for features and causes): 1. **S4** ➢ Extra sounds that may follow S1 (see theoretical notes for features and causes): 1. **Ejection click** 2. **Opening click of prosthetic AV** 3. **Non-Ejection Click of MVP** ➢ Extra sounds that may follow S2 (see theoretical notes for features and causes): 1. **S3** 2. **Opening Snap** 3. **Opening Click of prosthetic MV** 4. **Pericardial knock** 5. **Split S2** ➢ **Pericardial rub** (occupies both systole and diastole; quality is noisy) ➢ **Murmurs**: see the following theoretical notes: ▪ Innocent murmur ▪ Pathological murmurs ▪ The grades of murmurs ▪ Systolic murmurs ▪ Diastolic murmurs ▪ Continuous murmurs ▪ Differentiation between murmurs of TR and MR ▪ Secondary murmurs in valvular lesions

(9) ADDITIONAL SIGNS

- Lower limb:
  - Feel for **ankle edema**
  - Palpate a **peripheral pulse** (pedal or posterior tibial)
  - Check for **saphenous vein harvest** used in bypass surgery
  - Check for **differential cyanosis** (cyanosis in LL rather than the UL is seen in PDA with PH)
  - Glance at the groins for **angiography scar/bruising** and consider **auscultating the femoral artery** for Duroziez's sign (audible femoral bruits) and Traube's sign (pistol shots), if you suspect AR (see theoretical notes for peripheral signs of AR)
- Examine (or ask to examine) the **Blood pressure** particularly in patients with AS/AR (if you suspect AR ask for both the brachial and the popliteal BP):
  - Check that the sphygmomanometer is set at zero, and ensure you have the correct cuff size (standard cuff will give falsely elevated readings with obese subjects)
  - Support the patient's arm comfortably at about heart level, and apply the cuff to the upper arm with the centre of the bladder over the brachial artery
  - Inflate whilst palpating the brachial artery until the pulse is impalpable (this is the systolic pressure by palpation)
  - Inflate the cuff another 10-20 mmHg, and apply the stethoscope (bell or diaphragm) to the brachial artery
  - Deflate the cuff slowly (2 mmHg/sec.) whilst listening through the stethoscope over the brachial artery.
  - The point at which you hear the first Korotkoff sound is the systolic pressure, and the point at which sound disappears (fifth Korotkoff sound) is the diastolic pressure. The fourth Korotkoff sound (the point of muffling) is acceptable in patients in whom muffled sounds persist and do not disappear.
  - Record measurements to the nearest 2 mmHg
  - Tell the examiner that you would also check for a postural fall in blood pressure
  - Be alert to a wide pulse pressure (AR) and a narrow pulse pressure (AS)
  - Be aware of the significance of differences > 15-20 mmHg in BP between upper and lower limbs (coarctation of aorta)
- Palpate (or ask to palpate) **the liver** particularly in TR/CHF:
  - Check for Pulsatile liver in TR especially if you have seen a large v wave and heard a pan-systolic murmur over the tricuspid area, in such cases you may be able to demonstrate a pulsatile liver by placing your left palm posteriorly and the right palm anteriorly over the enlarged liver
  - Check for tender liver in CHF

(10) THANK THE PATIENT AND COVER HIM (HER)

THEORETICAL NOTES

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 revered 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)

GRADES OF EARLOBES CREASES (associated statistically with CAD in most population groups):

- Grade 3= a diagonal crease in the lobule of the auricle (Frank's sign)
- Grade 2A= crease more than halfway across the lobe
- Grade 2B= crease across the whole lobe, but superficial
- Grade 1= lesser degrees of wrinkling.

ARACHNODACTYLY: abnormally long and slender fingers; usually associated with excessive height and congenital defects of the heart and eyes in Marfan's syndrome

OSLER'S NODES: small, tender, purplish erythematous skin lesions due to infected micro-emboli and occurring most frequently in the pads of the fingers or toes and in the palms of the hands or soles of the feet.

JANEWAY LESIONS: slightly raised, non-tender haemorrhagic lesions in the palms of hands and soles of the feet

SPLINTER HAEMORRHAGES: are Janeway lesions occurring under the nail-beds

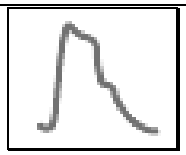
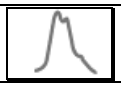
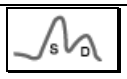
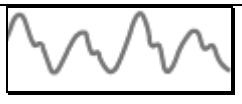
CAUSES OF ABSENT RADIAL PULSE:

- Congenital
- Traumatic
- Surgery (Blalock shunt, cardiac catheter)
- Systemic embolization (e.g. AF with MS)

COMPONENTS OF CAROTID PULSE:

Percussion wave: the shock wave transmitted up the elastic wall of the artery
Tidal wave: the forward-moving column of blood follows the percussion wave and is normally palpable separately
Dicrotic notch: occurs with aortic valve closure

ABNORMALITIES OF THE PULSE VOLUME AND CHARACTER:

Abnormality	Causes
Large volume (bounding or hyperkinetic pulse) 	Large stroke volume**:** ▪ **AR**, bradycardia (e.g., CHB) High cardiac output state**:** ▪ Physiological: exercise, emotion, heat, pregnancy ▪ Pathological: fever, sepsis, thyrotoxicosis, anaemia, peripheral A-V shunts, and Paget's disease of bone
Large volume collapsing pulse: a large volume pulse characterized by a short duration with a brisk rise and rapid fall (due to rapid diastolic run-off from the aorta)	▪ **Severe AR, PDA**
Small volume or hypokinetic pulse (pulsus parvus) 	▪ HF ▪ Obstructive valvular (**AS/MS**) or vascular (PAD) disease ▪ Hypovolaemia (thin thready pulse), ▪ Restrictive pericardial disease ▪ During tachyarrhythmias
Small volume , slow-rising, "plateau", or "anacrotic" pulse (pulsus parvus et tardus): slow rising pulse with a delayed percussion wave and sometimes a palpable judder "anacrotic notch" on the upstroke 	▪ **Severe AS**
Small volume collapsing pulse: there is a quickly rising percussion wave but it is small	▪ Ventricular run off states: **MR** (pulse in MR is sometimes described as jerky), **VSD**
Bisferiens "biphasic" pulse: has two systolic peaks separated by a distinct midsystolic dip 	▪ **Mixed AVD**, occasionally in HCM
Jerky pulse: sharp early percussion wave due to rapid ejection followed by jerky late systolic phase as the dynamic obstruction supervenes. This is followed by a smaller and more slowly rising wave "tidal wave" due to continued ventricular ejection and reflected waves from the periphery	▪ HCM (pulse in HCM is sometimes described as bifid or bisferiens)
Dicrotic pulse has two palpable waves, one in systole and one in diastole (in most normal persons, a dicrotic wave is not palpable) 	▪ Very low stroke volume states: DCM
Pulsus alternans: alternating large and small volume beats in the presence of a regular rhythm; often initially noted when taking the BP 	▪ Advanced HF (poor prognosis) ▪ During or following paroxysmal tachycardia ▪ For several beats following a premature beat in patients without heart disease
Paradoxical pulse: an exaggerated decrease in pulse volume on inspiration, corresponding to inspiratory decline of SBP > 10-15 mmHg	▪ pericardial tamponade ▪ constrictive pericarditis ▪ severe asthma

HOW TO DIFFERENTIATE JVP FROM ARTERIAL PULSATION

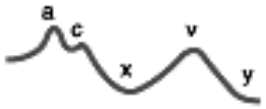
JVP	Arterial pulsation
Two peaks with rapid inward movement	One peak with rapid outward movement
Impalpable and diminished by pressure at the root of the neck	Palpable and unaffected by pressure at the root of the neck
Has a definite upper level, which falls during inspiration, rises with abdominal pressure, and varies with position of patient	Independent of respiration, abdominal pressure, and position of patient

MANOEUVRES TO CHECK FOR INVISIBLE JVP (LOW OR VERY HIGH JVP LEVELS)

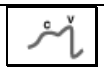
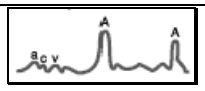
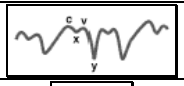
- If JVP is invisible, check for a low level by:
  - pressing firmly on the liver (or the centre of the abdomen) for a few seconds after explaining to patient (this transiently increases the JVP by 2-3 cm)
  - Lying patient more horizontally
- If JVP is still invisible, check for a very high level by:
  - Looking at earlobe (this may be oscillating with cardiac cycle)
  - Sitting the patient vertical
  - If the pressure is very high, the hand veins may be used as a manometer as they collapse when the hand is held at the appropriate height above the right atrium.

NORMAL JVP

a wave: atrial contraction
c wave: closure of TV
x descent: atrial relaxation
v wave: passive filling of RA against closed TV
y descent: emptying of RA into RV upon opening of TV



ABNORMALITIES OF THE JVP:

Abnormal JVP	Causes
Elevation (> 4 cm)	▪ Volume overload commonly HF (sustained abdomino-jugular reflux) ▪ Pericardial effusion (prominent "Y" descent) ▪ Pericardial constriction (Kussmaul's sign) ▪ SVC obstruction (non-pulsatile) ▪ Pulmonary embolism
Systolic V wave (cV wave): synchronous with the carotid pulse and sometimes oscillate the earlobe, and usually associated with peripheral edema and pulsatile liver 	▪ TR (rapid y descent)
Prominent a wave (comes before the carotid pulsation) 	▪ TS or PS ▪ Pulmonary hypertension (MVD, corpulmonale)
Cannon (giant a) wave (AV dissociation) 	▪ **Irregular:** CHB ▪ **Regular**: nodal rhythm, VT, or ventricular paced rhythm
absent a wave 	▪ AF
Steep x descent 	▪ Constrictive pericarditis ▪ Tamponade ▪ Restrictive CM
Inspiratory filling (Kussmaul's sign)	▪ Constrictive pericarditis ▪ Tamponade ▪ Severe asthma

ABNORMALITIES OF THE APEX BEAT:

Abnormal apex beat	Causes
Impalpable apex beat	▪ Overweight or muscular subjects ▪ Asthma or emphysema ▪ Pericardial effusion or dextrocardia
Displaced apex	▪ Chest deformity (scoliosis, pectus excavatum) ▪ Mediastinal shift: (in these situations trachea may also be deviated) ➢Large pleural effusion, tension pneumothorax (mediastinal shift away from the affected side) ➢Pneumonectomy or lung collapse (mediastinal shift towards the affected side) ▪ LV dilatation: ➢Volume overload (AR, MR, ASD) ➔ apex beat diffusely displaced inferiorly and laterally (hyperdynamic) ➢DCM
Hyperdynamic (lifting, Thrusting): vigorous but not sustained	▪ Volume overload: AR, MR, ASD (displaced inferiorly and laterally)
Sustained (heaving): vigorous and sustained	▪ Pressure overload: AS, hypertension (minimally displaced)
Tapping impulse (Palpable S1)	▪ MS
Double impulse (two apical pulsations with each heart beat)	▪ HCM: due to palpable presystolic atrial impulse "palpable LA gallop" (On occasion, a triple impulse may be palpable, due to a late systolic bulge that occurs when the dynamic obstruction is marked) ▪ Ventricular aneurysm: due to accentuated outward movement in late systole "LV dyskinesia"

SUGGESTING VALVE ABNORMALITIES ACCORDING TO APEX DISPLACEMENT AND PULSE VOLUME (BEFORE AUSCULTATION):

- Displaced apex + large volume pulse ➔ AR
- Displaced apex + small volume pulse ➔ MR
- Undisplaced apex + small volume pulse ➔ AS
- Undisplaced apex + small volume pulse + AF ➔ MS

CAUSES OF LEFT PARASTERNAL LIFT

1. RVH of any cause, e.g. PH (MVD, corpulmonale, ASD) or PS → RV sustained impulse (RV heave), which starts in early systole and is synchronous with the left ventricular apical impulse. It is frequently associated with prominent a wave (and giant v wave if there is secondary TR)
2. Anterior displacement of the right ventricle by an enlarged left atrium in severe mitral regurgitation causes a left parasternal lift, which occurs distinctly later than the left ventricular apical impulse

RECOGNITION OF S1 & S2:

1) Normally both are low pitched, best heard with the bell of the stethoscope
2) The interval S2—S1 (diastole) is longer than S1—S2 (systole)
3) Timing: S1 with the upstroke of carotid pulse & S2 with its descent
4) Quality: S1 is lower in pitch & longer in duration than S2

ABNORMALITIES OF S1:

- **Loud S1**: mobile MS, hyperdynamic states, tachycardiac states, short PR interval, loud tricuspid component (L-R shunt, Epstein's anomaly)
- **Soft S1**: immobile MS, hypodynamic states, MR, poor ventricular function (HF), long PR interval
- **Wide splitting** (normally single or narrowly split): RBBB, LBBB, VT, deep inspiration
- **Variable S1**: AF, CHB
- **Metallic S1**: metallic closing click of prosthetic MV

ABNORMALITIES OF S2:

- **Loud S2**: Hypertension, tachycardia states, loud P2 (PH, ASD)
- **Soft S2**: severe AS
- **Persistent splitting**: delayed P2 (RBBB, PS, deep inspiration), early A2 (MR)
- **Fixed splitting**: ASD
- **Single S2**: inaudible A2 (severe AS, large VSD), inaudible P2 (severe PS, F4, pulmonary atresia, elderly, complete TGA), synchrony of A2 & P2 (Eisenmenger's)
- **Reversed splitting**: delayed A2 (LBBB, AS, HCM), early P2 (RV pacing, PDA, WPW type B)
- **Metallic S2**: metallic closing click of prosthetic AV

EXTRA SOUND THAT MAY PRECEDE S1

Extra sound	Features	Causes
1. **S4:** produced in the ventricle during late ventricular filling, due to the atrial contraction that fills a stiff ventricle	▪ Precedes S1 (presystolic) ▪ Low pitched (best heard with the bell of the stethoscope) ▪ LV S4 is loudest at the apex ▪ RV S4 is loudest over the left sternal border ▪ S4 is Absent in patients with atrial fibrillation	▪ LV S4: HTN, AS, LVH, amyloid, HCM, IHD, acute MI, and acute MR. ▪ RV S4: RVH secondary to either PS or PH

EXTRA SOUND THAT MAY FOLLOW S1

Extra sound	Features	Causes
1. **Ejection click:** occurs due to semi-lunar valve stenosis or dilation of the aorta or pulmonary artery	▪ Closely follows S1 (in early systole) ▪ Sharp (clicky), high-pitched ▪ Aortic EC is heard best at the apex and the second right intercostal space (first aortic area) ▪ Pulmonary EC is loudest at the upper left sternal border (pulmonary area). ▪ Usually followed by ESM (lub-k-voo-dub)	▪ Aortic EC is heard in AS & some congenital heart disease ▪ Pulmonary EC is heard in PS & PH
2. **Non-ejection (mid systolic) click:** probably result from chordae tendineae that are functionally unequal in length on either or both AV valves	▪ May be single or multiple, and may occur at any time in systole but usually later than the EC ▪ Heard best along the lower left sternal border and at the apex. ▪ Occurs with or without a late systolic murmur	▪ MVP ▪ Tricuspid valve prolapse
3. **Opening click of prosthetic AV**		▪ Prosthetic AV

EXTRA SOUNDS THAT MAY FOLLOW S2

Extra sound	Features	Causes
1. **Split S2:** splitting occurs normally during inspiration, when the augmented inflow into the RV increases its SV and ejection time and thus delays closure of the pulmonic valve. Splitting that persists with expiration is usually abnormal.	▪ Heard best at the pulmonary area & may be along the left sternal border	▪ Normal finding with inspiration ▪ Persistent splitting that is wider during inspiration than during expiration occurs due to either: ➢ Delayed P2 (RBBB, PS, PE, RVF, ASD) ➢ Early A2 (MR) ▪ Wide fixed splitting (ASD)
2. **S3:** produced in the ventricle due to rapid ventricular filling	▪ Low pitched (more muffled than S2 - best heard with the bell of the stethoscope) ▪ S2—S3 interval is longer than A2—P2 interval (help in differentiating S3 from split S2) ▪ LV S_3 is best heard at the apex ▪ RV S4 is best heard at the left sternal border or just beneath the xiphoid and is often accompanied by the systolic murmur of functional TR.	▪ Normal finding in children ▪ abnormal in adults & indicates either: ➢ HF ➢ The ventricle fills rapidly with a large volume (L-R shunt, MR, AR, TR)
3. **Pericardial knock:** occurs due to abrupt cessation of diastolic filling that occurs when further ventricular relaxation is impeded by the rigid pericardium	▪ Earlier and higher-pitched than S3	▪ Constrictive pericarditis
4. **Opening snap**	▪ Brief, high-pitched (snappy) ▪ Early diastolic ▪ Best heard at the lower left sternal border and radiates well to the base of the heart. However, it may be heard all over the precordium (wide propagation) ▪ Nearly always followed by a diastolic rumble (lub-ta-ta-roo) ▪ Distance from S2 is variable according to severity of the MS, but always longer than A2—P2 and shorter than S2—S3 ▪ In the second intercostal space, an OS is often confused with P_2. However, careful auscultation will reveal both components of S_2, followed by the OS (lub-ta-ta). ▪ The OS of TS occurs later in diastole than the mitral OS and is often overlooked in patients with more prominent mitral valve	▪ MS ▪ TS
5. **Opening click of prosthetic MV**	▪	▪ Prosthetic MV

INNOCENT MURMUR:

- Ejection systolic
- Between LSE and pulmonary area, occasionally apical
- No thrill, added sounds, or cardiomegaly
- Normal ECG, CXR and echocardiography

PATHOLOGICAL MURMURS are either **organic** (valvular or subvalvular) or **functional** (dilated valve ring or increased flow through the valve). Characteristics of pathologic murmurs include

- A sound level of grade 3 or louder
- A diastolic murmur
- An increase in intensity when the patient is standing

THE GRADES OF MURMURS: murmurs are graded on a scale from I to VI.

- A grade I is soft intermittent murmur that is usually heard only with special manoeuvres
- A grade IV is a palpable murmur (accompanied by a thrill)
- A grade VI is a murmur that can be appreciated without a stethoscope.

SYSTOLIC MURMURS

Site of maximal intensity	Cause	Features
Systolic murmur with maximal intensity over the **apex** and propagated to the axilla more than to the sternum	1. **MR**	▪ **Timing:** pan-systolic; starts at S1 (S1 may be muffled by the murmur) and reaches up to S2 (not a must) ▪ **Quality:** blowing (high pitched & clear); of uniform intensity ▪ MR caused by MVP ➔ late systolic murmur, usually preceded by mid systolic click
Systolic murmurs with maximal intensity over the **lower left sternal border** and may propagate to the axilla	1. **TR**,	▪ **Timing:** pan-systolic ▪ **Quality:** high pitched; blowing ▪ increased with inspiration (due to negative intra-thoracic pressure that suck more blood to the RA & RV)
	2. **VSD**	▪ **Timing:** pan-systolic but sometimes short (early to mid-systolic) as in cases of VSD associated with pulmonary hypertension or small VSD in the muscular part of the septum ▪ **Quality:** harsher & usually associated with thrill
	3. **Innocent murmur of childhood (Still's murmur)**	▪ **Timing:** short (early to mid-systolic) ▪ **Quality:** buzzing (musical vibratory), soft (grade 2) with uniform medium pitch
Systolic murmurs with maximal intensity over the **Aortic area** (2nd right ICS)	1. **AS**	▪ **Timing:** mid-systolic of long duration ▪ **Quality:** Harsh diamond-shape (crescendo-decrescendo) ▪ In early cases, cusps are mobile (although thickened & fibrosed) ➔ ejection click precedes the ESM ▪ Increased severity of AS ➔ increased duration of the murmur with muffling of S2 (due to rigid calcified valve) ▪ Murmur is selectively propagated to the neck & also to the apex
	2. **Functional ESM** as in case of: ▪ Hyperdynamic circulation ▪ Hypertension ▪ Aortic aneurysm.	▪ **Timing:** mid-systolic ▪ **Quality:** diamond-shape (crescendo-decrescendo) ▪ In hypertension & aortic aneurysm, it's associated with accentuated, ringing S2
Systolic murmurs with maximal intensity over the **pulmonary area** (2nd left ICS)	1. **Congenital PS**	▪ **Timing:** mid-systolic of long duration ▪ **Quality:** Harsh diamond-shape (crescendo-decrescendo) ▪ Associated with split S2 and muffled P2 ▪ When PS is a part of TOF, it's associated with single S2 (A2 only).
	2. **Functional ESM** as in case of: 1. Hyperdynamic circulation 2. increased flow across pulmonary valve (e.g. ASD) 3. Pulmonary hypertension	▪ **Timing:** med-systolic; very short murmur ▪ **Quality:** diamond-shape (crescendo-decrescendo) ▪ Associated with normal S2

DIASTOLIC MURMURS

Site of maximal intensity	Cause	features
Apical mid-diastolic/pre-systolic murmurs (heard with the bell of the stethoscope using light pressure)	1. **Organic MS** (due to narrowing of the valve)	▪ **Timing:** mid-diastolic; separated from S2 ▪ **Quality:** Always of low pitch (rumbling) ▪ Preceded by opening snap and accentuated S1 ▪ Tight lesion → increased duration of the murmur, till it reaches S1, with presystolic accentuation due to atrial contraction ▪ If associated with AF → no effective atrial contraction → no presystolic accentuation & the murmur is variable in length from beat to beat
	2. **Relative (functional) MS** (due to increased blood flow across the valve) as in case of: ▪ MR ▪ L-R shunt (VSD, PDA) ▪ AR (Austin Flint murmur)	▪ **Timing:** mid-diastolic; separated from S2 ▪ **Quality:** Always of low pitch (rumbling) ▪ No opening snap and S1 in normal (not accentuated)
Diastolic murmurs with maximal intensity over the **aortic areas** (2^{nd} right ICS & 3^{rd} left ICS)	1. **Aortic regurgitation**	▪ **Timing:** early diastolic ▪ **Quality:** very high pitched; decrescendo murmur ▪ Associated with peripheral signs of AR (see below) ▪ if AR is due to Syphilis, S2 will be ringing
Diastolic murmurs with maximal intensity over the **pulmonary area** (2^{nd} left ICS)	1. **Pulmonary regurgitation** (more commonly caused by aortic regurgitation murmur propagated from the aortic area)	▪ **Timing:** early diastolic ▪ **Quality:** very high pitched; decrescendo murmur ▪ Associated with signs of pulmonary hypertension (see below)

CONTINUOUS MURMURS

Continuous murmurs should be differentiated from combined ESM & early diastolic murmur associated with double aortic valve disease; especially if murmurs of AS & AR are prolonged & fill the whole cardiac cycle (To & fro murmur)

1. **PDA**: gives continuous murmur differentiated from those of double AVD by being only one murmur continuous all over the cardiac cycle with maximal intensity at S2 and minimal intensity at S1. It sounds like a machine, so called machinery murmur
2. **Venous hum**: only heard over the neck and disappears on pressure over the root of the neck
3. **Mammary soufflé**: only heard over the lactating breast due to associated A-V shunting.

DIFFERENTIATION BETWEEN MURMURS OF TR AND MR: murmur of TR can mimic that of MR (particularly in case of severe pulmonary hypertension, when a very large RV displaces the LV posteriorly). However, the murmur of TR is differentiated by:

- Best heard at the lower left sternal border, and not heard at the axilla or over the spine.
- Increases with inspiration
- Giant v wave in the neck and pulsatile liver

SECONDARY MURMURS IN VALVULAR LESIONS:

Valvular lesion	Basic Murmur	Secondary murmur
Aortic **I**ncompetence	basal early **diastolic** murmur	apical mid **diastolic** murmur (**A**ustin fl**i**nt) due to the aortic regurgitant jet striking the AML, restricting the mitral inflow
MS	apical mid **diastolic** murmur	basal early **diastolic** murmur (Graha**m** **S**teel) due to pulmonary artery dilatation in pulmonary hypertension complicating MS
sever mitral **regurgitation** (without MS)		mid-diastolic flow murmur (without OS) might be audible at the apex
significant aortic **regurgitation** (without AS)		Systolic flow murmur (without EC) might be audible at the heart base.

PERIPHERAL SIGNS OF AORTIC REGURGITATION: these signs are present only in severe chronic aortic incompetence and are usually not clinically helpful.

Head	1. **De Musset sign**—head nodding in time with the heartbeat 2. **Müller sign**—pulsation of the uvula in time with the heartbeat
Neck	3. **Corrigan sign**—forceful carotid upstroke with rapid decline
UL	4. **Collapsing radial pulse** (water hammer pulse) (Corrigan's pulse) 5. **Quincke sign**—marked capillary pulsation in the nail beds, with blanching during diastole with mild nail pressure
LL	6. **Duroziez sign**—systolic and diastolic bruit over the femoral artery (to and fro murmur) on gradual compression of the vessel by the stethoscope bell 7. **Traube sign**—a double sound heard over the femoral artery on compressing the vessel distally; this is the "pistol-shot" sound that may be heard with very severe aortic regurgitation
LL in relation to UL	8. **Hill sign**—increased blood pressure in the legs compared with the arms (≥30 mm Hg discrepancy)

AUSCULTATORY SIGNS OF PULMONARY HYPERTENSION

- Closely split S2 with accentuated P2
- Ejection click
- Functional ESM
- Early diastolic murmur at the LLSB due to dilatation of the pulmonary valve ring

MIXED MVD: WHICH IS DOMINANT?

Dominant MR	Dominant MS
Apex beat displaced, and thrusting S1 muffled S3 rather than OS ECG: LVH, LAD	Apex beat is not displaced, and tapping S1 Loud OS rather than S3 Loud dominant mid-diastolic murmur Evidence of severe PH

MIXED AVD: WHICH IS DOMINANT?

Dominant AR	Dominant AS
Pulse is Collapsing Apex is thrusting, displaced High systolic BP and wide pulse pressure	Pulse is slow rising Apex is heaving, not displaced much Systolic thrill and loud, harsh systolic murmur Low systolic BP and narrow pulse pressure

CAUSES OF SPECIFIC VALVULAR LESIONS

	Mitral	Aortic
Stenosis	▪ RHD, and rarely IE ▪ Congenital (rare) ▪ Carcinoid ▪ Connective tissue disease (SLE) ▪ Mucopolysaccharidoses (glycogen deposits on cusps) ▪ Senile degeneration	▪ RHD, and rarely IE ▪ Congenital bicuspid valve (usually male; presents in sixth decade) ▪ Degenerative calcification (elderly) ▪ Sub-valvular**:** HCM, sub-aortic membrane stenosis ▪ Supra-valvular**:** coarctation of aorta, and conditions of accelerated calcification, e.g. William's syndrome, Paget's disease, ESRD.
Regurgitation	▪ RHD, IE ▪ MVP ▪ IHD (papillary muscle dysfunction) ▪ Dilated LV (functional MR) ▪ Connective tissue diseases (Marfan's) ▪ Infiltration (amyloid) ▪ Associated with ASD (primum) or HCM	▪ RHD, IE ▪ Connective tissue disease**:** Marfan's, rheumatoid, SLE, ankylosing spondylitis, Reiter's, Hurler's, pseudoxanthoma elasticum ▪ Syphilitic aortitis ▪ Aortic dissection / trauma ▪ Hypertension ▪ Bicuspid AV ▪ Ruptured sinus of valsalva aneurysm ▪ VSD with prolapse of right coronary cusp

SIGNS OF SEVERITY OF SPECIFIC VALVULAR LESIONS

	Mitral	Aortic
Stenosis	▪ Early OS (< 60 msec from S2) ▪ Long diastolic murmur (extends throughout diastole) ▪ Signs of pulmonary hypertension (loud P2, RV heave, TR, PR) ▪ SPAP > 50 mmHg ▪ Valve area < 1 cm ▪ Pressure gradient > 10 mmHg	▪ Slow rising pulse with narrow pulse pressure ▪ Systolic thrill ▪ Soft Single S2 or paradoxically split ▪ Late peaking long murmur ▪ Signs of LVF ▪ Orifice area < 1 cm ▪ Mean pressure gradient > 40 mmHg
Regurgitation	▪ Displaced apex ▪ Systolic thrill ▪ S3 ▪ Mid-diastolic flow murmur ▪ Signs of pulmonary hypertension (loud P2, RV heave, TR, PR), or pulmonary congestion	▪ S3 ▪ Lengthening diastolic murmur ▪ Austin Flint murmur ▪ Diastolic blood pressure < 40 ▪ Pulse pressure > 100

INDICATION OF SURGERY IN SPECIFIC VALVULAR LESIONS

	Mitral	Aortic
Stenosis	**1) <u>Severe MS</u>** (valve area < 1 cm) + any of the following: ▪ symptoms: dysponea NYHA III or IV ▪ Pulmonary hypertension (SPAP > 60 mmHg) ▪ MV repair may be considered in patient with recurrent embolic events while receiving adequate anticoagulation and who have valve morphology favorable for repair **2) <u>Moderate MS</u>** (valve area < 1.5 cm) + either symptoms or recurrent embolic events	**1) <u>severe AS</u> +** any of the following: ▪ Symptoms ▪ Patient undergoing CABG or surgery on the aorta or other heart valves ▪ EF < 50% ▪ May be considered in asymptomatic patient if valve area < 0.6 cm, mean gradient > 60 mmHg or abnormal response to exercise **2) <u>Moderate AS</u>** in patient undergoing CABG or surgery on the aorta or other heart valve
Regurgitation	**1) <u>Severe MR</u>** + either: • Symptoms (NYHA II or greater) • EF 30 - 60 % (implies marked LV dysfunction as it should be above normal in chronic MR) – less favourable if EF < 30 % • LVESD ≥ 40 mm (less favourable if LVESD > 55) • Acute sever MR • It is reasonable for asymptomatic patient in experienced surgical centre • SPAP > 50 mmHg • New onset AF	**1) <u>Severe AR</u> +** either: • Symptoms • Patient undergoing CABG or surgery on the aorta or other heart valve • EF 25-50 % (less favourable if EF < 25%) • LVESD > 50 - 55 mm • LVEDD > 70 - 75 mm • Acute severe AR **2) <u>Moderate AR</u>** in patient undergoing CABG or surgery on the ascending aorta

<u>INDICATIONS OF AORTIC BALLOON VALVULOPLASTY</u>

- Paediatric congenital AS
- Palliative for elderly with co-morbidities or before non cardiac surgery

<u>TREATMENT OF MS WITH MITRAL BALLOON VALVULOPLASTY REQUIRES ALL THE FOLLOWING</u>:

- Leaflet tips and chordae are not heavily distorted, thickened or calcified
- Cusps are mobile at the base
- No or mild MR
- No LA thrombus on TEE