Back to Bedside Basics - Pulse, Blood Pressure and Jugular Venous Pulse

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INTRODUCTION

In the era of electrocardiogram and echocardiography, clinical bedside examination is a forgotten art. However accurate these investigations may be, evaluation of the patient is incomplete without a detailed physical examination. In this chapter we shall discuss the basic bedside art and clinical relevance of examination of the arterial pulse, blood pressure and jugular venous pulse.

THE ARTERIAL PULSE

Physiology - The arterial pulse begins with aortic valve opening and ejection of blood from the left ventricle into the aorta. The nature of arterial pulse depends on left ventricular stroke volume, ejection velocity, compliance, distensiblity and capacity of arterial system. The pulse contour is a result of frequency waves produced by antegrade blood flow and reflection of the waves returning from the peripheral circulation.

The central aortic pulse wave is described as early systolic, late systolic and diastolic component (Figure 1). The early systolic component (percussion wave) has a rapid upstroke. It is due to early systolic ejection of blood which is stored in central aorta. The normal pulse has a brief crest which is slightly sustained and somewhat rounded. The mid and late systolic component (anacrotic notch and shoulder) has a rounded summit or peak. It is due to propagation of blood from central aorta to periphery and reflection of the waves from the upper limbs. Diastole is initiated by a negative wave (dicrotic notch). The nadir of dicrotic notch coincides with aortic leaflet closure (A2 component of S2) and the positive wave is due to reflection of waves from the lower limbs. Normally only the systolic

peak is palpable. Percussion wave is more prominent than the tidal wave. Anacrotic notch, tidal wave, dicrotic notch and dicrotic wave are not palpable.

As the pulse wave travels from aortic valve to the peripheral arteries; i) the upstroke becomes steeper, ii) systolic peak becomes high, iii) systolic upstroke time becomes shorter, iv) ejection time increases, v) systolic pressure increases, vi) pulse pressure increases, vii) diastolic pressure decreases, viii) mean pressure decreases, ix) anacrotic shoulder disappears, x) sharp incisura is replaced by a smoother and latter dicrotic notch, followed by a dicrotic wave. The carotid artery is a large artery close to the aortic valve and its contour resembles that of central aortic pulse. Hence the carotid pulse and not the peripheral pulse is used to assess the volume and contour (Table 1).

With aging, arteriosclerosis and hypertension, there is decreased compliance, increased vascular resistance and vasoconstriction of the arterial tree. The noncompliant arterial tree contributes to increased pulse wave velocity and the tidal wave becomes more sustained.

Examination - Varying degrees of pressure is applied with the finger pads of the thumb or first two fingers to assess upstroke, systolic peak and diastolic slope of the pulse (trisection method). Attempt should be made to assess the rate, rhythm, volume, amplitude, contour and stiffness of the arterial wall. Focus on the speed and quality of early rise, peak and drop off of the arterial pulse.

A.   Rate and Rhythm - This information is derived from the radial artery. Count the arterial pulse for 15 s and multiply by 4 to get the pulse rate. The heart rate should be compared with the pulse rate. A pulse deficit of >6 beats/min is suggestive
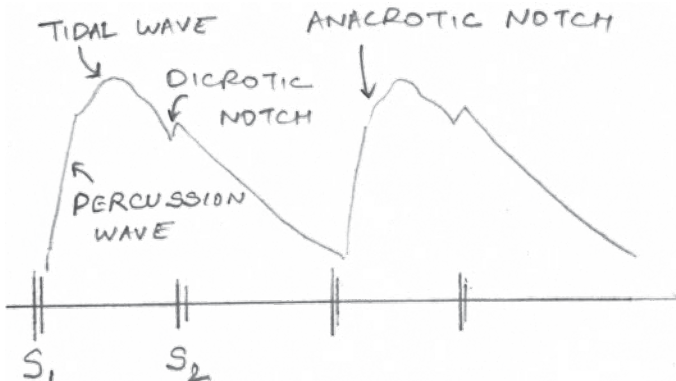
Fig. 1: Normal arterial pulse. Note the rapid upstroke of percussion wave, rounded peak of tidal wave and fall off in late systole. The dicrotic notch coincides with S2

Table 1: Comparison of the pulse in central aorta and peripheral artery	
Central artery (Aorta)	**Peripheral artery (Brachial)**
i. Upstroke has a rounded dome	i. Steep upstroke
ii. Anacrotic notch on ascending limb	ii. Disappearance of anacrotic notch on ascending limb
iii. Descending limb has incisura followed by replaced dicrotic wave by dicrotic wave	iii. Incisura in descending limb is by dicrotic notch followed

| Table 2: Causes of tachycardia and bradycardia	
Tachycardia (>100 beats per minute)	Bradycardia (<60 beats per minute)
a. Sinus tachycardia	a. Sinus Bradycardia
i. Physiological - Infancy, Early childhood, exercise, anxiety, excitement	i. Physiological - Athletes, during sleep
ii Pharmacological - Amyl nitrate, amiodarone, epinephrine, isoproterenol, ephedrine, atropine, alcohol, nicotine, caffeine	ii. Pharmacological - Beta blockers, propafenone, Lithium
iii. Pathological - Cardiac - Heart failure, acute myocardial infarction, pulmonary embolism, myocarditis, shock Non cardiac - Fever, thyrotoxicosis, anemia, hemorrhage, hypotension, hypoxia	iii. Pathological - Cardiac - Inferior wall myocardial infarction, vasovagal syncope, sinoatrial block, post cardiac transplant Non cardiac - myxedema, raised intracranial pressure, hypothermia, obstructive jaundice, enteric fever, sepsis, Chagas disease
b. Tachyarrhythmia - Atrial fibrillation, atrial tachycardia, supraventricular tachycardia, ventricular tachycardia	b. Bradyarrhythmia - complete heart block, second degree AV block.

Table 3: Irregular pulses	
Regularly irregular	Irregularly irregular
i. Sinus arrhythmia	i. Atrial fibrillation
ii. Pulsus bigeminy	ii. Multifocal atrial tachycardia
iii. Pulsus alternans	iii. Frequent premature ventricular contractions
iv. 1st and 2nd degree heart block	

Differentiating between irregular pulses

Ventricular premature complex - There is a large pause following the premature beat.

Atrial premature complex - There is a short pause following two beats

Atrial fibrillation - Irregular in rate and rhythm with apex pulse deficit of >6 beats/min.
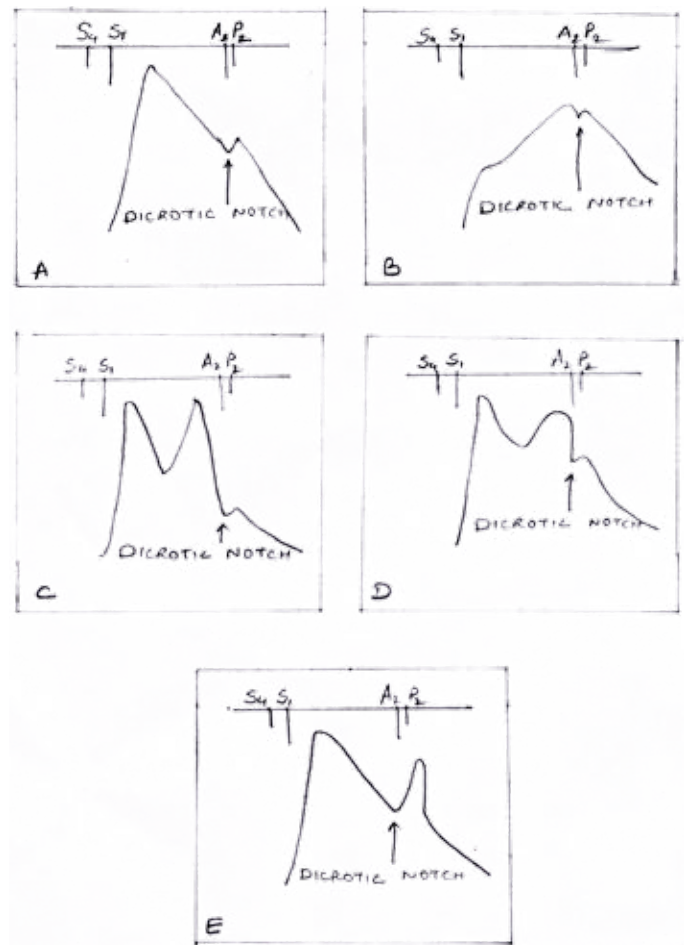
Fig. 2: Carotid pulse waveforms and heart sounds. A - Normal, B - Severe Aortic stenosis - anacrotic pulse with slow upstroke, C - Severe aortic regurgitation - bifid pulse with two systolic peaks, D - Hypertrophic obstructive cardiomyopathy - bifid pulse with spike and dome pattern, E - Dicrotic pulse - one peak in systole, another in diastole

of atrial fibrillation and <6 beats/min is suggestive of premature ventricular contraction. The normal pulse occurs at regular intervals. When the cycle length shortens with inspiration and lengthens with expiration by >120 ms during quiet breathing, it is called sinus arrhythmia. It is associated with autonomic dysfunction and is a risk factor for sudden cardiac death. Normal sinus rhythm varies between 60-100 beats per minute (Tables 2, 3).

B.   Character and volume of the pulse (Figure 2)

a.   Hyperkinetic (Bounding) Pulse - It has larger pulse wave amplitude. It is due to increase in left ventricular ejection, stroke volume, arterial pressure, sympathetic activity or decreased arterial compliance. It is seen in i) elderly subjects with arteriosclerosis and systolic hypertension, ii) anxiety, iii) anemia, iv) thyrotoxicosis, v) exercise, vi) hot and humid environment, vii) alcohol intake and viii) high output states with increased distal arterial runoff like aortic regurgitation, patent ductus arteriosus, large A-V fistula, Paget's disease and severe cirrhosis.

b. Hypokinetic Pulse - A small or diminished pulse is due to low cardiac output with reduced left ventricular stroke volume, shorter left ventricular ejection time or intense vasoconstriction. An unsustained pulse suggests decreased stroke volume without left ventricular outflow obstruction, whereas a slow rising sustained pulse of small volume suggests aortic stenosis. It is seen in i) severe left ventricular dysfunction, ii) congestive cardiac failure, iii) hypotension and iv) left ventricular outflow tract obstruction.

c. Pulsus parvus et tardus - It is a slow rising pulse with delayed systolic peak and upstroke. It is best appreciated with simultaneous auscultation and carotid palpation. In aortic stenosis, it is associated with a carotid thrill (carotid shudder).

d. Water Hammer (Collapsing pulse) or Corrigan pulse or Pulsus Celer - This term was coined by Thomas Watson after a Victorian toy which comprised of a glass vessel partly filled with water and vacuum. It produces a slapping impact on being turned over. In aortic regurgitation, there is an early, brief peak and a swift descent without dicrotic notch which gives a collapsing sensation. To elicit this, the patient's arm is suddenly raised above the head and the wrist grasped with the examiner's hand so that the palm faces the anterior aspect of wrist. The collapsing nature can be felt after each systole. The brief peak is due to rapid ejection of increased stroke volume. The swift descent is due to diastolic run off (back flow into left ventricle), rapid run off to the periphery due to decreased systemic vascular resistance and reflex vasodilatation mediated by carotid baroreceptors. It may also be seen in hyperkinetic circulatory states like patent ductus arteriosus, aortopulmonary window, arteriovenous fistula, rupture of sinus of valsalva into right heart and tetrology of Fallot with bronchopulmonary collaterals.

e. Double beating pulse - Simultaneous auscultation and palpation are necessary to delineate the timing of the twice beating pulse.

i. Bisferiens pulse - The double peaked pulse occurs in systole and is best detected by light but firm compression of the brachial artery with a single finger. It is seen in pure aortic regurgitation, combined aortic stenosis with predominant aortic regurgitation and high output states with normal heart. It is likely to disappear after onset of congestive cardiac failure.

ii. Bifid pulse - A bifid or spike and dome pattern in systole is recorded, but rarely palpable in significant hypertrophic obstructive cardiomyopathy.

iii Dicrotic pulse - The first wave occurs in systole and the second accentuated component is a diastolic reflection wave occurring in diastole. On simultaneous auscultation, S2 separates the two pulse waves. It is better appreciated during inspiration. It is seen in young patients having cardiomyopathy with severe left ventricular dysfunction, low cardiac output, low blood pressure, high systemic vascular resistance, tachycardia, during inspiration in pericardial tamponade, post valve replacement for aortic or mitral regurgitation with left ventricular dysfunction and occasionally with fever in young.

e. Pulsus alternans - It is present during sinus rhythm when patient's peak systolic arterial pressure and pulse volume are alternately strong and weak. It occurs due to beat to beat alteration in left ventricular ejection pressure and signifies severe left ventricular dysfunction. (It is not related to electrical alternans which has a beat to beat variation in the amplitude of QRS complex as seen in massive pericardial effusions). It is best appreciated clinically in the radial or brachial arteries. It may be associated with signs of heart failure like S3 gallop. It can also be detected by slow decompression of the sphygmomanometer cuff while listening to the alteration of Korotkoff sounds. When systolic pressure alternates by >20 mm Hg it can be detected by palpation of the peripheral pulse with patient's breath held in deep expiration. It is accentuated after a PVC, Valsalva maneuver, abrupt upright posture or deep inspiration.

f. Pulsus paradoxus - This term was coined by *Kussmaul*. There is marked and exaggerated inspiratory fall in systolic blood pressure in which palpable peripheral arterial pulse and audible Korotkoff sounds disappear in inspiration. The blood pressure cuff is inflated beyond the peak systolic pressure and slowly deflated. The degree of paradoxus is the difference between the systolic pressure at which the Korotkoff sounds are first heard during expiration and the point at which all beats are well heard during both phases of respiration. The word paradoxus is a misnomer because systolic pressure normally falls during inspiration by 4-6 mm Hg. In pulsus paradoxus the difference is >10 mm Hg. The patient must be breathing quietly and not deep breathing or performing Valsalva maneuver. It is seen in pericardial tamponade, constrictive pericarditis, emphysema, asthma, severe congestive cardiac failure and marked obesity.

Mechanism - Cardiac tamponade is a continuum from effusion to full blown circulatory collapse. The hemodynamic effects depend on the amount of effusion and the pericardial pressure volume relationship. As fluid accumulates in pericardial sac, there is increased left and right sided atrial and ventricular pressures which equalize at a pressure similar to intrapericardial pressure. Inspiration increases the venous return to the right side of the heart at a period when the total heart volume is

Table 4: Grading of pulses
Grading of pulses
Grade 0 - Absent pulse
Grade 1 - Feeble
Grade 2 - Palpable but diminished
Grade 3 - Normal
Grade 4 - High volume/bounding

fixed. This increases the right ventricular diastolic dimensions pushing the interventricular septum to the left. This reduces left ventricular dimensions, compliance and filling. Also inspiratory pooling of blood into the pulmonary circulation causes under filling of the left atrium and left ventricle. The under filled left ventricle in tamponade operates on the steep ascending limb of Starling curve, so inspiratory reduction of left ventricle filling causes a marked depression of stroke volume and systolic pressure.

Reverse pulsus paradoxus may be seen in cardiac tamponade with positive pressure ventilation and isorrhythmic AV dissociation.

Pulsus paradoxus may be absent in cases of cardiac tamponade with atrial septal defect/ ventricular septal defect/ aortic regurgitation/ pericardial adhesions.

C.   Condition of the arterial wall - The examiner must roll the vessel wall between the index and second finger. In young age, it seems to merge with surrounding tissue, in middle age it is palpable and in old age it feels like a cord. Also try to note the irregularity of surface seen in arteriosclerosis and tortuosity.

D.   All pulses should be palpated and compared bilaterally (Tables 4 and 5).

Unequal/ diminished/ absent pulse
i.   Takayusu arteritis
ii.   Thoracic outlet syndrome - cervical rib, scalenus anticus syndrome
iii.   Subclavian steal syndrome
iv.   Atherosclerosis of innominate, left subclavian artery
v.   Acute micro embolism
vi.   Coarctation of aorta
vii.   Dissection of aorta

Radiofemoral Delay - The central aortic pulse reaches the carotids at 40 ms, brachials at 60 ms, femorals at 75 ms and radials at 80 ms. Radiofemoral delay is seen in coarctation of aorta and obstructive diseases of aortic bifurcation, common iliac or external iliac artery. Coarctation should be suspected in patients with hypertension or infants with heart failure. The brachial and carotid pulses are

Table 5: Sites of palpation of various pulses
i.   Superficial temporal artery - It is palpable over the zygomatic process anterior to the tragus.
ii.   Common carotid artery - It terminates at C4 level at the upper border of thyroid cartilage. There it bifurcates into the external and internal carotid artery. It is best felt in the lower half of the patient's neck. Rotating the neck slightly towards the examiner relaxes the sternocleidomastoid. The left thumb is used to palpate the right artery and vice versa.
iii. Subclavian artery - It is effectively compressed against the first rib with shoulder depressed with pressure exerted down back and medially in the angle between the sternocleidomastoid and clavicle.
iv.   Axillary - It is felt against the middle of humerus
v.   Brachial - It is felt against the humerus just above the antecubital fossa medial to biceps tendon.
vi.   Radial - It is felt with the tip of index, middle and ring fingers compressing against the head of the radius with the forearm slightly pronated and wrist slightly flexed.
vii. Femoral - It is felt midway between the iliac crest and pubic ramus below the inguinal ligament against the head of the femur.
viii. Popliteal - The patient lies supine with the knees flexed at 120 degrees. The fingertips of both hands are placed in the popliteal fossa with thumbs resting on the patella.
ix.   Posterior Tibial - The patient's foot should be relaxed between plantar and dorsiflexion. It is felt 1 cm behind the medial malleolus of the tibia.
x.   Dorsalis Pedis - It is best felt between the tendons of extensor hallucis longus and extensor digitorum (between first and second toe) against the first metatarsal. It may be impalpable in 2% of normal individuals.

bounding, whereas lower extremity pulses have a slow rate of rise, late peak and a low pulse pressure. This can be demonstrated by placing the patient's wrist over the femoral artery so that the examiner's fingers at the radial pulse are above the fingers at the femoral pulse. Vary the compression pressure till both pulses are felt equally with both hands.

The radiofemoral delay may be absent when coarctation is associated with bicuspid aortic valve + aortic stenosis/ aortic regurgitation, supravalvular aortic stenosis and mitral regurgitation.

BLOOD PRESSURE
Physiology - The blood pressure is the measure of potential energy or lateral force per unit area of the vessel wall. Systolic blood pressure is the maximum pressure exerted by the heart during systole. It is the amount of

Table 6: Sphygmomanometer cuff size			
Sphygmomanometer cuff size (cm):			
Cuff type	Mid arm circumference	Bladder width	Bladder length
Child	13-20	8	13
Small adult	17-26	11	17
Standard adult	24-33	12-13	22-24
Large adult	33-42	15-17	32-33
Thigh	42-50	18-20	35-42

work done by the heart. Systolic arterial pressure depends on the stroke volume, velocity of left ventricular ejection, distensiblity of vessels and volume of blood at end diastole. Diastolic blood pressure is the minimum pressure exerted during diastole. It is the load against which the heart has to work. The diastolic arterial pressure depends on peripheral arteriolar resistance, cardiac cycle length and compliance of the arterial tree. Cardiac output is the product of heart rate and stroke volume (CO=HRxSV). Arterial blood pressure is the product of cardiac output and peripheral resistance (BP=COxPR). The pulse pressure is the difference between systolic and diastolic blood pressure (approximately 40 mm Hg). Mean blood pressure is the sum of diastolic blood pressure and 1/3rd pulse pressure (approximately 95-100 mm Hg).

Measurement of blood pressure - The blood pressure is measured with an aneroid or mercury manometer. The bladder length/width should be 80%/40% of the arm circumference. The ratio of cuff width to length should be 1:2 (Table 6). The center of the rubber bladder should be on the brachial artery. The blood pressure cuff is wrapped tightly around the arm. The diaphragm or bell of the stethoscope is firmly placed at the brachial artery, so that the upper edge of the stethoscope is in contact with the distal edge of the cuff. The patient should be seated comfortably, back supported, bared upper arm, legs uncrossed with the arm at level of heart. The cuff is inflated 20 mm Hg above the point when the radial pulse is no longer palpable. This palpatory method prevents underestimation of blood pressure due to auscultatory gap. The systolic and diastolic pressures are then estimated by auscultatory method. Cuff should be deflated at <3 mm Hg/sec. The column should be read to the nearest 2 mm Hg. The level of peak systolic pressure is the point at which two consecutive Korotkoff sounds are heard. The disappearance of the Korotkoff sounds is the true diastolic pressure. After every cuff inflation, deflate the cuff completely and allow sufficient time for venous return. There should be no talking between the subject and observer. In severe aortic regurgitation and hyperkinetic circulatory states, the diastolic pressure should be recorded in both phase IV and V. In atrial fibrillation, there is beat to beat variation in blood pressure; hence an average of three readings is taken as a blood pressure. For measurement of lower limb pressure, the patient lies prone; the thigh cuff is wrapped around and auscultate the popliteal fossa. If a thigh cuff is not available, an arm cuff can be wrapped around the lower leg and auscultate

the posterior tibial artery or dorsalis pedis artery with a pediatric bell chest piece.

Korotkoff sounds arise as a result of oscillation from distension of arterial walls with every cardiac impulse due to partial occlusion of the artery by the blood pressure cuff. Shock, low stroke volume and peripheral vasoconstriction cause weak Korotkoff sounds. This can be enhanced by hand elevation or isometric handgrip exercises for 5-10 times.

Phases of Korotkoff sounds

I	Onset of auscultatory sound	Represents peak systole Initially low intensity
II	Swishing sound or murmur	Occurs 10-15 mm Hg below peak systolic pressure Auscultatory gap occurs when phase 2 sounds are soft, absent or missed by examiner
III	Crisp, easily heard	15-20 mm Hg below phase II
IV	Abrupt damping or muffling of tapping sounds	It occurs 5-8 mm Hg above the diastolic sounds
V	Disappearance of sound	Used as true diastolic pressure

Orthostatic hypotension - In a normal individual, systolic blood pressure falls by 10-12 mm Hg and diastolic blood pressure remains same or may increase on standing. Orthostatic hypotension is fall in systolic blood pressure by >20 mm Hg and/or diastolic blood pressure by >10 mm Hg on moving from supine to standing position within 3 minutes. It may or may not be associated with compensatory tachycardia. It manifests as postural giddiness or syncope. It is seen in hypovolemia, baroreflex dysfunction, autonomic insufficiency, diabetics, elderly and with vasodilators (alpha blockers, beta blockers, nitrates, diuretics).

Ambulatory BP monitoring - It gives a record of 24 hours of blood pressure. Blood pressure is higher when awake, alert, mentally or physically active and early morning. It is lower during rest and sleep. Patients in whom the blood pressure does not fall by 10-20% at night or an average greater than 135/85 mm Hg are at a higher risk of cardiovascular events. It is indicated in

Table 7: Differential blood pressure	
Differential BP in upper limbs (>10 mm Hg) mm	**Differential BP in arm and leg (>20 Hg)**
i. Arterial occlusion - embolism, atherosclerosis, Takayusu's arteritis, post cardiac catheterization ii. Dissecting aortic aneurysm iii. Supravalvular aortic stenosis iv. Thoracic outlet syndrome v. Subclavian steal syndrome vi. Error in technique, Physiologic variations	i. Coarctation of aorta ii. Aortic dissection iii. Aortic arch syndrome iv. Subclavian steal syndrome v. Severe aortic regurgitation (Hill's sign)

i.   Drug resistant hypertension

ii.   Drug induced hypotension

iii.   White coat hypertension

iv.   Autonomic dysfunction

v.   Episodic hypertension

Ankle brachial index - It is the ratio of systolic blood pressure in ankle divided by the higher of the two arm systolic pressures (normal >0.9). Values <0.9 is suggestive of peripheral vascular disease, and <0.3 indicates rest pain and critical ischemia.

Blood pressure should be measured in both arms. Normally the difference in both arms is less than 10 mm Hg. Systolic leg pressures exceed arm pressures by <20 mm Hg (Table 7).

A wide pulse pressure arises due to increase in stroke volume and decreased peripheral resistance. A narrow pulse pressure arises due to increased peripheral resistance and decreased intravascular volume (Table 8).

Sources of error in blood pressure measurement

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i. Failure to detect peak systolic pressure	Insufficient inflation, too rapid initial inflation, failure to recognize auscultatory gap
ii. Cuff applied too loose	Falsely elevated readings
iii. Fat arm with small cuff	Pseudohypertension
iv. Thin arm with large cuff	Pseudohypotension
v. Excessive venous congestion in arm	Decreased intensity of Korotkoff sounds
	Widened auscultatory gap
vi. Arm below heart level	Overestimation of blood pressure

Table 8: Causes of narrow and wide pulse pressure	
Wide pulse pressure	**Narrow pulse pressure**
i. Hyperkinetic circulation - pregnancy, exercise, anemia, hyperthyroidism, arteriovenous fistula, hot weather ii. Aortic regurgitation iii. Patent ductus arteriosus iv. Truncus arteriosus v. Complete heart block	i. Heart failure ii. Severe aortic stenosis iii. Diabetic ketoacidosis

vii. Arm above heart level	Underestimation of blood pressure

Jugular venous pulse

Physiology - Careful examination of the venous pulse can provide useful information about the right sided cardiac physiology (Figure 3). When the right atrium contracts, its pressure rises pushing the blood from right atrium to the right ventricle at end of ventricular diastole. It also causes blood to flow retrogradely into the superior vena cava and jugular veins. This produces a positive wave called the A wave. The A wave begins at the peak of the P wave of ECG, immediately prior to S1 and the carotid upstroke. As the right atrium starts relaxing, pressure falls causing the early portion of X descent. Simultaneously, the right ventricular systole commences causing the intraventricular pressure to rise above the atrial pressure. This leads to closure of the tricuspid valve. The upward bulging motion of closed tricuspid valve during isovolumic systole produces a positive wave called C wave. The onset of C wave corresponds to the tricuspid component of S1. It can be confused with transmitted carotid pulsations. It is usually not visible as a separate wave. As the right atrial relaxation continues, right atrial pressure falls during early right ventricular systole. During this phase the tricuspid valve ring is also pulled downwards. The latter part of X descent, X' reaches its lowest point. The X descent begins during systole and ends before S2. The great veins empty into the right atrium during ventricular systole with a closed tricuspid valve. This leads to a rise in the right atrial pressure causing a second positive wave called the V wave. It begins in late systole and ends in early diastole. It is synchronous with the carotid pulse and peaks after S2. At this point of early ventricular diastole, the ventricular pressure falls below the intra atrial pressure causing the tricuspid valve to open and blood flows from right atrium to right ventricle. This causes a negative wave called the Y descent. The H (H from Hirschfelder) wave arises due to passive right heart filling during diastole. In normal individuals, the A wave is larger than the V wave and X descent is more prominent than the Y descent. During inspiration, negative intrathoracic pressure causes increased venous
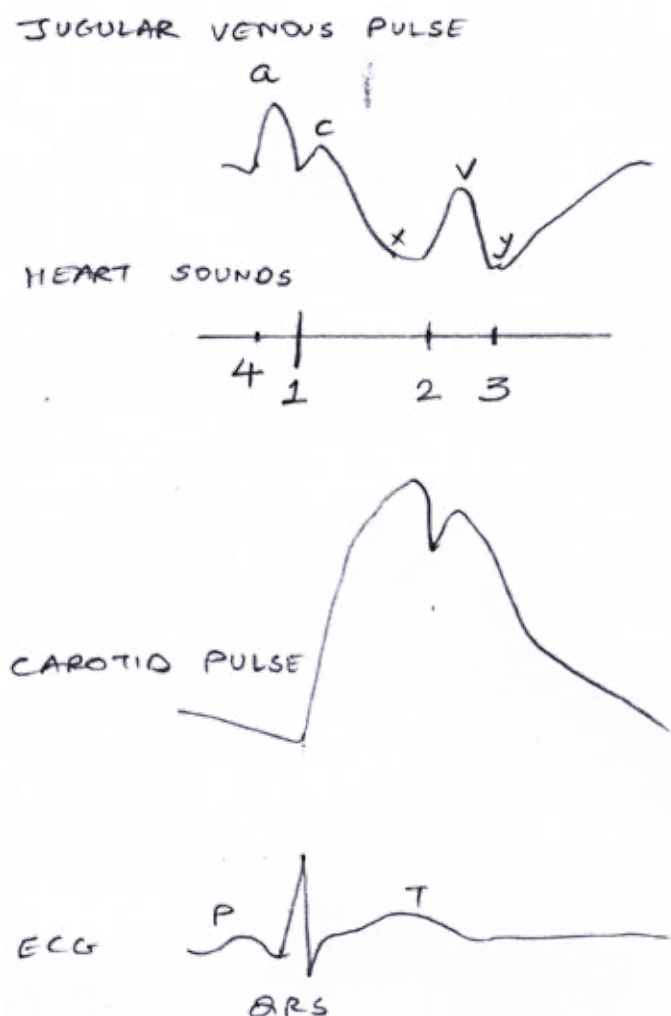
Fig. 3: Normal jugular venous pulse. The large A wave almost coincides with S1 and the V wave coincides with S2. The X descent occurs in systole and Y descent in diastole

	Jugular venous pulse	Carotid arterial pulse

Table 9: Differences between Jugular venous pulse and Carotid arterial pulse

	Jugular venous pulse	Carotid arterial pulse
Location	Low in neck, lateral Better seen than felt	Deep in neck, Medial Better felt than seen
Contour	Two crests and two troughs Descent more obvious than crest	Single brief visible upstroke
Inspiration	A and V waves more prominent	No change
Upright position	Decrease in mean pressure	No change
Compressibility	Can be obliterated with gentle pressure	Not easily compressible
Abdominal compression	Transient increase in pressure	No change

pooling of blood which leads to prominent X and Y descent. During expiration, A wave diminishes and V wave becomes the dominant positive reflection.

Examination - The patient should be reclining comfortably without any tension on neck tissues. The chin is elevated and head rotated to the left. It is preferable to have tangential lighting. Lean over to the left side of the patient while examining the right side of the neck. The sternal angle of Louis is 5 cm above the mid right atrium whether supine, 45 degree or 90 degree position is given. The venous pressure is measured from the angle of Louis. The thorax should be positioned at an angle where the peak of the venous column is well identified. If the venous pressure is too low, place the patient supine with leg elevation and ask him to take deep breaths. If the pressure is too high, the pulsations may be behind the angle of the mandible, so keep the patient at 90 degree and examine. The height of the A and V wave during inspiration is taken as the venous pressure. Two scale method is used. A horizontal scale at the peak of the venous column cuts the vertical scale kept at the angle of Louis. For supine 2 cm is the upper limit of normal and for 45 degree 4.5

cm is the upper limit of normal for venous pulsations. By adding 5 cm, we can obtain the actual venous pressure. If it is not visible, deep inspiration can bring out the waves. The normal level is 4 cm above the angle of Louis which is equal to 9 cm of water or 6 mm Hg.

It is recommended to use the X and Y descent to time the venous pulse. The negative X descent is between S1 and S2 and X' is simultaneous with radial pulse. The A wave is visible as a flickering pulsation just before the carotid pulse is felt. During auscultation, the A wave coincides with S4 and is almost simultaneous with S1. The V wave peaks just after S2 and Y descent begins after the V wave.

Internal jugular pulsations are visible but not palpable. The indirect pulsations of the jugular bulb and internal jugular veins are transmitted to the overlying skin and soft tissue. The internal jugular veins are preferred to the external jugular veins - i) the internal jugular veins have no valves. ii) The external jugulars communicate with superior vena cava with two bends each of nearly 90 degrees whereas internal jugulars communicate directly. iii) External jugular vein passes through many fascial planes and is easily affected by extrinsic compression. iv) Increased sympathetic stimulation in heart failure causes vasoconstriction of external jugular veins. Right internal jugular vein is preferred because it runs a straight line course to the superior vena cava. Left internal jugular vein runs into the left innominate vein which is not a straight line.

Abdominojugular (Hepatojugular) reflux - The patient is positioned such that the upper level of venous column is at mid neck level. Gentle but firm compression is applied with the hand in the right upper quadrant for at least 10
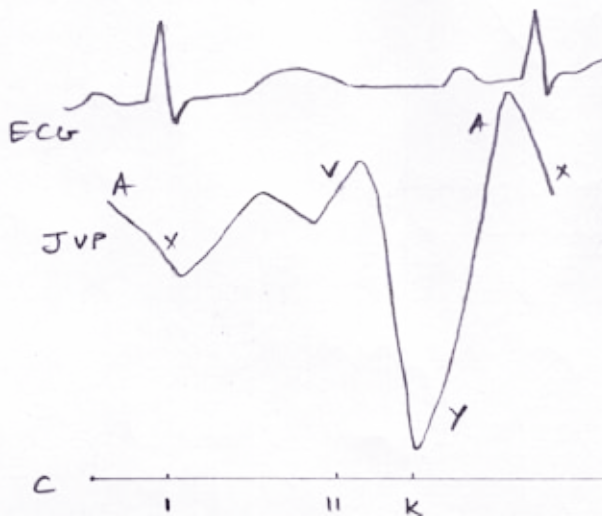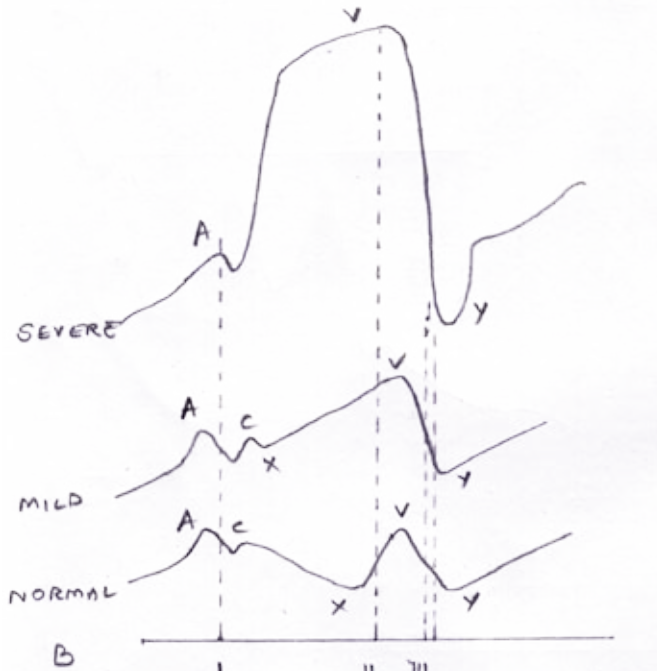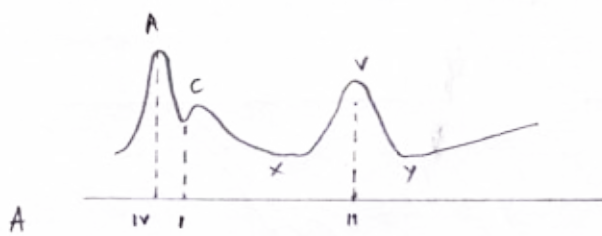
Fig. 4: Abnormal jugular venous waveforms. A - Large A wave associated with reduced RV compliance and elevated RV end diastolic pressure. It corresponds to right sided S4. B - Compared to the normal waveform, in mild TR, there is a prominent V wave. In severe TR, there is ventriculization with a rapid Y descent. C - Constrictive pericarditis with a prominent Y descent which corresponds to the pericardial knock (K)

seconds. The patient is instructed not to strain and breathe comfortably with an open mouth. In a normal person, prolonged pressure will not cause a sustained elevation of venous pressure. Elevation of the venous pressure more than 3 cm for at least 15 seconds is a positive response. It will be positive in latent right ventricular failure, silent tricuspid regurgitation, hypervolemia and systemic vasoconstriction. It predicts heart failure and PA wedge pressure greater than 15mm Hg. Patients with chronic obstructive lung disease may have a false positive test.

Elevated venous pressure can also be estimated by looking at the veins of the dorsum of the hand and under surface of tongue (May's sign)

Differences between Jugular venous pulse and Carotid arterial pulse (Table 9)

ABNORMALITIES OF JUGULAR VENOUS PULSE (FIGURE 4)
Increased jugular venous pressure
i. Reduced right ventricular compliance - pulmonary stenosis, pulmonary artery hypertension, right ventricular failure following left ventricular failure. In right ventricular infarction myocardial necrosis may cause the right ventricle to stiffen leading to elevation of right ventricular filling pressure

ii. Right ventricular inflow impedance - Tricuspid stenosis, right atrial myxoma, constrictive pericarditis

iii. Fluid overload, renal failure

Decreased jugular venous pressure
i. Hypovolemia, dehydration

ii. Hypotension, shock

INCREASED A WAVE AMPLITUDE
Large A waves
i. Increased resistance to right atrial emptying causes increased right atrial contraction - Tricuspid stenosis, right atrial myxoma, tricuspid atresia

ii. Reduced right ventricular compliance is seen in right ventricular hypertrophy. It leads to increased right ventricular end diastolic pressure - severe pulmonary arterial hypertension, pulmonic stenosis, pulmonary vascular disease, acute pulmonary embolism, right ventricular infarction, right ventricular cardiomyopathy

iii. In left ventricular hypertrophy, the hypertrophied interventricular septum alters the pressure volume relationship of right ventricle causing the *Bernheim effect* - seen in hypertrophic cardiomyopathy (symmetric and asymmetric) and valvular aortic stenosis,

Giant or Cannon A waves - Intermittent giant A waves called cannon A waves occur when the right atrium contracts against a closed tricuspid valve. Paul Wood described giant A waves as venous Corrigan.

i. Regular cannon waves - Junctional rhythm,

Ventricular tachycardia with 1:1 retrograde conduction, isorrhythmic AV dissociation

ii. Irregular cannon waves - complete heart block, A-V dissociation, ventricular pacing, ventricular extrasystoles and ventricular tachycardia.

Decreased A wave amplitude - Right atrial injury during surgical cannulation in open heart surgery can cause reduction in A wave amplitude.

Absent A waves - In atrial fibrillation, the atrial kick is lost.

X DESCENT
Absent X descent
i. As severity of tricuspid regurgitation increases, the x descent gets blunt and is replaced by a large regurgitant wave.

ii. In atrial fibrillation, the X trough is reduced in size, but preserved.

Prominent X descent
i. The x descent becomes deeper when vigorous right ventricular contraction occurs - cardiac tamponade, constrictive pericarditis.

ii. In right ventricular overload states the x descent may be prominent - atrial septal defect.

V WAVE
Prominent V wave - In severe cases it may cause head bobbing, systolic pulsations of earlobe, pulsatile exophthalmos and pistol shot sound over the internal jugular veins

i. In tricuspid regurgitation, increased right atrial blood during ventricular systole causes blunting or obliteration of x descent with a prominent v wave called systolic or regurgitant wave (Lanci's sign)

ii. In atrial fibrillation, loss of A waves causes the V waves to become more prominent

iii. Other - large atrial septal defect, ventricular septal defect with left ventricle to right atrial shunt (Gerbode's defect), severe heart failure, cor pulmonale.

Diminished V waves - It is seen in hypovolemic states and hypotension following use of nitrates

Equal A and V waves may be seen in non restrictive atrial septal defect, constrictive pericarditis with increased venous pressure, right ventricular failure in sinus rhythm with increased venous pressure

Y DESCENT
Rapid Y descent (Diastolic collapse) - The Y descent trough is exaggerated due to elevated venous pressure.

i. In constrictive pericarditis, a prominent Y descent is associated with a diastolic filling sound (pericardial knock). This is called Fredrich's sign.

ii. Severe tricuspid regurgitation has a prominent Y descent following the large regurgitant wave

iii. Severe right ventricular failure

iv. Atrial septal defect with mitral regurgitation

The Y descent is diminutive when right atrial emptying or right ventricular filling is hampered - pericardial tamponade, tricuspid stenosis and right atrial myxoma

Kussmaul's sign - Normally during inspiration, due to negative intrathoracic pressure, there is fall in the jugular venous pressure by at least 3 mm Hg. A rise in venous pressure or failure to decrease with inspiration is called Kussmaul's sign. In states of right sided volume overload and reduced right ventricular compliance, the right ventricle cannot accommodate the increased volume and the pressure rises. It is seen in constrictive pericarditis, restrictive cardiomyopathy, pulmonary embolism, right ventricular infarction, and advanced systolic heart failure.

Cervical venous hum - The hum is a continuous murmur, loudest in diastole produced by turbulence in neck veins when head is turned to the left. It occurs due to mild compression of the internal jugular veins by the transverse process of the atlas. It is detected by auscultating the right supraclavicular area and base of the neck in sitting position. It is common in children, young adults, high output states and patients undergoing hemodialysis. It can be confused with murmurs of aortic regurgitation, patent ductus arteriosus, A-V fistula and carotid arterial bruits.

CARDIAC DISORDERS
Aortic stenosis - Pulse - In aortic stenosis, there is obstruction to left ventricular ejection and is best detected in the carotid artery. The classic pulse is called pulsus parvus et tardus or anacrotic pulse. The hallmark of carotid pulse in valvular aortic stenosis is slow rising with delayed upstroke (pulsus parvus), delayed peak which is a smooth prolonged peak with a gradual drop off (pulsus tardus or plateau pulse), small volume due to hemodynamic obstruction at the valve, palpable thrill due to turbulence caused by ejection of blood across a narrow orifice and a prominent anacrotic notch due to jet effect produced by ejection of blood across a narrow valve or decreased velocity of blood flow during early ejection (anacrotic pulse). Palpation cannot detect the anacrotic notch. The severity of valvular obstruction is proportional to the degree of abnormality of the carotid pulse. A lag between onset of apical impulse and carotid impulse predicts a valve area of <1 sq cm (100% specific). The severity of aortic stenosis may be masked with high cardiac output states in children, increased vessel stiffness in elderly, associated aortic regurgitation, hypertension, low stroke volume and congestive heart failure. The severity of aortic stenosis is exaggerated by impaired left ventricular function, hypovolemia and mitral stenosis. A bisferiens pulse may be seen when aortic stenosis is associated with moderate to severe aortic regurgitation and preserved left ventricular function. Bisferiens pulse disappears with onset of congestive heart failure. Pulsus

alternans may be seen in severe aortic stenosis with left ventricular dysfunction.

Blood pressure - As the severity of aortic stenosis increases, the systolic arterial pressure decreases and pulse pressure narrows. Patients with hypertension can have high blood pressure associated with aortic stenosis. If there is associated aortic regurgitation, depending on severity there would be lower diastolic pressure and widening pulse pressure.

Jugular venous pulse - In most of the cases it will be normal. In presence of left ventricular hypertrophy and hypertrophied septum, there would be decreased left ventricular compliance leading to prominent A wave *(Bernheim effect)*. It may be elevated when aortic stenosis is associated with mitral stenosis and pulmonary artery hypertension or tricuspid stenosis.

Supravalvular aortic stenosis - Patients have selective jet streaming of blood in the right innominate vessels which causes greater pulse amplitude in right carotid, subclavian and brachial arteries with left carotid pulse having features of aortic valve obstruction. The blood pressure in the right arm may be 10-20 mm Hg higher than the left arm.

Aortic regurgitation - Pulse - The pulse of aortic regurgitation has a collapsing quality. The large stroke volume increases the force and amplitude of ejection followed by an abrupt fall or collapsing pulse caused by low systemic vascular resistance and early diastolic reflux of blood into the left ventricle. Arterial pulses all along the arterial tree have a bounding quality giving rise to the nonauscultatory signs of aortic regurgitation. The presence of these signs indicates moderate or severe chronic aortic regurgitation. These signs may be attenuated with low cardiac output, heart failure, arterial vasoconstriction and tachycardia in congestive heart failure.

Blood pressure - As the severity of aortic regurgitation increases, the diastolic pressure decreases and pulse pressure widens. The degree of decrease of diastolic pressure is a better indicator to assess the severity of aortic regurgitation, than the increase in systolic blood pressure. Though the sphygmomanometer measured pressure approaches zero, true diastolic pressure is never lower than the maximal left ventricular end diastolic pressure. Hence diastolic pressure is measured at muffling of Korotkoff sounds and disappearance of sound. Tachycardia shortens the time for diastolic reflux and increases the diastolic pressure, whereas bradycardia increases duration of diastolic reflux and decreases the diastolic pressure.

PERIPHERAL SIGNS OF AORTIC REGURGITATION

i.   Bisferiens pulse - A double peaked systolic impulse, best appreciated in brachial artery

ii. Water hammer pulse

iii. Hill's sign - A difference in systolic blood pressure between the brachial and popliteal arteries is used to assess the severity of aortic regurgitation - 20-40 mm Hg - angiographic 2+ AR, 40-60 mm Hg - angiographic 3+ AR, >60 mm Hg - angiographic 4+ AR

iv. Palmar click - A palpable, abrupt flushing of the palms in systole

Eyes

v.   Landolfi's sign - Contraction and dilatation of pupil in systole and diastole respectively

vi. Becker's sign - Prominent retinal artery pulsations

Head and Neck

vii. De Musset's sign - Visible oscillation or bobbing of the head with each heart beat

viii. Corrigan's sign - Visible pulsations of carotid artery (dancing carotids)

ix.   Muller's sign - Visible pulsations of the uvula

x.   Minervi's sign - Strong lingual pulsations demonstrated by up and down movement of the tongue depressor

xi.   Logue's sign - Pulsatile sternoclavicular joint when aortic regurgitation is associated with aortic dissection

Upper limb

xii. Locomotor brachialis - Pulsations of the brachial artery

xiii. Quincke's pulse - Exaggerated sequential reddening and blanching of the fingernail when light pressure is applied to the tip of fingernail. A similar phenomenon can be observed by pressing a glass slide against the lips.

xiv. Palfrey's sign - Pistol shot sounds over the radial artery

Lower limb

xv.   Pistol shot of Traube - A large systolic sound with the stethoscope lightly placed on the femoral artery

xvi. Durozeiz's sign - Light pressure applied to the femoral artery distal to the edge of the stethoscope produces a to and fro bruit caused by exaggerated reversal of flow in diastole.

A systolic murmur is perceived by pressing the femoral artery proximal to the stethoscope.

Abdomen

xvii. Rosenbach's sign - Pulsatile liver

xviii. Gerhardts's sign - Pulsatile spleen

xix.   Dennison's sign - Pulsatile cervix

Acute aortic regurgitation - In acute aortic regurgitation, there will be sinus tachycardia, slightly low systolic blood pressure with a near normal diastolic blood pressure with pulsus alternanas. Peripheral signs may be absent. The mean jugular venous pressure may be elevated.

Hypertrophic cardiomyopathy - Pulse - The carotid pulse

has a rapid, jerky, sharp upstroke which taps against the fingers, followed by a midsystolic dip or collapse which is followed by a second late diastolic wave (spike and dome or pointed finger pulse). The rapid upstroke is due to early, exaggerated emptying of the left ventricle and the second peak is probably a reflected wave or rebound phenomenon. The magnitude of midsystolic dip correlates with the left ventricular - aortic pressure gradient. In a normal individual, the postextrasystolic pulse after a long pause is larger than normal. In obstructive variety of hypertrophic cardiomyopathy, after an extrasystolic pulse, there is Starling effect which causes increase contractility and increase obstruction. So, the pulse stays the same or reduces. This is called Brokenborough phenomenon.

Jugular venous pulse - The A wave is prominent and appears as a flicking motion prior to carotid upstroke. It is due to stiff RV myocardium and strong right atrial contraction. A more prominent A wave indicates right ventricular outflow tract obstruction.

Mitral stenosis - Pulse - The carotid arterial pulse is normal or has decreased pulse volume and normal contour. If associated with atrial fibrillation, the pulse will be irregular with variable pulse volume. If associated with mitral or aortic regurgitation, there is an increase in the carotid pulse amplitude and rate of rise.

Jugular venous pulse - It may be normal in uncomplicated mitral stenosis. There will be a prominent A wave in patients with pulmonary artery hypertension in sinus rhythm as a result of right ventricular hypertrophy and decreased right ventricular compliance. In presence of right ventricular failure, mean pressure will be elevated. In atrial fibrillation, A wave disappears, X descent is attenuated with irregular V waves.

Mitral regurgitation - Pulse - In moderate or severe mitral regurgitation, the carotid pulse is brisk or jerky with decreased pulse volume or quick rising, poorly sustained and low amplitude. It is due to decreased forward stroke volume because of regurgitation.

Jugular venous pulse - It is normal in uncomplicated cases. As right heart failure sets in, pulmonary artery hypertension causes rise in mean venous pressure. This causes a prominent A wave suggestive of an elevated right ventricular end diastolic pressure. Patients with functional tricuspid regurgitation have a prominent V wave which increases on inspiration.

Mitral valve prolapse - Majority of patients have no abnormality of the arterial or venous pulse. When associated with severe mitral regurgitation, the arterial pulse is brisk and collapsing. A retraction notch coincident with midsystolic click has been recorded but is not palpable.

Tricuspid regurgitation - Pulse - Hemodynamically significant tricuspid regurgitation may result in low amplitude arterial pulse. Many patients may have associated atrial fibrillation.

Jugular venous pressure - It has more diagnostic importance than auscultation. Sometimes, in severe tricuspid regurgitation, the mean venous pressure is elevated. In such a situation, it may be necessary to examine the patient in a sitting or standing position. Do not conclude that the jugular venous pulse is normal unless the upper level of the wave form is identified in supine, 45 degree or sitting position. As the severity increases, X descent is attenuated and ultimately disappears. The V wave is augmented and Y descent is more prominent. In severe tricuspid regurgitation, there is a rounded or plateau like severe regurgitant C-V wave, V wave or S wave. This V wave is a systolic wave which is followed by a sharp, steep trough called the Y descent. In severe cases, prominent eyeballs and pulsatile earlobes have been observed. With inspiration, due to increase in right ventricular inflow, the V wave has a higher peak and a prominent Y descent. In atrial fibrillation, there is disappearance of A wave with presence of a dominant V wave which may simulate the V wave of tricuspid regurgitation.

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