Medical

GROUP
NEET SS

Volume 1
2nd Edition

Based on Video
Lectures on
DocTutorials App



TUTORIALS

LOGIN FOR SUCCESS

DocTutorials Hard Copy Notes are transcript-based notes from the video lectures by our esteemed Faculty. The latest edition of notes has been carefully designed for a comfortable learning experience in all the subjects.

Our notes are a valuable asset to your NEET SS preparation. We have added images, diagrams, charts and tables to the well-organized, clear and concise study content, wherever required. The coloured notes give you much-needed relief during continuous reading.

The notes has been carefully reviewed by our video lecture faculty and has been curated to provide you a helping hand during your NEET SS preparation.

Team DocTutorials wishes you the best of luck for your preparation.

Published by
DocTutorials Edutech Pvt Ltd,
Hyderabad-500034.

This book is made out of recycled paper material. Join us in our commitment to a greener future as we take a step towards a more sustainable and mindful approach to publishing!

TUTORIALS

LOGIN FOR SUCCESS

Dear User,

Accuracy Disclaimer:

- While reasonable care has been taken to ensure accuracy, neither the faculty nor DocTutorials assumes responsibility for any liability or damages resulting from the application of information in this workbook.

Copyright Notice:

- All rights reserved. No part of this publication may be reproduced, copied, transmitted, adapted, modified, or stored in any form or by any means, electronic, photocopying, recording, or otherwise, without prior written permission.

- We hope this workbook enhances your learning experience with DocTutorials. For any queries or assistance, please reach out to our support team.

Happy Learning!

INDEX

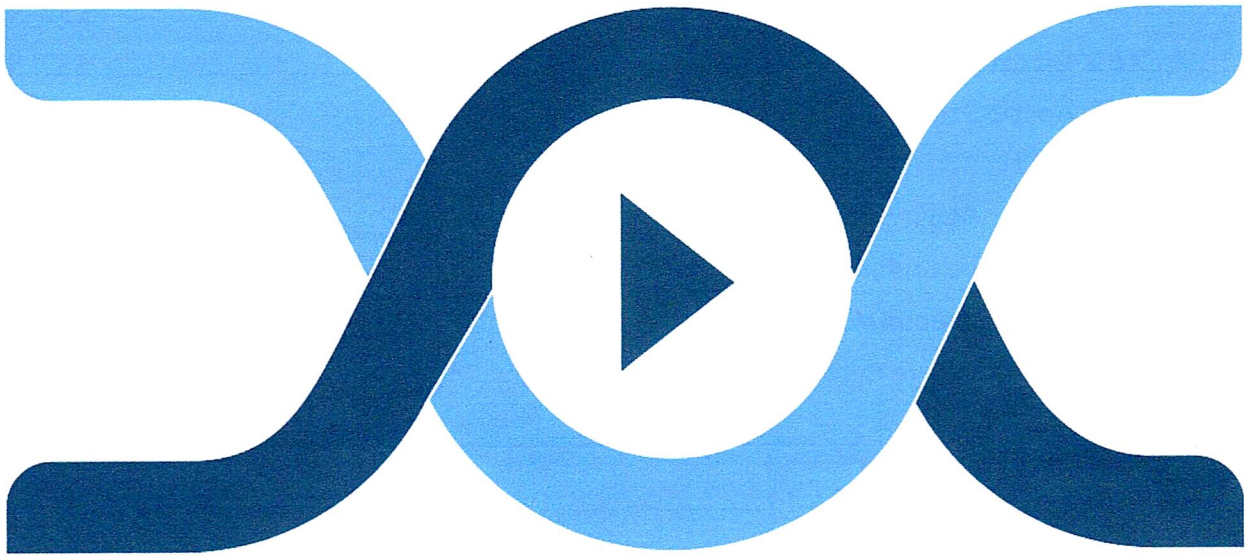
S.No.	Topic	Page No.
1.	**Cardiology**	
	1.1 Arterial Pulse	1-6
	1.2 JVP	7-12
	1.3 Cardiovascular Examination Part -1	13-30
	1.4 Cardiovascular Examination Part -2	31-43
	1.5 Heart sounds	44-48
	1.6 Chest X- Ray In Cardiology	49-73
	1.7 Cardiac Stress Testing	74-85
	1.8 Stress Testing, TMT	86-95
	1.9 Cardiac Pharmacological Stress Testing	96-102
	1.10 Catheter Tracings - Pathological and Physiological	103-119
	1.11 Cardiac MRI	120-135
	1.12 Hypertension	136-144
	1.13 Management of Hypertension Beyond Pharmacology	145-154
	1.14 Pulmonary Hypertension Classification, Diagnosis & Treatment	155-174
	1.15 ECG Part -1	175-183
	1.16 ECG Part -2	184-222
	1.17 ECG Part -3	223-235
	1.18 Introduction-Tachyarrhythmias	236
	1.19 Classification of SVT	237
	1.20 Sinus Tachycardia	238-239
	1.21 Focal Atrial Tachycardia	240-242
	1.22 AVNRT	243-245
	1.23 AVRT	246-250
	1.24 Atrial Fibrillation	251-254
	1.25 Ventricular Tachyarrhthmias	255-258
	1.26 Bradyarrhythmias	259-263
	1.27 Antiarrhythmic Drugs	264-288
	1.28 Genetic Channelopathies	289-297
	1.29 Atherosclerosis	298-301
	1.30 Chronic Stable Angina	302-313

INDEX

S.No.	Topic	Page No.
1.31	Basic and Introduction to ACS	314-318
1.32	Types of Myocardial Infarction	319-323
1.33	Evaluation and Management of NSTEMI	324-335
1.34	Management of STEMI	336-347
1.35	Mechanical Complication of MI	348-357
1.36	Risk Scores in CAD	358-370
1.37	MINOCA	371-383
1.38	Anti-Anginal Drugs in Management of CAD Beyond Nitrates	384-393
1.39	Novel Oral Anticoagulants (NOACs)	394-406
1.40	Dilated Cardiomyopathy	407-410
1.41	Hypertrophic Cardiomyopathy	411-414
1.42	Restrictive Cardiomyopathy	415-416
1.43	Arrhythmogenic Right Ventricular Cardiomyopathy	417
1.44	Left Ventricular Non-Compaction	418
1.45	Takotsubo Cardiomyopathy	419-420
1.46	Cath and Hemodynamics of Constriction Vs Restrictive Cardiomyopathy	421-432
1.47	Introduction and Etiopathogenesis	433-435
1.48	Symptoms & Signs	436-437
1.49	Evaluation	438-439
1.50	Treatment of Heart Failure	440-443
1.51	Acute Pericarditis	444-446
1.52	Cardiac Tamponade	447-449
1.53	Constrictive Pericarditis	450-452
1.54	Atrial Septal Defect	453-457
1.55	Patent Ductus Arteriosus	458-467
1.56	Tetralogy of Fallot	468-479
1.57	Ventricular Septal Defect	480-481
1.58	Practical Approach to Congenital Heart Diseases	482-489
1.59	Cardiac Tumors	490-505

INDEX

S.No.	Topic	Page No.
1.60	Acute Rheumatic Fever	506-510
1.61	Disorder of Mitral valve (Introduction)	511-512
1.62	Mitral Stenosis	513-517
1.63	Evaluation and Management of MS	518-523
1.64	Mitral Regurgitation	524-531
1.65	Evaluation & Management of MR	532-536
1.66	Mitral Valve Prolapse	537-539
1.67	Aortic Stenosis Part -1	540-542
1.68	Aortic Stenosis Part -2	543-545
1.69	Aortic Regurgitation	546-549
1.70	Infective Endocarditis	550-563
1.71	Percutaneous Valvular Intervention During Pregnancy	564-570
1.72	Preoperative Evaluation From Cardiovascular View Point	571-582
2.	**Rheumatology**	
2.1	APLA Syndrome	583-585
2.2	Systemic Sclerosis	586-595
2.3	Sjogren's Syndrome	596-599
2.4	Sarcoidosis	600-604
2.5	IgG4 Related Disease	605-606
2.6	Rheumatoid Arthritis	607-615
2.7	Seronegative Spondyloarthritis	616-623
2.8	Crystal Associated Arthropathies	624-628
2.9	Vasculitis	629-644
2.10	Inflammatory Myopathies	645-649
2.11	SLE	650-661



TUTORIALS

LOGIN FOR SUCCESS

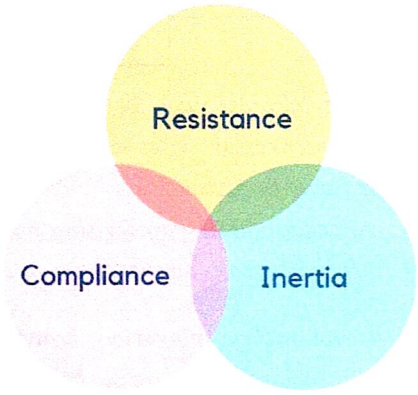
ARTERIAL PULSE

Definition 00:00:22

- Pulse: Intermittent expansion of arterial wall due to transmitted pressure changes in left ventricle occurring prior to actual expansion of ejected blood
  - It represents the expansile impulse produced by ventricular ejection and transmitted along arteries.
  - Represents the performance of left ventricle and response of arterial system to left ventricular ejection.
  - Transmitted along aorta to periphery at speed of 5m/sec
  - But intraluminal blood travels at speed of 40-50 cm/sec
- Genesis: LV pressure exceeds aortic pressure

  ↓

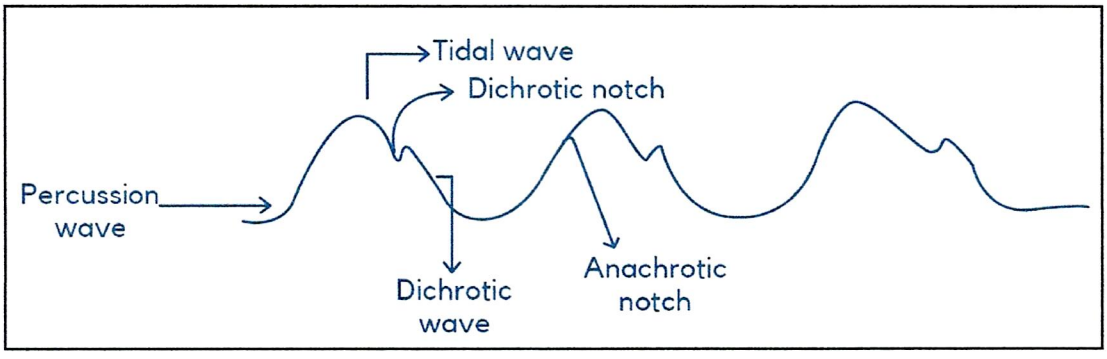
  Drives the blood into ascending aorta
- Determinants of arterial impedance:



Resistance ↓	Compliance ↓	Inertia ↓
- Related to viscosity of blood and geometry of vasculature - Oppose flow and unaffected by change in heart rate	- Related to elasticity of vessel wall	- Related to mass of columns of blood, opposes the rate of change of arterial blood flow and depends on HR

Normal pulse 00:03:45

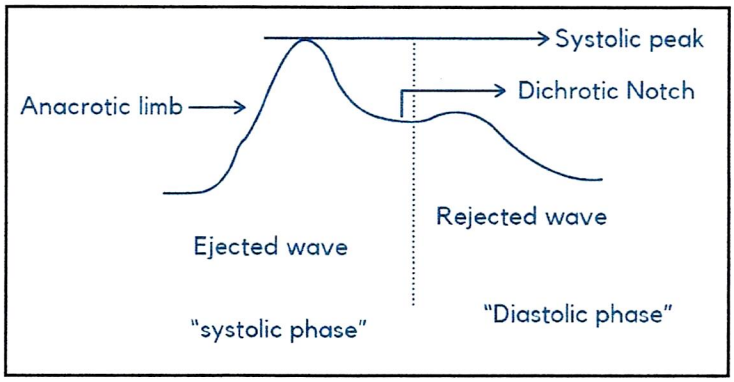
- Normal central aortic pulse wave is characterized by a fairly rapid rise to a rounded peak



- Anachrotic shoulder - Present on Ascending limb, occurs at time of peak rate of aortic flow just before maximum pressure is reached
- Incisura - Step descending limb, interrupted by a sharp downward deflection, associated with AV. Closure called incisura.

Compliance

- Related to distensibility of vascular walls. Which opposes the changes in arterial blood volume and also depends on HR.
- Inertia and compliance are important determinants of character of ventricular ejection, especially in early systole when flows and pressures are changing rapidly.



- Arterial pulse wave begins with Aortic valve opening and onset of LV ejection.
- Aortic pressure rises very rapidly in early systole because LV. Stroke volume enters the aorta faster than it flows to distal site.
- Anacrotic limb: rapid rising portion arterial pressure curve
- After its peak, aortic pressure declines as LV. Ejection slows and peripheral blood flow continues

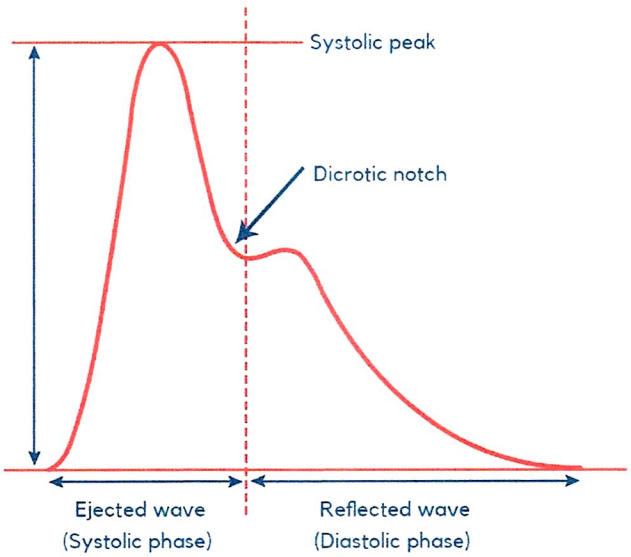
↓

Isovolumetric relaxation, transient reversal of flow from central arteries just prior to aortic valve closure is associated with incisura on descending limb of aortic pressure pulse.

∴ subsequent smaller positive wave (second wave) has been attributed to elastic recoil of aorta and aortic valve, but it is partially caused by reflected wave from distal arteries → "DICROTIC – WAVE"

Contour of pulse:

Percussion wave ↓	Tidal wave ↓	Dicrotic wave ↓
- Initial systolic portion of pressure pulse - Begins just after S1 - Predominant pulse wave appreciated at bedside	1. Later systolic portion of pressure pulse, smaller Peak 2. presumed to represent effected Wave from periphery, often recorded not palpable	- Following dicrotic notch - Usually, diastolic - Corresponding to S2



- Normal pulse as it travels along peripherally
  - Initial upstroke becomes steeper
  - Anacrotic shoulder becomes less apparent
  - Incisura replaced by smoother dicrotic notch

Evaluation 00:07:38

- Rate
- Rhythm
- Volume
- Character
- Vessel wall
- Radio-radial, brachio-femoral, brachio brachial delay
- Grade the palpability
- Bruit over artery
- Peripheral pulses
- Allen's test

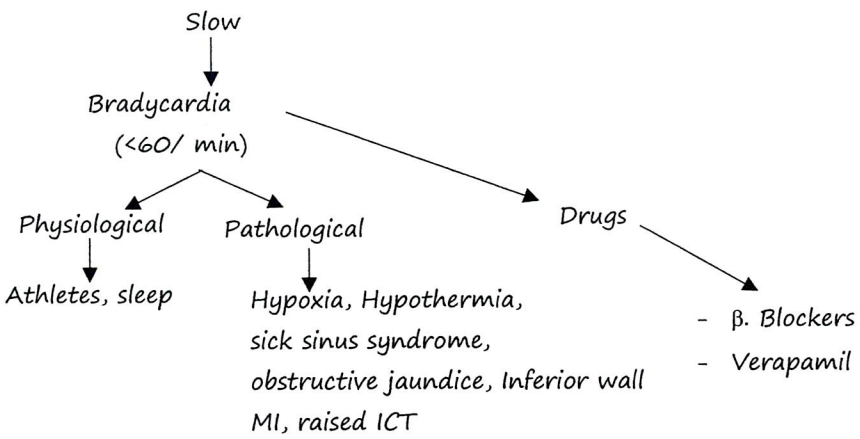
For Evaluation:

Ring finger ↓	Middle finger ↓	Index finger ↓
To prevent transmitted pulsations in radial artery from palmar arch	To perceive the lift of pulse ⇓ ∴ Estimate volume + tension	To estimate the force required to obliterate the pulse so that it is not felt by middle finger ∴ Estimates force of pulse

- Carotids are just medial to sternocleidomastoid mastoid and delay is just 30m sec as compared to cardiac output
- Dorsalis pedis= felt just lateral to tendon of extensor hallucis longus
- Pimento's point for posterior tibial: behind medial malleolus, midway b/n. medial malleolus and Tendo Achilles.

i. Rate: beats/ minutes

= Resting = 72/ min



Fast

↓

Tachycardia

(> 100 /min)

Physiological ↓	Pathological ↓	Drugs ↓
- Infants/ children/ emotion exertion	- Arrhythmia SVT/VT - High output state	- Atropine, Nifedipine, Nicotine, coffee

- Relative Bradycardia → aka FAGET'S SIGN

=↓
TYPHOID
= HAEMORRHAGIC FEVER
= LYMPHOCYTIC
= CHORIOMENIGITIS

- Every 1° is temperature Increase beats by 10/min
  - If not = relative bradycardia

ii. Rhythm:

It can be

Regula — Irregularly

Irregularly → Regular irregular / Irregularly irregular

Regular irregular → Recurring pattern of irregularity → E.g.: - Trigeminy, AV Blocks

Irregularly irregular → No specific pattern → E.g., Atrial fibrillation / multifocal VPC

- Sinus arrythmia: - Irregular in healthy individual
  - Inspiration = acceleration in HR
  - Expiration = slowing down of HR
- Secondary to variations in vagal tone
- Non-respiratory sinus arrythmia→ digitalis toxicity.
- Differentiation between heart block and ectopic:

↓

Check response with exertion

Irregularity Changes with exertion ↓	Irregulating does not change with exertion ↓
Extra systole / ectopic	Heart block

iii). Volume: Amplitude of movement of vessel wall due to passage of pulse wave.

- Indicator of stroke volume
- It can be

High volume ↓	Low volume ↓
- Elderly - Emotional excitability - Anxiety - High cardiac output (Thyrotoxicosis, Anemia) - Systemic hypertension	- "PULSUS PARVUS" ↓ - Shock - Low cardiac output states - Myocardial disease - Valvular disease Pericardial disease - Hypovolemia

Arterial pulse comparison:

0 ↓	1+ ↓	2+ ↓	3+ ↓
Absence of pulse	Small / reduced pulses	Normal/ Average pulses	Large/ bounding

State of vessel wall:

- Normal arterial wall= compressible and has elastic feel
- Diseased arteries may feel inelastic and even hard in case of calcification

  ↓
- "Mockenberg's degeneration of arterial wall"
- OSLER'S SIGN: Inflate BP cuff above the systolic BP, and if you can still feel the arterial wall.

  ↓
- Suggestive of diseased arterial with calcification

Radio-femoral delay:

- Usually both the radial pulses come simultaneously and femoral comes 5 m sec before the ipsilateral radial pulse.
  - Delay in femoral → Obstruction to aorta - coarctation,
    → Aortoarteritis
- Brachio-femoral delay: It appears more sensitive in diagnosis of coarctation as there is delay of 15m sec from brachial to femoral. In normal subject any further delay is easy to defect.
  - Unless the delay is more than 20 msec, it is not appreciable clinically.
- A radio radial or brachio-brachial delay is suggestive of vascular obstruction anywhere from
  - Innominate artery on right or subclavian artery on the left of palpation
  - Carotid: 30 msec
  - Brachial = 60 msec
  - Radial = 80 msec
  - Femoral =75 msec

  ] Time taken to reach

Allen's test:

- Test is indicated to test the patency of ulnar artery to maintain circulation of hand if radial artery is occluded

Pulse- Apex Deficit:

- Difference between heart rate and pulse rate, when counted simultaneously for one minute.
- Apex and pulse rate comparison:
  - >10/min is suggestive of Atrial fibrillation
  - <10/min = VPC

Character:

a) Anacrotic pulse= AS
b) Bisferiens pulse: moderate AS with severe AR, HOCM.
c) Corrigan pulse: rapid distal run off
   → AR, PDA, Anemia, thyrotoxicosis.
d) Dicrotic pulse: Fever like enteric fever, shock, like CCF, cardiac tamponade
e) Pulse palvus et tardus: severe AS
f) Pulsus alternans: DCM, As with LV Dysfunction, CCF
g) Pulsus celer: AR
h) Pulsus tardus: AS

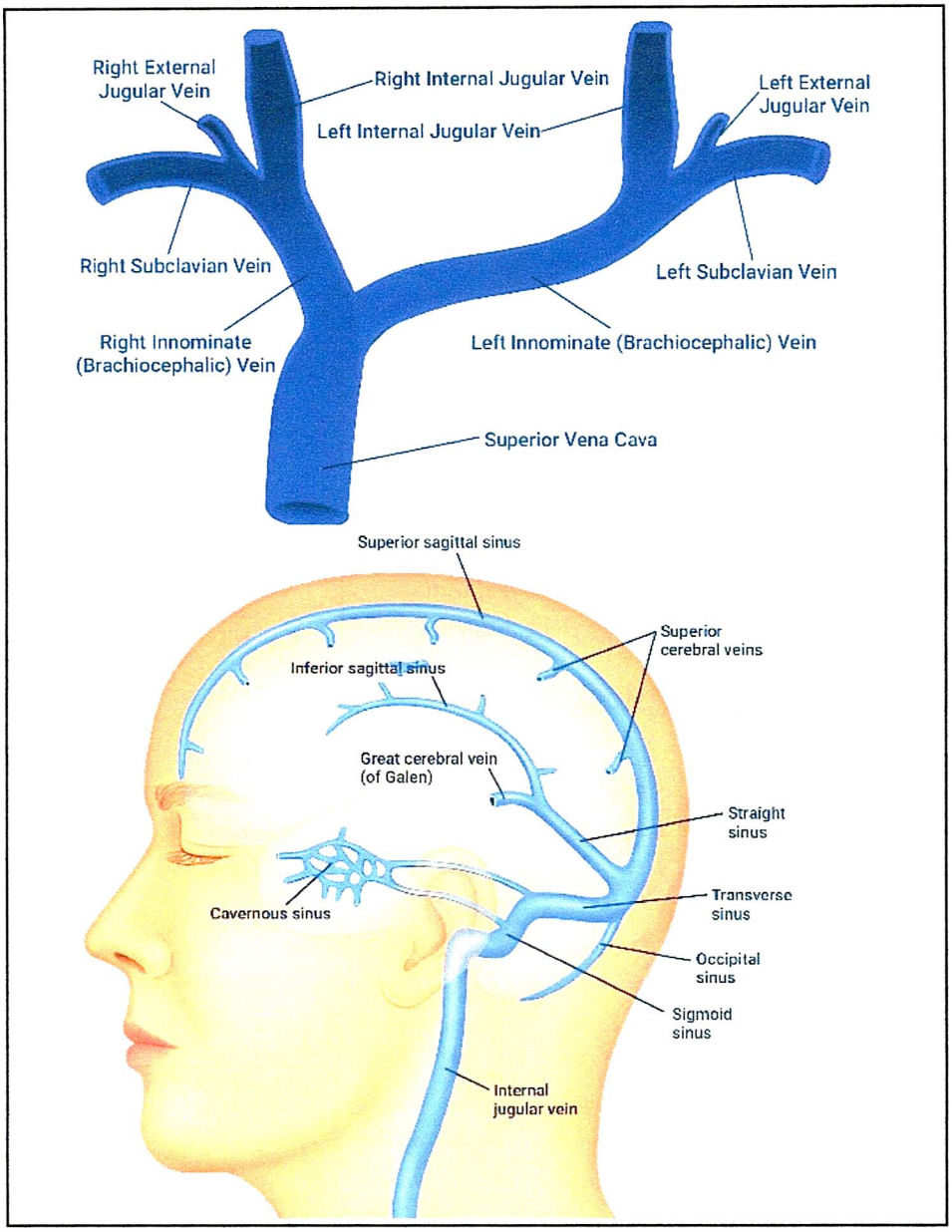
- Carotids are most suitable for evaluating the character of arterial pulse
  ↓
  If not= brachial is second choice
  - Radial artery = suitable when attempting to appreciate a collapsing pulse/ dicrotic pulse
  - Femoral artery: Pulsus paradoxus is best felt, as elevated JVP hamper the carotid palpation.
- Bisferiens pulse: twice beating pulse
  ↓
  Two positive waves during systole.
  - Percussion Wave
  - Tidal wave.
- Eg: - Pure AR and combined AS with severe AR
  - Also, in association with rapid ejection of increase SV from LV> (exercise, fever)
  - HCM
- Dicrotic Pulse: two peak pulses
  1. One in systole
  2. One in diastole (after 2nd heart sound).
     - Usually suggests poor prognosis
     - Common in young/ middle aged patients with poor LV performance
     - Associated with cardiac output state, markedly diminished smoke volume, elevated LV end diastolic pressure, high systemic arterial resistance.

Pulsus paradoxus 00:29:35

- Exaggerated reduction in strength of arterial pulse during normal inspiration due to exaggerated fall in systolic blood pressure during infraction. (>10mm of Hg)
- >20mm of Hg → detected by palpating brachial artery
- Common in cardiac tamponade.
  ↓
  Demonstrate= Blood pressure wH is inflated above systolic pressure and cuff is deflated at 2mm Hg/sec
  ↓
  Initial sound only during expiration, late on both inspiration and expiratory sound heard
  → Difference between two pressure is degree of paradox
- Tamponade w/o Pulsus Paradoxus ASD/VS /AR

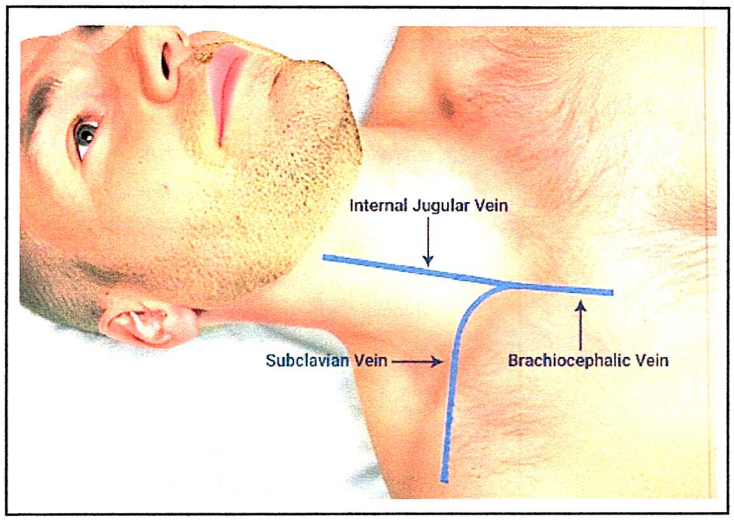
JVP

- Jugular venous pressure: - Refers to the pressure of the venous column
- Jugular venous pulse: - Pulsations of the venous column
- Provides a rough estimate of the central venous pressure
- Also provides an estimate of volume status
- Internal jugular vein → direct continuation of the sigmoid sinus



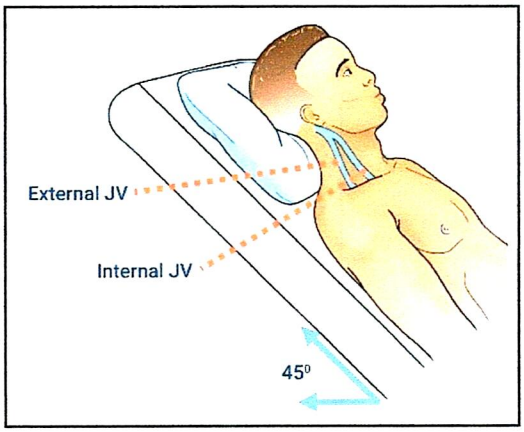
- R and L Internal jugular vein join with subclavian vein and it results in the formation of R and L brachiocephalic vein (innominate vein)
- R and L Brachiocephalic veins join to form SVC
- Superior Vena cava into the Right atrium
- No valves in Brachiocephalic vein and Superior Vena cava
- When there is a R atrial contraction it causes pulsations in SVC, brachiocephalic vein and IJV
- Study of Pulsations of IJV will give an idea about the hemodynamic of the right side of the heart

- JV Pulsations are Oscillating top of the distended IJV
- 2 important observations while studying JVP are
  - Study different waves of jugular venous pulsations
  - Study/measure jugular venous pressure
- Right IJV is used for the purpose
- IJV lies lateral to the Carotid artery and deep to Sternocleidomastoid between sternal and clavicular head
- EJV cannot be used to measure JVP as it gets compressed by structures in the neck and upper thorax and the presence of Valves. These factors interfere with the accurate transmission of pulsations
- R- IJV more in line in Right atrium
- L Brachiocephalic vein is not in line with right atrium and is more likely to be compressed by structures like Aorta and sternum



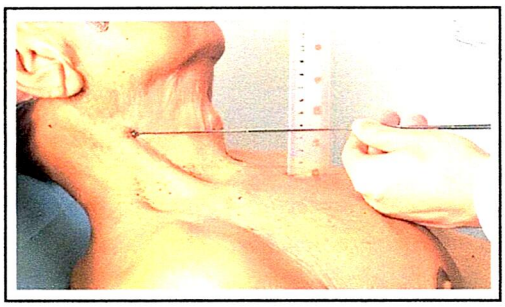
Position of the patient:

- Patient should lie supine
- Elevation of upper part of the body by 30-45° from the bed
- Head and trunk are in the same line
- Normally, JV Pulsations cannot be seen in sitting, standing positions as pulsations are occurring below the clavicle inside the thorax
- If Pulsations of IJV are seen in sitting or standing position- pressure is high
- JVP does not change with change in position
- Position at which pulsations are best seen depends on pressure in the IJV
- Low Venous pressure: - position <15 °
- Normal venous pressure: - 30-45°
- High volume pressure: - 60-90°



Jugular Venous Pressure 00:17:04

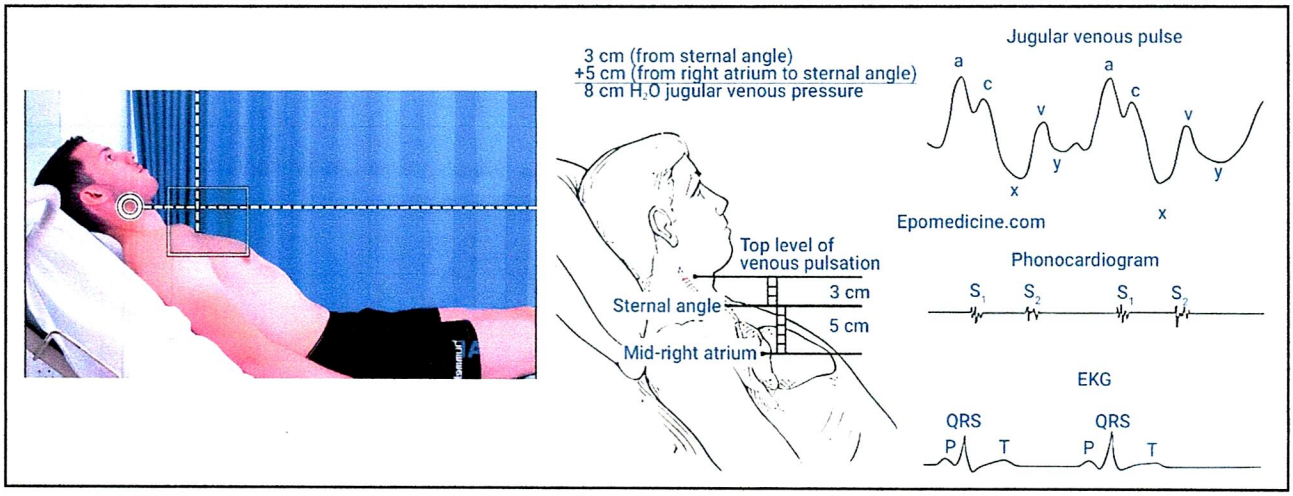
- Vertical height in cms of the oscillating venous column
- Anatomical landmark: - **sternal angle (angle of Louis)**
- Sternal angle is at a constant distance of 5cm from centre of RA
- Sternal angle: - point where the costal cartilage of 2nd rib will articulate with sternum
- The distance between angle of Louis and R atrium can vary, especially in obese persons



2 object method

Vertical object → Always Scale Horizontal object → Scale

- Vertical distance from the sternal angle to lower border of horizontal object
- In normal individuals, the stop column is maximum 3 cm from angle of Louis (3+5=8cms)
- >9 cm H2O → Abnormal
- 1.36 cm H2O = 1mm Hg



Distinguishing jugular venous pulse from carotid pulse:

Feature	Internal jugular vein pulse	Carotid artery pulse
Appearance of pulse	• Superficial and more lateral • Undulating two troughs and two peaks for every cardiac cycle (Biphasic) • Descents are more prominent than crests	• Deeper and more medial • Single brisk upstroke (Monophasic)
Response to inspiration	Height of column falls and troughs become more prominent	No respiratory change to contour
Palpability	Generally, not palpable (Except in severe TR)	Palpable
Effect of pressure	Can be obliterated with gentle pressure at base of vein/clavicle	Cannot be obliterated

- Hepatojugular reflex: -also called as, Abdominojugular reflex is used to elicit venous hypertension
- Useful in Impending heart failure
- Pressure applied over R upper quadrant at least 10 seconds
- Rise > 3 cm in venous pressure which is sustained at least for 15 seconds -Positive Hepatojugular reflex
- Patient should not hold breath
- Gives indication if PAWP > 15 mm Hg

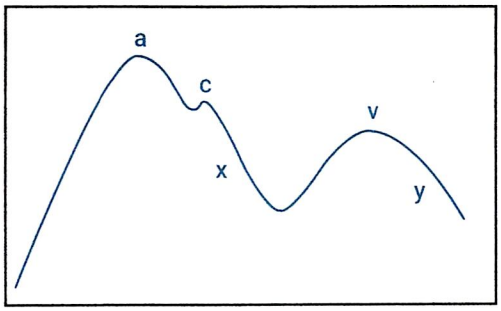
Kussmaul's sign

- Normal JVP fall by 3 mm Hg with inspiration
- Rise in venous pressure (or) a failure to decrease of JVP during inspiration is Kussmaul's sign
- Seen in
  - Constrictive pericarditis
  - Restrictive cardiomyopathy

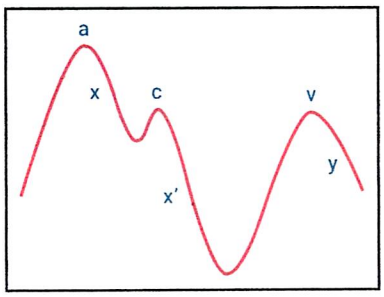
- Pulmonary embolism
- RV infraction

- Seen in R sided volume overload& reduced RV compliance

Waves of JVP 00:35:05

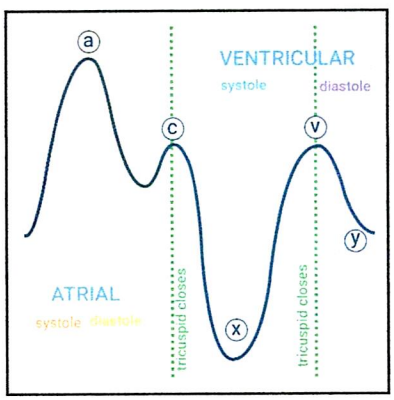


- 3 positive waves
  a wave → most prominent wave
  c wave
  v wave
- 2 Negative waves
  - x descent → most prominent wave
  - y descent
- x' →x prime



JVP waves in relation with cardiac cycle

- A wave: (a- atrial contraction) produced by R atrial contraction happens in atrial systole, which is in other words, happens during the Late phase of ventricular diastole (late filling phase)
- The atrial contraction produces increase pressure in RA, the backpressure is transmitted to jugular wave resulting in a wave
- Comes before carotid pulse and first heart sound (presystolic wave)
- Comes after P wave of ECG

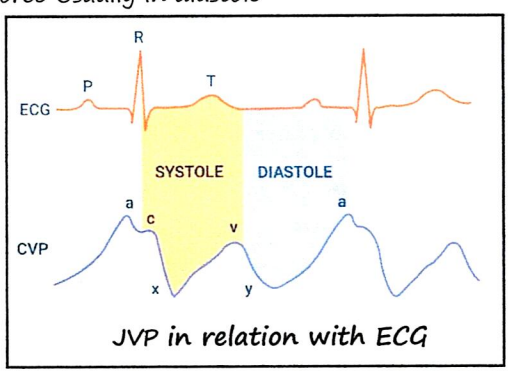


Abnormalities in a wave:

- Absent a wave is seen in atrial fibrillations
- Giant a wave occurs when R atrium contract with increased force Usually in diastole
  - RVH
    ↓
    Decreased compliance
    ↓
    Occurs 2° to pulmonary hypertension
  - TS
  - LVH (severe aortic stenosis)

Canon Wave

- Occurs when R atrium contracts with closed tricuspid valve
  - Irregular cannon waves in complete heart block, VT



JVP in relation with ECG

- Regular cannon waves in Junctional rhythm

- Differentiating point between giant and canon a wave:
  Canon- usually Systolic in timing
  Giant – usually diastolic in timing

C Wave

- It is due to tricuspid valve bulging into RA during isovolumetric contraction
  (To remember, C=cusp wave)

X- Descent

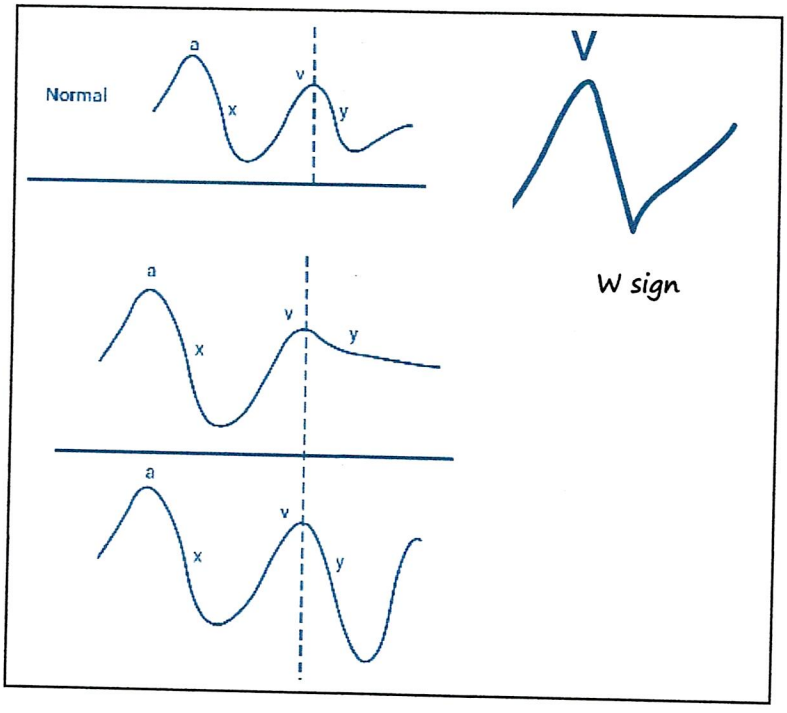
- Due to Fall in RA pressure
- Due to atrial relaxation, downward movement of the tricuspid valve during RV systole
- Atrial diastole
  (To remember, X- relax)
- More prominent during inspiration
- Ends before S2
- X' (x prime)- the part subsequent to c wave is sometimes referred to as x' (arbitrary division by c wave)
  - Absent X Descent →in Tricuspid regurgitation → Atria is not able to relax
  - Prominent x descent → in Cardiac tamponade
  - In cardiac tamponade:
    Fluid around the ventricles
    ↓
    Vigorous RV Contraction
    ↓
    Floor of the RA, Tricuspid valve pulled down
    ↓
    Significant decrease in RA Pressure

V wave

- Begins in late systole and ends in early diastole
- Roughly corresponds to S2
- Because of venous return
- A wave – carotid pulse – V wave
  Abnormalities of v wave:
- Hypovolemia– absent V wave
- Hypervolemia – prominent V wave
- Giant V wave – Occurs in TR
  - During RV systole – These is regurgitation of blood from RV to RA through the incompetent tricuspid valve, leads to significant increase in RA pressure
  - In severe TR- Positive regurgitant wave fuses with v wave
    ↓
    Ventricularization of waveform
    ↓
    giant v wave
  - D/d: Carotid pulsation
  - Lancisi's sign – refers to large systolic JVP Caused by TR replacing the normally occurring X descent
  - Also referred as C-V waves

y Descent

- Tricuspid valve Opens
  ↓
  Rapid flow of blood from RA to RV in early diastolic filling phase
  ↓
  Fall in RA pressure – y Descent
- Abnormalities of y descent:
- Prominent y descent:
  - Constrictive pericarditis –mid, late diastolic resistance and Normal early diastolic filling
  - TR
- Absent y descent
  - Cardiac tamponade: No diastolic filling
- Friedreich sign- sharp & steep y descent seen in constrictive pericarditis



- Slow y descent:
  - Tricuspid stenosis
  - R atrial myxoma

H wave

- Brief positive wave
- Follows y descent
- Prior to a-wave
- Seen when diastole is too long
- In SVC Obstruction
  - Raised, non – pulsatile JVP