

HANDWRITTEN NOTES

DAMS

α

MEDICINE VOL. 1

CRISP, CONCISE, CONCEPTUAL

Integrated Edition





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Phone : 011-4009 4009, 9899664533

<http://www.damsdelhi.com>

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HOW TO MAKE BEST USE OF NOTES?

A Message by Mentor Duo Specially for you,



- Read the notes thoroughly, they are absolutely concise, crisp & conceptual and hence it is best advised not to add a lot of extra information to them as that will dilute the quality.
- Images have been provided alongside to aid in better understanding and also help you solve image-based questions, these images have been specially picked by the faculty so have a high probability of being asked in exams.
- Notes are handwritten in a way to help make them easier to retain, a lot of tables, graphs and algorithms have been used to simplify the learning.
- While reading notes try and use the CFAQ technique —
 - A. Use the C to denote concept part in the notes and ensure you are clear with this part in the first go if not then it's advisable to listen to this part of the video from your course.
 - B. Use the F To denotes facts in your notes, it is okay if you can't remember them in first go but will need repeat reading. But these facts are important for exams as they could be integrated to clinical questions.
 - C. Use A to denote applied parts, this is how concepts and facts are asked indirectly in exams. This will also help you develop MCQ solving skill.
 - D. Use Q to denote areas where faculty has said it's a direct question or a PYQ or a potential question.
- This technique will help you summarize your notes In way that your second reading will become easy and faster.
- Active space has been provided with these notes to make your own annotations alongside and this will help you maintain one single notebook for one subject.
- Try and solve MCQs with every topic from DQB. Your goal should be to start with at least 30 MCQs every day and then increase to at least 50 MCQs every day. Also, when you do a topic wrong write it alongside the notes that this topic needs to be read again but mark only the specific area that you have done wrong not the whole topic.
- After the topic is covered then in the active space try and summarize the topic in the form of mind map. This will help in active recall and make your revision easier.

Best Wishes & Happy Learning!!!!



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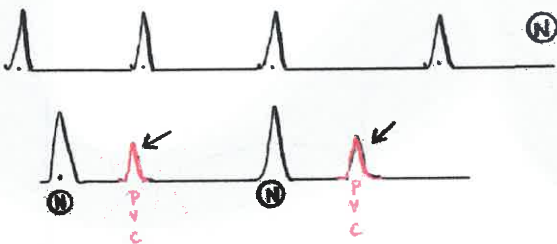
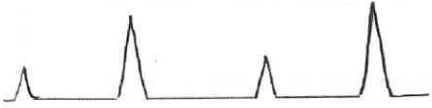
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**CARDIOVASCULAR SYSTEM
GASTROENTEROLOGY
ENDOCRINOLOGY**

MEDICINE CVS

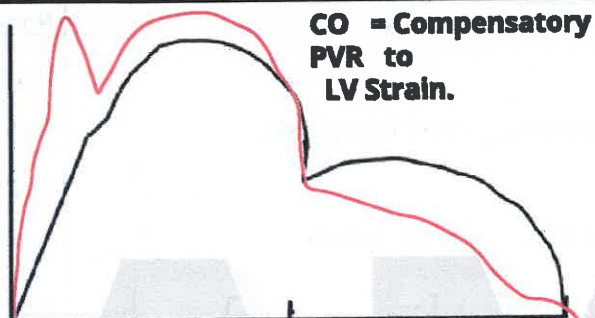
Pulse: Rate, Rhythm, Waveform, Pulse deficit, Pulsus Paradoxus

Abnormality	Description	Causes
<p>Faget's sign</p> <p>Relative Bradycardia</p> <p>(Normally for every 1 F increase in body temperature- PR increases by <u>10/min</u>)</p>	<p>PR does not Increase In Proportion to Temp.</p> <p>Step ladder fever →</p> <p>MCC of atypical nosocomial pneumonia →</p> <p>Q fever →</p> <p>Undulating fever →</p>  <p>Self induced fever</p> <p>Thermometer manipulation</p> <p>3-10 days fever—3-10 days afebrile—cycle repeat</p>	<p>Infectious - SA Node</p> <ol style="list-style-type: none"> 1. Typhoid 2. Legionella 3. Coxiella Burnetti 4. Brucella <p>5. Viral fevers</p> <p>Non-infectious</p> <ol style="list-style-type: none"> 1. Drug induced - Beta Blocker 2. Factious Fever 3. Fraudulent Fever 4. Pel Ebstein[Hodgkin's Dis]
<p>Pulsus bigeminy</p> <p>Regularly = Predictable</p> <p>Irregular = Variation</p> <p>Rhythm</p>	<p>Every alternate pulse is due to premature ventricular contraction(PVC)</p> 	<p>Digoxin Toxicity</p> <p>↓</p> <p>+ Ventricle Fiber Excitability</p>
<p>Pulsus alternans</p> <p>Key Word for Diagnosis of LVF.</p>	 <p>Regular Alteration in Pulse Amplitude.</p>	<p>LV systolic failure</p>

<p>Normal pulse waveform</p> <p>Carotid Pressure Change</p> <p>Percussion Wave</p>	<p>Best assessed in carotids</p> <p>Tidal Wave</p> <p>CO [SV]</p> <p>Dicrotic Notch</p> <p>PVR</p> <p>Dicrotic Wave</p> <p>S1</p> <p>S2</p> <p>S1</p> <p>Systole</p> <p>Diastole</p> <p>Clinically: only one systolic wave palpable</p> <p>Dicrotic wave not palpable</p>	<p>Percussion wave= LV pressure transmission to carotids</p> <p>Tidal wave= Blood ejection into carotids</p> <p>Dicrotic wave= Back pressure transmission from small vessels to carotids</p>
<p>Parvus et Tardus</p> <p>Low Amplitude</p> <p>Late Peak</p> <p>Keyword for Diagnosis for AS.</p>		<p>Severe Aortic Stenosis</p>
<p>Dicrotic pulse</p> <p>2 Peaks - 1 Systole</p> <p>1 Diastole</p>	<p>↓ CO - ++ Sympathetic Activity -↑ PVR</p>	<p>Severe Shock Typhoid</p>

Bisferiens pulse
2 Peaks Both in Systole.

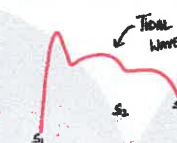
Better Felt in Radial Artery.



Increased LV force of contraction

LV pressure transmission rapid to carotids

Percussion wave will come Early
(gets separated from tidal)



Pointed Finger Pulse

1. Severe AR

2. Severe AR - Tidal Wave
+ AS Late

3. HOCM
[↑LV force of Contraction]

Pulse deficit

(Normally
HR-PR=0)

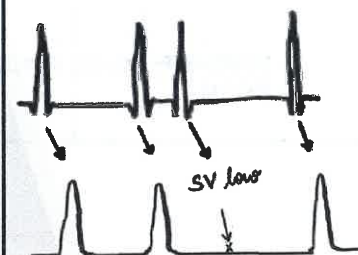
Ventricle Contraction Rate
Arterial Pulsation Due to
Adequate Stroke Volume

If HR-PR = +

When ventricle contracts with low stroke volume—
that time pulse will not be palpable

ECG =

PULSE =



1. Atrial Fibrillation
with Irregular HR.

2. Early PVC

If Pulse Deficit is > + 10/min d/t AF only.

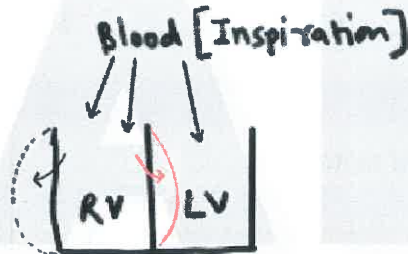
Pulsus paradoxus

$$(SBP_{exp} - SBP_{ins} = 0 \text{ to } +10 \text{ mmHg})$$

$$SBP_{exp} - SBP_{ins} > 10 \text{ mmHg}$$

Due to SBP_{ins} ↓↓ more than physio.

LV blood filling ↓↓ in inspiration



② RV wall dilates during inspiration

If unable to dilate → IVS bulge into LV
RV Wall

Reverse Pulsus Paradoxus

$$SBP_{insp} > SBP_{exp}$$

- ↓
- Positive Pressure Ventilation
 - HOCM

Non-CV causes

Hyperventilation

(More deep inspiration)

↑ pulmonary vasodil

↑ blood pooling in lungs

↓ blood to LV

MCC

1. $PaCO_2 \uparrow$ - COPD/ Asthma Exacer.
2. $PaO_2 \downarrow$ - Pulm Embolism
3. $pH \downarrow$ - Metabolic Acidosis
4. Progesterone \uparrow - Pregnancy
 - Other
 - SVC Obstruction
 - Obesity
 - Hypovolemia

CV causes

1. Tamponade → MC CV Cause

(pericardial effusion compressing RV)

"Failure of relaxation" due to stiff

Constrictive = Pericarditis

Pericardium = 2.

Restrictive = Cardiomyopathy

Myocardium = 3.



Golden points

Clinical disease	Pulse
Aortic stenosis	Anacrotic Pulse
Aortic regurgitation	Water Hammer Pulse
DCMP	Pulsus Alternans
HOCM	Bisferiens Pulse
RCMP	Pulsus Paradoxus [Low Volume Pulse]
Constrictive pericarditis	Pulsus Paradoxus
Tamponade	Pulsus Paradoxus

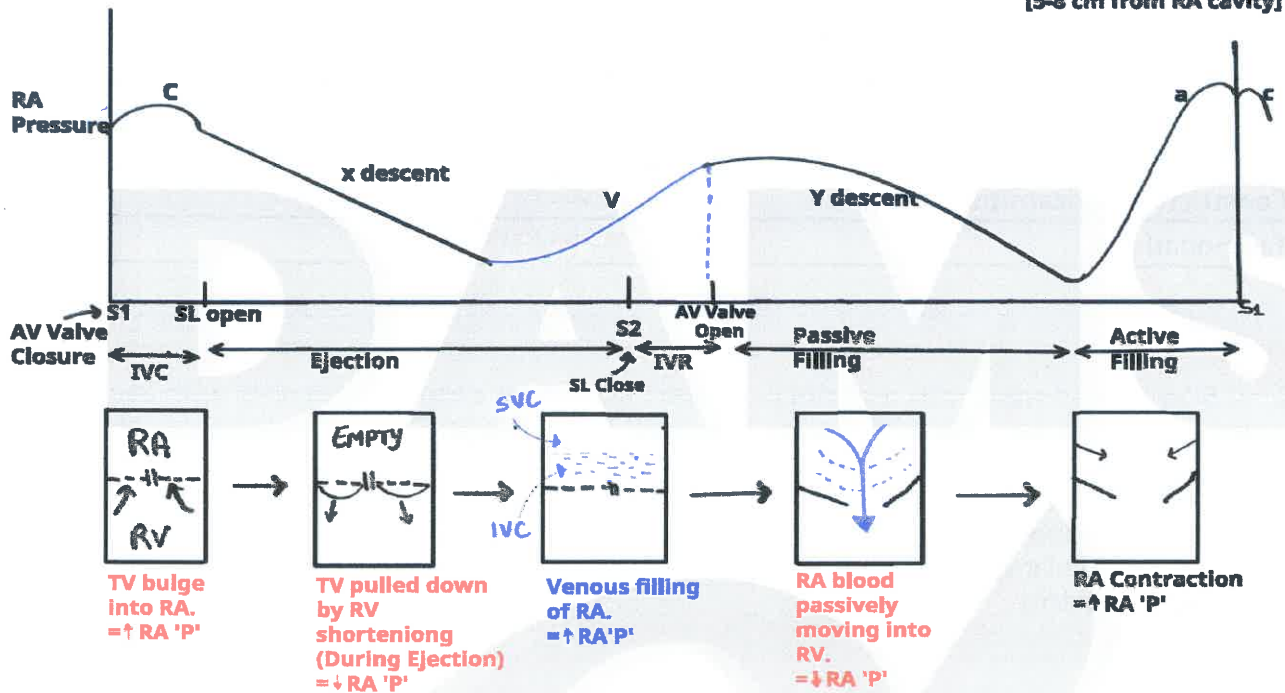
Q. A 58-year-old man with a history of heart failure on digoxin presents with increasing dyspnea. On examination, pulse has beat to beat variation of amplitude, regular in rhythm. Which of the following is the most likely underlying cause of this finding?

- A. Aortic stenosis
- B. Cardiac tamponade
- ☒ C. Left ventricular systolic dysfunction
- D. Digoxin toxicity

N height = 0-3 cm above Sternal

Angle

[5-8 cm from RA cavity]

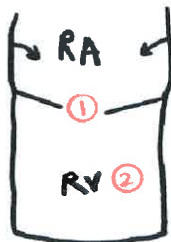
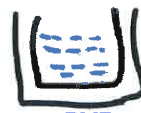
IVP examination : RA pressure Best seen in right internal Jugular Vein

A wave abnormality

1. a wave Absent = if RA (effective) contractions are absentKeyword Cause- **Atrial Fibrillation**2. a wave Large = if RA is contracting against resistanceDiastolic Event
[just Before S1]

Causes-

1. TS

2. \uparrow RV Pressure d/tRVH
PS
Pulmonary HTN

[Abnormal Increase in Blood retention in RV Cavity]

RVMI, Pulm. Embolism

RV Compression
Tamponade

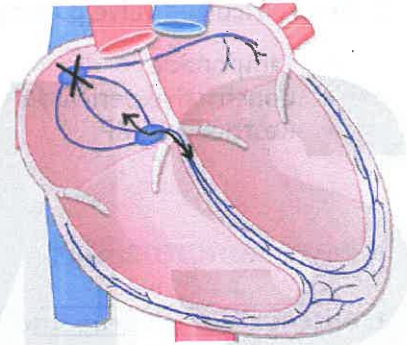
3.a wave **Cannon** = If RA is contracting against closed tricuspid valve
 Systolic Event (just After S1) Occur when RA & RV Stimulation Contraction

Causes-

1. Junctional rhythm = AVN is pacemaker

SAN Arrest

Cannon a = 40 - 60/min



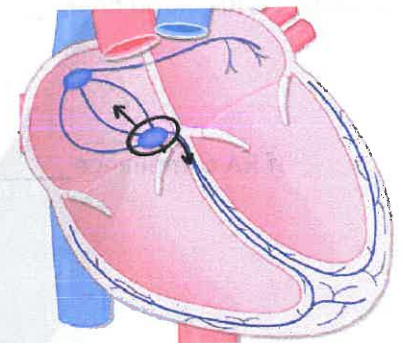
2. **PSVT** =

Episodic activation of accessory pathway making re-entry loop around AVN [Conduct Faster]

Above Ventricle

Cannon a = > 100/min
Rate

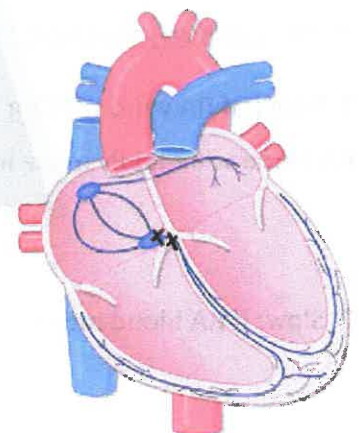
= Frog Sign



3. Complete AV block=

Occasionally Atria & Ventricle can Depolarise Simultaneously.

Cannon a : Intermittently



X descent abnormalities

1. Absent- RA pressure doesn't fall as it contains blood during this phase due to **TR**
or
Clot d/t Blood Stasis = **AF**

2. Deep- Tricuspid valve pulled **More** downward due to **More** vigorous RV contractions

- Tamponade
- Constrictive pericarditis
- Restrictive CMP

V wave abnormalities

1. Absent : **SVC Obstruction**

2. Large : If RA blood filling 

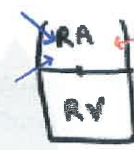
If RA compliance  = **Failure of Relaxation of RA**

Constrictive Pericarditis
RCMP

[Have same JVP Abnormality]



TR



ASD



AF induced
RA Thrombus

Y descent abnormalities

1. Rapid: If RA venous filling is with a high pressure → as soon as tricuspid valve opens → RA blood will rapidly move into RV. **Causes of Large v = Large y = Freidrich's Sign**

2. Slow: If RA blood moves into RV slowly with resistance. → 1. TS
2. ↑ RV Pressure



3. **Absent**: If RA blood doesn't move into fully compressed RV

Keyword for Diagnosis of - Tamponade



JVP sign	Description	causes
1. Kussmaul sign (normally JVP \downarrow during inspiration)		RA compliance decreases
2. Hepatojugular reflex Pre-requisite: JVP Normal before this test	Abdomen is compressed for 10 seconds \downarrow If JVP remain elevated for >15 sec even after release of compression	

Golden points

<u>JVP abnormality</u>	<u>Causes</u>
Absent a	
Large a	
Cannon a	
Absent V	
Absent X	
Absent Y	

Q. On examination, jugular venous pressure shows prominent outward going wave just before S1. Which of the following is the most likely cause?


- A. Tricuspid regurgitation
- B. Atrial fibrillation
- ☒ C. Tricuspid stenosis
- D. Complete heart block

Heart sounds

S1- Closure of **AV** valve due to **Ventricle** contractions against **Atria**
 = **M1T1**

Best site: **Mitral area = Apex = \odot 5 I/C , MCL**



Factors affecting intensity	Soft S1	Loud S1
1. Force of ventricular contraction	Weak due to - LVF - RVF - VSD	Strong due to \uparrow LA pressure \leftarrow MS \uparrow RA pressure \leftarrow TS
2. Condition of AV leaflets	Fail to strike due to - MR - TR calcification	-
3. 	\uparrow ventricle blood due to - AR - PR LVH \leftarrow AS RVH \leftarrow PS Pericardial effusion	\downarrow ventricle blood
4. Heart rate	Bradycardia \uparrow ventricle blood filling time	Tachycardia \downarrow ventricle blood filling time
5. PR interval in ECG	Prolong	Short

All valvular heart diseases cause SOFT S1 except

MS , TS

S2- Closure of **SL** valves as soon as ventricular ejection is complete

= $\overbrace{A_2 \quad P_2}^{\text{LV ejection time is less than RV}}$
(Normally S2 split : 30-60 msec)

Best site: A_2 @ aortic area \rightarrow \odot 2 I/C

P_2 @ Pulmonary area \rightarrow \odot 2 I/C

S_2 @ Pulmonary area

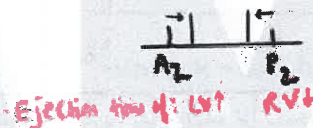
During **inspiration** = Split **Increases**



During **expiration** = Split **Decreases / Expires**

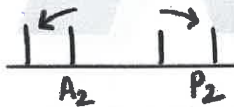


Inspiration- blood flow **increases** on **right** and **decreases** on **left** side

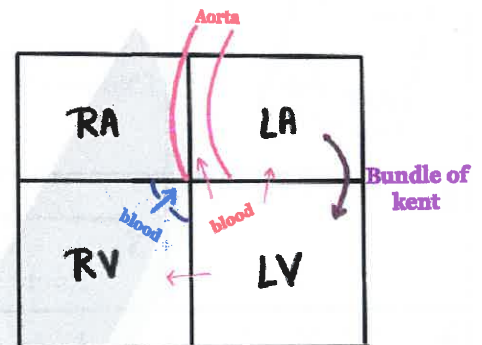
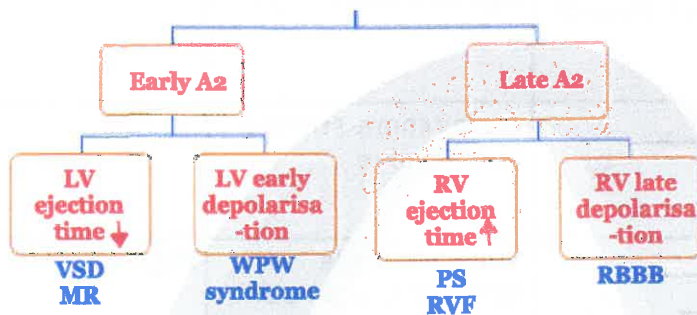


Expiration- blood flow **decreases** on **right** and **increases** on **left** side

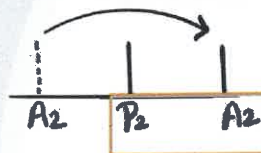
S2 split abnormalities:



1. Wide and variable =
(>60 msec)

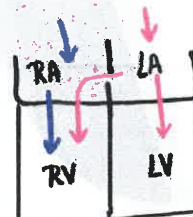


2. Reverse/paradoxical split



Late A2:
- LV ejection time increase = AS, LVF, Systemic HTN
- LV late depolarisation = LBBB

3. Wide and fixed split - Atrial septal defect
(doesn't change with respiration)












Inspiration = \uparrow + Blood vol. constant
Expiration = \downarrow + Blood vol. constant

Q. A patient has S2 split widely separated which further increases on inspiration. All the following can be the causes except

- A. RBBB
 - B. RVF
 - C. Pulmonary stenosis
 - D. LBBB
- } P_2 late
- } A_2 late = reverse split

Any abnormal HS in systole = "click"
If 2 HS at same time = their pitch will be different

Abnormal sound	Timing	Cause	Pitch
1. Ejection Click 	Early Systole 	High pressure opening of SL valve due to High LV Pressure \rightarrow AS High RV Pressure \rightarrow PS	High Aorta/Pulm Artery Aneurysm
2. Non-Ejection Click	 Mid-Late Systole	MVP	High
3. Opening Snap 	Early Diastole  AV Valve Opening [Normally, Not Heard]	High pressure \rightarrow opening of AV valve due to High LA pressure \rightarrow LA Myxoma High RA pressure \rightarrow MS High RA pressure \rightarrow TS	High
4. Tumor Plop	 Early Diastole	LA myxoma striking mitral valve	Low
5. Pericardial Knock	 Passive Filling = Mid-Diastole	Ventricle walls striking(knock) stiff pericardium Constrictive Pericarditis	High
6. S3 = Ventricle Gallop	 I High CO states II Ventricular systolic failure \rightarrow	Increased blood in the ventricle due to High CO states Ventricular systolic failure \rightarrow	Low
7. S4 = Atrial Gallop	 Late Diastole Active Filling	Atrial contractions against stiff ventricles \rightarrow LVH, RVH, HOCM, RCMP = Diastolic Failure	Low

All heart sounds are high pitch except
Tumor Plop, S3, S4