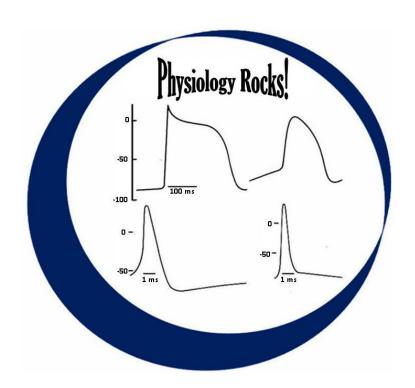
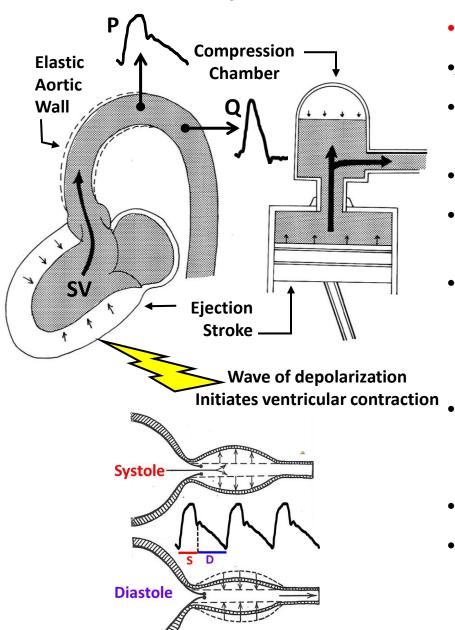
Lecture 29 Arterial Pressures



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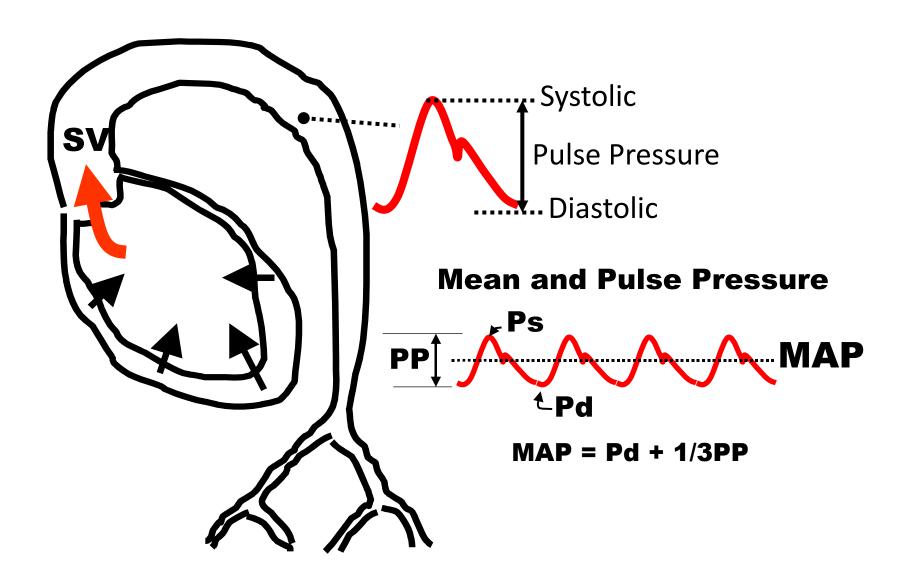
Cardiac Ejection Generates Pressure and Flow



- Excitation causes ventricular contraction
- Ejected volume = Stroke Volume
- Ejected volume/time is pulsatile aortic blood flow that rapidly rises
- Volume entry into aorta → Aortic BP
- Elastic aorta absorbs volume → Expands transiently
- Aortic wall acts like a compression chamber that initially expands during systole and then in response to stretch helps propel volume distally during diastole
- Heart is a pump governed by two parameters
 RATE and STROKE or Heart Rate (HR) and
 Stroke Volume (SV)
- HR x SV = Cardiac Output (L/min)
- After passing through the organs and supplying their needs blood returns to the heart and the cycle repeats

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Arterial Blood Pressure Basics



Arterial Blood Pressure (ABP): Major Factors

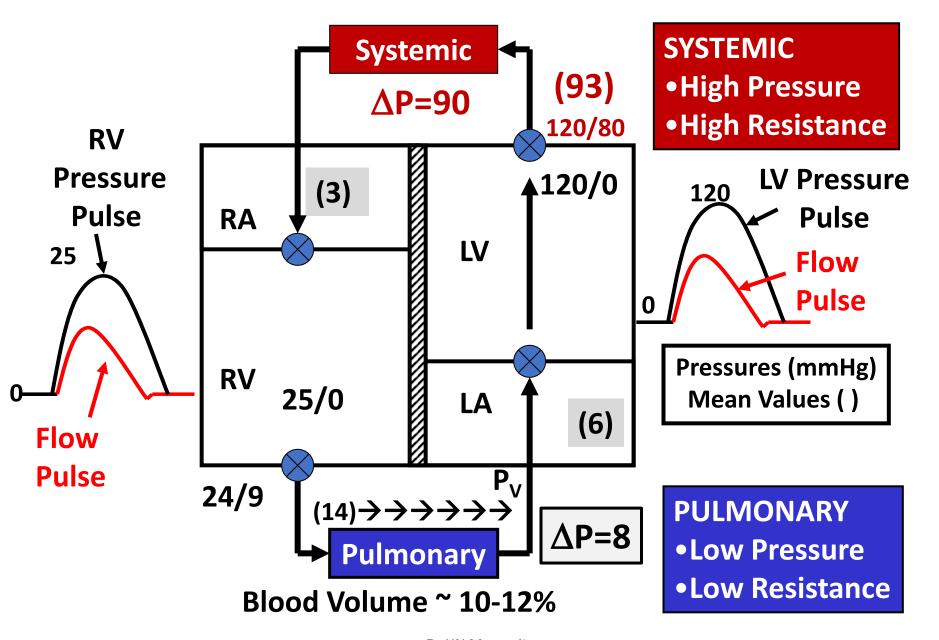
Arteriole
$$\leftarrow$$
 Sympathetic Hormones

MAP \sim Q x TPR + f (V / C)

 \uparrow \uparrow \uparrow Renal

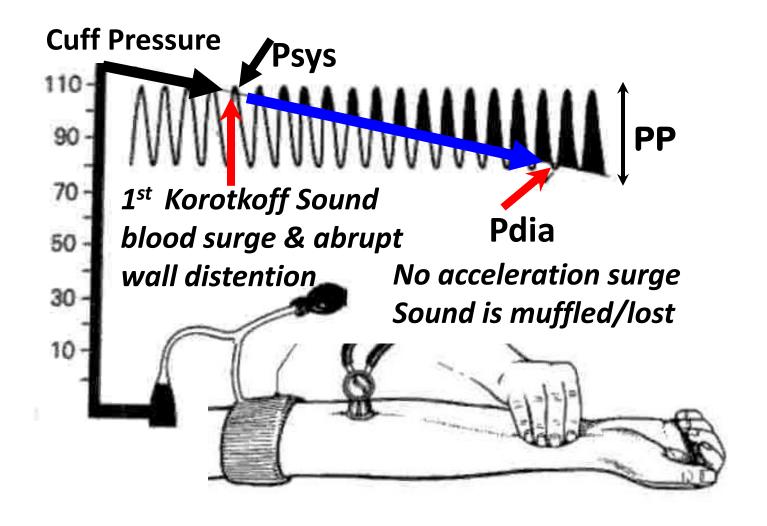
SBP/PP

Systemic vs. Pulmonary Hemodynamic Features



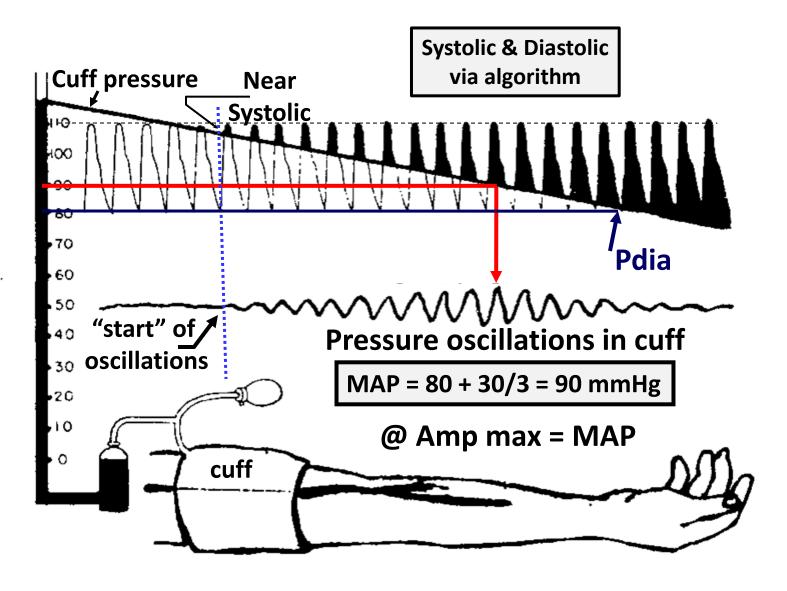
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BP Measurement: Sphygmomanometer



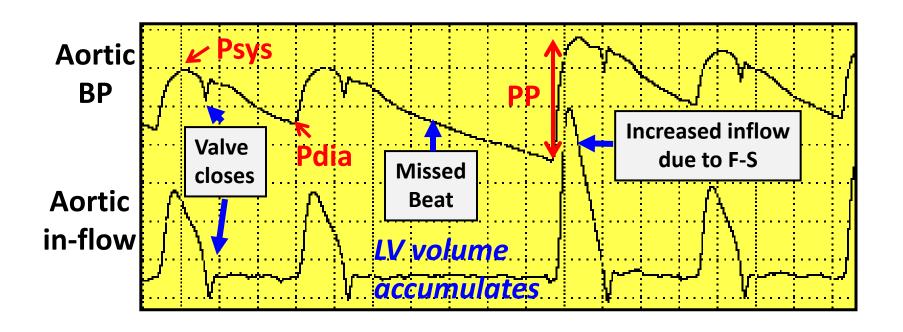
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BP Measurement: Oscillographic Method



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Pulse Pressure Determinants



Increase in pulse pressure (PP)

- Increased rate of LV ejection → + dV/dt
- Increased stroke volume → + SV
- Decreased aortic compliance → C

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Hypertension = High Blood Pressure

So What's High?

Hypertension

BP CLASSIFICATION	SBP (mmHg)	DBP (mmHg)
Normal	< 120	AND <80
Elevated	120-129	AND < 80
Stage 1 Hypertension	130 - 139	OR 80 - 89
Stage 2 Hypertension	>= 140	OR >= 90
Hypertensive Crisis	> 180	AND/OR > 120

If DBP is normal but SBP is high then may be called Isolated Systolic Hypertension (e.g. 145/75, also stage 2 HTN)

Decreased Arterial Compliance

Hypertension

- Secondary (Known organ dysfunction related)
- Primary (Essential Hypertension HTN)
 - + TPR
 - Vasoconstriction
 - Structural wall changes
 - Microvessel Rarefaction

- + CO → Early phase of HTN
- Compliance → Systolic HTN

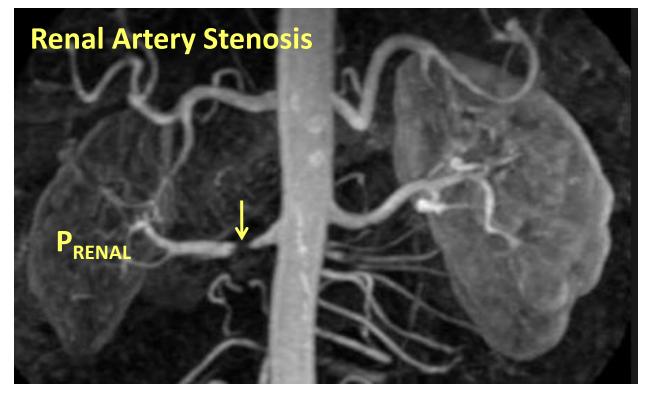
Secondary Hypertension

~ 5 - 15 % of Hypertension Patients

- Renal artery stenosis (kidney*)
- Chronic renal disease (kidney)
- Primary hyperaldosteronism (adrenal cortex)
- <u>Hyperthyroidism/Hypothyroidism (thyroid gland)</u>
- Pheochromocytoma (adrenal medulla)
- Preeclampsia / Gestational (pregnancy)

Remainder -> Essential HTN

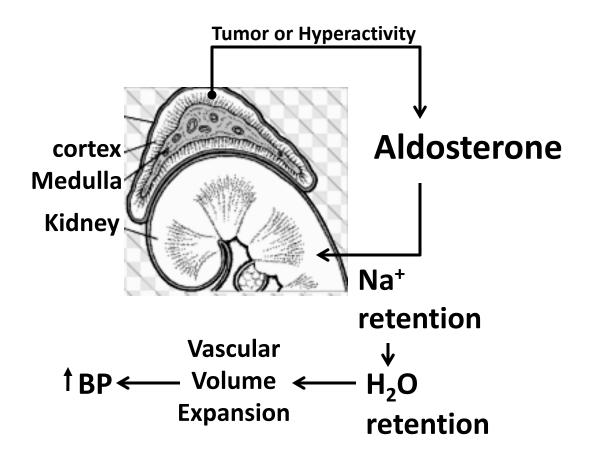
Renal Artery Stenosis -> Hypertension



 $\downarrow P_{RENAL} \rightarrow \uparrow Renin \rightarrow \uparrow Angiotensin II \rightarrow \uparrow TPR$

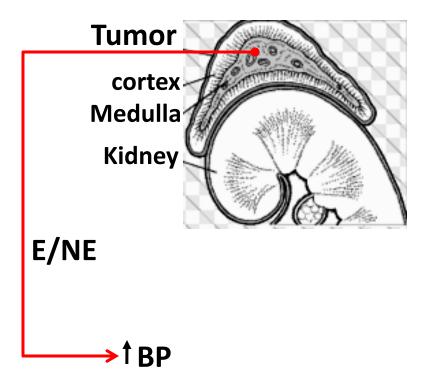
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Primary Hyperaldosteronism -> Hypertension



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Pheochromocytoma -> Hypertension



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Interactive Questions

Which (is) are normotensive?

Which (is) are stage 1 hypertension

Which (is) are isolated systolic hypertension

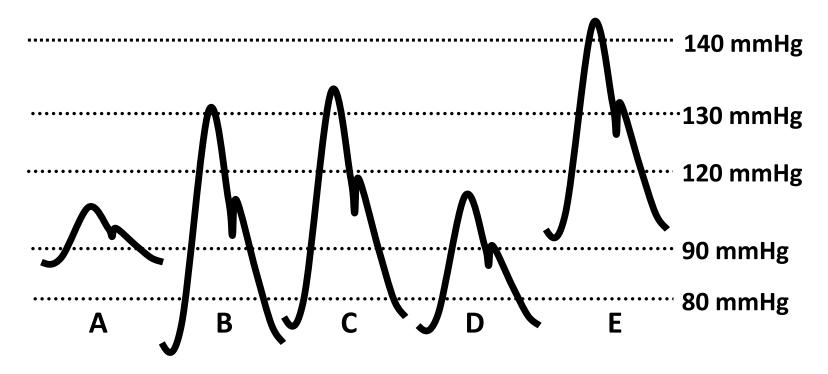


Figure shows artery Pulses of 5 patients

Interactive Question

R. Heart

CVP ~ RAP

Brain

Heart

Liver & GI

Kidney

Muscle

Skin

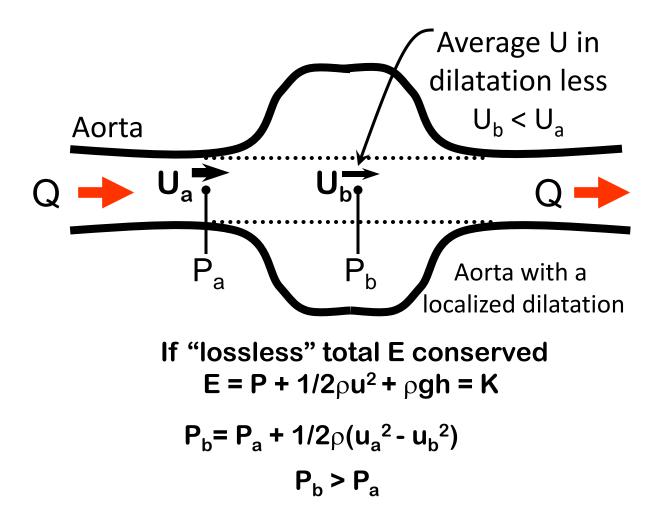
Skeleton, Bone Marrow, Fat

Bill has the following hemodynamic data Arterial Blood Pressure = 150/60 mmHg Central venous pressure (CVP) = 3 mmHg Stroke Volume (SV) = 100 ml HR = 50

What is his TPR in Wood units?

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"Lossless" Pressure Energy Conversion



So - P within expansion is greater than upstream A case where flow is not from higher to lower pressure

Interactive Question



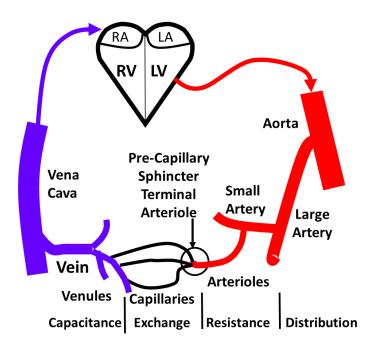
Reynolds Number:

- A. Has the units of inverse seconds
- B. Is an index of resistance to flowing blood
- C. Is directly proportional to the viscosity of flowing blood
- D. Is an index of inertial to viscous forces that operate in flowing blood
- E. Assumes a high value in capillaries because of their small diameter

Interactive Question

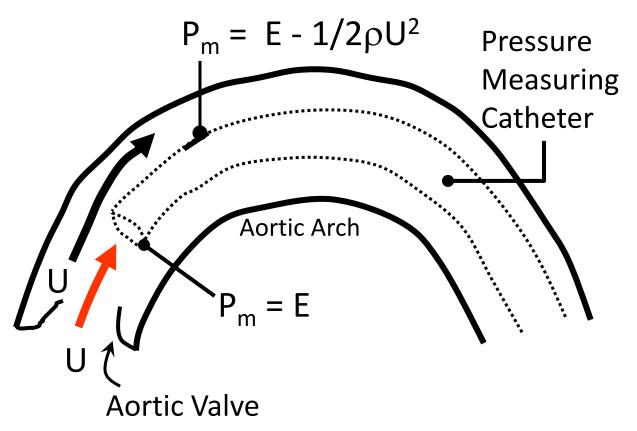
If all arterioles of an organ vasoconstrict then:

- A. Blood flow to the organ increases
- B. Blood pressure in the organ's capillaries decreases
- C. Total resistance of the organ decreases
- D. Blood flow within arterioles increases
- E. Total peripheral resistance decreases



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Measuring Aortic Pressure



Measuring "end" pressure gives a pressure value that is higher than aortic wall truly experiences

End of Lecture 29