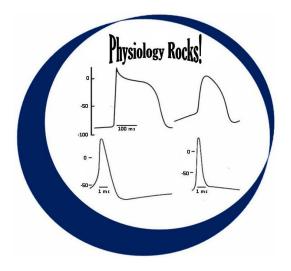
# Lecture 12 Arterial Pressures, Pulses and Propagation



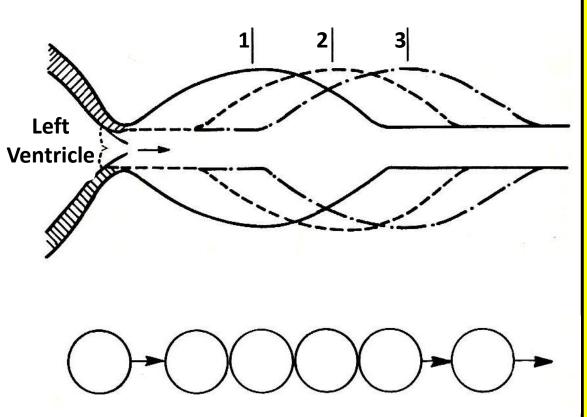
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# Topics

- Arterial pulse wave process
- Arterial pulse wave transmission and reflection: Introduction
- Pressure gradient produces pulsatile blood flow
- Pressure vs. pressure gradient features
- Pulse wave interactions
- Pulsatile pressure and flow variations
- Ankle-brachial systolic pressure index (ABI)
- Pulse pressure determinants
- Central pressure concept and augmentation index
- Clinical correlations

# **Arterial Pulse Wave Transmission Process**

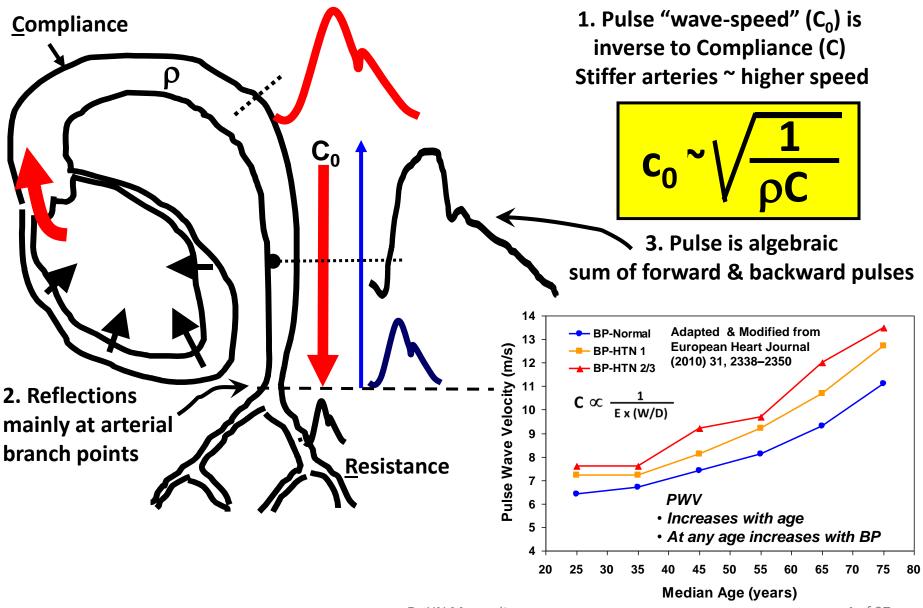
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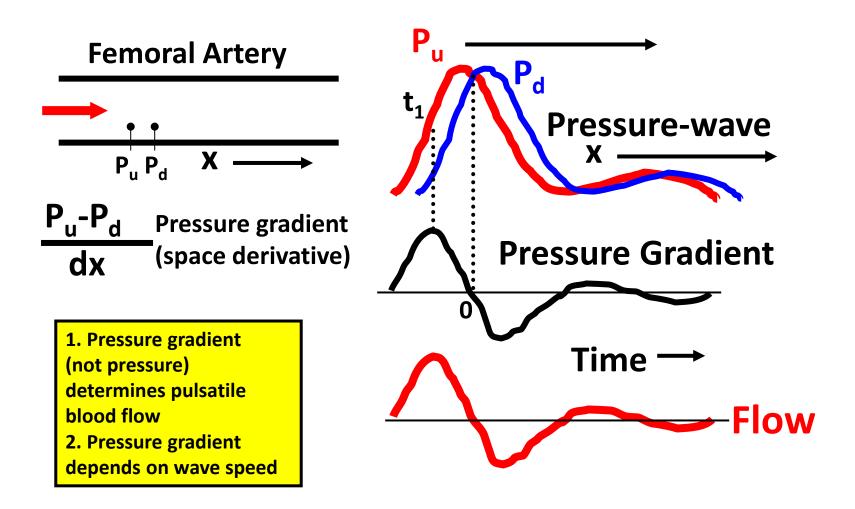
#### **Billiard Balls with Impact Transmission**

- Volume ejected initially distends aortic wall at 1 and progresses to site 2 and then to site 3 as a pulse wave
- Wave speed much greater than speed that blood cells themselves move
- Pulse wave speed vs blood velocity concept can be compared to the impact on a billiard ball combination
- The end ball moves off quickly (high speed transmission) but the group hardly moves
- Pulse wave speed is inverse to the square root of the vessel compliance wall in which the wave is travelling

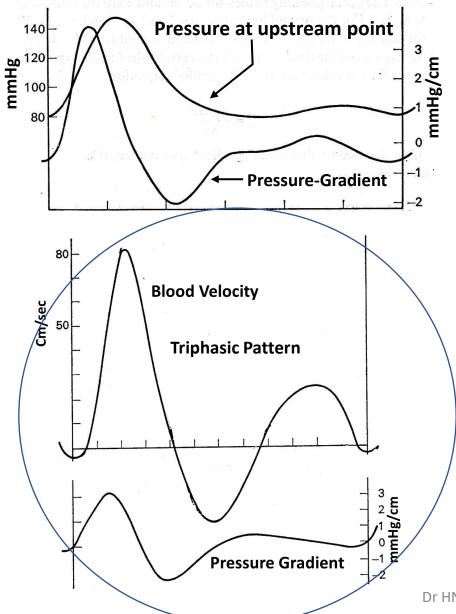
# **Transmission and Reflection of Pulses**



## **Pressure Gradient Produces Pulsatile Blood Flow**

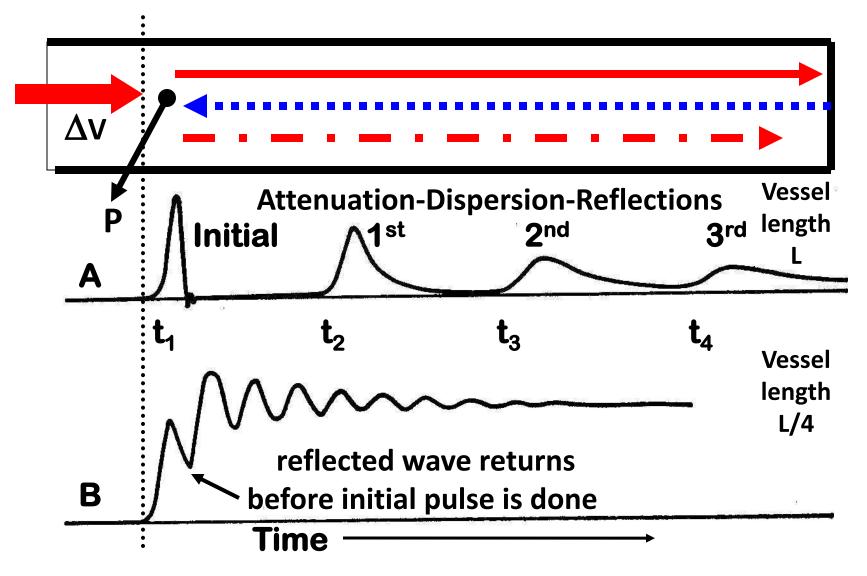


# **Arterial Pressure vs. Pressure Gradient: Summary**

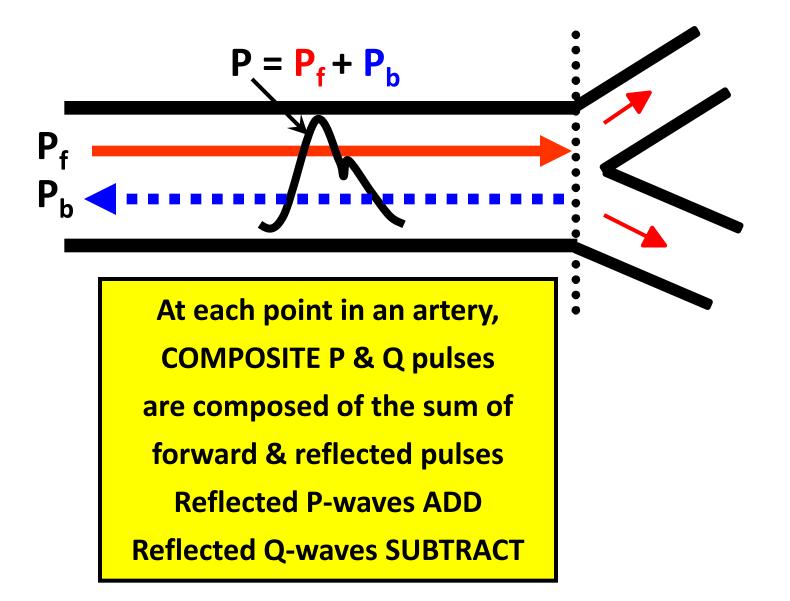


- Pressure is measured in a femoral artery at two sites separated by 5 cm
- Pressure difference (upstream downstream) is calculated and the difference divided by the separation → this is the pressure-gradient
- It is pressure-gradient that is the driving force to cause pulsatile blood flow NOT the pressure!
- The intensity and pattern of the pulsatile flow depends on the intensity and pattern of the pressure gradient
- Note the similarity of the patterns of blood velocity and pressure gradient

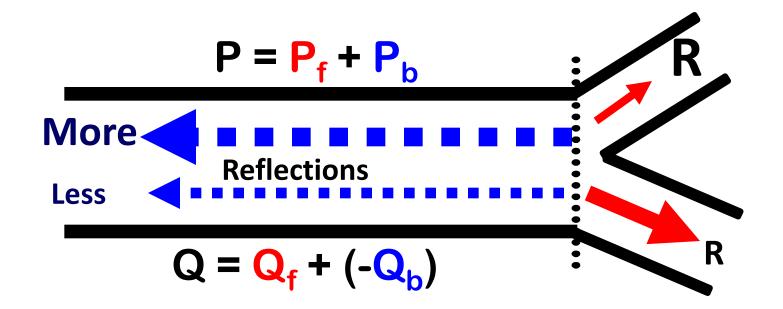
## **Multiple Pulse Wave Interactions**



#### **Forward and Backward Arterial Pressure Waves**

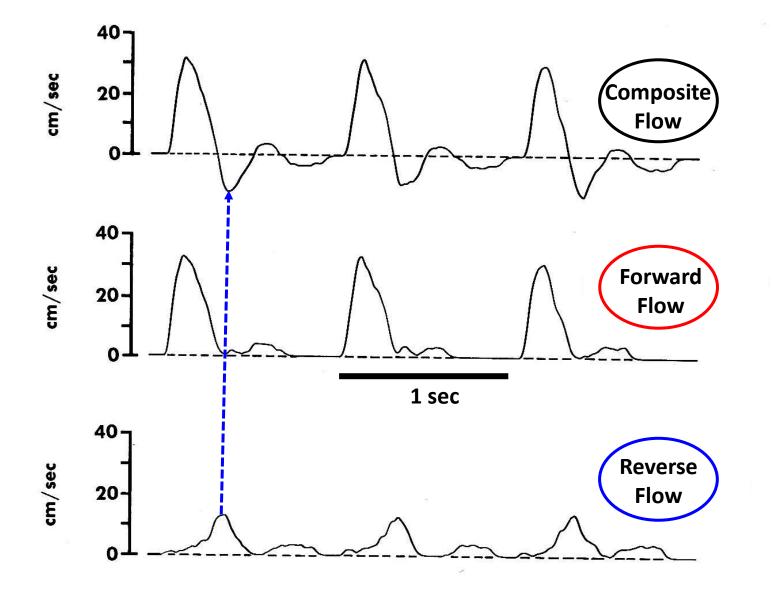


# **Vascular Resistance Effects on Reflection**

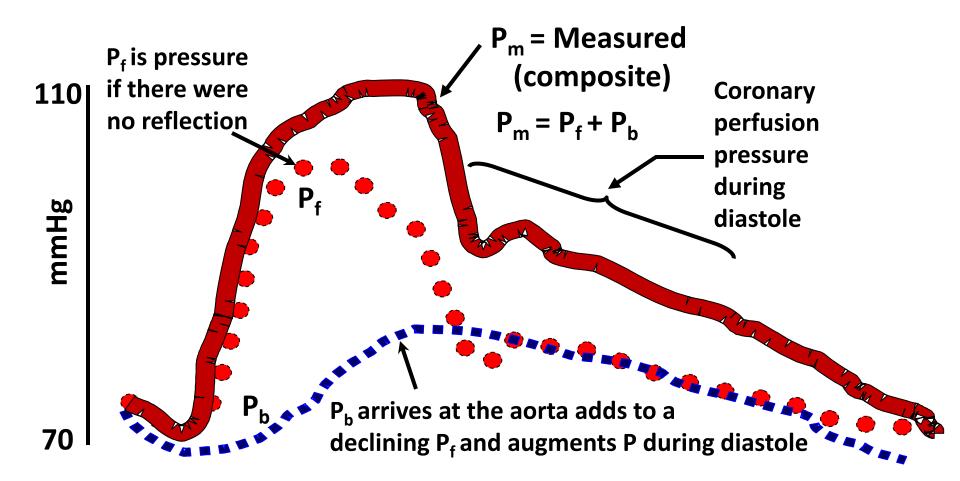


Reflected energy (P & Q) is less if branch pathways lead to vascular beds that have less Resistance. Reflection is greater if these have greater R.

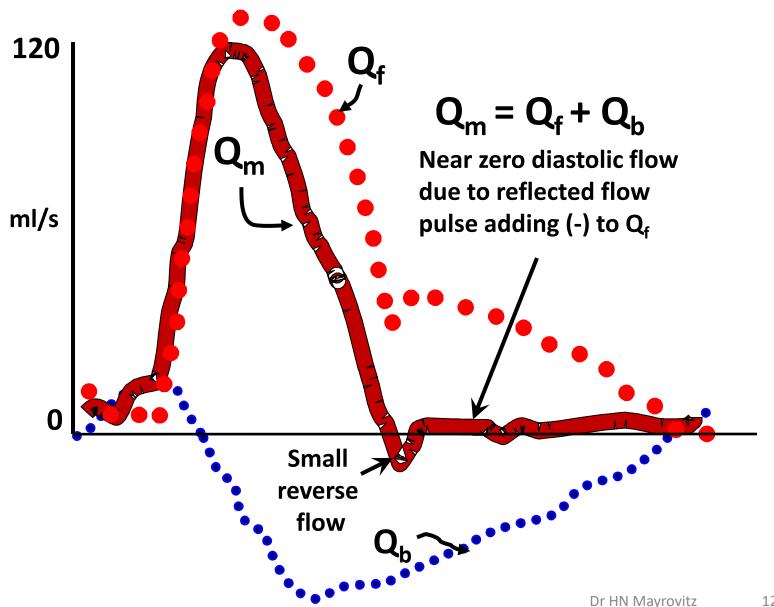
## **Forming Normal Femoral Arterial Pulsatile Flow**



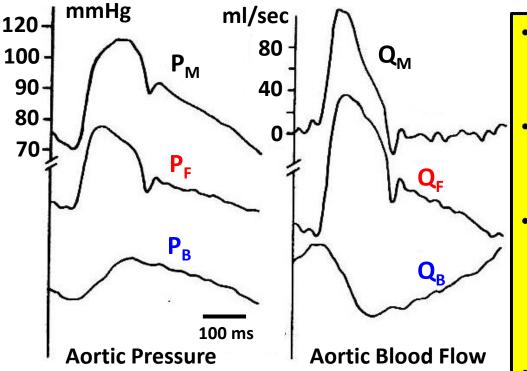
## **Forming Normal Ascending Aortic Pressure**



#### **Forming Normal Ascending Aortic Blood Flow**



# **Aortic Pressure and Flow Reflections: Summary**

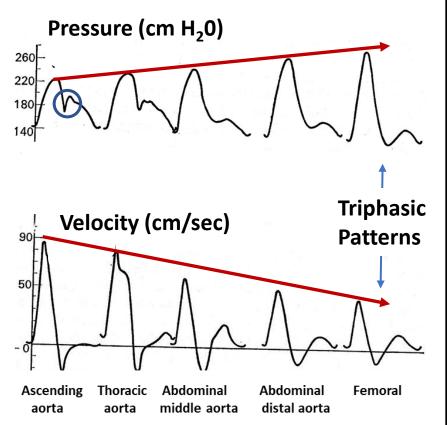


Subscripts F and B denote forward and backward wave components. Subscript M denotes measured value that depends on both forward and backward waves

#### **Take Home Points**

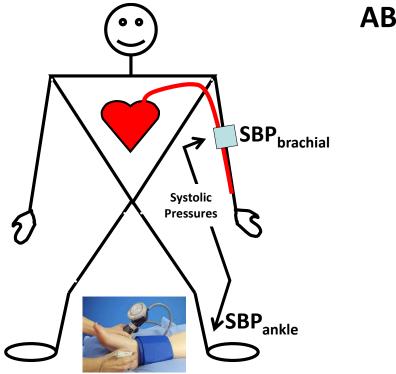
- Forward and backward pressure waves add to form measured (P<sub>M</sub>) pressure.
- Backward *flow* wave *subtracts* from the forward wave to form the measured flow (Q<sub>M</sub>)
- A greater pulse wave speed causes reflected waves to arrive back to the aortic site earlier causing an elevation in early systolic pressure and a reduction in diastolic pressure
- These effects cause an increase in cardiac afterload and decreased coronary perfusion respectively.
- The effect of earlier flow pulse return is to decrease net measured blood aortic pulse flow

# **Pulse Pressure Variations with Arterial Position**



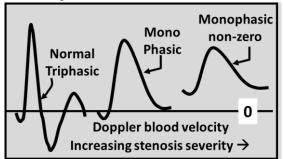
 Pressure pulses increase and smooths from aorta toward periphery Factors involved are due to combined effects of (1) high frequency filtering because arterial system acts a low frequency filter (2) greater transmission speed of higher frequency components (fast upstroke part) because wall does not expand rapidly enough (3) wave reflections due to interactions Peak blood velocity decreases with distance but the smoothing of the waveforms occurs for the same reasons as the pressure effects Pulse wave velocity increases in more peripheral arteries due to compliance decrease

# Ankle Brachial Systolic BP Index (ABI)

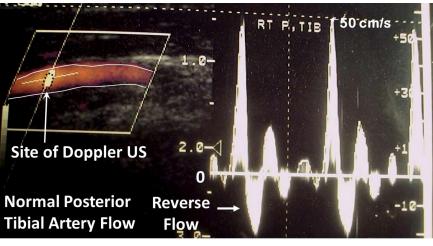


BI = (Ankle/Brachial) Systolic Pressure		
	Normal $\rightarrow$ 1.00 – 1.40 Borderline $\rightarrow$ 0.90 – 0.99 < 0.90 $\rightarrow$ Abnormal	
	0.80 – 0.89 Mild Blockage	
Claudication $\rightarrow$	0.50 – 0.79 Moderate Block	
Rest Pain →	< 0.50 Severe Block	

Triphasic pattern lost due to upstream stenosis

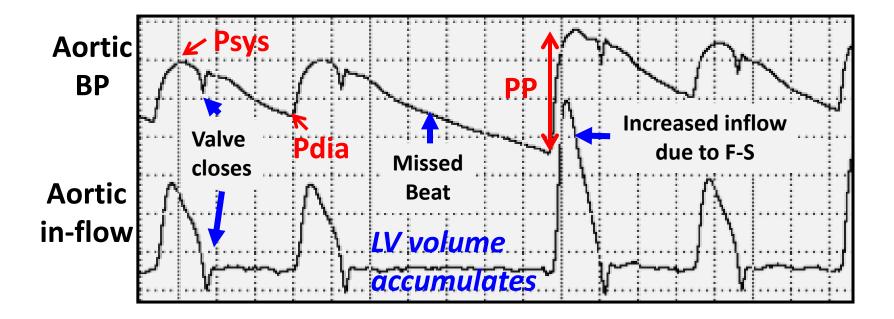


Normal Velocity Pulse Pattern is Triphasic



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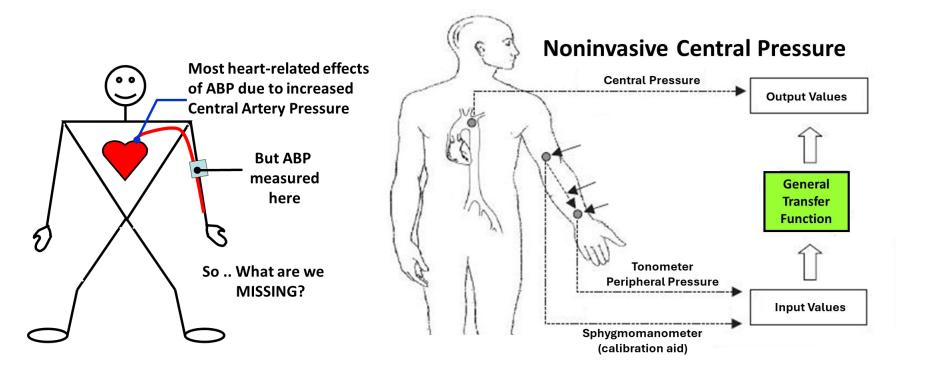
#### **Pulse Pressure Determinants**

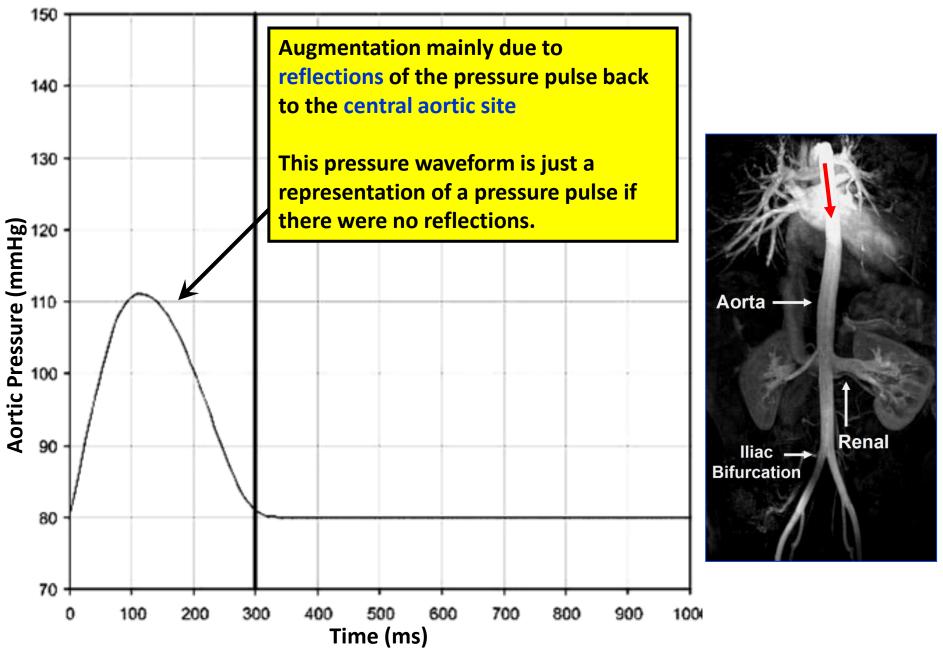


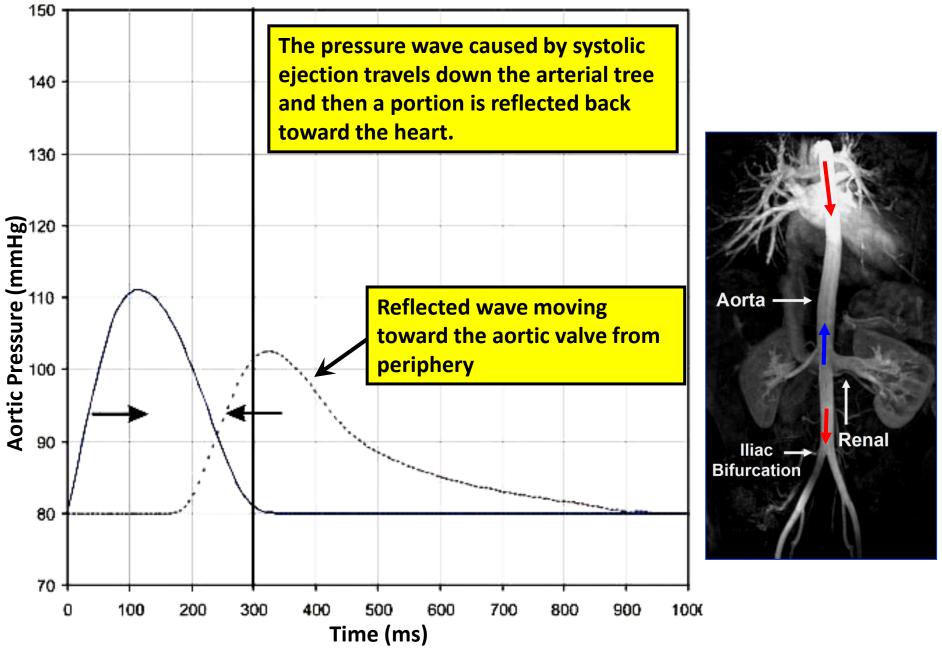
**Increase in arterial pulse pressure** 

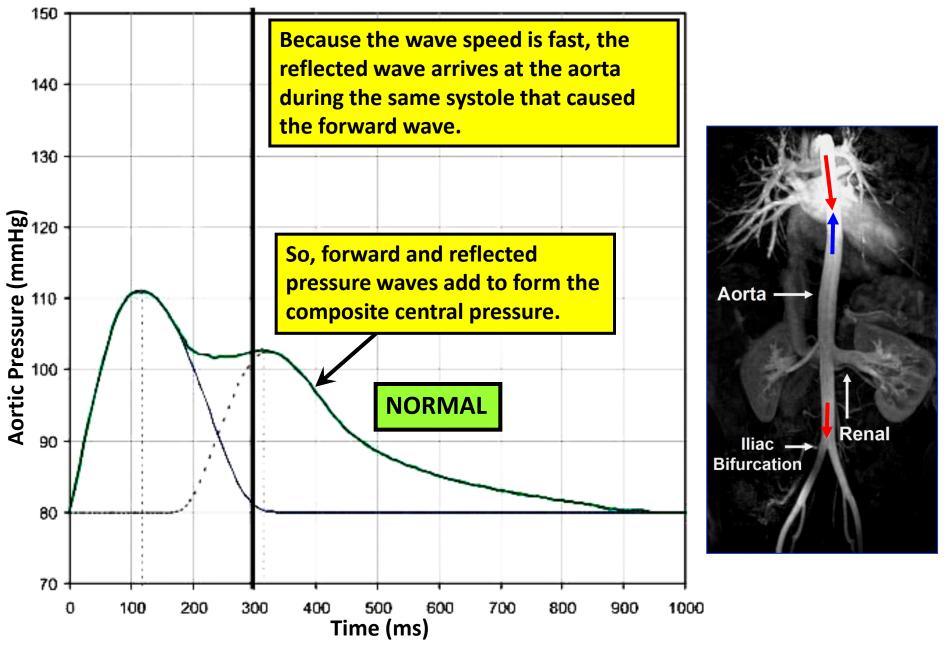
- Increased rate of LV ejection  $\rightarrow$  + dV/dt
- Increased stroke volume → + SV
- Decreased aortic compliance  $\rightarrow$  C

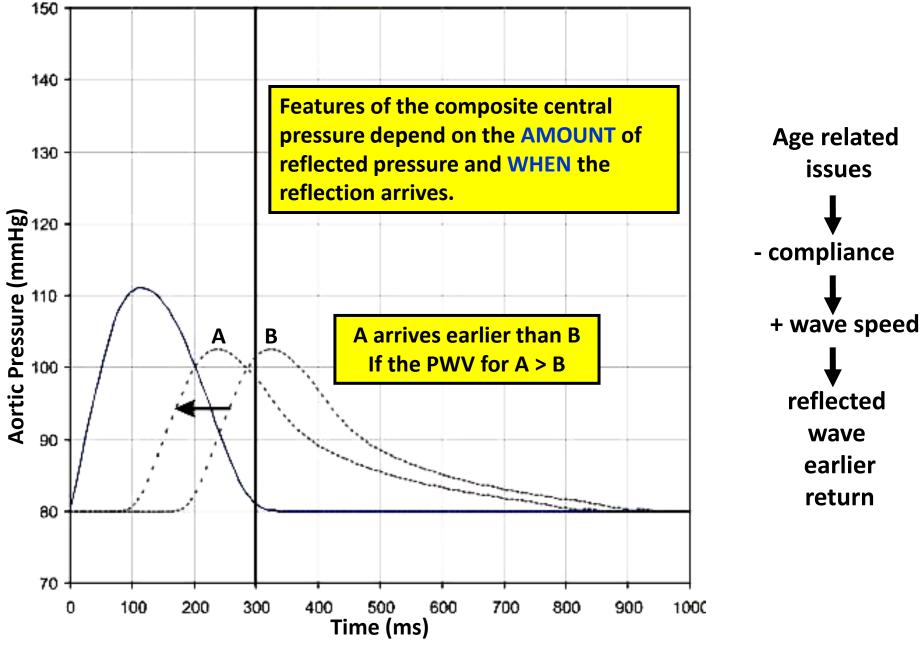
# **Central Pressure Augmentation via Pulse Reflection**



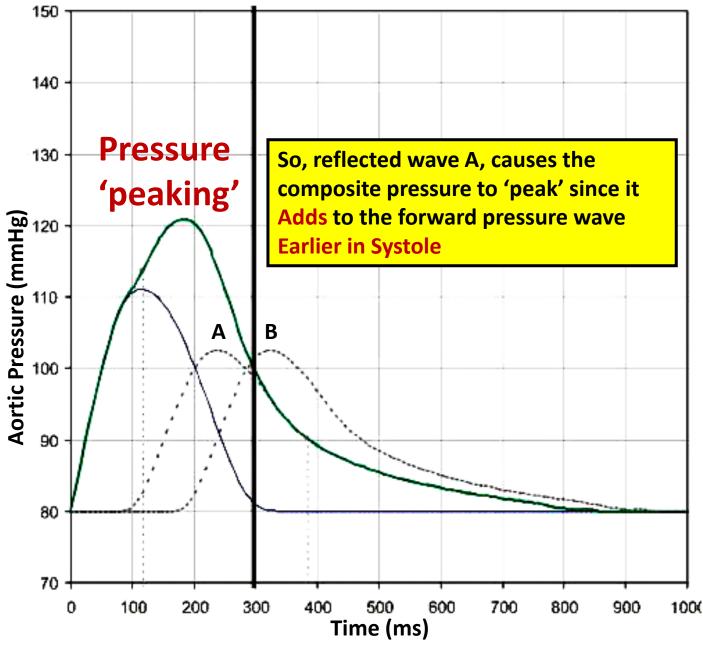


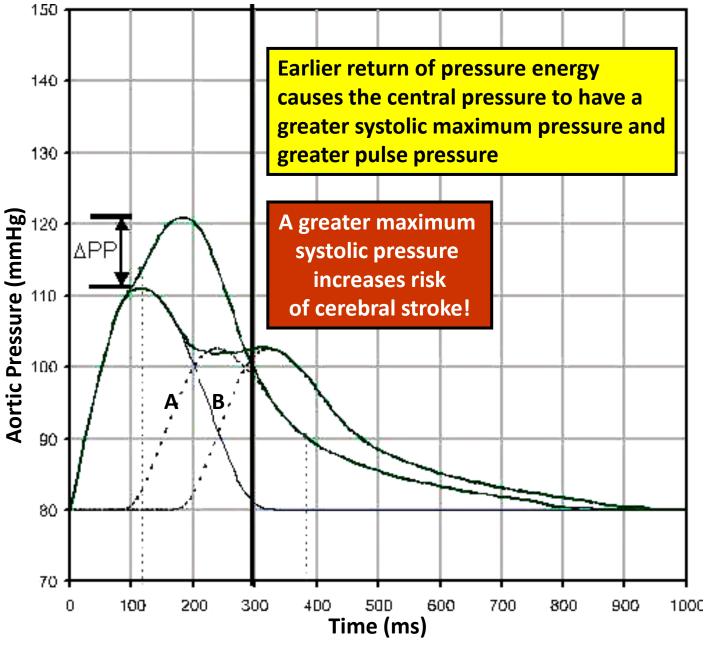


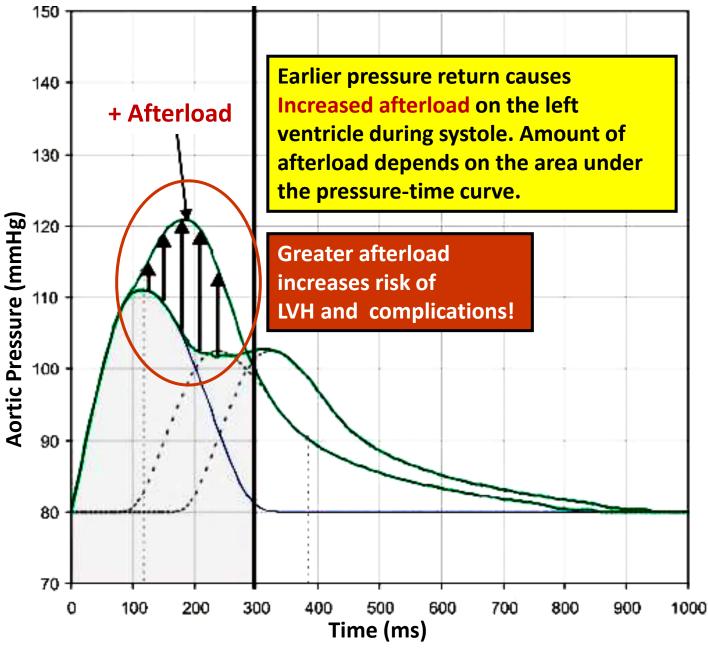


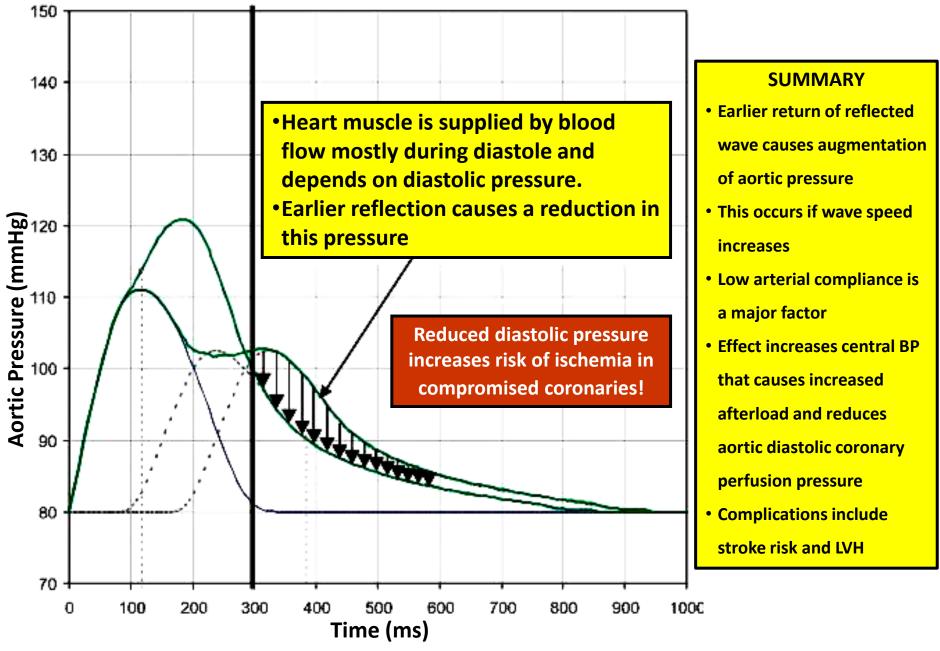


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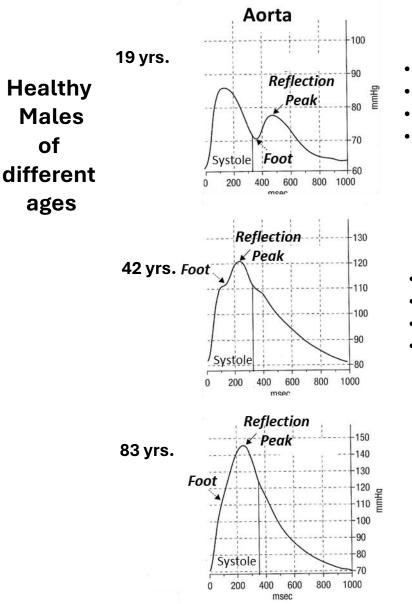








## **Central Pressure Augmentation Examples: AGE**

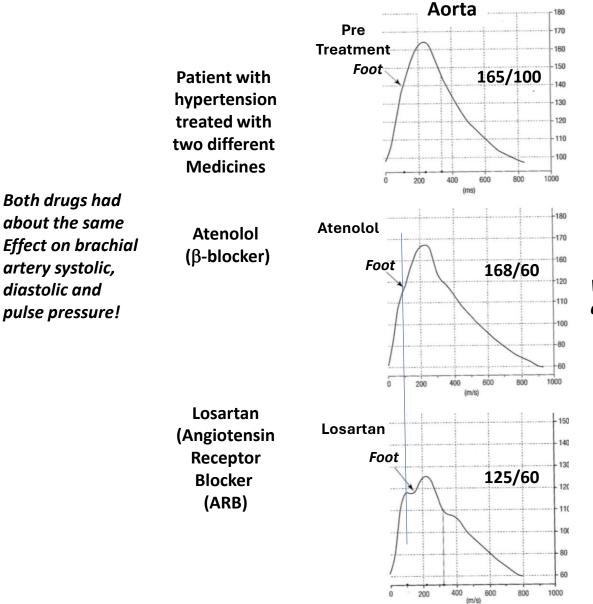


- Reflection in diastole only
  - Contributes to coronary perfusion
- No augmentation of systolic pressure
- Does not increase systolic afterload

- Reflection in mid-systole
- Augments systolic pressure
- Increases systolic afterload
- AI = 25%

- Reflection in early-systole
- Greatly augments systolic pressure
- Large increase in systolic afterload
- AI = 46%

# Central Pressure Augmentation: Which Drug to use?



#### How did drug effects on central systolic pressure compare?

Losartan more reduction in systolic

# What accounts for the BP reduction associated with Losartan?

Reduced AI due to later reflection

# **End CV Physiology Lecture 12**

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