Regulation of SV and CO

Stroke Volume, Cardiac Output and Ejection Fraction

- **Stroke Volume (SV)**
- SV = The volume (ml) of blood ejected during one ventricular systole (it is the same for RV and LV if no valve regurgitation, or shunt exists) = EDV-ESV
- **SV x HR =** Total ml ejected in 1 minute (ml/min) **= Cardiac Output (CO, ΚΛΟΑ)**
- **CI (Cardiac Index) = CO/BSA**
- **Ejection Fraction (EF)** = SV/EDV = (EDV-ESV)/EDV

Stroke Volume, Cardiac Output and Ejection Fraction

- **SV x HR = (CO, ΚΛΟΑ):** Critical circulatory parameter
- **Ejection Fraction (EF):** Powerful predictor of prognosis
  - Normal = 60%
  - When reduced ➔ **HFrEF** (Heart Failure with reduced EF), approximately 50-60% of HF pts
  - When severely reduced **(< 35%)** ➔ risk of SCD (Sudden Cardiac Death) ➔ Reduced survival. Need for AICD (Automatic Implantable Cardioverter Defibrillator)

Regulators of SV (CO) and EF

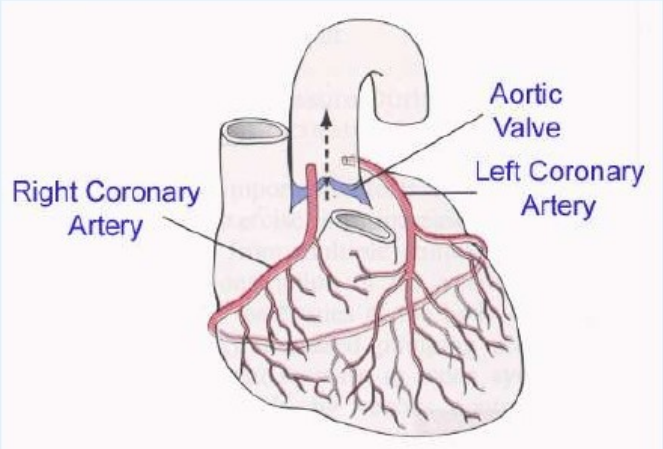
- Preload
- Afterload
- Contractility

EDV & ESV

- **Preload** is the volume at which the heart is most filled with blood at the end of diastole; this value is equal to end-diastolic volume **(EDV)**.
  - End Systolic Volume **(ESV)** is the volume of blood remaining in the ventricles after systole.

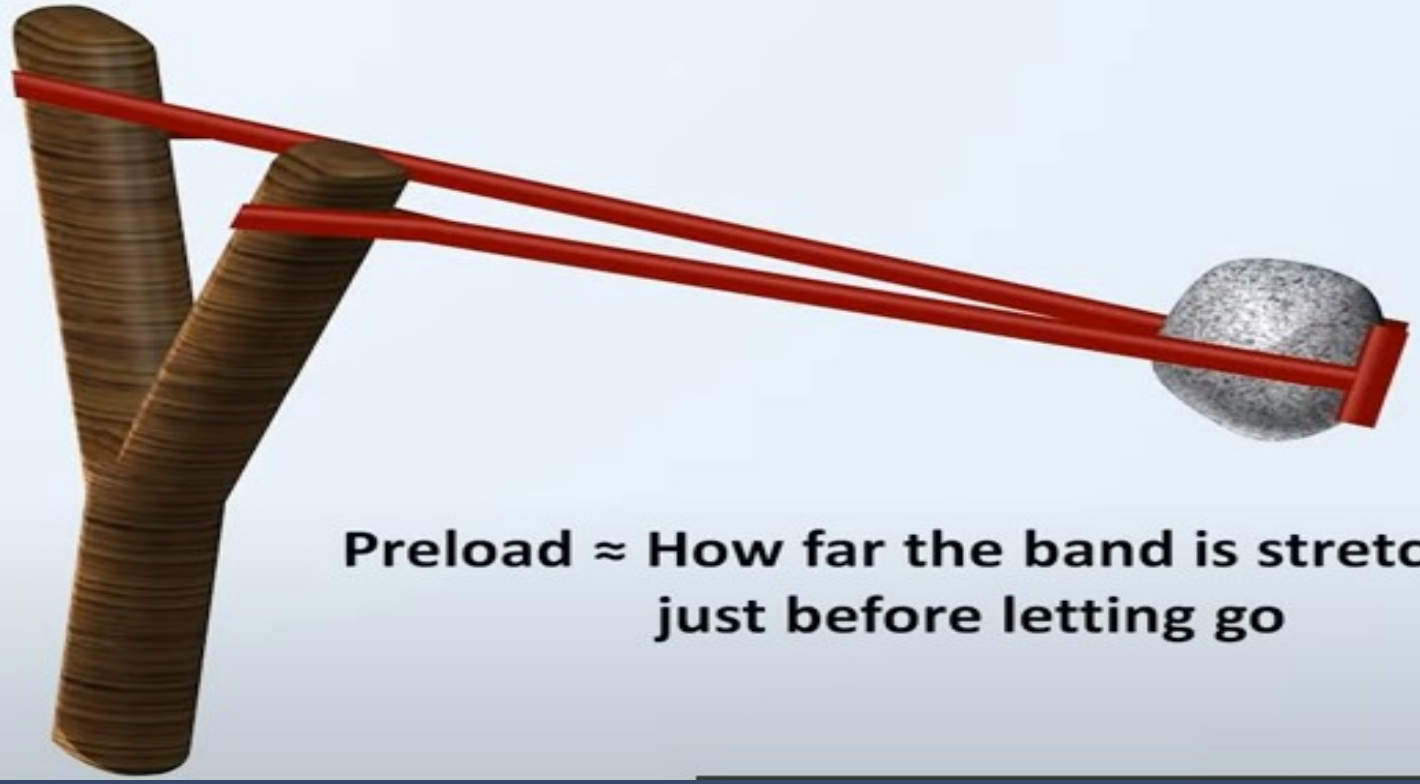
Starling's Law of the Heart

- The greater the stretch of the myocardial fibers, the stronger the force of the contraction.

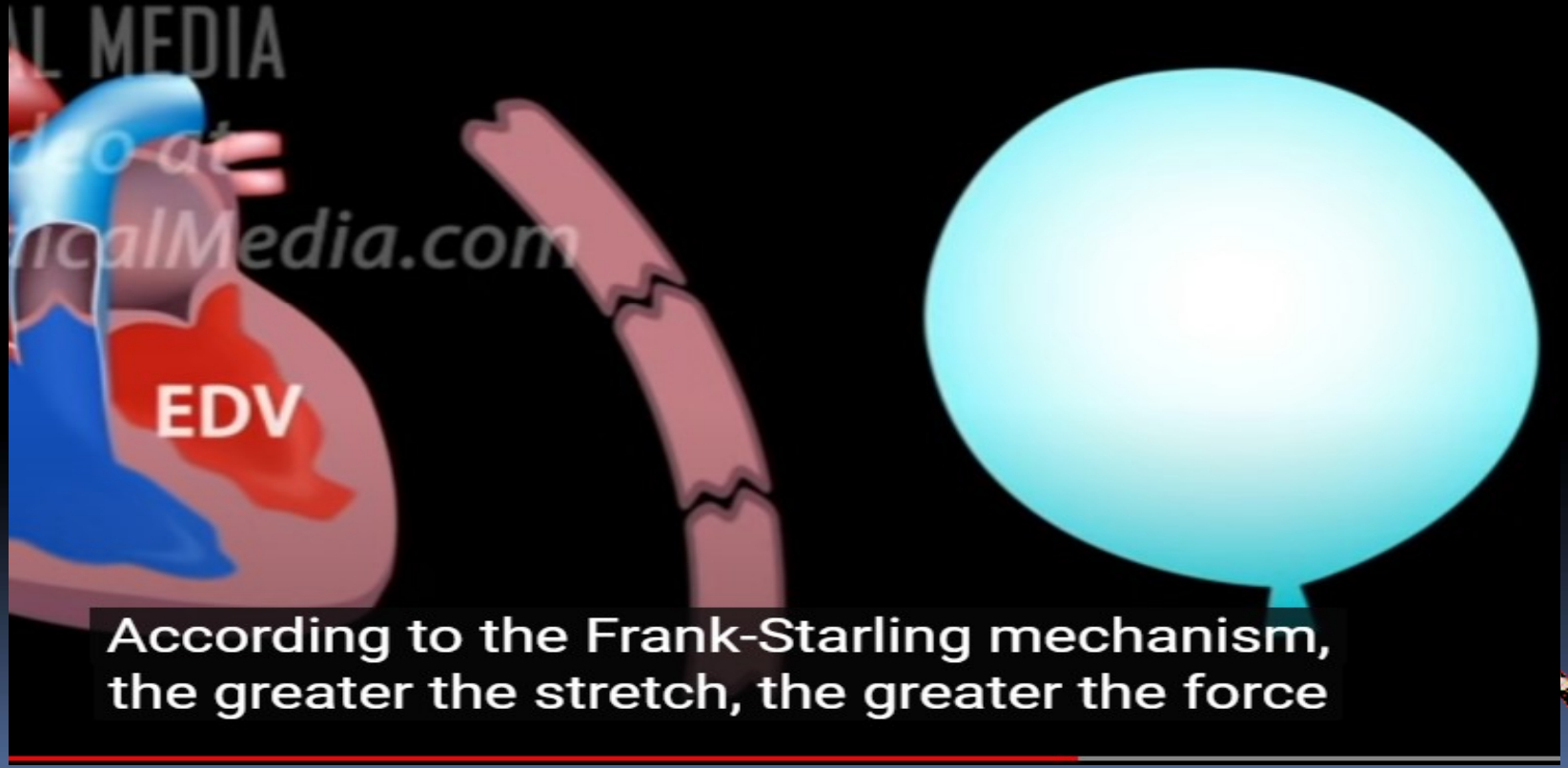


Mechanism of Starling Law:

An increase in preload ➔ increase in the sarcomere length ➔ increases **troponin C** calcium sensitivity ➔ increases the rate of cross-bridge attachment and detachment, and the amount of tension developed by the muscle fiber ➔ **This increases SV**…


Preload ≈ How far the band is stretched
just before letting go

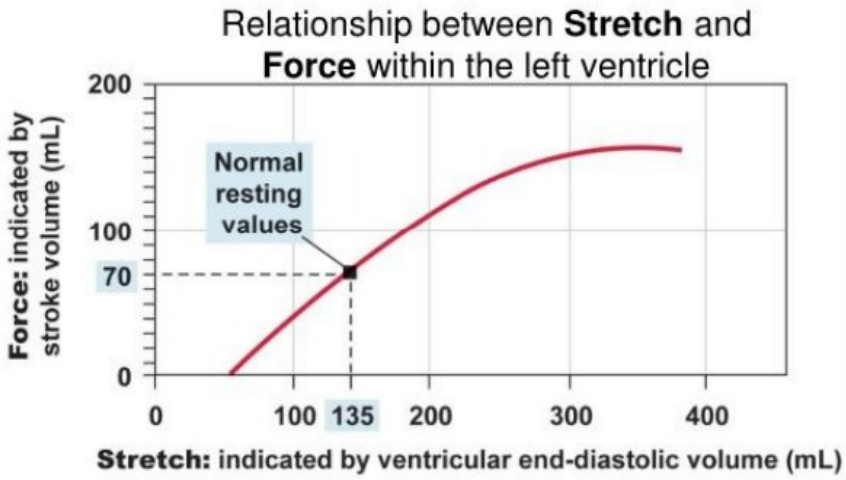
Preload


EDV
According to the Frank-Starling mechanism,
the greater the stretch, the greater the force

Cardiovascular Physiology

Cardiac Output

- Influencing stroke volume
  - Pre Load
    - The amount of stretch within the contractile myocardial fibers
    - Represents the "load" placed on the muscle fibers before they contract
    - They respond according to length-tension patterns observed in muscle tissue by Frank, then by Starling
      - Became known as the Frank-Starling Law of the Heart
      - "The heart will pump all the blood that is returned to it"

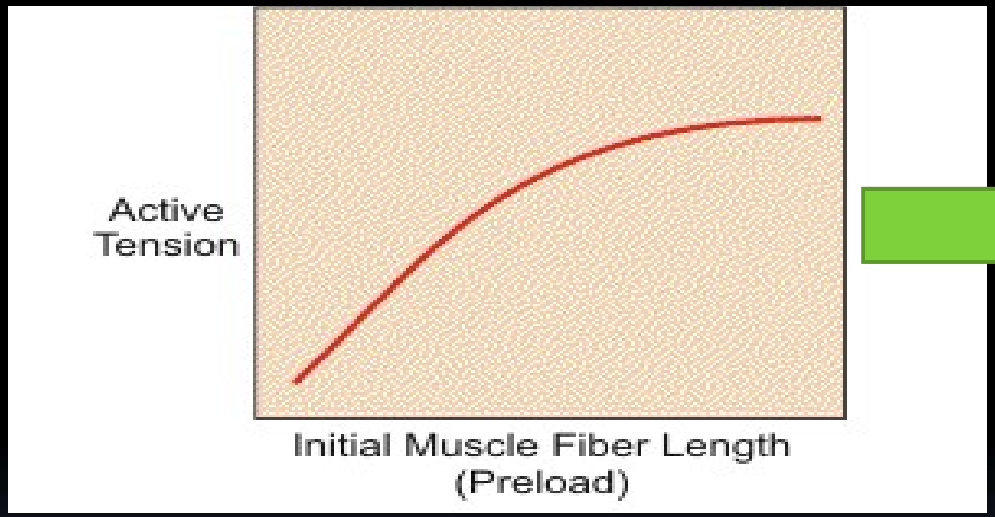


Starling's Law of the Heart

- The heart adjusts its pumping rate to the rate of blood return. How?
  - More blood returning stretches atria and ventricles more
    - Stretching SA node muscle causes faster rhythmicity
    - Stretching heart muscle causes faster conduction
    - Stretching heart muscle **causes stronger**, more complete contraction…

Εκπαιδευτικός Στόχος (ΕΣ): Η αρχή και το τέλος της ΚΔ λειτουργίας, ο νόμος FS

Preload and SV…



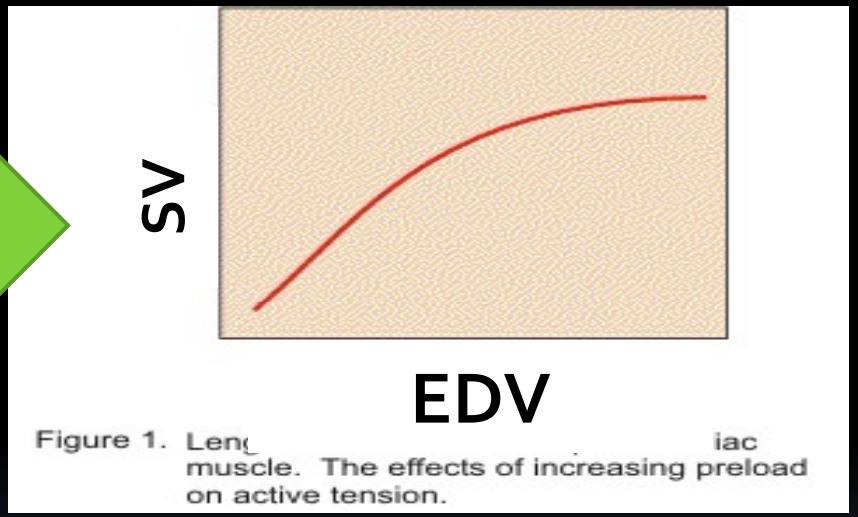
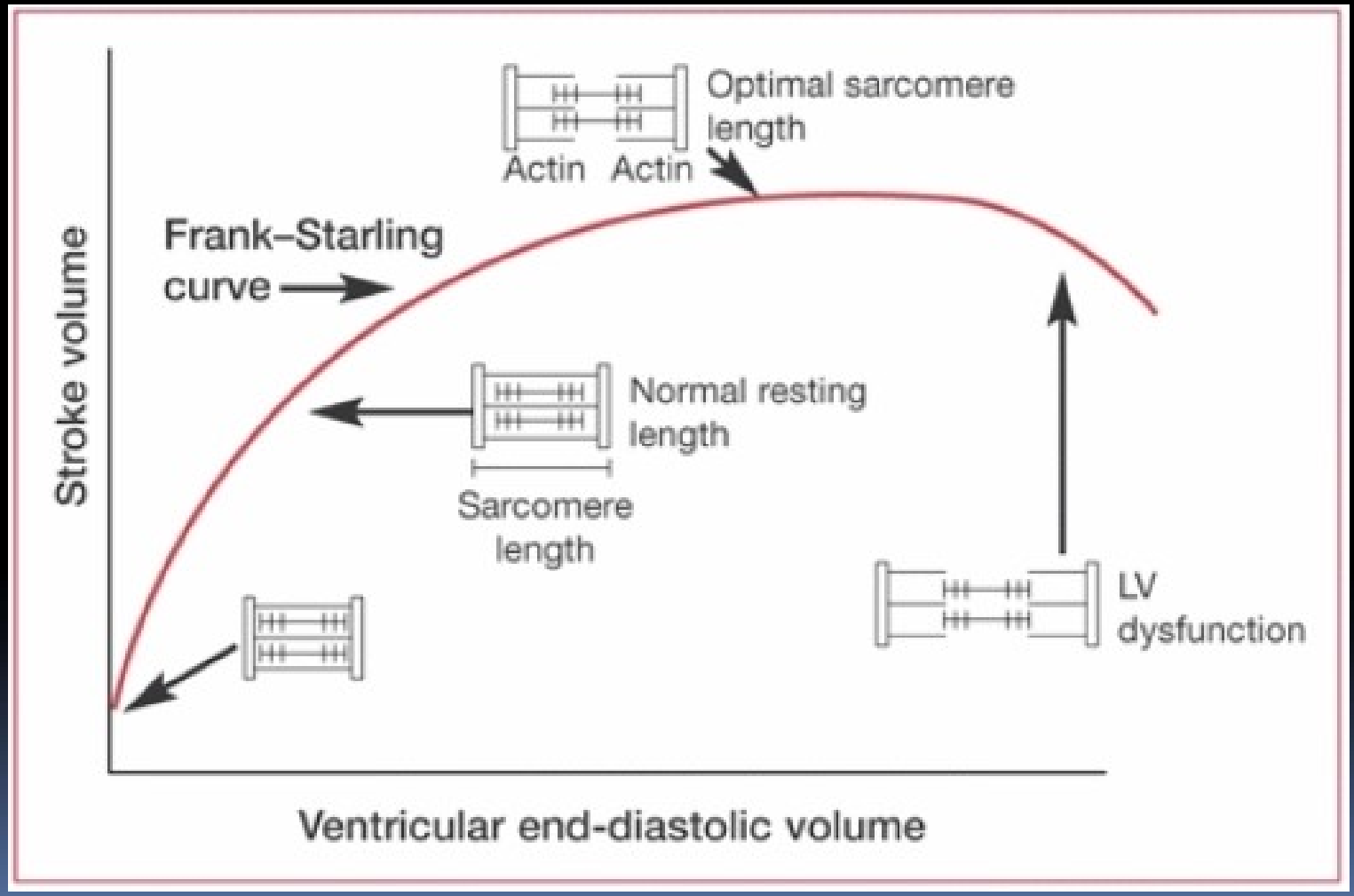
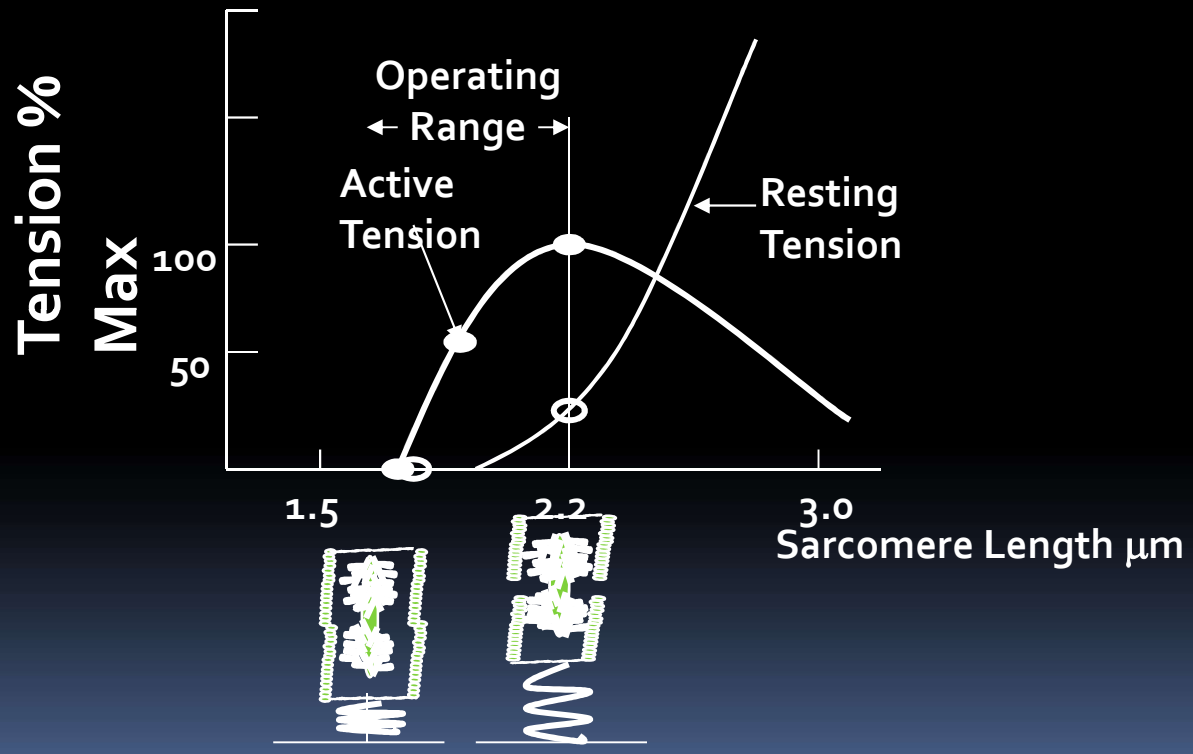


Figure 1. Leng iac muscle. The effects of increasing preload on active tension.

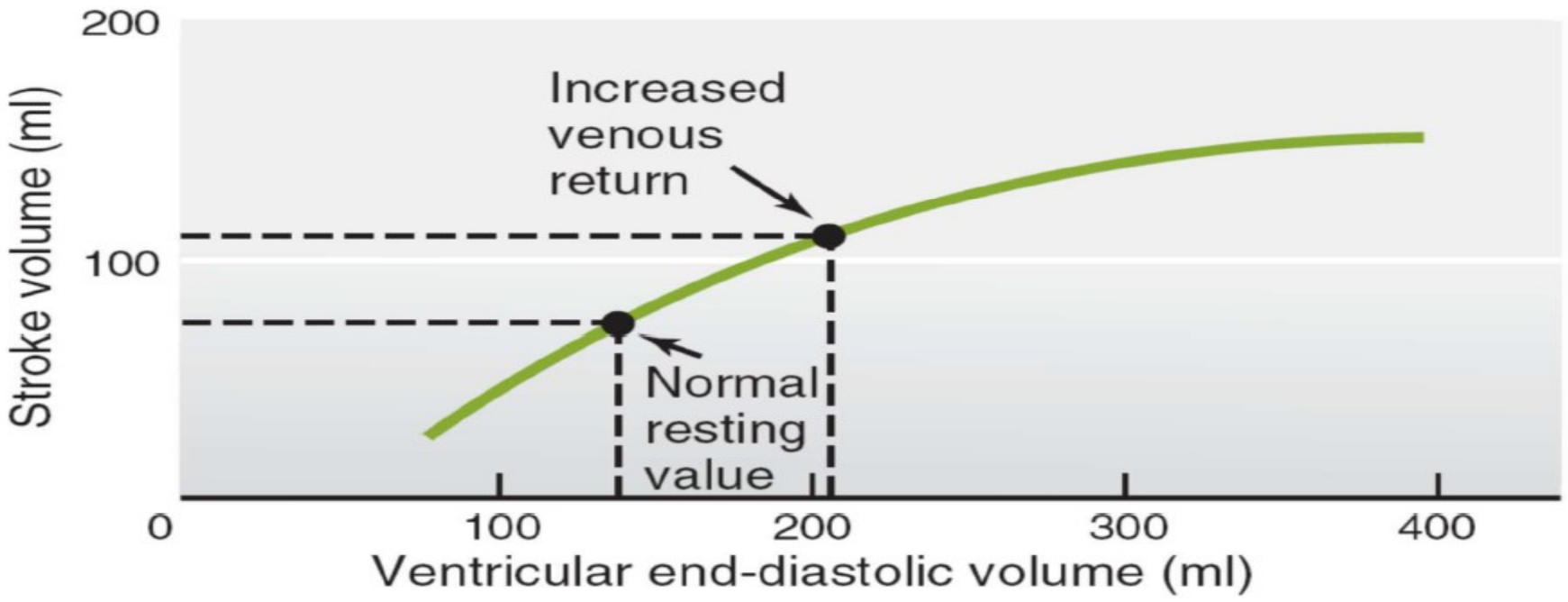
Starling's Law of the Heart


Optimal sarcomere length
Actin
Actin
Frank–Starling curve
Stroke volume
Normal resting length
Sarcomere length
LV dysfunction
Ventricular end-diastolic volume

Length Tension Relationship

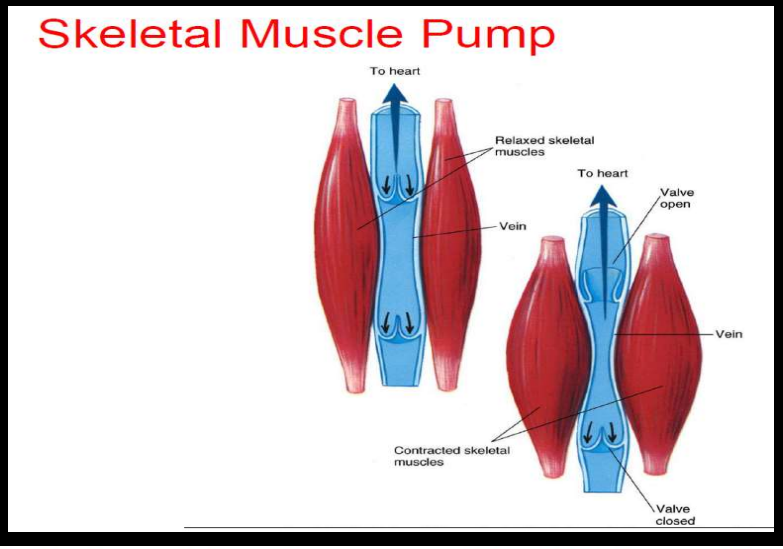


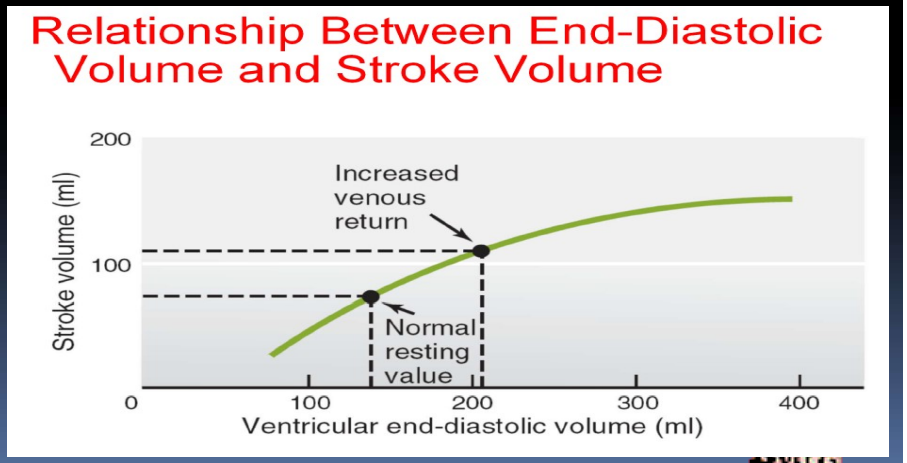
Relationship Between End-Diastolic Volume and Stroke Volume



Example: Exercise

- The EDV and Stroke Volume
  - At rest
    - EDV is low
    - Myocardium stretches less
    - Stroke volume is low
  - With exercise
    - EDV increases (venus return)
    - Myocardium stretches more
    - Stroke volume increases





Εκθετική σχέση διαστολικής πίεσης με όγκο: EDV vs. EDP…

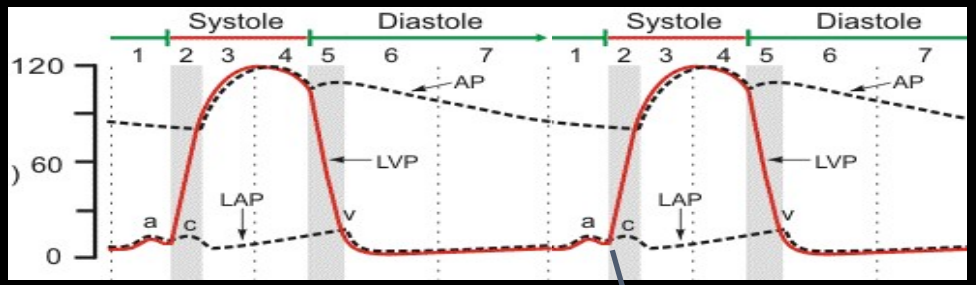
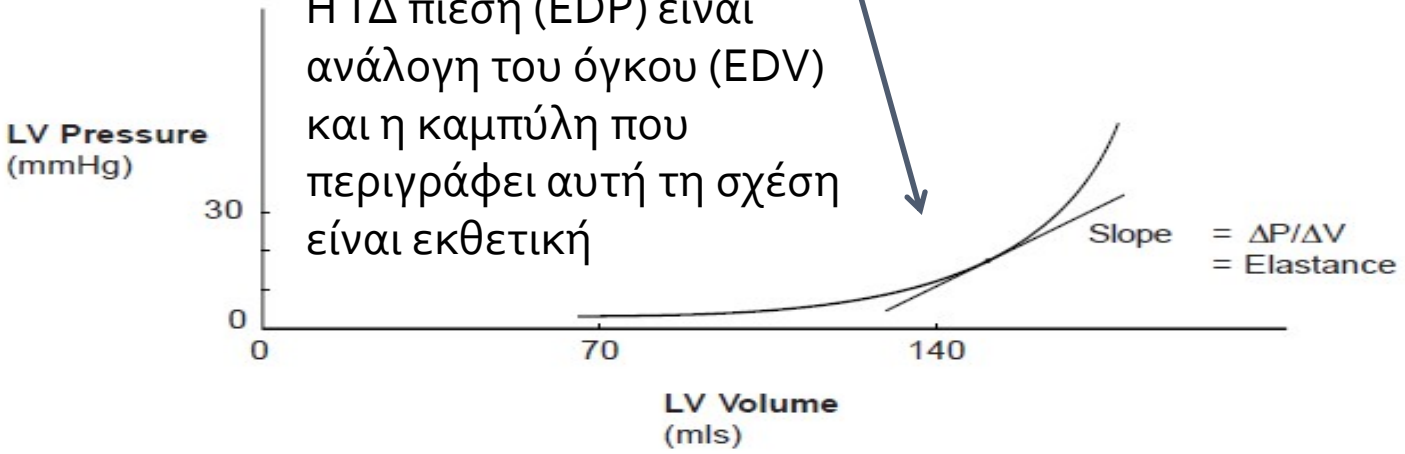
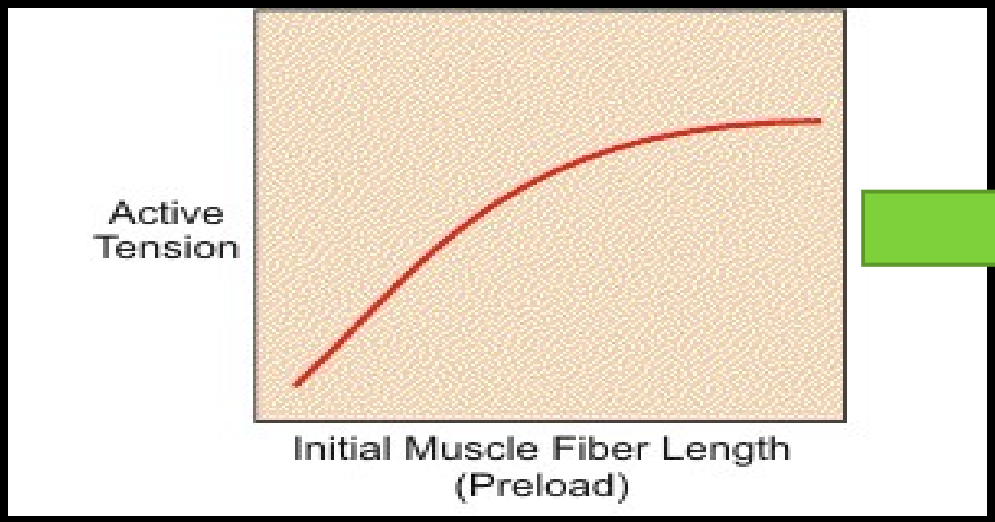
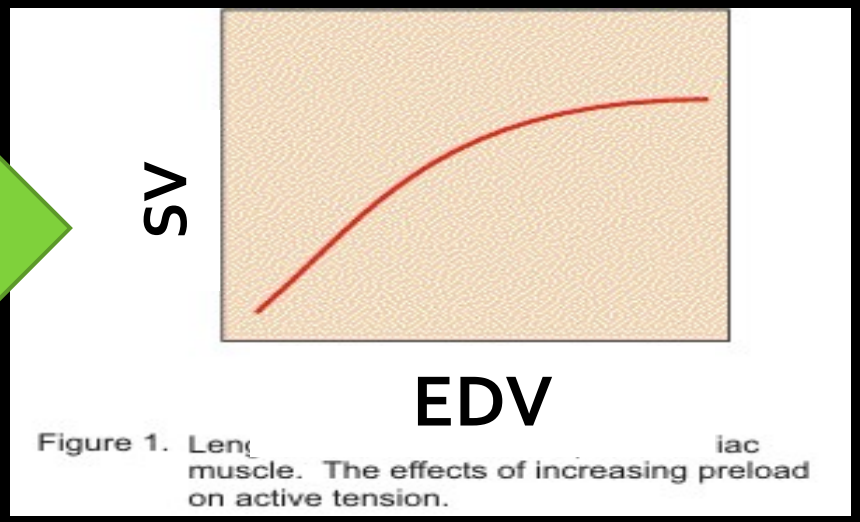


Fig 3.3 LV Pressure-Volume Relationship during Diastole

Η ΤΔ πίεση (EDP) είναι ανάλογη του όγκου (EDV) και η καμπύλη που περιγράφει αυτή τη σχέση είναι εκθετική




Active Tension
Initial Muscle Fiber Length (Preload)


SV
EDV
Figure 1. Leng iac muscle. The effects of increasing preload on active tension.

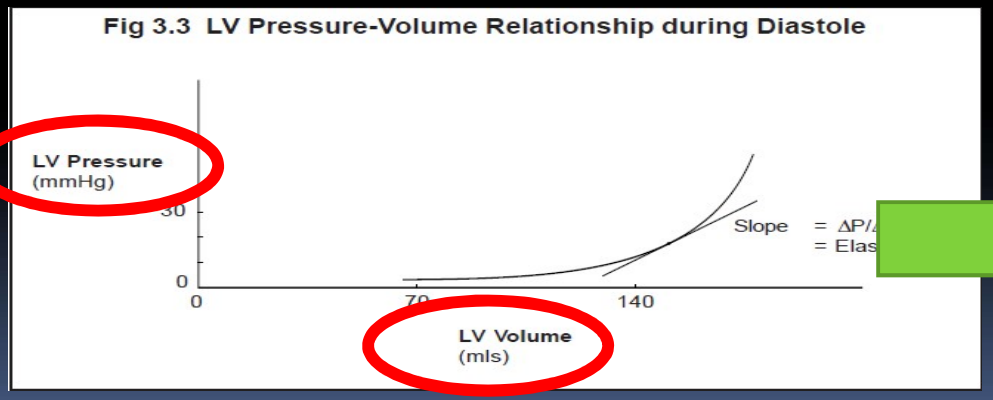
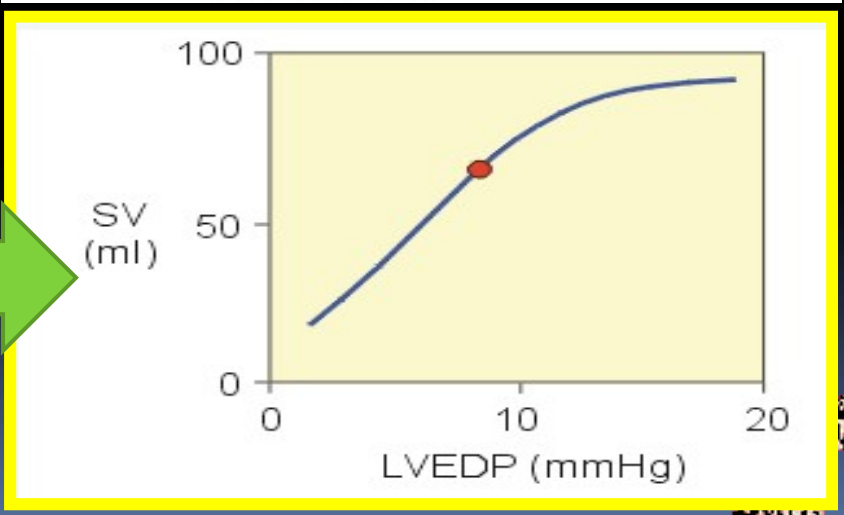


Fig 3.3 LV Pressure-Volume Relationship during Diastole
LV Pressure (mmHg)
30
0
0
70
140
Slope = ΔP/
= Elas
LV Volume (mls)


SV (ml)
100
50
0
0
10
20
LVEDP (mmHg)

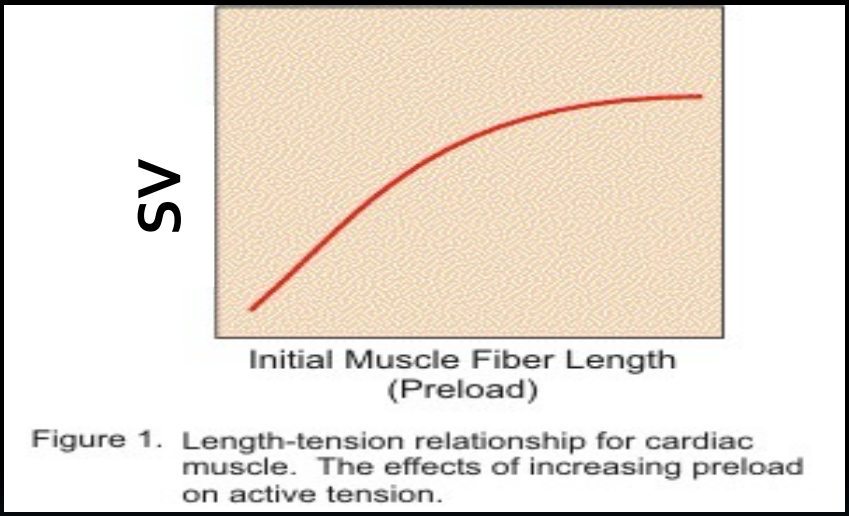
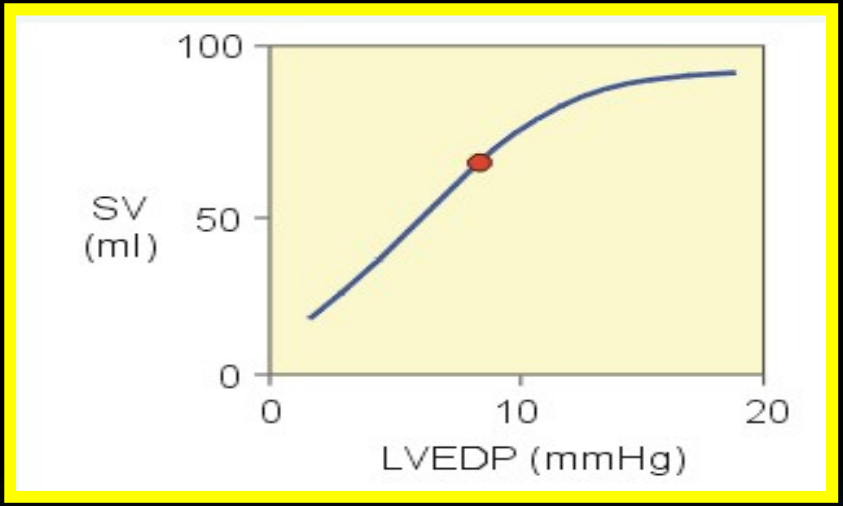
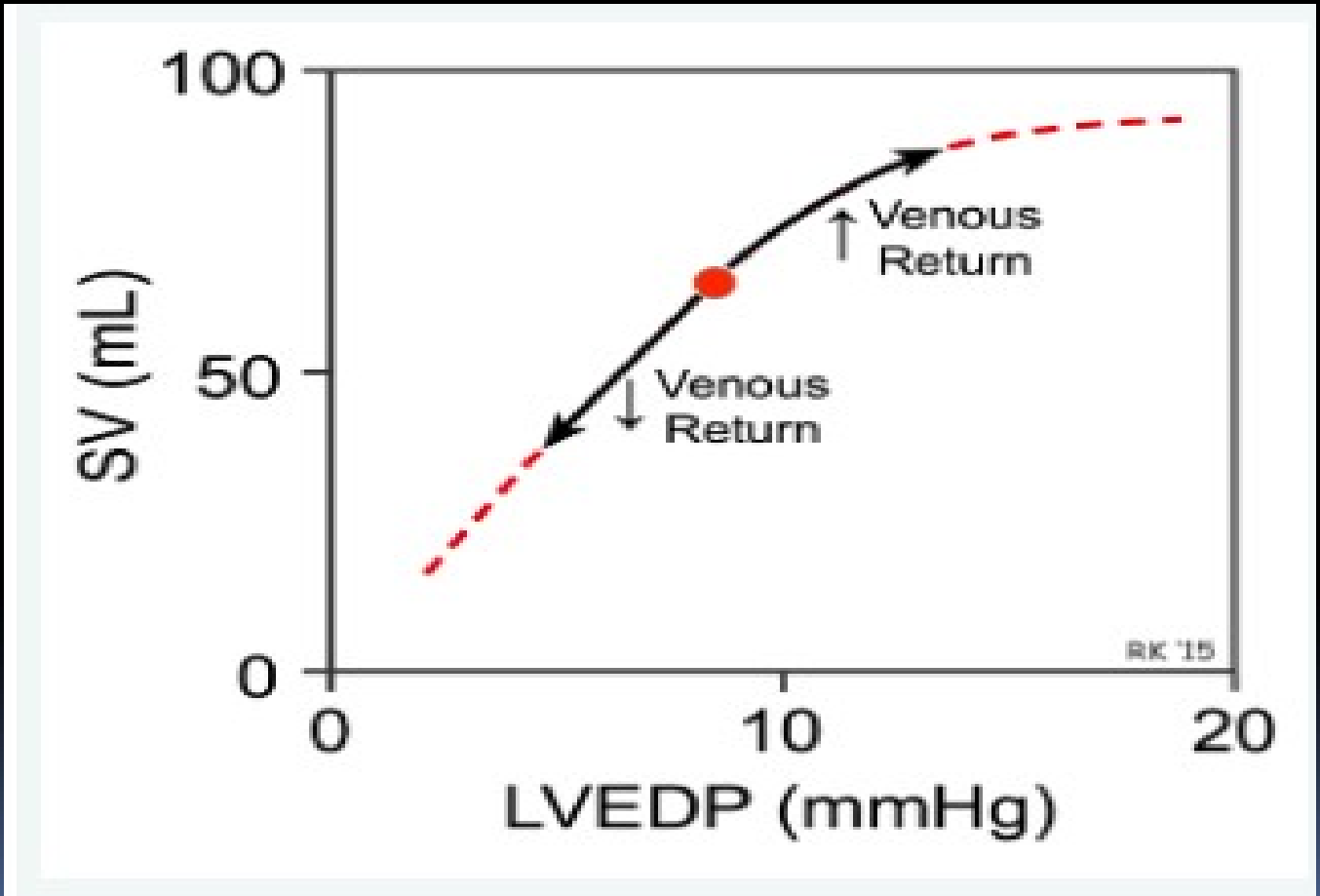


Figure 1. Length-tension relationship for cardiac muscle. The effects of increasing preload on active tension.



Starling's Law of the Heart

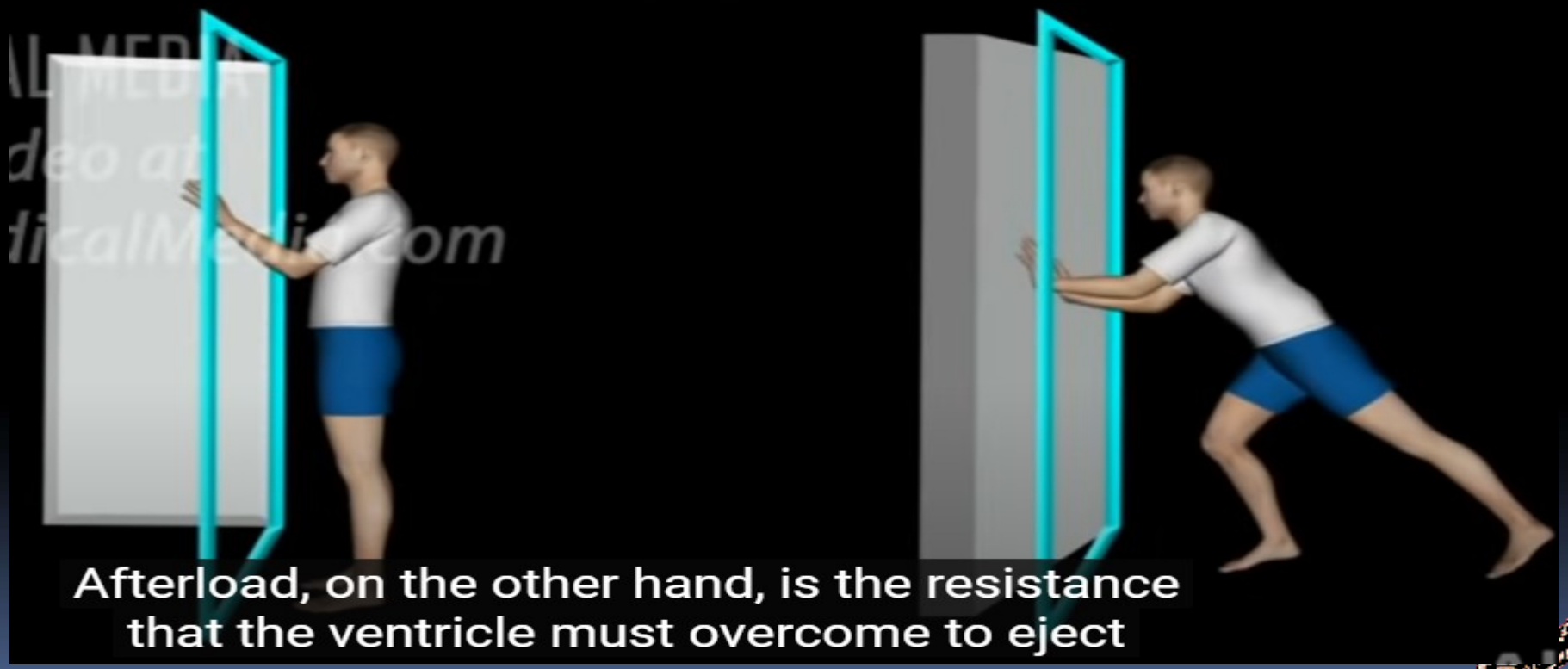


Μείωση προφορτίου (φλεβικής επιστροφής, VR) π.χ. αφυδάτωση΄-διουρητικά, φλεβοδιαστολή-μορφίνη, όρθια θέση, κτλ ➔ μείωση LVEDP ➔ μείωση SV και αντιστρόφως

Afterload

- Afterload is the pressure the ventricle must overcome to eject blood during systole…
  - Afterload can be defined as the "load" that the heart must eject blood against.
  - The afterload is closely related to the **blood vessel pressure, and accordingly to resistance (Systemic SVR, & Pulmonary PVR)**.

Afterload


Afterload, on the other hand, is the resistance
that the ventricle must overcome to eject

Afterload

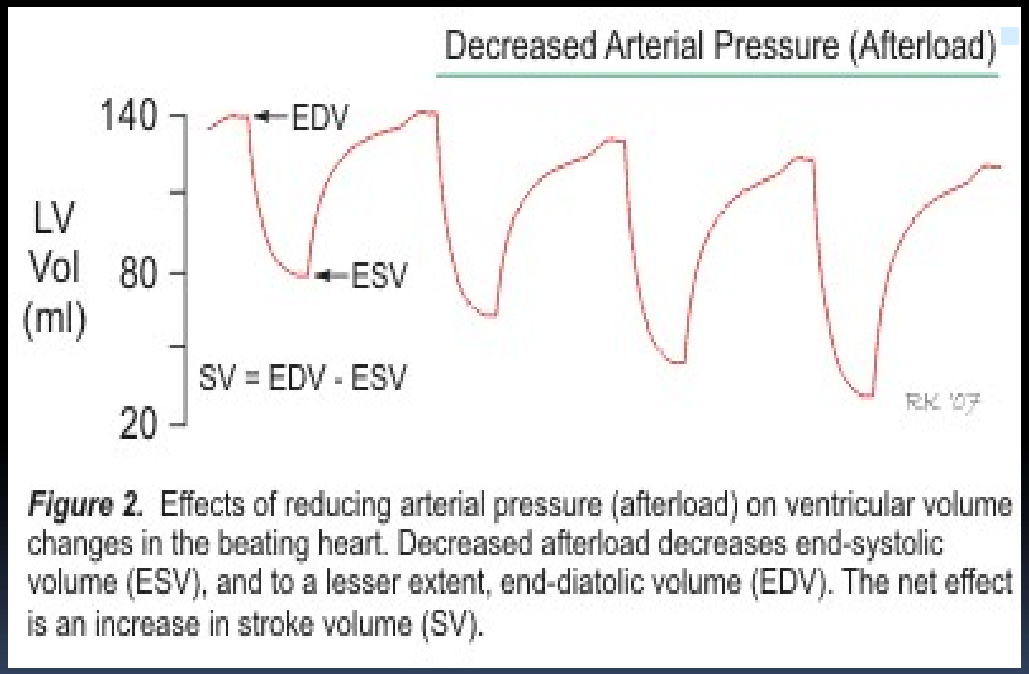


Figure 2. Effects of reducing arterial pressure (afterload) on ventricular volume changes in the beating heart. Decreased afterload decreases end-systolic volume (ESV), and to a lesser extent, end-diatolic volume (EDV). The net effect is an increase in stroke volume (SV).

When arterial pressure is reduced, the ventricle can eject blood more rapidly, which increases the stroke volume and thereby decreases the end-systolic volume.

Effect of an increase in afterload on SV

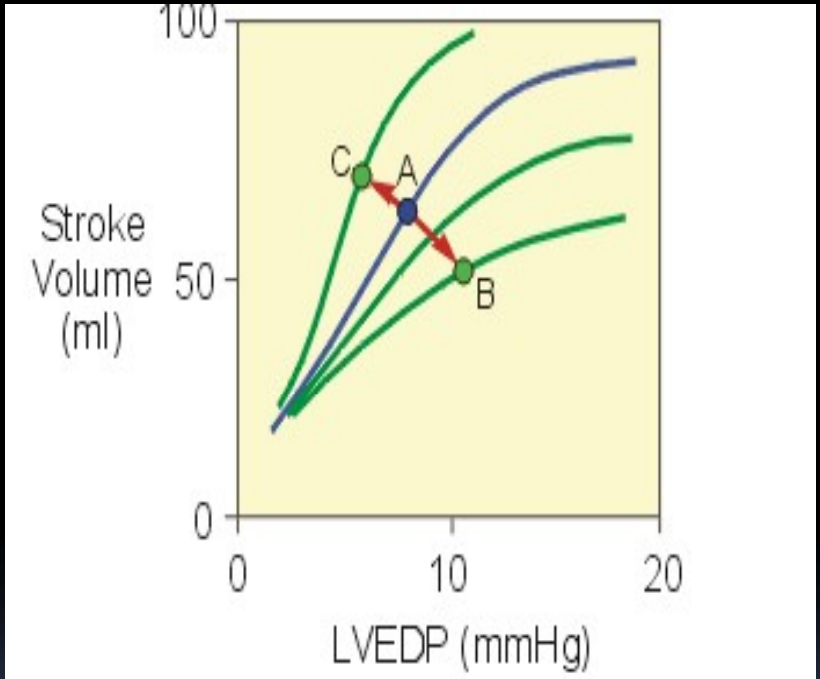


Figure 1. Effects of changes in afterload on Frank-Starling curves. A shift from A to B occurs with increased afterload, and from A to C with decreased afterload.

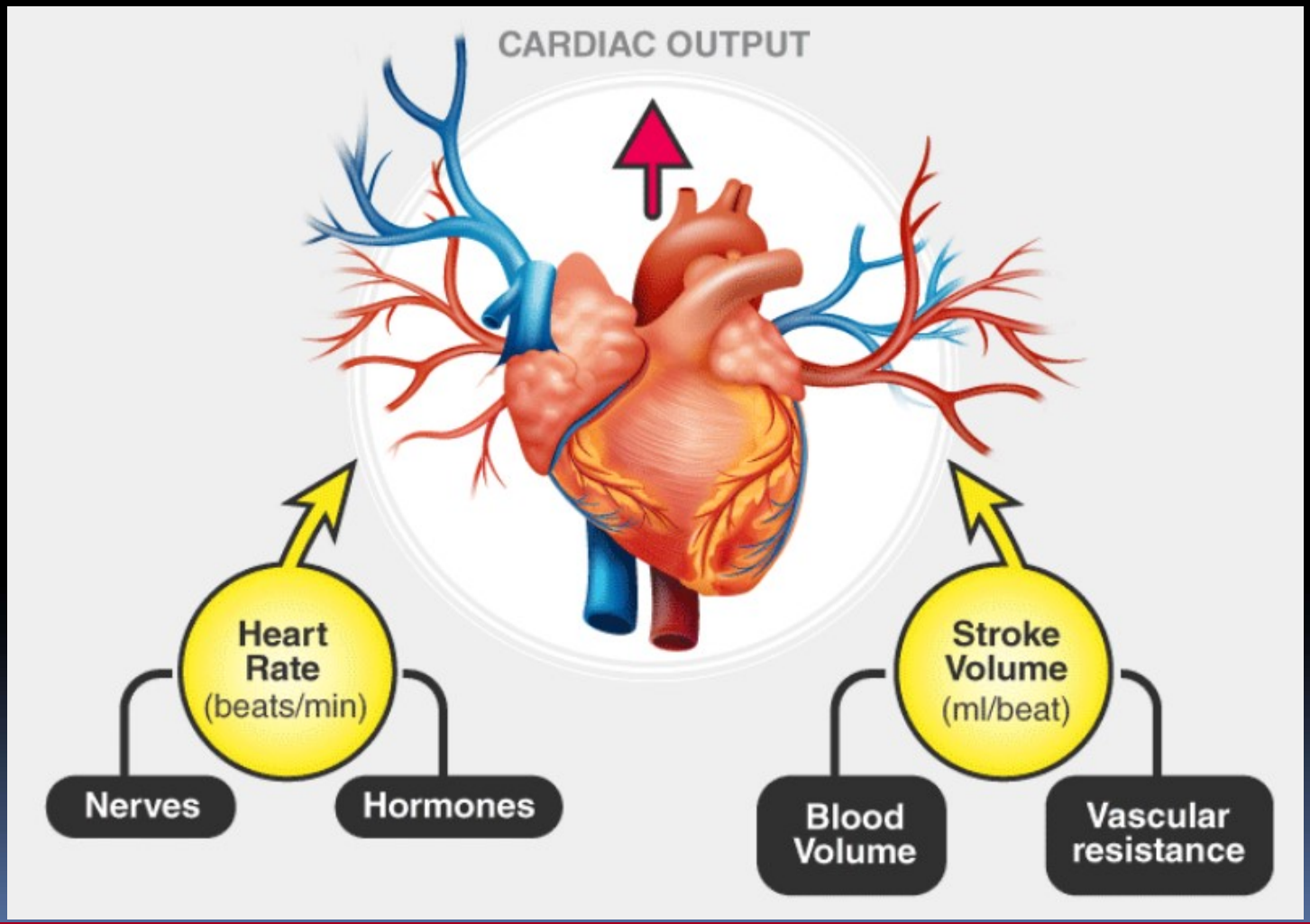
- An increase in afterload ➔ increase in ESV and a decrease in SV.
- An increase in afterload shifts the Frank-Starling curve down and to the right (from A to B).
- Explanation: increase in afterload decreases the velocity of fiber shortening. This reduces the rate of volume ejection so that more blood is left within the ventricle at the end of systole (increase ESV)
- A decrease in afterload shifts the Frank-Starling curve up and to the left (A to C).

Preload and Afterload

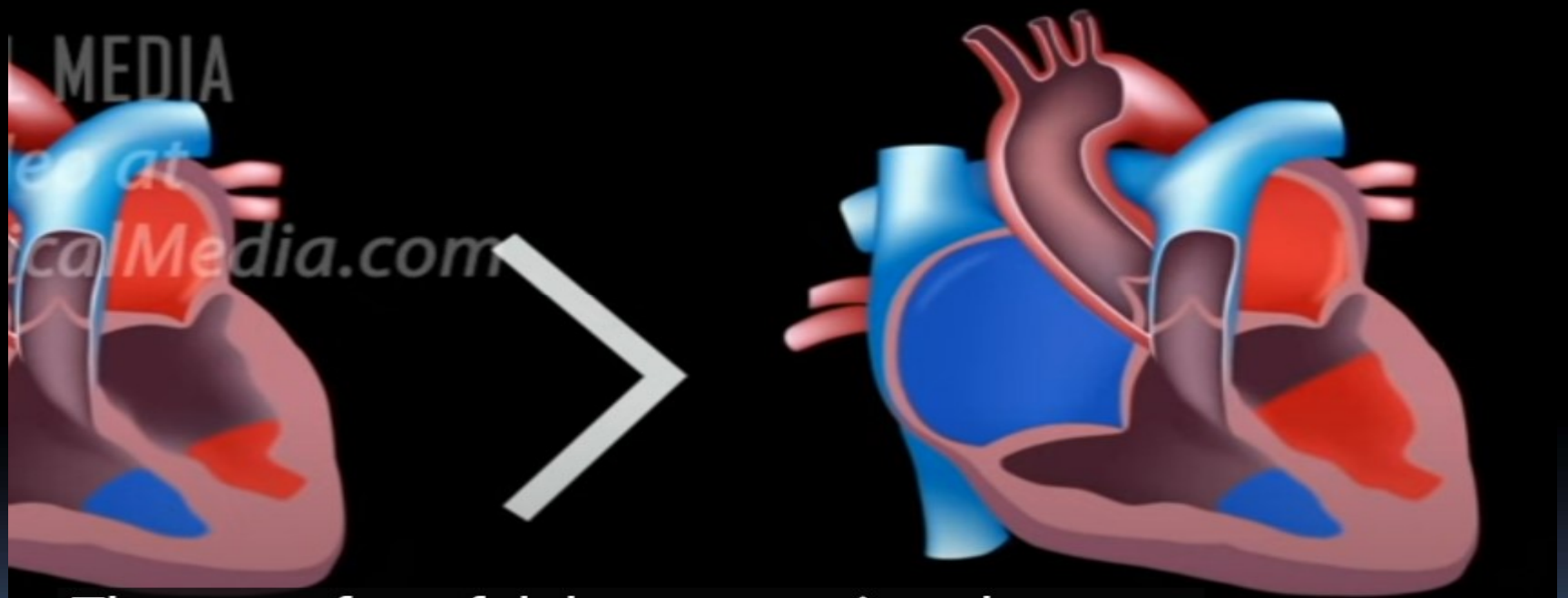
Preload: volume entering ventricles

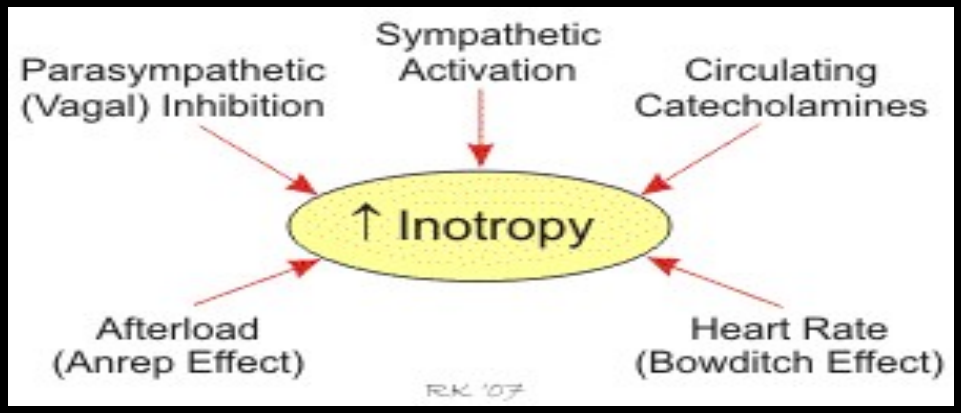
Afterload: resistance left ventricle must overcome to circulate blood

CARDIAC SYSTEM
Memory Notebook of Nursing


CARDIAC OUTPUT
Heart Rate (beats/min)
Nerves
Hormones
Stroke Volume (ml/beat)
Blood Volume
Vascular resistance

Contractility





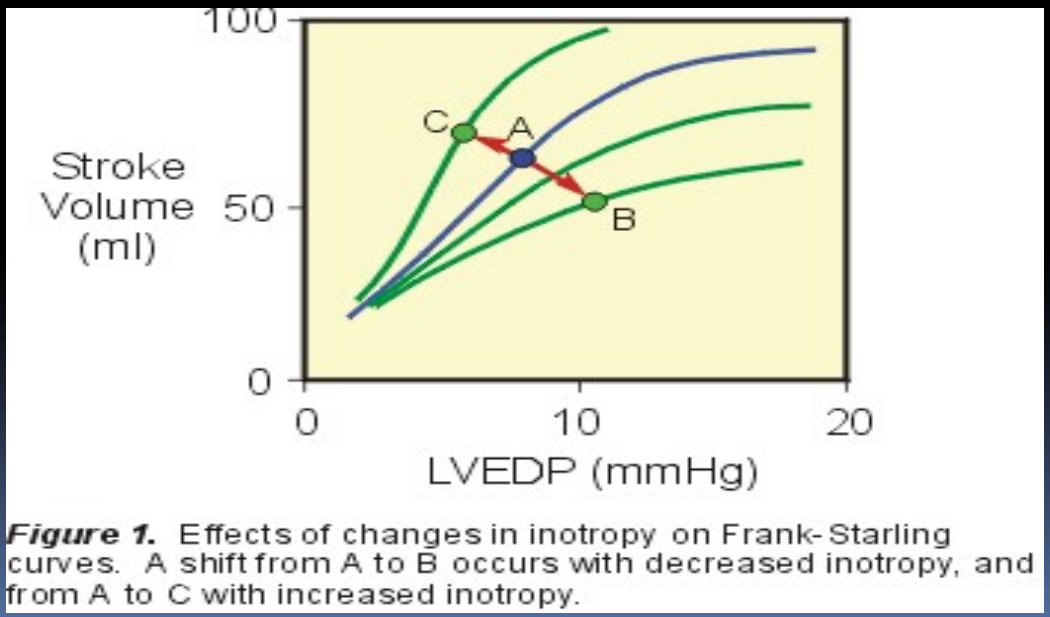
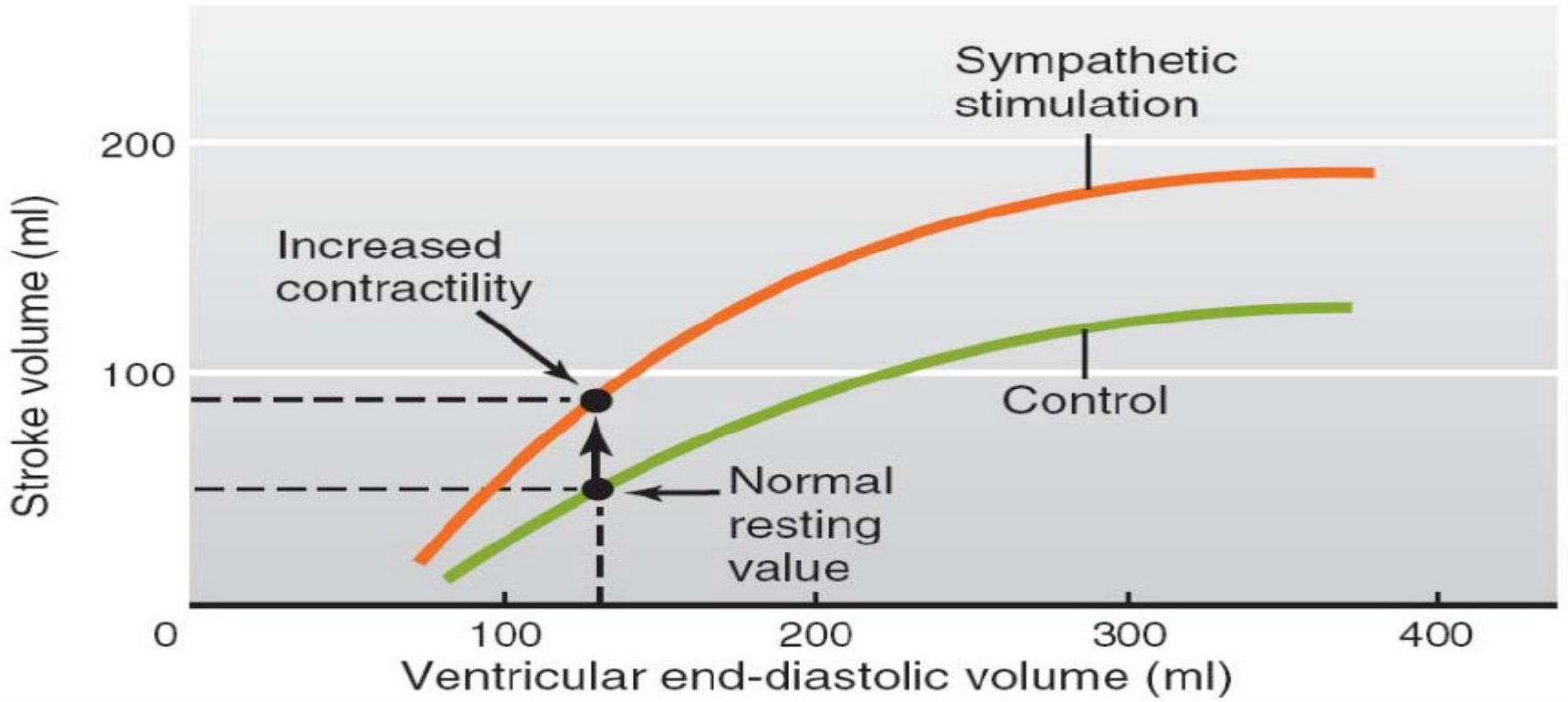
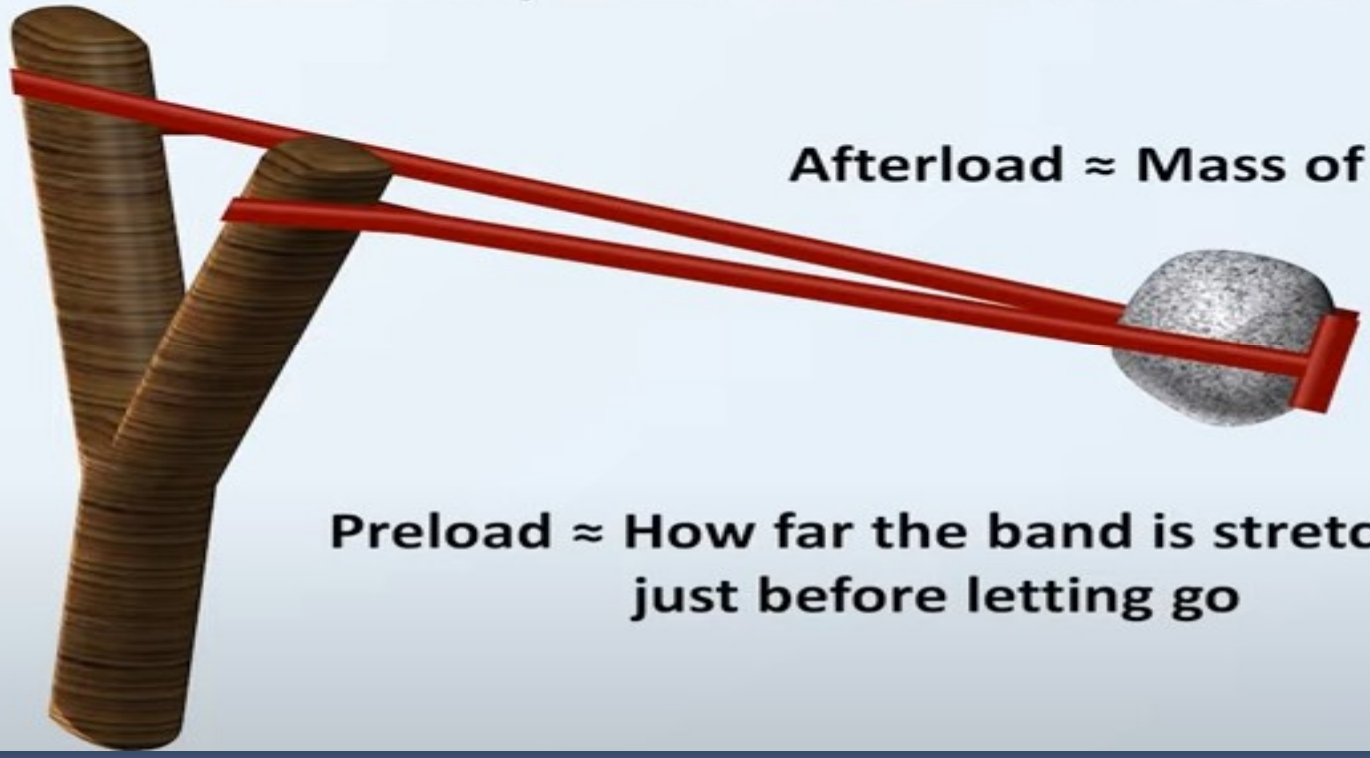


Figure 1. Effects of changes in inotropy on Frank-Starling curves. A shift from A to B occurs with decreased inotropy, and from A to C with increased inotropy.

Effects of Sympathetic Stimulation on Stroke Volume



Contractility ≈ Intrinsic recoil of the band



Afterload ≈ Mass of the rock

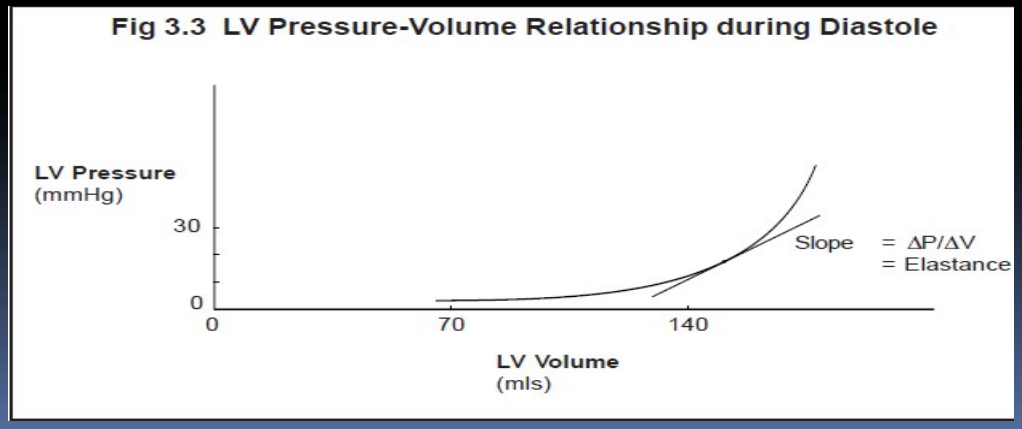
Preload ≈ How far the band is stretched just before letting go

Determinants of RV & LV-EDP

- **Preload** → Volume of Fluids (Venus Return) = EDV
- **Afterload** → Impedance to ejection (Large vessel pressure → Resistance…)
- **Contractility** → Force of Contraction

Regulation of RV & LV-EDP

- ↑ **Preload** ➔ ↑ **EDV** ➔ ↑ EDP
- ↑ **Afterload** ➔ ↑ **ESV** ➔ ↑ **EDV** ➔ ↑ EDP
- ↑ **Contractility** ➔↓ **ESV** ➔ ↓ **EDV** ➔ ↓ EDP



$$SV = EDV - ESV$$

$$EF_{(\%)} = \frac{SV}{EDV} \cdot 100$$

Normal EF = 55-70%

$$CO = SV \cdot HR$$

Το ΚΕ (Ejection Fraction, EF)

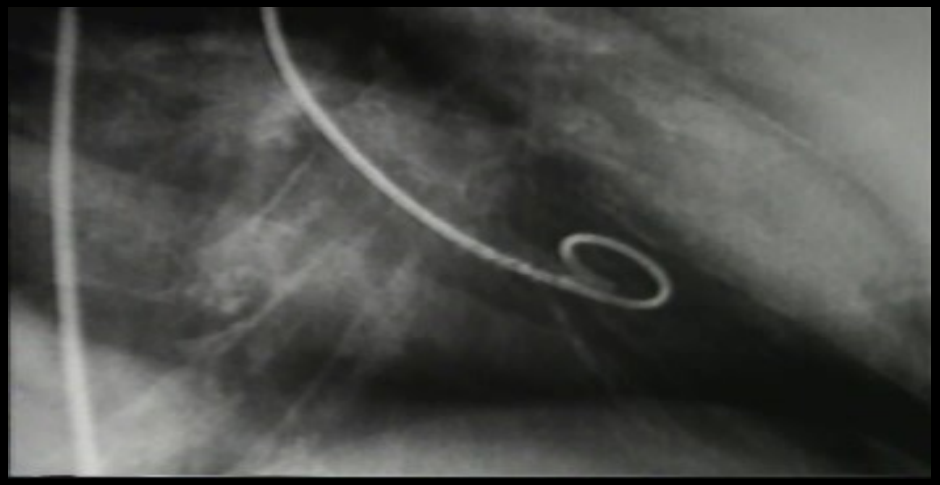
- Η πιο σημαντική κλινική παράμετρος της καρδιακής λειτουργίας

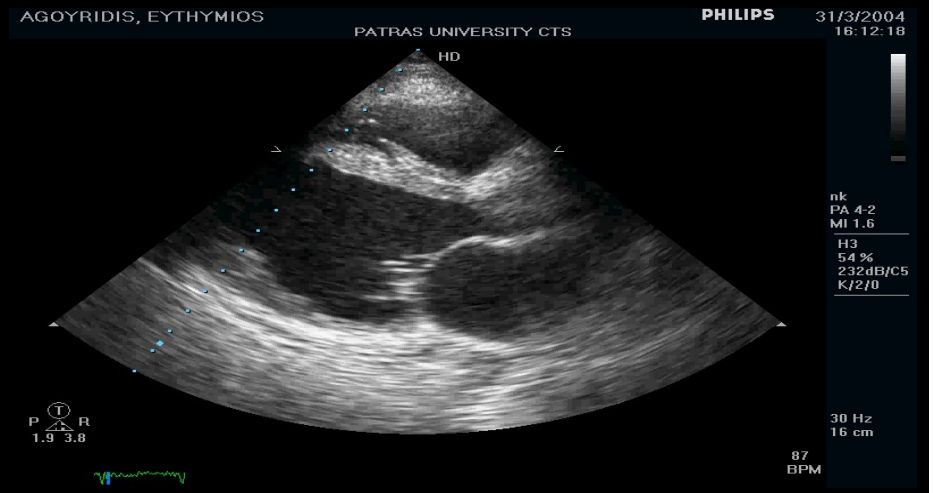
Το ΚΕ (Ejection Fraction)

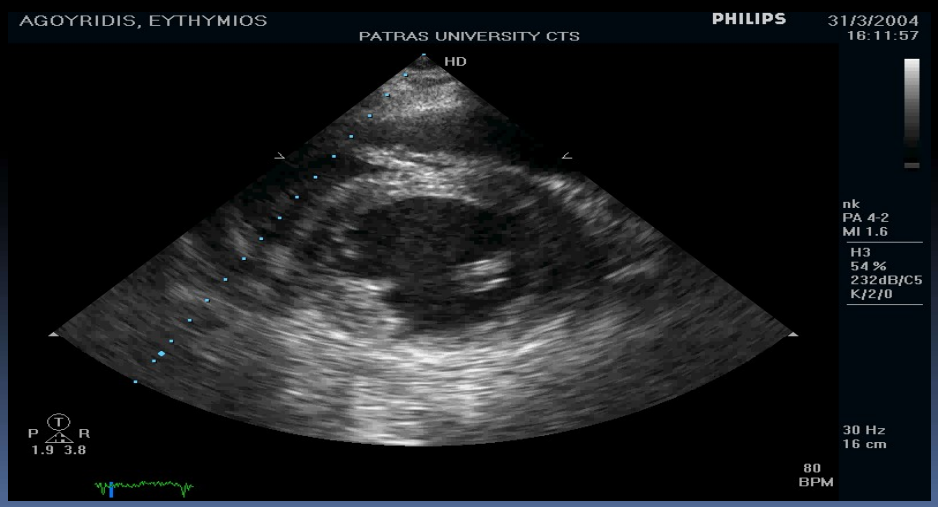
- **Προσδιορίζει την πρόγνωση των καρδιολογικών ασθενών**
  - **Φυσιολογικό: 60%**
  - **Χαμηλό: Συστολική καρδιακή ανεπάρκεια**

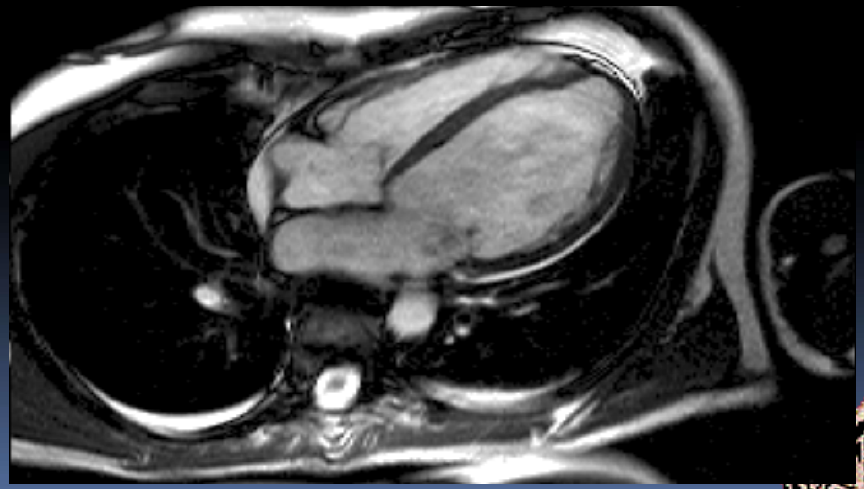
Το ΚΕ (Ejection Fraction, EF)

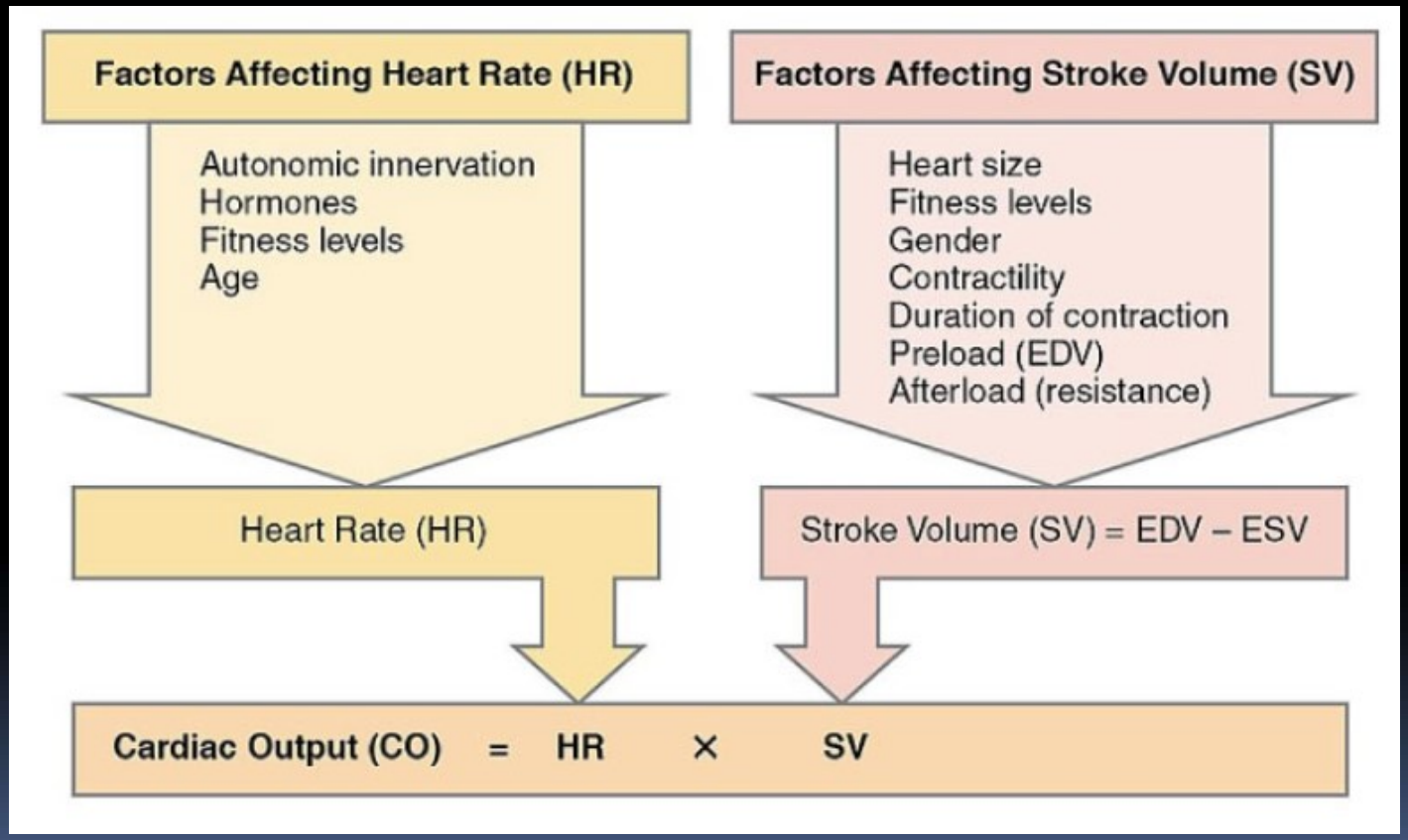
- **Προσδιορίζεται με απεικονιστικές μεθόδους (γεωμετρικές παραδοχές)**
  - **Υπερηχογράφημα**
  - **Αγγειογραφία (κοιλιογραφία)**
  - **Μαγνητική τομογραφία**
  - **Αξονική τομογραφία**



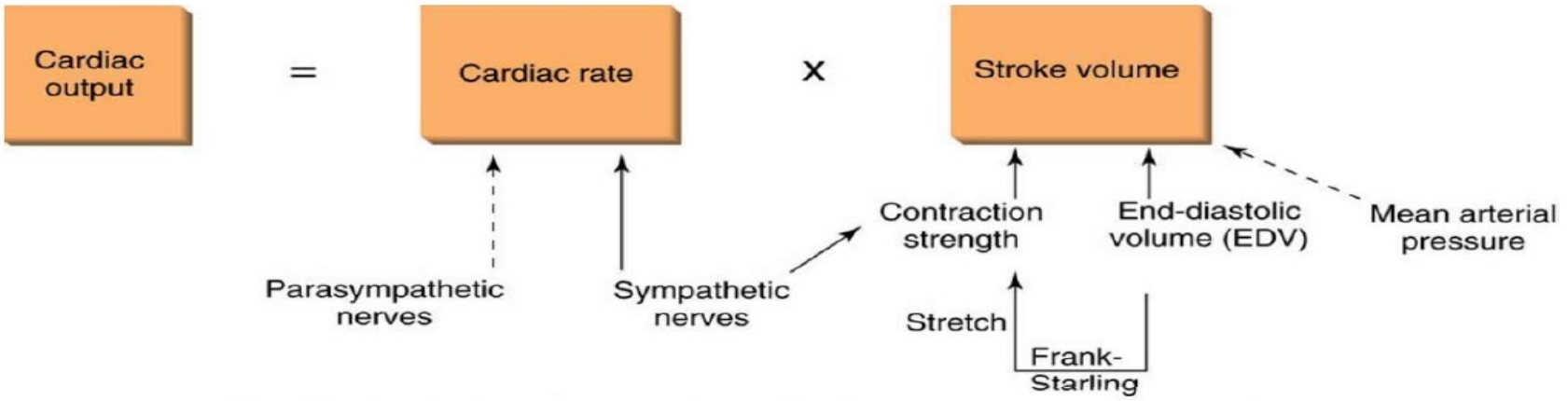

AGOYRIDIS, EYTHYMIOS
PHILIPS
31/3/2004
16:12:18
PATRAS UNIVERSITY CTS
HD
nk
PA 4-2
MI 1.6
H3
54 %
232dB/C5
K/2/0
30 Hz
16 cm
87
BPM
1.9 3.8


AGOYRIDIS, EYTHYMIOS
PHILIPS
31/3/2004
16:11:57
PATRAS UNIVERSITY CTS
HD
nk
PA 4-2
MI 1.6
H3
54 %
232dB/C5
K/2/0
30 Hz
16 cm
80
BPM
1.9 3.8

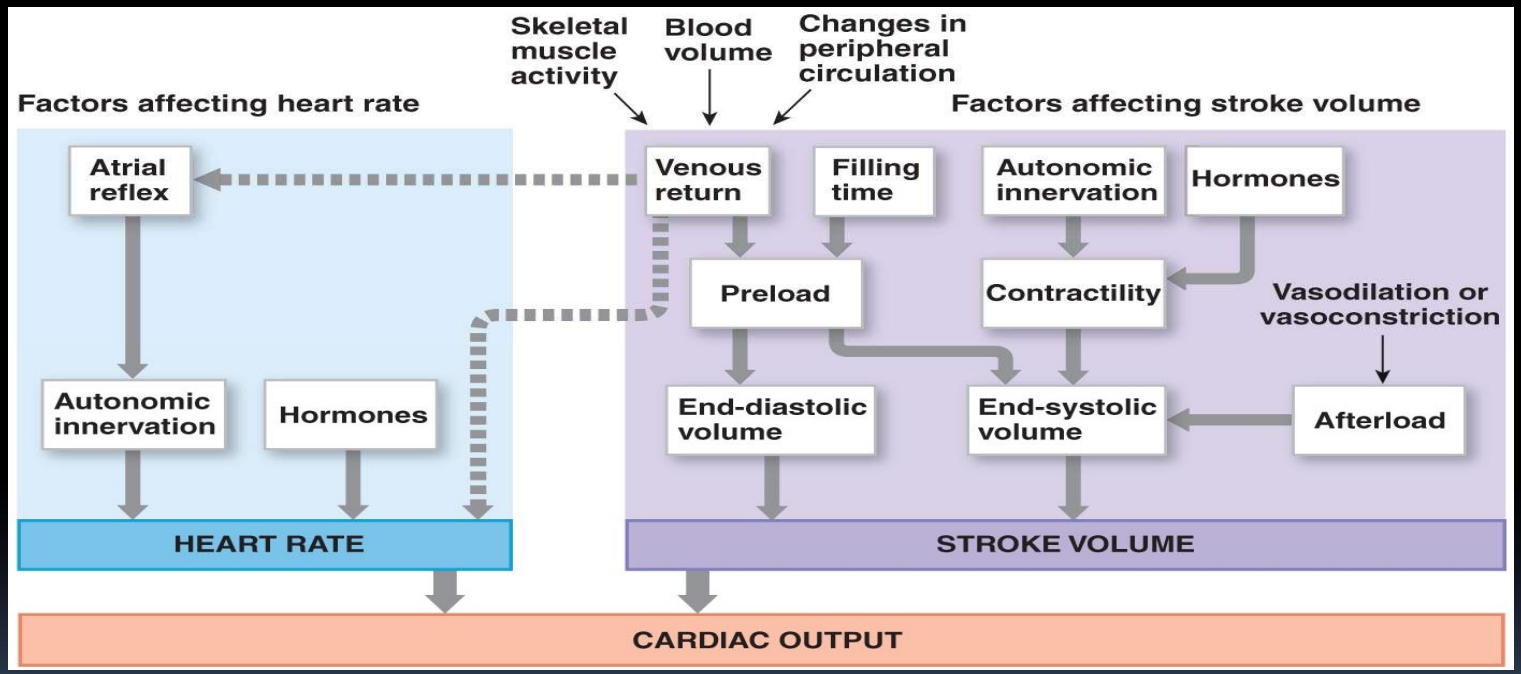



Factors Affecting Heart Rate (HR)
Autonomic innervation
Hormones
Fitness levels
Age
Factors Affecting Stroke Volume (SV)
Heart size
Fitness levels
Gender
Contractility
Duration of contraction
Preload (EDV)
Afterload (resistance)
Heart Rate (HR)
Stroke Volume (SV) = EDV – ESV
Cardiac Output (CO) = HR × SV

Factors that Regulate Cardiac Output

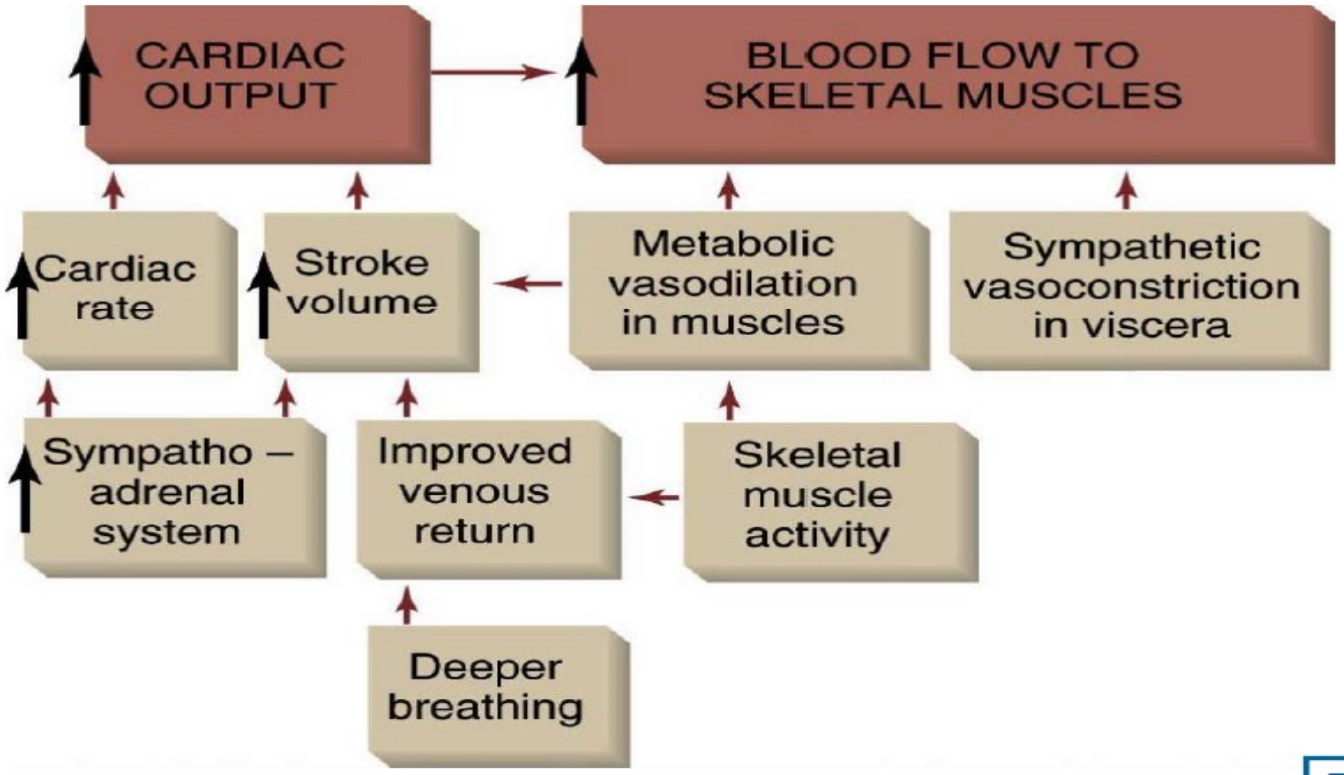


Cardiodynamics

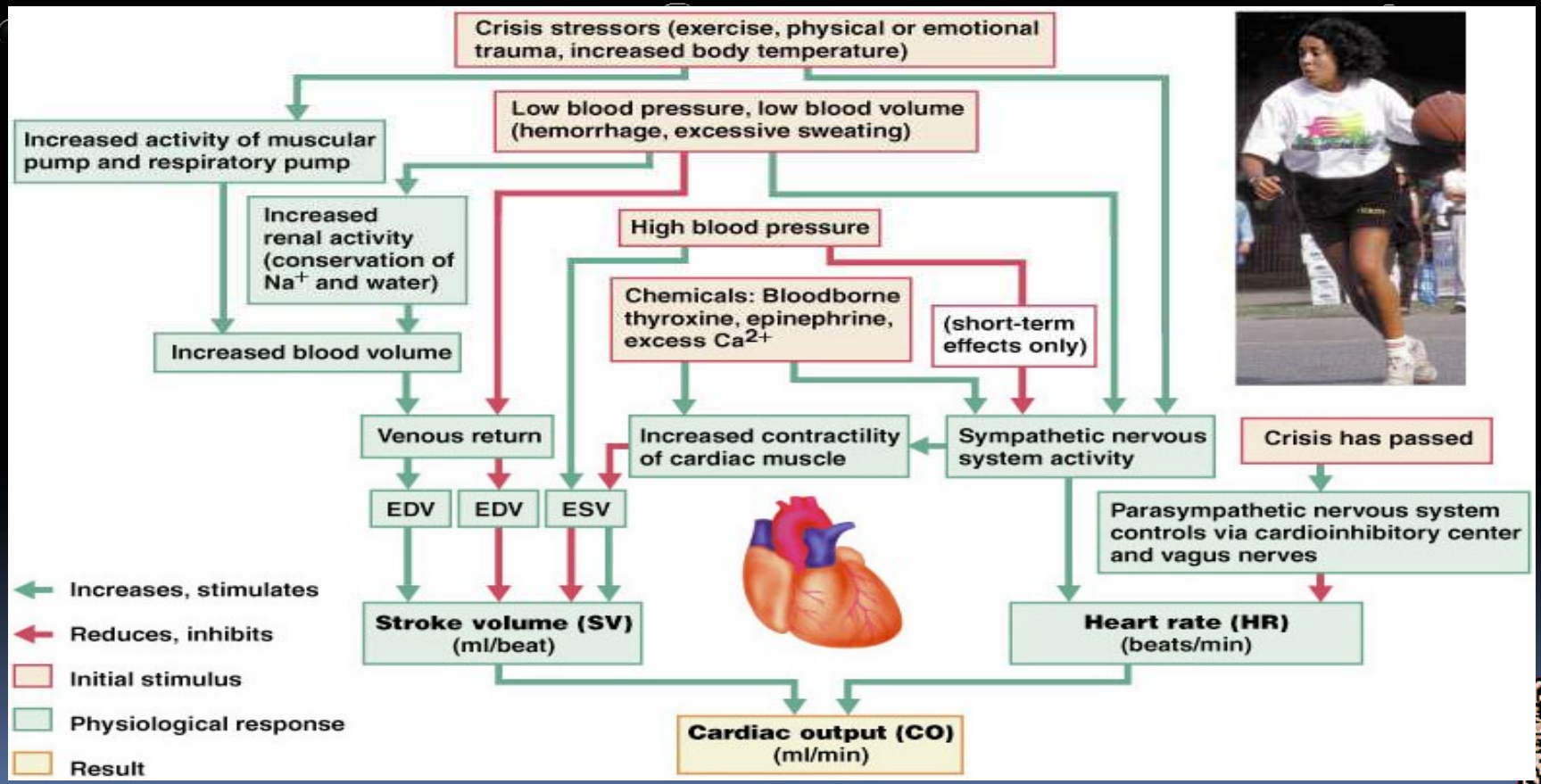


A Summary of the Factors Affecting Cardiac Output

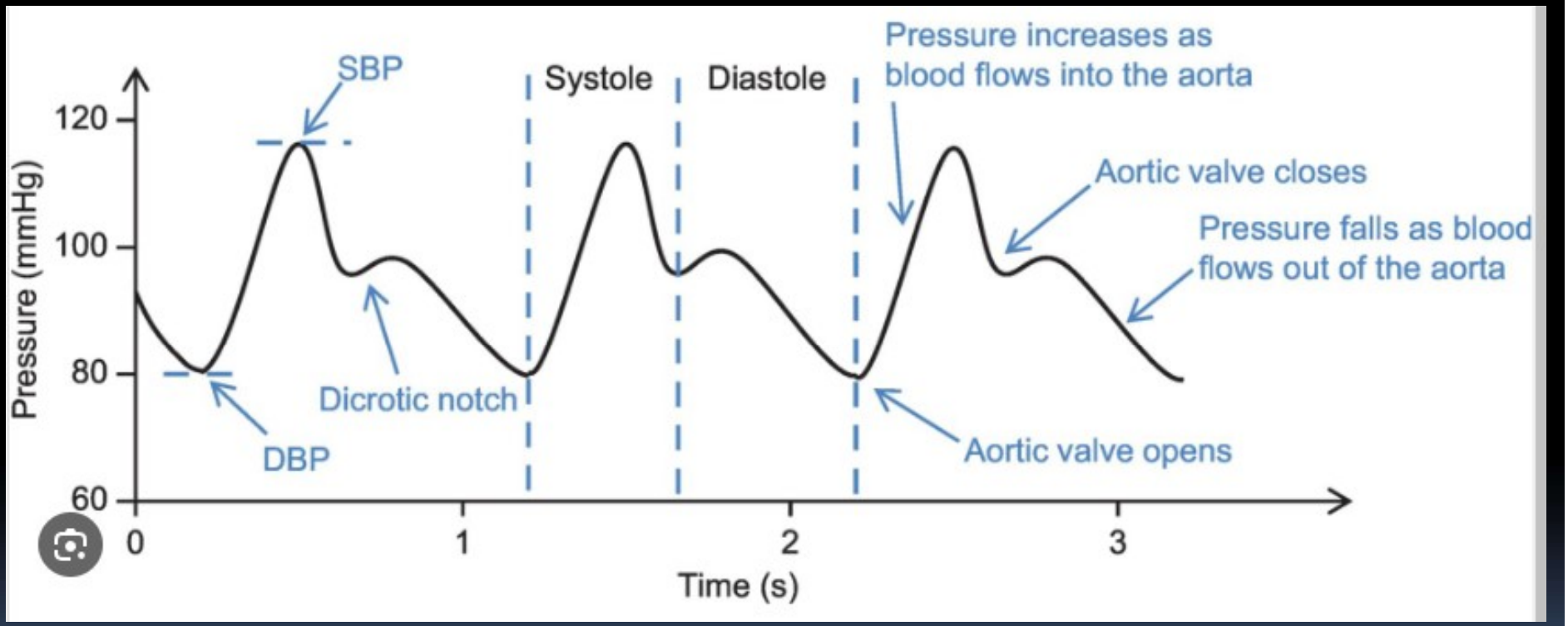
Summary of Cardiovascular Responses to Exercise

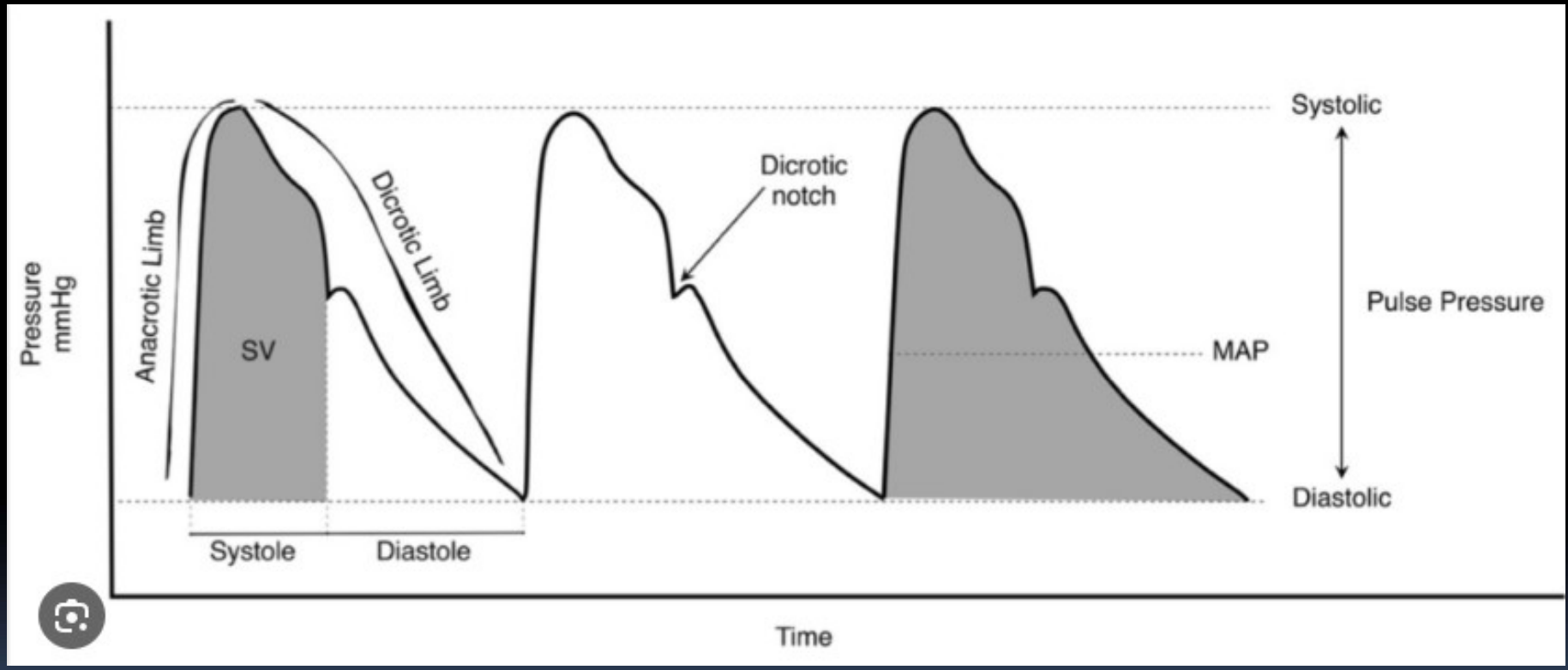


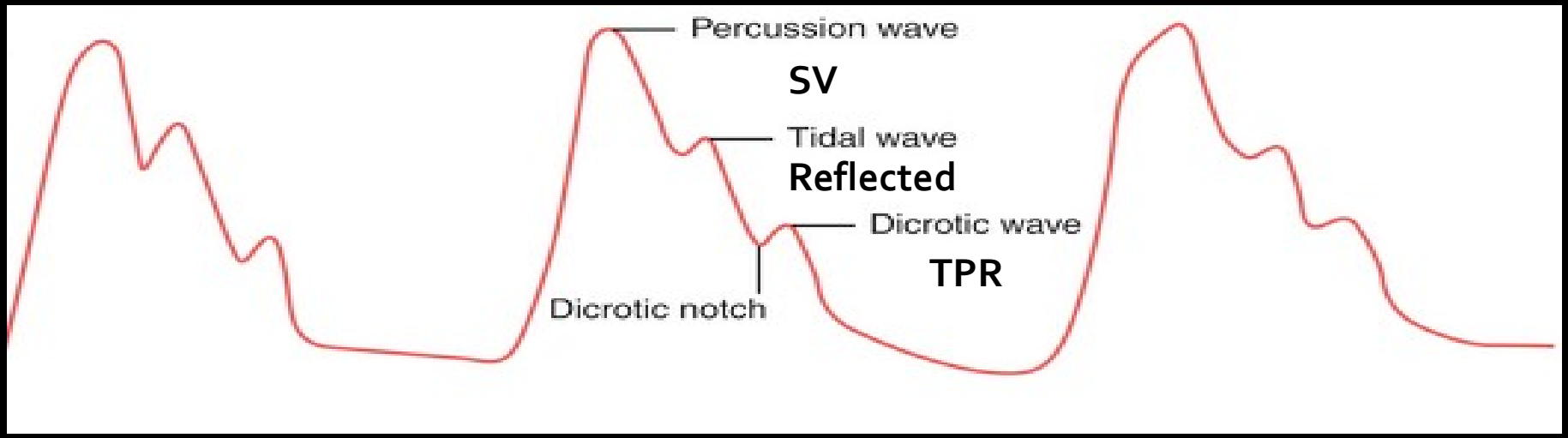
Factors Involved in Regulation of Cardiac Output



Blood Pressure


SBP
Systole
Diastole
Pressure increases as blood flows into the aorta
Aortic valve closes
Pressure falls as blood flows out of the aorta
Dicrotic notch
DBP
Aortic valve opens
Pressure (mmHg)
120
100
80
60
0
1
2
3
Time (s)


Pressure
mmHg
Anacrotic Limb
SV
Dicrotic Limb
Dicrotic
notch
Systolic
MAP
Pulse Pressure
Diastolic
Systole
Diastole
Time



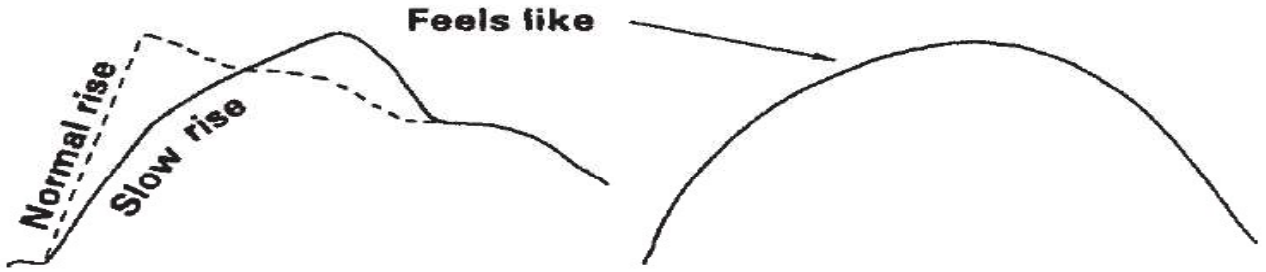
Percussion Wave = rise due to LV ejection;
Tidal Wave = echo of the percusion wave by the arterial system;
Dichrotic notch is the abrupt closure of the aortic valve; dicrotic wave is the reflected wave from the peripheral vasculature

Pulsus parvus et tardus…

The first indication that the rise is slow is the **absence of a tap**.

Then you may notice that the sensation is one of a caressing lift.

The normal rise is a tap; the slow rise is a caress.

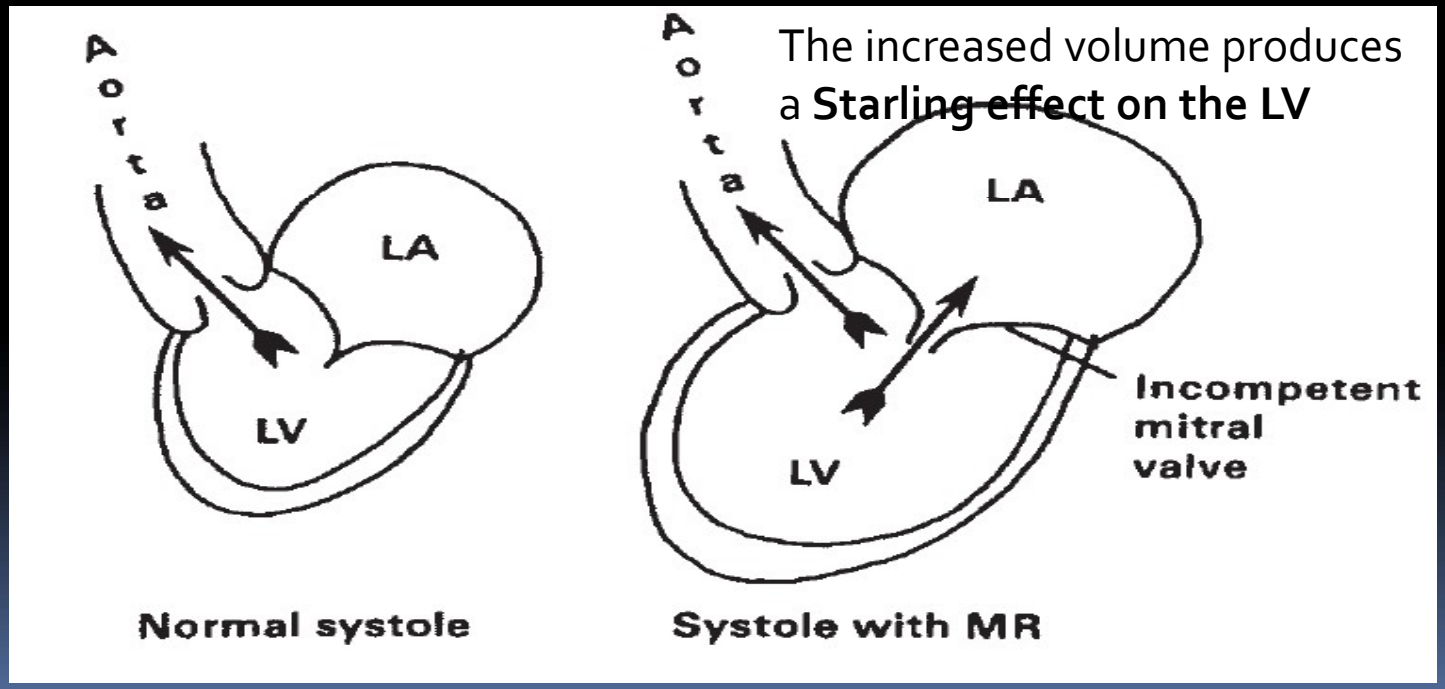


SOS: If you feel a tap followed by a push, then you may be feeling an anacrotic shoulder followed by a late slow-rising tidal wave

A lag between the onset of the apical impulse and the carotid impulse predicts a valve area < 1 cm^2 (100% specific).

Rapid Rates of Rise (Brisk Pulse) with Normal Pulse Pressures

- If there are two outlet orifices for ejection, as in MR or VSD



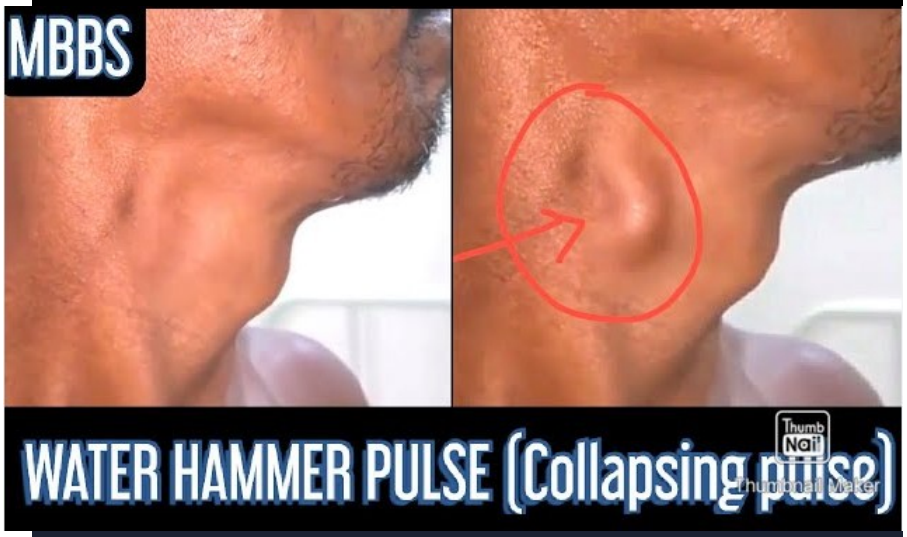
Collapsing pulse

Normal Pulse | Water hammer pulse

Collapsing pulse also known as **Watson's water hammer pulse** is described in aortic regurgitation with a rapid upstroke and descent.

Causes of collapsing pulse

1. Aortic regurgitation
2. Patent ductus arteriosus



- **Bounding pulse/ water hammer pulse**

This type of pulse rises suddenly followed by a quick fall.

Quick rise is due to increased stroke volume, and the quick fall is due to quick passage of blood from aorta to ventricle.

Seen in aortic incompetence.

What is Corrigan's pulse?

ANS: It is the bounding carotid seen by eye.

2. What are the most common cardiac causes of a bounding pulse?

   ANS: Aortic regurgitation (AR), **persistent ductus arteriosus**, and coarctation.

3. What are the most common noncardiac causes of a rapid rise with increased pulse pressure?

   ANS: Thyrotoxicosis, pregnancy, and severe anemia.

4. Why is the pulse of AR bounding?

   ANS: a. There is a high systolic pressure because a large volume is ejected. The large volume is from two sources: the diastolic AR flow plus the mitral diastolic flow. The Starling effect caused by stretching the LV creates the rapid rise.
   b. There is a low diastolic pressure.

Rapid Rates of Rise with Increased Pulse Pressure

- Most common cardiac causes of a bounding pulse
  - Aortic regurgitation (AR), persistent ductus arteriosus, and coarctation.
- Most common noncardiac causes of a rapid rise with increased pulse pressure
  - Thyrotoxicosis, pregnancy, and severe anemia.

Corrigan pulse…

- The loud sounds heard when the stethoscope is placed over the rapidly rising large pulsations of the femoral artery are called Traube's sign, or pistol-shot sounds

Corrigan pulse…

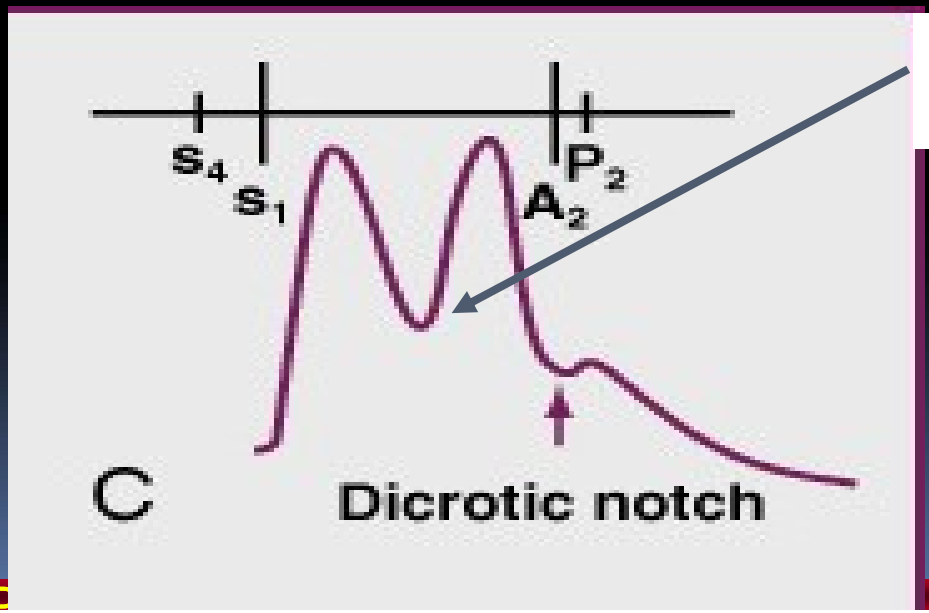
- The lower diastolic pressure in AR is only partly due to backflow into the LV during systole.
- It is mostly due to the reflex decrease in TPR caused by the large stroke volume stretching the carotid and aortic sinuses.
  - *Although sAR will generally have a diastolic pressure of about 50 mmHg or less, if the patient goes into heart failure the resultant reflex increase in peripheral resistance caused by low output may raise the diastolic pressure to normal values.*

Duroziez double murmur…

- Duroziez' double murmur:
- 1st ➔ systolic murmur heard by gradually compressing the femoral artery with a finger proximal to the stethoscope
- 2nd ➔ diastolic murmur produced by gradually compressing the artery distal to the stethoscope (backflow in all the large arteries in diastole).
- *Note: Traube's sign and Duroziez' double murmurs are more of historical than practical interest because no more information is gained from them than by palpating the pulses or taking a blood pressure*

Δικόρυφος (Bisferiens) σφυγμός

- Μεγάλος όγκος παλμού που εξωθείται ταχέως
- Μεσοσυστολική έμφραξη

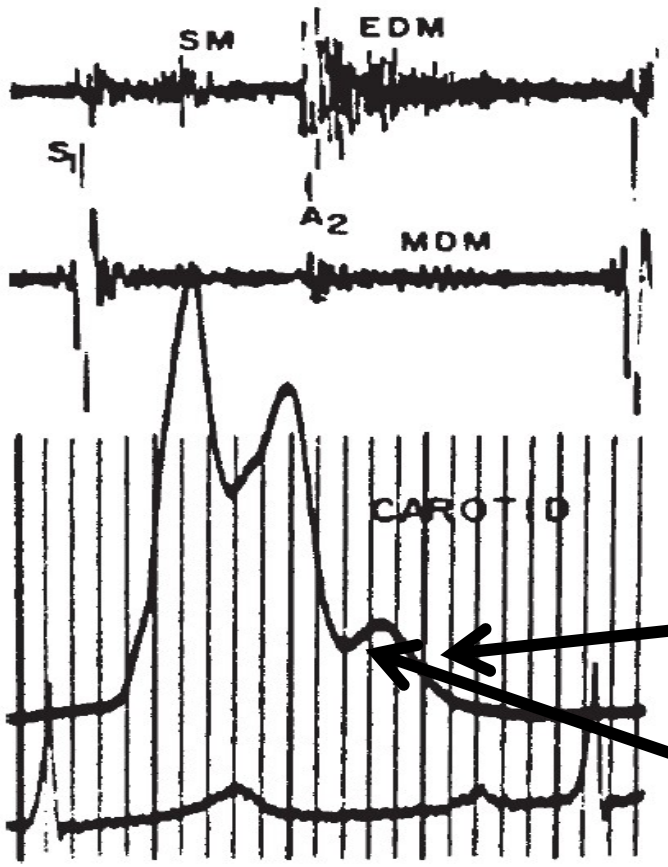


Bernoulli effect…or
Midsystolic LVOTO

Δικόρυφος σφυγμός: Percussion

& tidal waves.

HOCM …

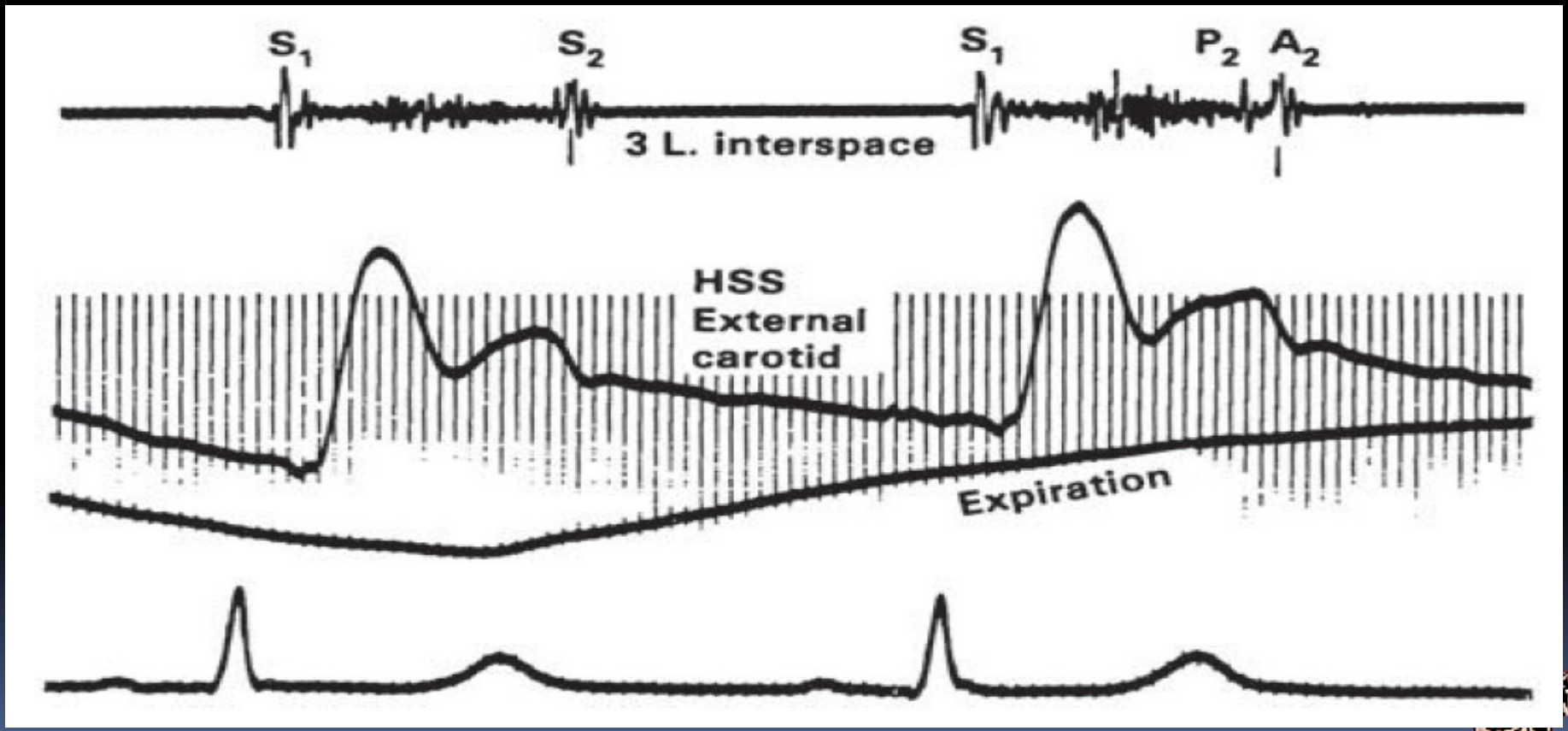


Bisferiens pulse in a patient with severe AR. (EDM = early diastolic murmur of AR at left sternal border; MDM = mid-diastolic murmur at apex [Austin Flint murmur]).

Δικροτικό κύμα

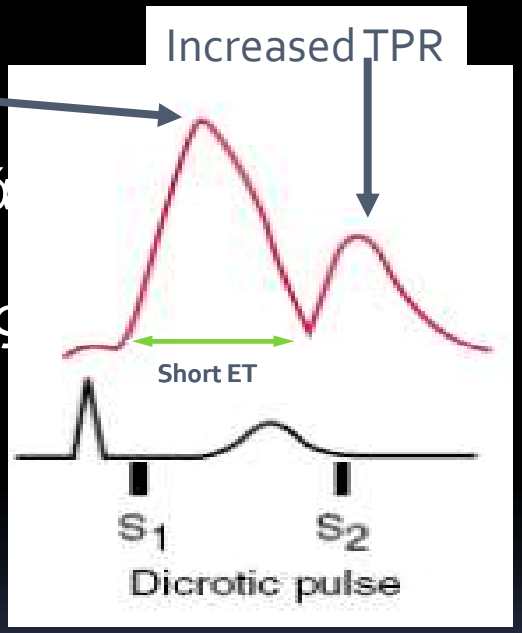
Δικροτική εντομή
Incisura

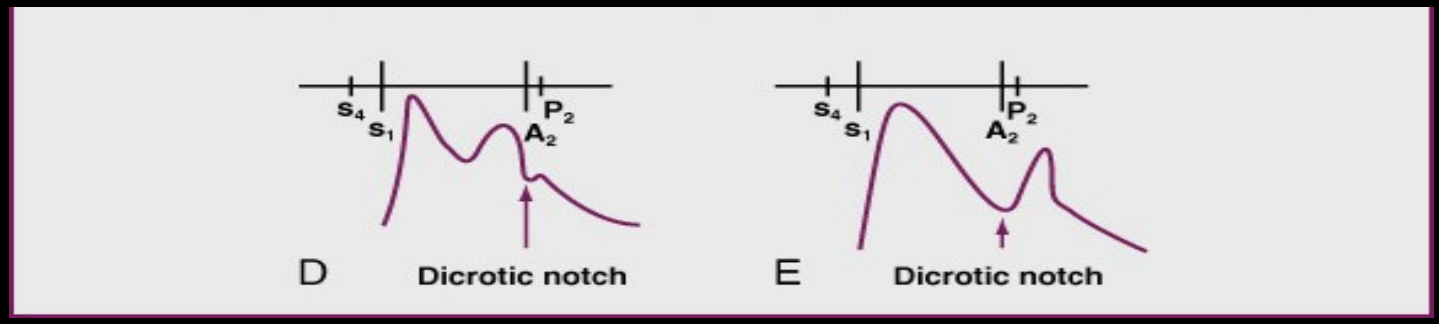
The rate of rise in HOCM is among the fastest in cardiology



Δίκροτος σφυγμός

- Η δεύτερη κορυφή **στην διαστολή** αμέσως μετά τον S_2 (υπόταση με αυξημένες αντιστάσεις όπως σε πυρετό, tamponade, σοβαρή οξεία καρδιακή ανεπάρκεια, υπογκαιμικό shock, post-AVR)





D, Δικόρυφος σφυγμός σε HOCM. Σπάνια ψηλαφητός.

Ε, Δικροτικός σφυγμός: Ενισχυμένο δικροτικό κύμα όπως σε σήψη, σοβαρή ΚΑ, υπογκαιμικό shock, tamponade, AVR.

Pulsus paradoxus

- Tamponade, συμπιεστική ΠΚ, βρογχικό άσθμα, υπογκαιμικό shock, πνευμονική εμβολή...

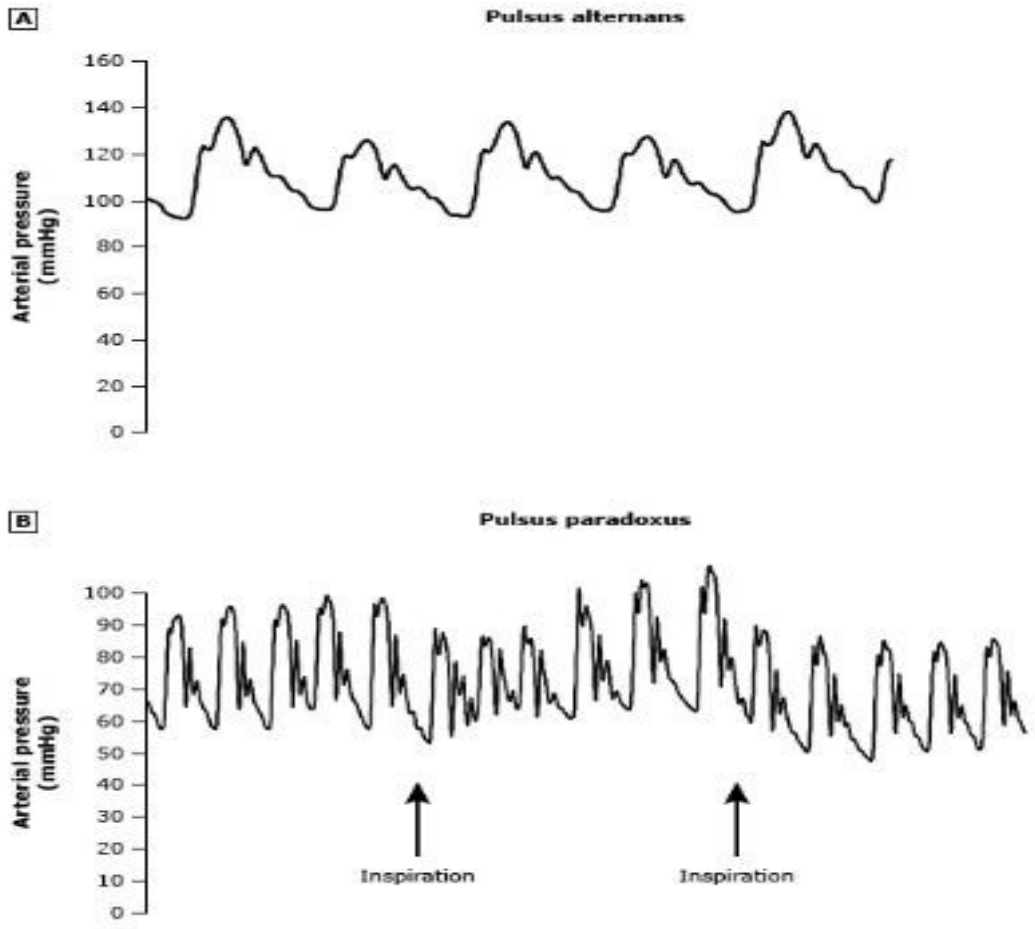
1. Why do some asthmatic patients seem to have a marked inspiratory fall in blood pressure?

   ANS: Bronchospasm may raise the intrathoracic pressure very high (similar to a Valsalva maneuver), and inspiration will, by contrast, seem to lower the systolic pressure excessively. Actually, it is an expiratory rise in blood pressure, not an inspiratory fall.

Pulsus alternans

- Σοβαρή μυοκαρδιακή δυσλειτουργία, εκτακτοσυστολές (ΔΔχ σφυγμός διδυμίας pulsus bigeminus). Με πιεσόμετρο (<10 mmHg διαφορά)...
- Έκλυση με διούρηση, όρθια θέση...(μετατροπή σε ↓ output)

Pulsus alternans (q2 beats, LV systolic faiure) versus Pulsus paradoxus (lower during inspiration)



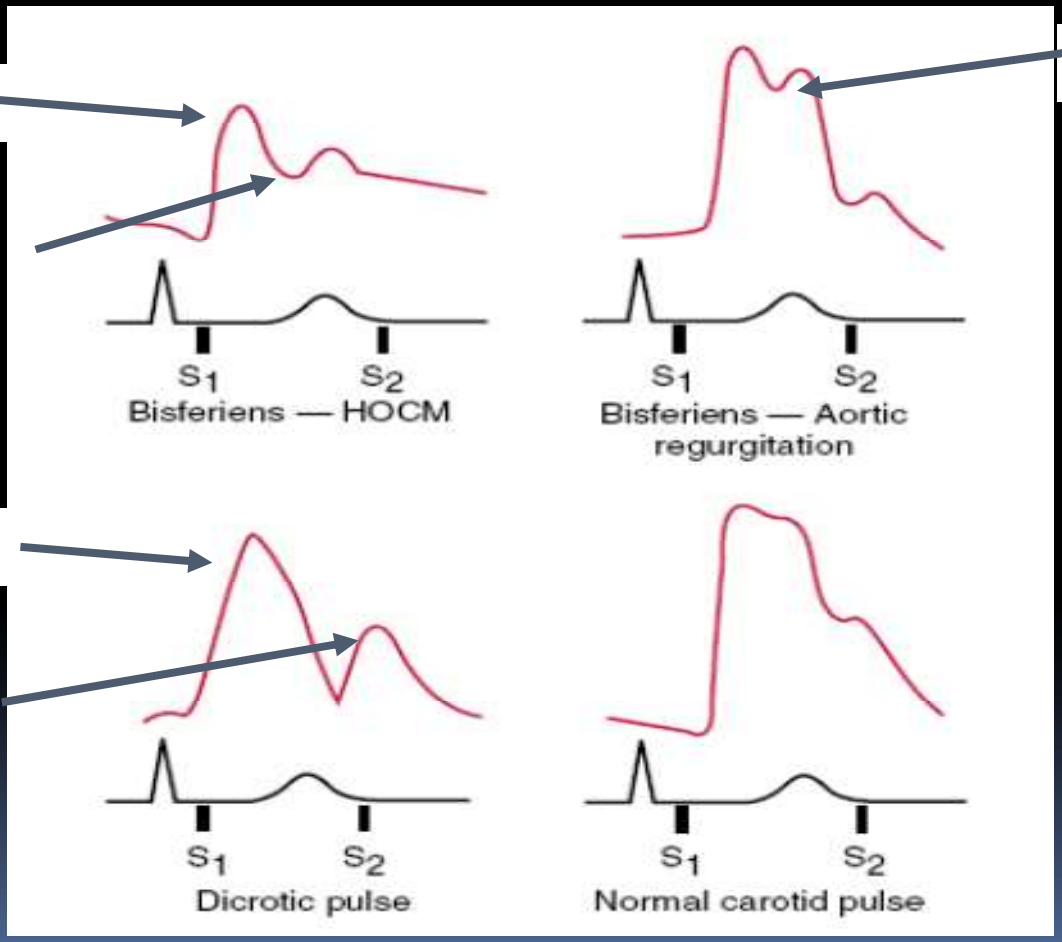
80% SV!

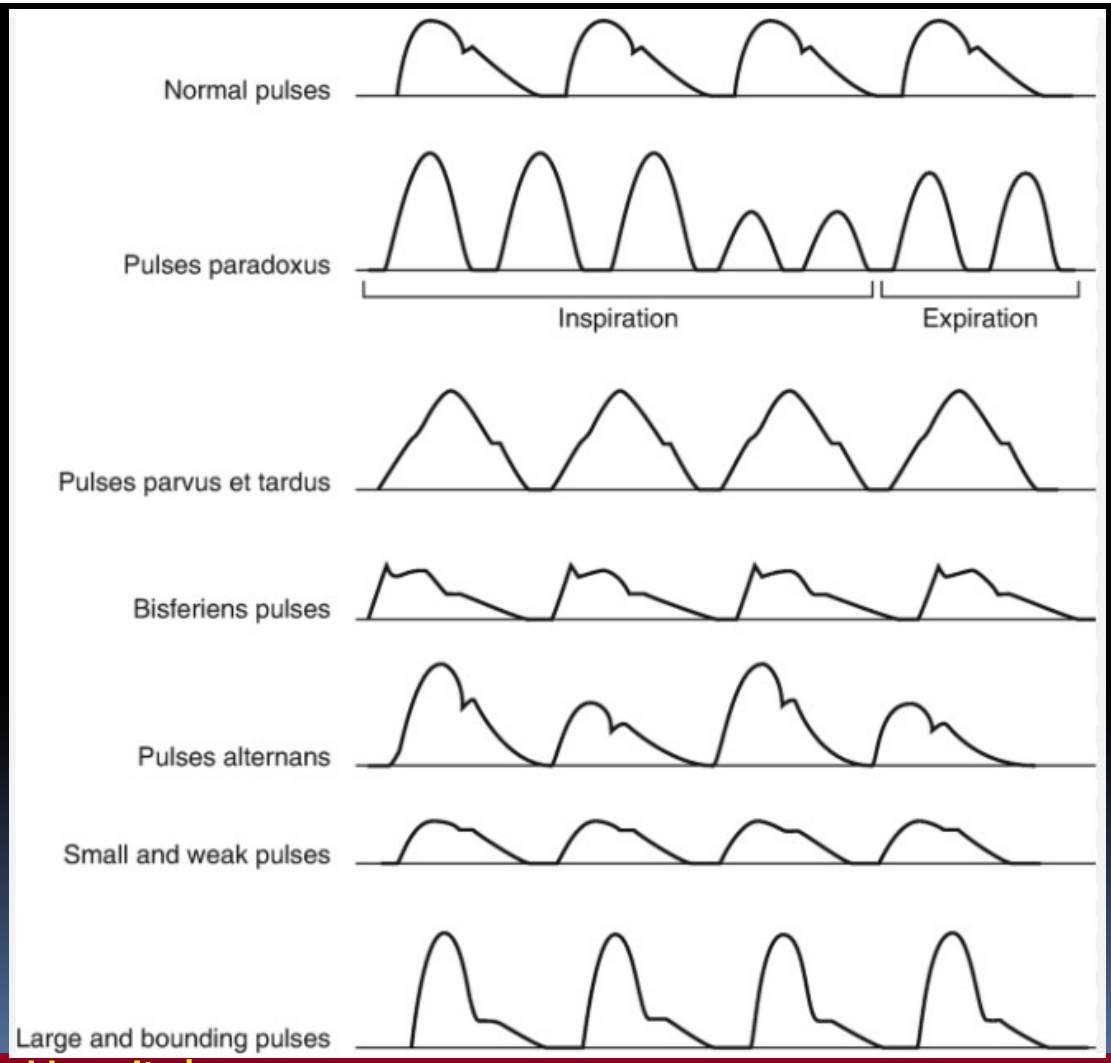
Midsystolic LVOTO

Reduced SV

Increased TPR

Bernoulli effect




Normal pulses
Pulses paradoxus
Inspiration
Expiration
Pulses parvus et tardus
Bisferiens pulses
Pulses alternans
Small and weak pulses
Large and bounding pulses

Παθολογικός σφυγμός

- Pulsus tardus: Fixed obstruction to left ventricular outflow (valvular aortic stenosis, congenital fibrous subaortic stenosis). Βραδεία άνοδος, ροίζος, βραδύς και παρατεταμένος με μικρό ύψος. Anacrotic notch & acrotic pulse.
- Pulsus parvus: σφυγμός μικρού ύψους λόγω ελαττωμένου όγκου παλμού.
- Pulsus parvus et tardus: σοβαρή στένωση αορτής.
- Έντονος σφυγμός:Υπερκινητική κυκλοφορία & LV volume loading (VSD, MR, AR)

Παθολογικός σφυγμός

- Corrigan or water-hammer pulse: σοβαρή AR; Αιφνίδια άνοδος, ταχεία πτώση χωρίς dicrotic notch. (χαμηλή αντίσταση αορτής και μεγάλος όγκος παλμού). Στην οξεία AR, πρώιμη σύγκλιση της MV παρεμποδίζει αυτά τα φαινόμενα.
- Bounding arterial pulses: PDA, AV fistulas, thyrotoxicosis, pregnancy, fever, and anemia, severe bradycardia, arteries proximal to CoA.
- Hill's sign: AR (ή καταστάσεις με αυξημένο όγκο παλμού) η συστολική πίεση στα κάτω άκρα είναι μεγαλύτερη από τα άνω άκρα > 20 mm Hg.
- Καταστάσεις με αυξημένο όγκο παλμού: Becker's sign (ορατές σφύξεις στον αμφιβληστροειδή) and Mueller's sign (ορατές σφύξεις σταφυλής).

Παθολογικός σφυγμός

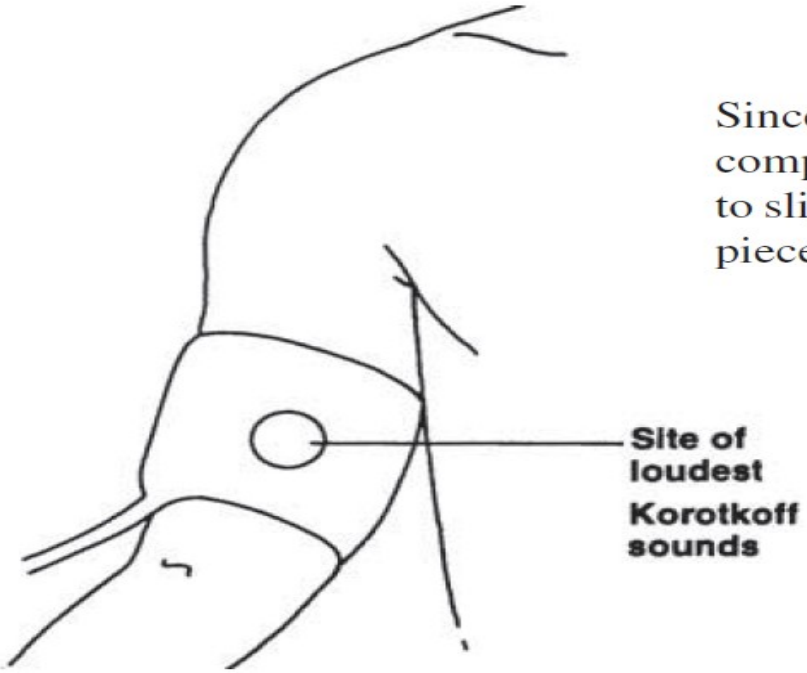
- AV dissociation: διακύμανση σφυγμού (επί ταχυκαρδίας, υποψία κοιλιακής ταχυκαρδίας)
- Bisferiens pulse: μεγάλος όγκος παλμού που εξωθείται ταχέως, ή μεσοσυστολική απόφραξη (AR, AR & AS, σπανίως σε νεαρούς ενήλικες, HOCM).
- Dicrotic pulse: Η δεύτερη κορυφή στην διαστολή αμέσως μετά τον S_2 (υπόταση με ελαττωμένες αντιστάσεις όπως σε πυρετό, tamponade, σοβαρή καρδιακή ανεπάρκεια, υπογκαιμικό shock)
- Pulsus alternans: Σοβαρή μυοκαρδιακή δυσλειτουργία, εκτακτοσυστολές (ΔΔχ σφυγμός διδυμίας pulsus bigeminus)
- Pulsus paradoxus: tamponade, συμπιεστική ΠΚ, βρογχικό άσθμα, υπογκαιμικό shock, πνευμονική εμβολή.

Quick Diagnoses Possible from the Carotid Pulse

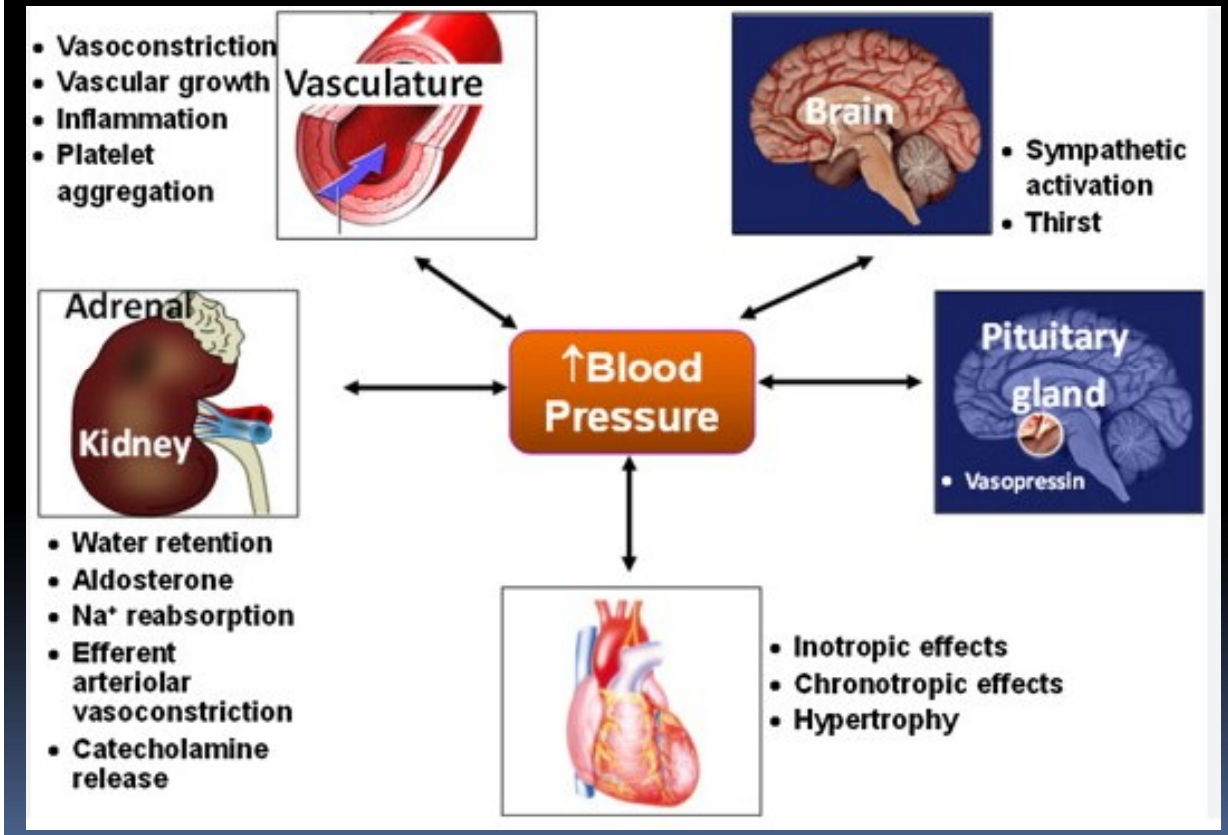
- Feel for a **gentle tap** → carotid with a normal amplitude and rate of rise.
- If you feel **no tap** but only a **"push"** you should assume that AS is probably present.
- If you feel a **sharp tap** (brisk pulse) due to a rapid rate of rise, consider MR, VSD, or HCM if the pulse volume or pressure is normal.
  - If the **pulse amplitude is increased**, consider **AR**, PDA, or CoA

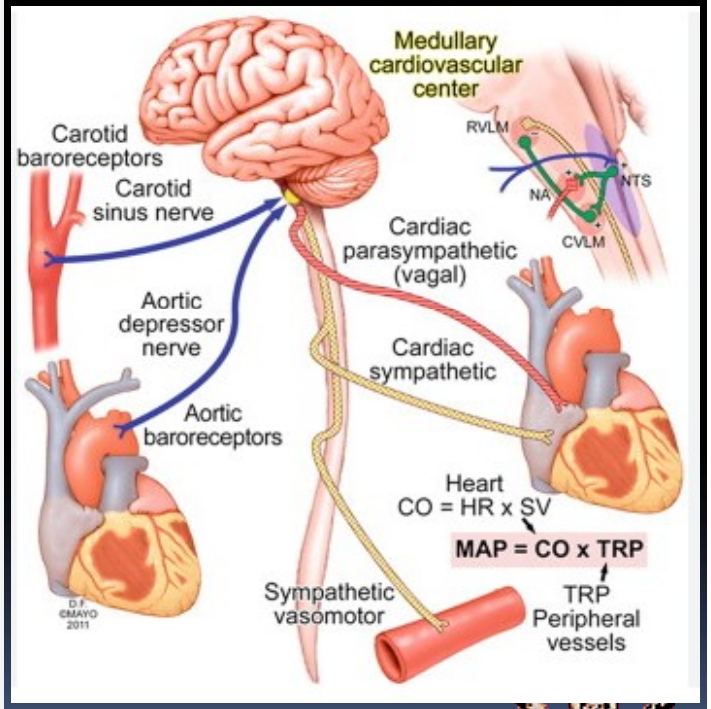
Where are Korotkoff sounds loudest? (a) In the center of the cuff, (b) at the edge of the cuff, or (c) a few centimeters distal to the cuff edge?

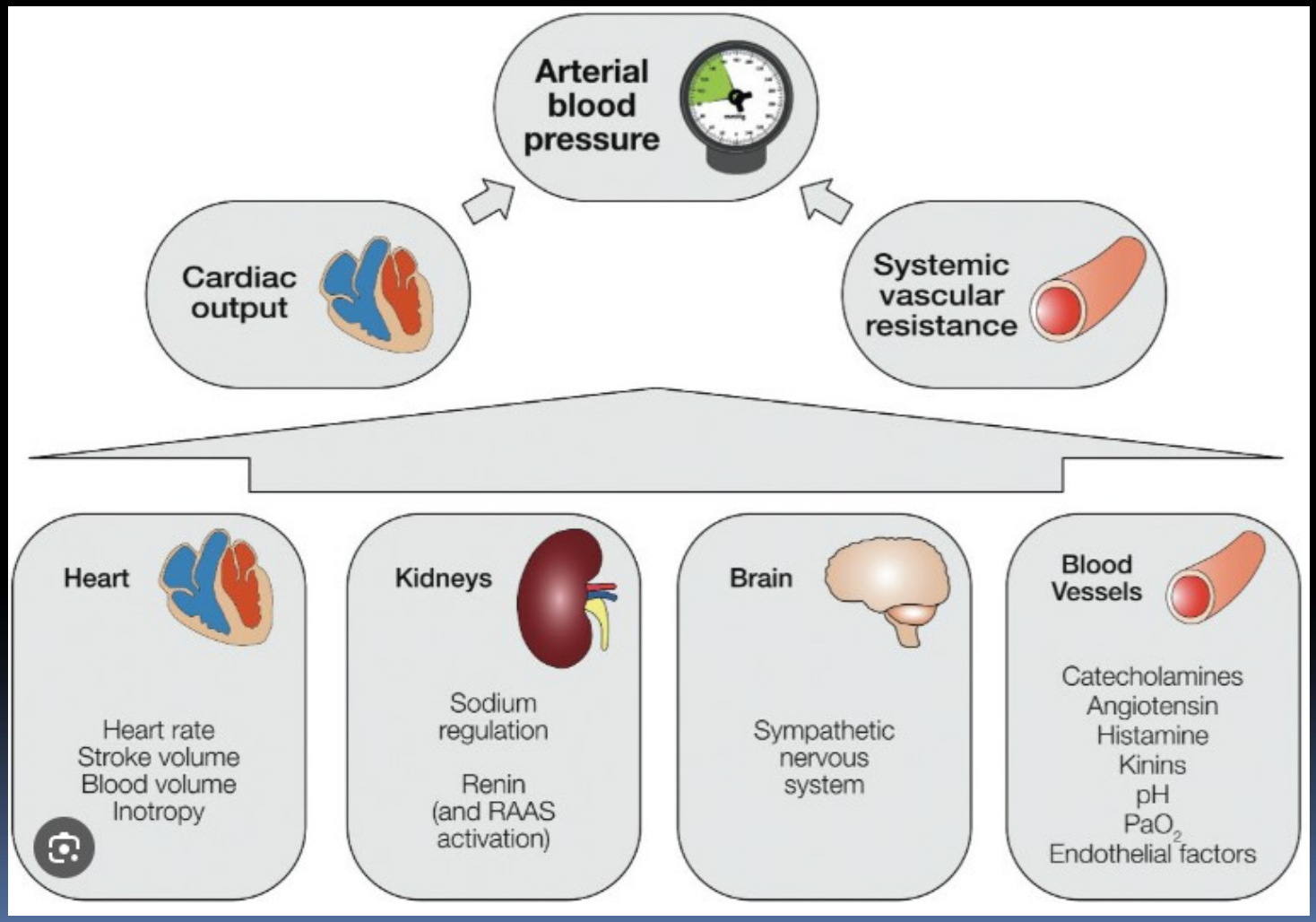
ANS: In the center of the cuff.



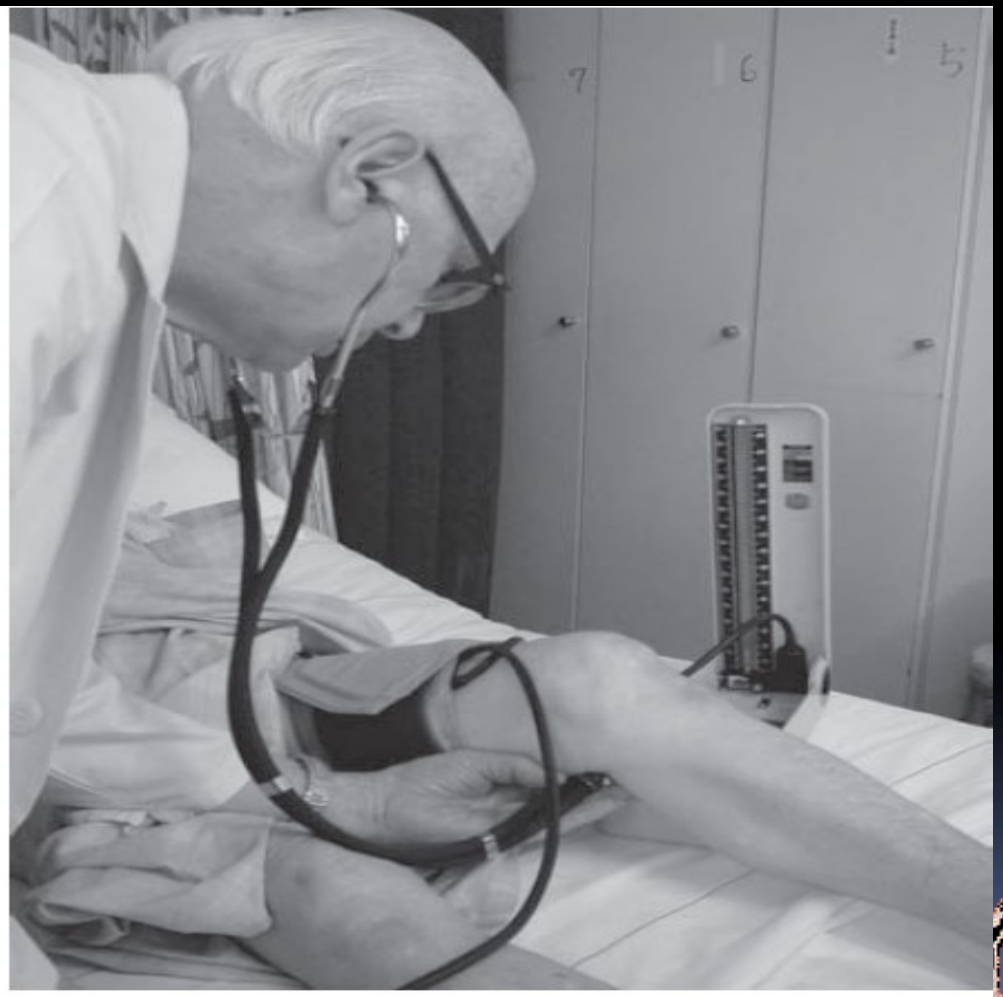
Since a stethoscope chest piece cannot be placed completely under a cuff, the best compromise is to slip as much as possible of the diaphragm chest piece under the distal edge of the cuff.

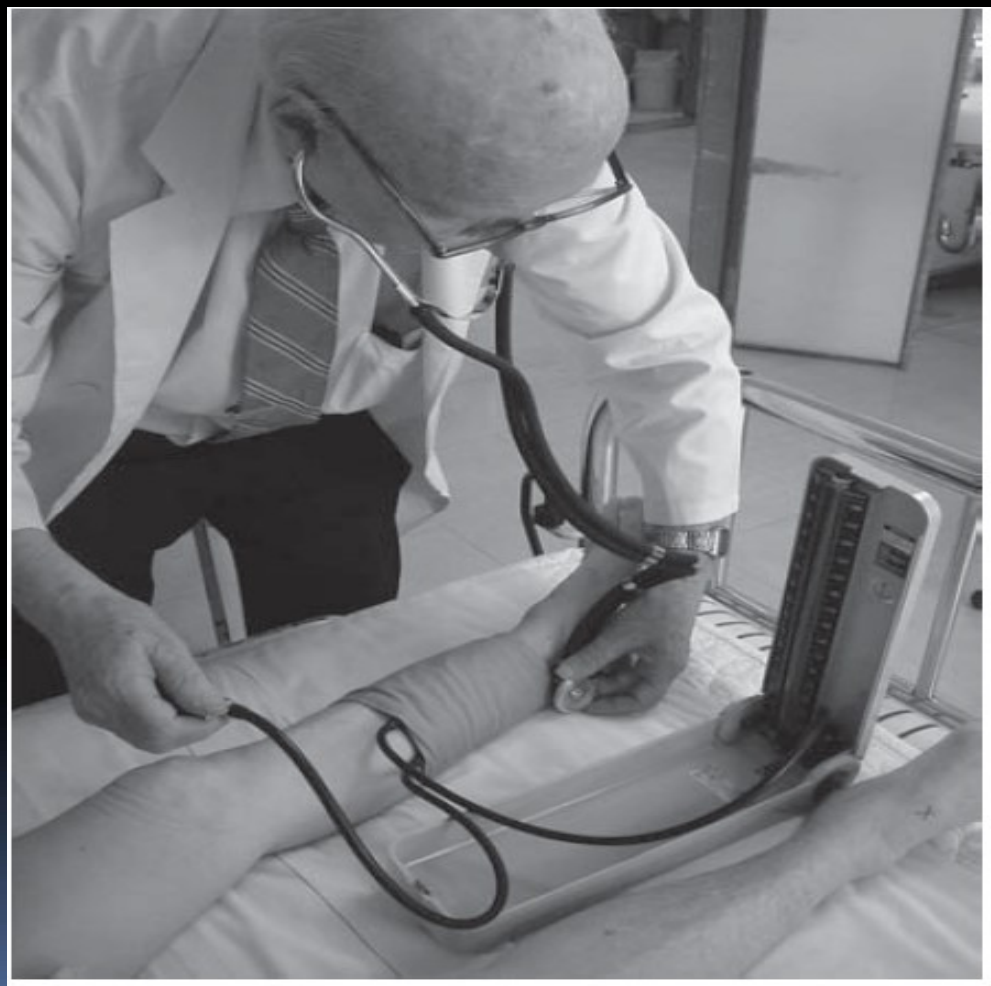

Vasoconstriction
Vascular growth
Inflammation
Platelet aggregation
Vasculature
Brain
Sympathetic activation
Thirst
Adrenal
Kidney
↑Blood Pressure
Pituitary gland
Vasopressin
Water retention
Aldosterone
Na+ reabsorption
Efferent arteriolar vasoconstriction
Catecholamine release
Inotropic effects
Chronotropic effects
Hypertrophy


Medullary cardiovascular center
RVLM
NA
NTS
CVLM
Carotid baroreceptors
Carotid sinus nerve
Cardiac parasympathetic (vagal)
Aortic depressor nerve
Cardiac sympathetic
Aortic baroreceptors
Heart
CO = HR x SV
MAP = CO x TRP
Sympathetic vasomotor
TRP
Peripheral vessels


Arterial blood pressure
Cardiac output
Systemic vascular resistance
Heart
Heart rate
Stroke volume
Blood volume
Inotropy
Kidneys
Sodium regulation
Renin (and RAAS activation)
Brain
Sympathetic nervous system
Blood Vessels
Catecholamines
Angiotensin
Histamine
Kinins
pH
PaO_2
Endothelial factors

The usual commercial leg cuff, such as that shown here, must be rolled diagonally around the thigh, to keep the edges snug against the skin. The systolic blood pressure in the legs should not be over 20 mmHg higher than in the arms





A convenient method of taking a leg pressure if you do not have a thigh cuff. A pediatric bell should be used to achieve an easy air seal behind the medical malleolus.

Leg Blood Pressure in Aortic Regurgitation

1. How does AR affect the blood pressure in the legs in comparison with that in the arms? What is this sign of AR called?

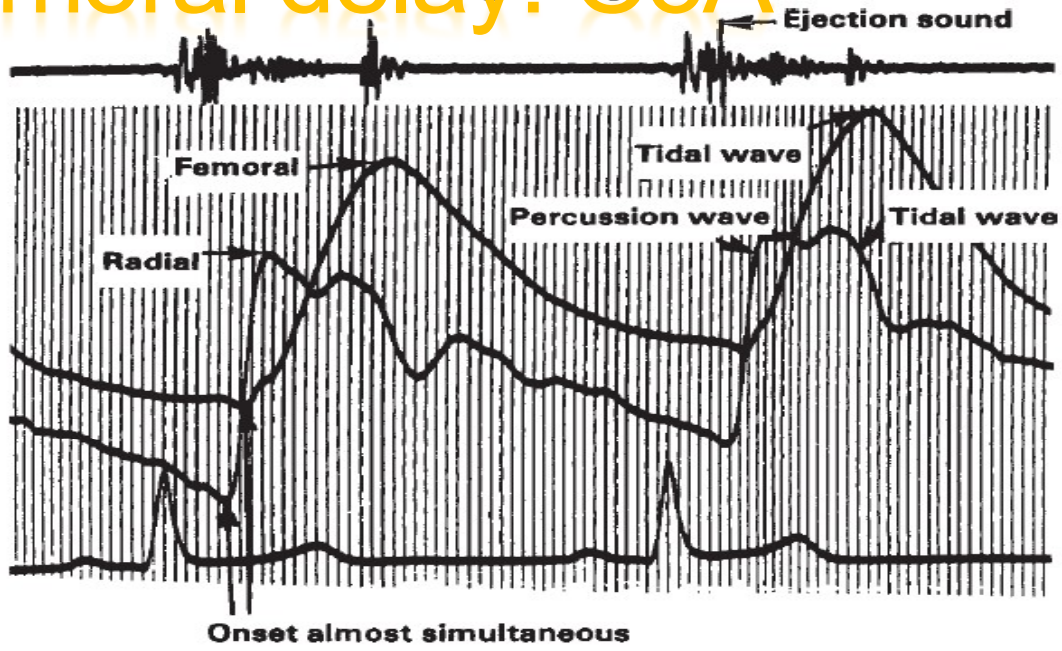
   ANS: AR exaggerates the tendency for the leg systolic pressure to be higher than that in the arms. If the difference is greater than normal, i.e., more than 20 mmHg, it is known as a positive Hill's sign [7].

   Note: It is easy to remember because blood pressure increases, i.e., goes "uphill" in AR as the examiner goes down the body.

2. Why is the cuff systolic pressure in AR higher in the legs than in the arms?

   ANS: One theory is that reflected waves from the periphery sum with forward waves. These summed waves are known as standing waves.

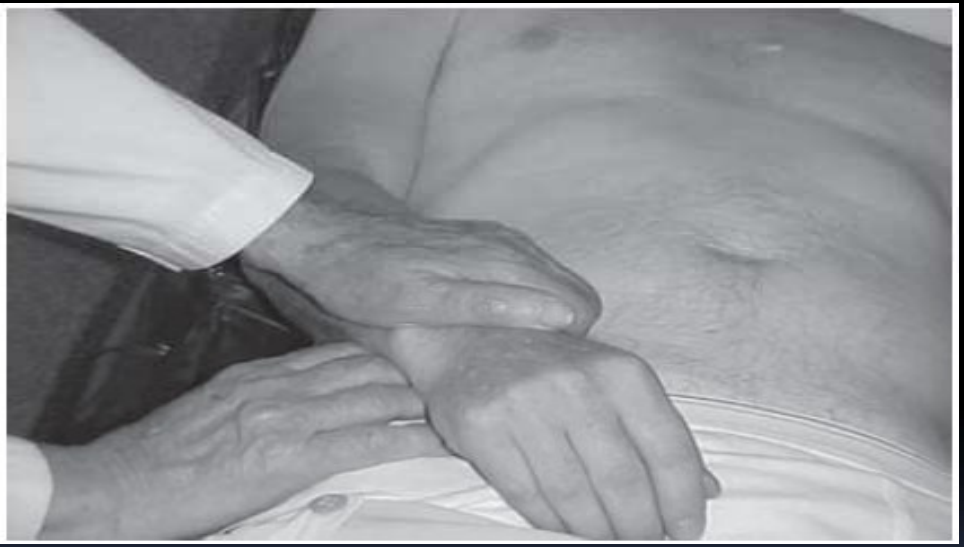
Radio-Femoral delay: CoA



Intra-arterial pressure tracings in a patient with coarctation of the aorta show that the onsets of the femoral and radial pulses remain almost simultaneous. In coarctation, a delay in the femoral is felt on palpating both arteries simultaneously because the percussion wave distal to an obstruction is obliterated by an anacrotic shoulder, which is imperceptible. Thus only the tidal wave is felt in the femoral artery, whereas the earlier percussion wave is felt in the unobstructed radial artery.

Radio-Femoral delay: CoA

By placing the patient's wrist over his or her femoral artery as you palpate both, you can best perceive the obvious delay of the femoral pulse peak over that of the arm. In using the radials rather than the brachials to test for differences between the arm and leg, you take advantage of the increased rapidity of pulse rise as you palpate more peripherally down the arm.



Orthostatic hypotension

- If a patient has a history of presyncope or syncope on standing (orthostatic hypotension), how long should the patient stand before checking for a fall in blood pressure?
- ANS: If the blood pressure does not fall immediately, you should recheck after 3–5 min of standing.
- *Note: A drop of more than 15 mmHg in systolic pressure or any fall in diastolic* pressure suggests hypovolemia or autonomic dysfunction…