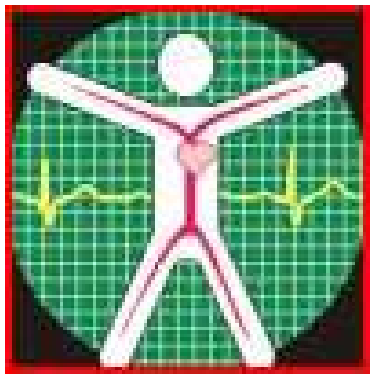
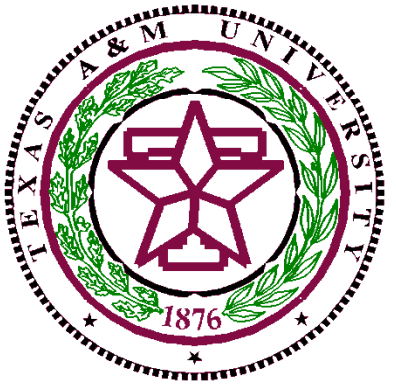


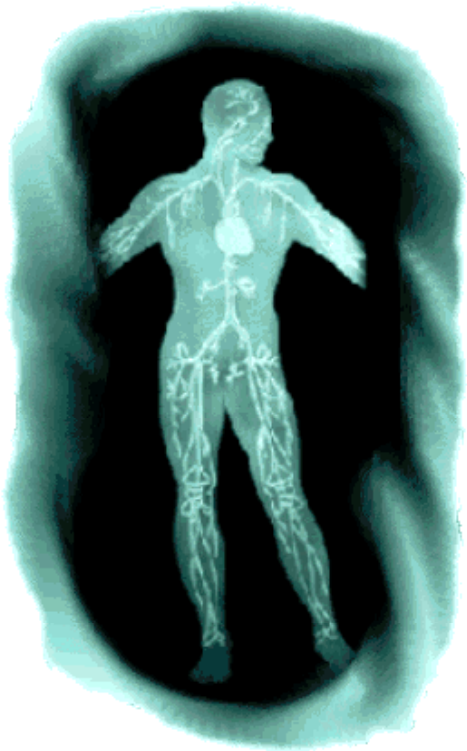
KINE 639 - Dr. Green

Section 1

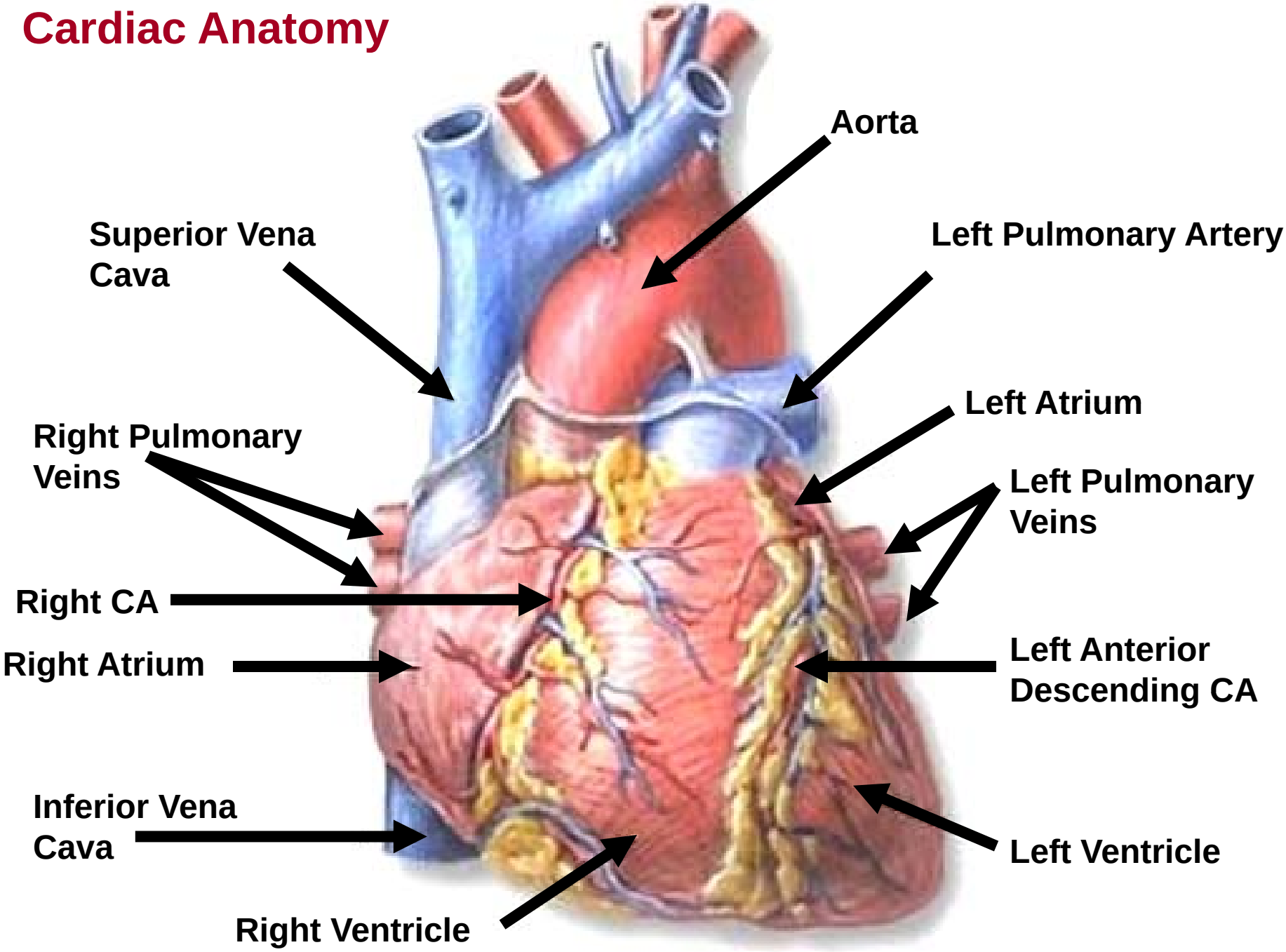


Clinical Physiology I, II, III, IV

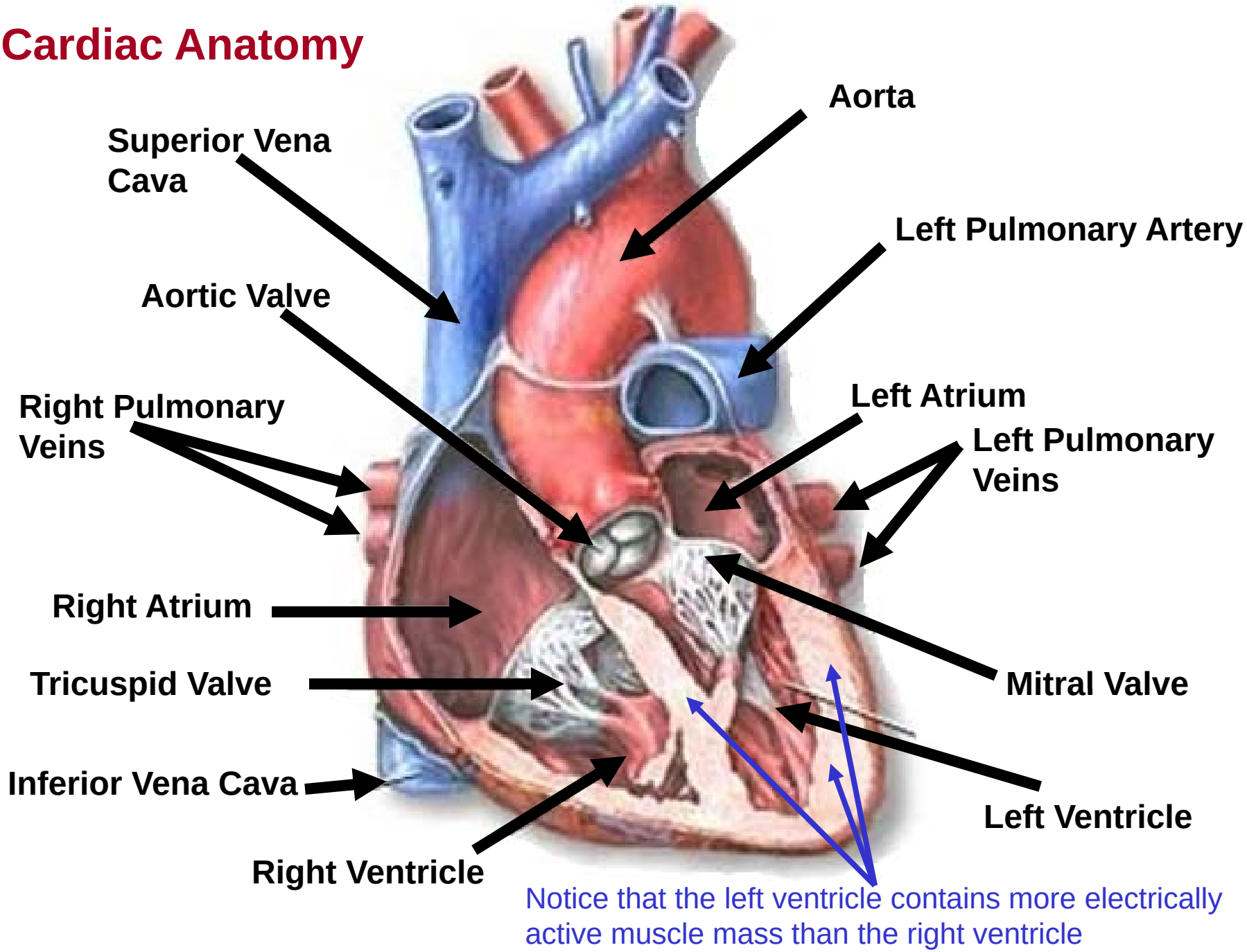
Definitions, Concepts, and Hemodynamics



Cardiac Anatomy

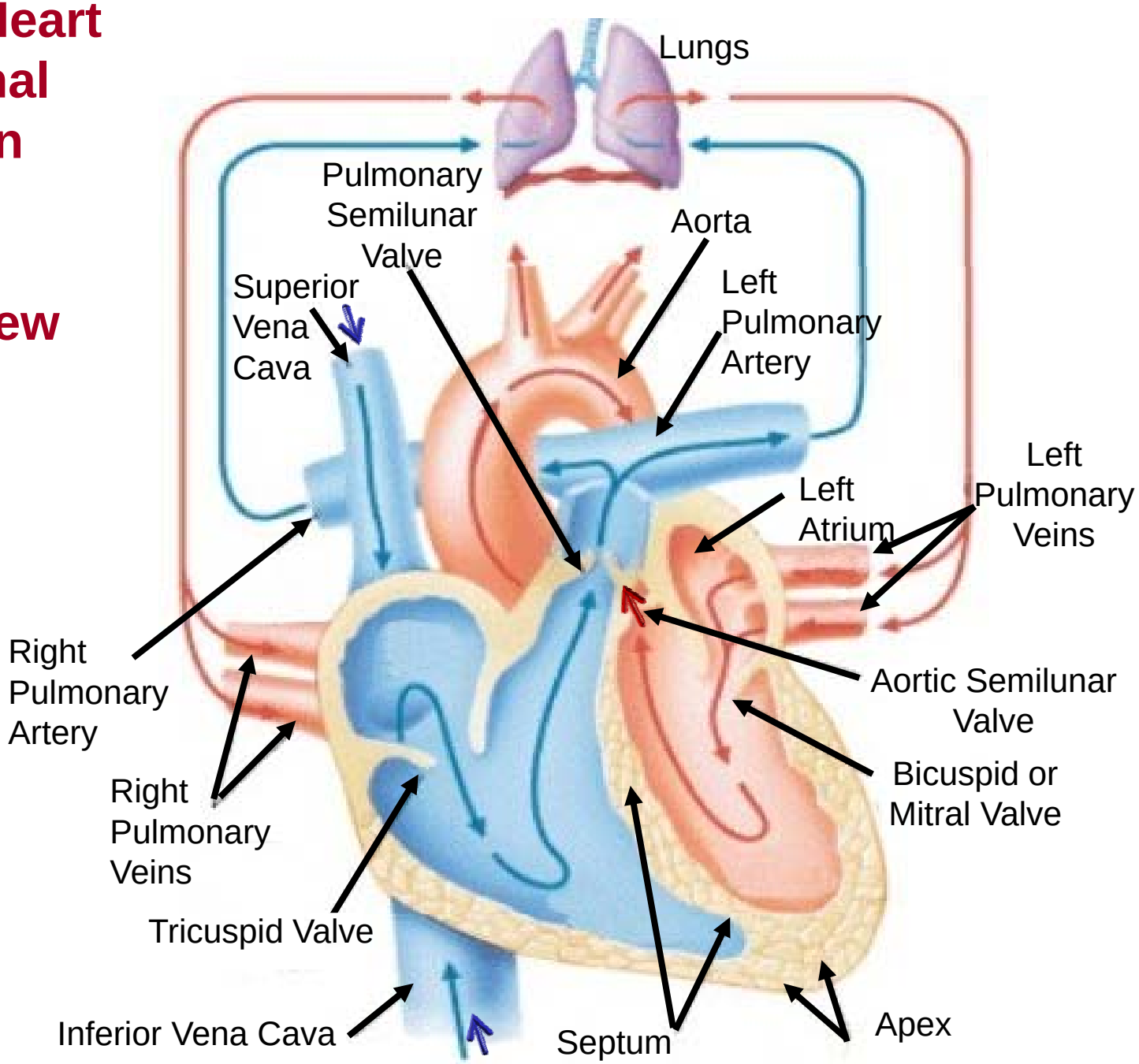


Cardiac Anatomy

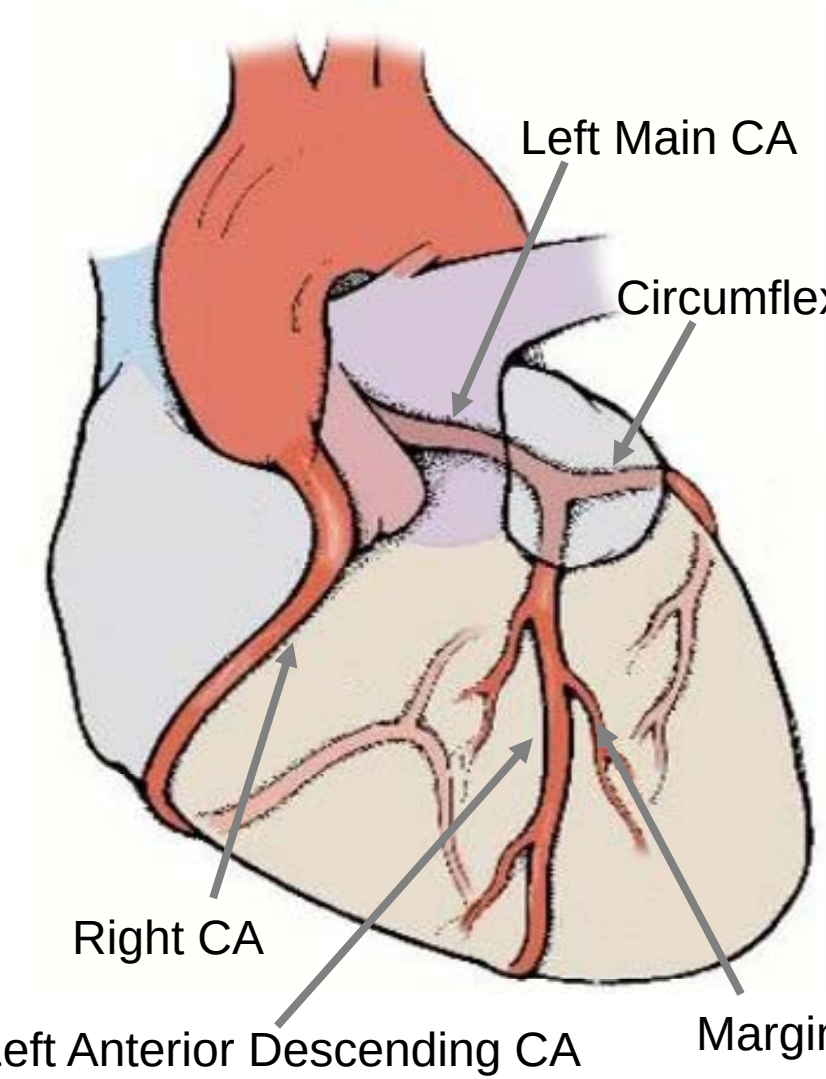


The Normal Heart and Regional Circulation

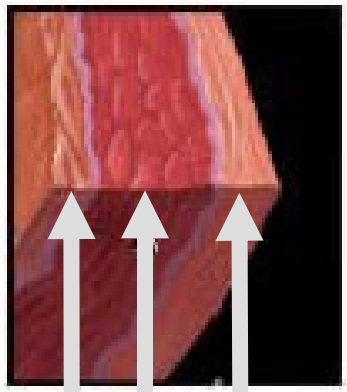
Anterior Cutaway View



The Normal Heart - Coronary Artery Anatomy

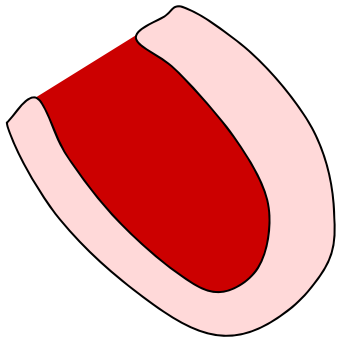


Layers of the Arterial Wall

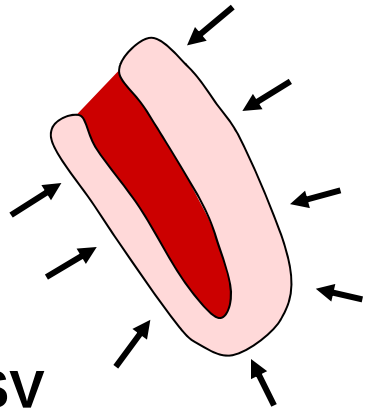


Left Ventricular Volumes - Definitions

End Diastolic Volume (EDV)
Volume at the end of diastole (end of ventricular filling). In a healthy heart this is directly proportional to **venous return**



End Systolic Volume (ESV)
Volume at the end of systole (end of **ventricular contraction**)



Stroke Volume (SV) = EDV - ESV

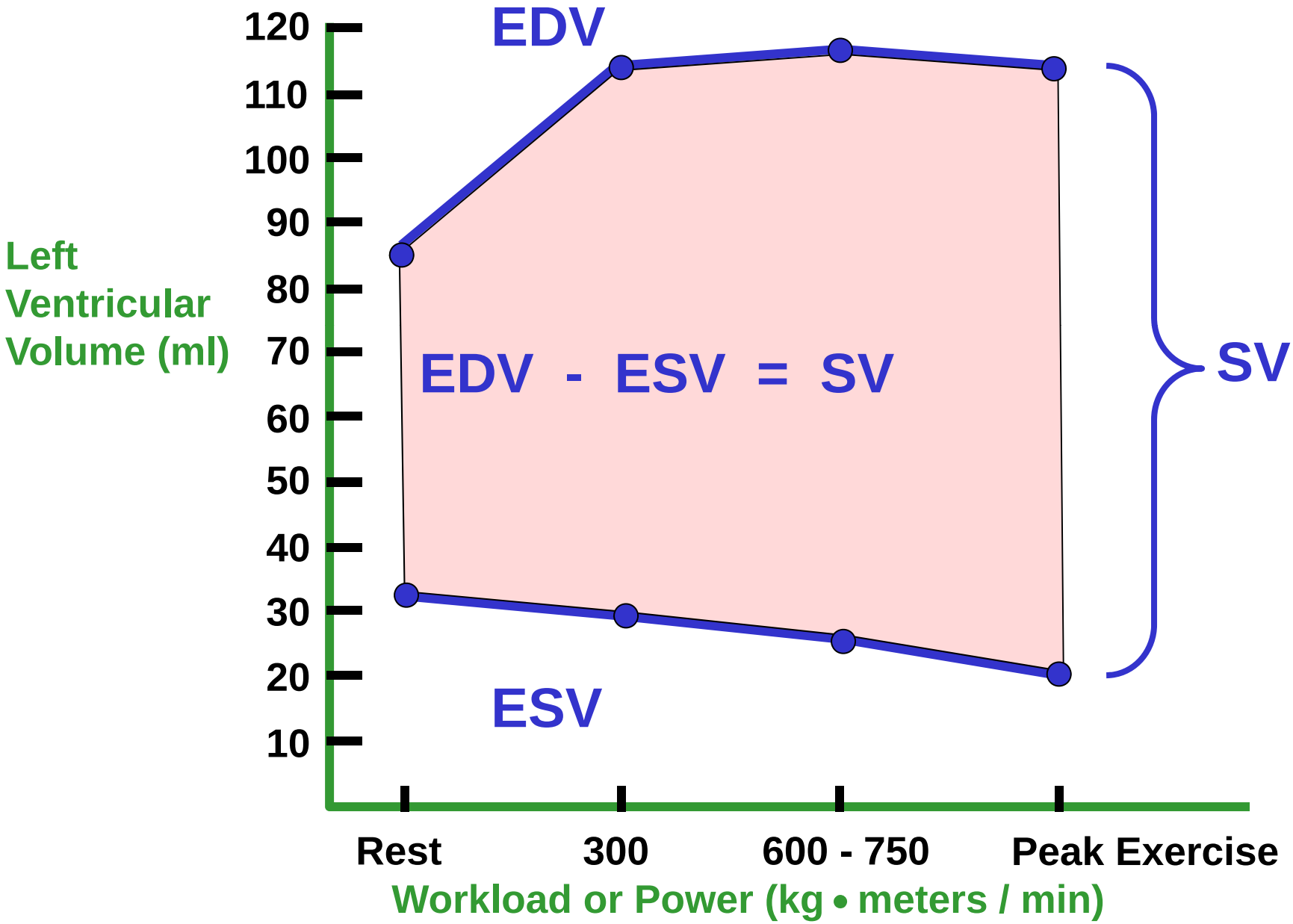
Ejection Fraction (EF) = $\frac{SV}{EDV}$

NOTE: Resting Ejection Fraction (EF) is the best indicator of both heart performance and heart disease prognosis

Left ventricular norm for EF at Rest: approximately 62%

Left Ventricular norms for Max Exercise: approximately 80%

Changes in Left Ventricular Volumes with Exercise of Increasing Intensity

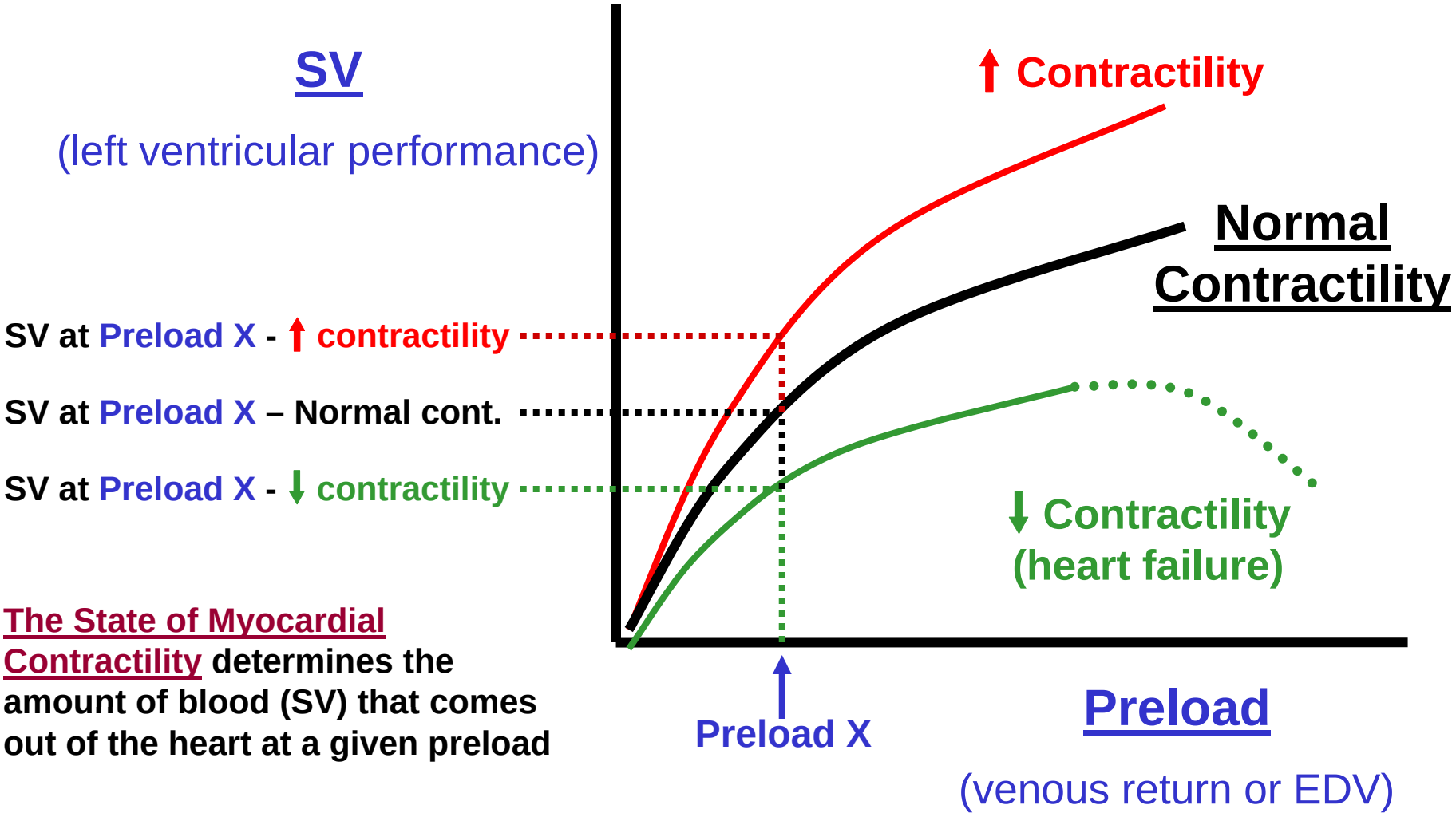


Definitions

- **Cardiac Output: (Q̇)** = HR X SV
- **Cardiac Index** = Q̇ / body surface area
- **Preload: (EDV)** volume of the left ventricle at the end of diastole
  dependent on **venous return** & **compliance ("stretchability") of ventricle**
- **Afterload:** resistance to ventricular emptying during systole or the amount of pressure the left ventricle must generate to squeeze blood into the aorta. In a a healthy heart this is synonymous with **Aortic Pressure** & **Mean Arterial Pressure (MAP)**
- **Frank Starling Law of the Heart:** the heart will contract with greater force as preload (EDV) is increased ➡ more blood in more blood out
- **Myocardial Contractility:** the squeezing contractile force that the heart can develop at a given preload
  - **Regulated by:**
    - Sympathetic nerve activity (most influential)
    - Catecholamines (epinephrine norepinephrine)
    - Amount of contractile mass
    - Drugs

Starlings Law of the Heart and Contractility

Starling's Law:
The greater the EDV (blood going in the heart), the more blood comes out of the heart



The State of Myocardial Contractility determines the amount of blood (SV) that comes out of the heart at a given preload

Influences on Myocardial Contractility

↑ Contractility related to :

Exercise: - ↑ β sympathetic adrenergic nerve output
Catecholamines: - Epinephrine & Norepinephrine
Excitement or Fear: - Fight or flight mechanism
Drugs: - Digitalis & Sympathomimetics

↓ Contractility related to:

Loss of contractile mass: - Most likely due to heart attack
Myocardial muscle disease: - Cardiomyopathy
Drugs: - Anesthetics, Barbiturates

Definitions

- **Arteriovenous Oxygen Difference (AVO_2D)** the difference in oxygen content between arterial and venous blood
  - measured in ml% - ml O_2 / 100 ml blood
- **Oxygen Consumption ($\dot{V}O_2$)** - the rate at which oxygen can be used in energy production and metabolism
  - "absolute" measures: L O_2 / min , ml O_2 / min
  - "relative" measures: ml O_2 / kg body wt. / min
  - Fick equation: $\dot{V}O_2 = \dot{Q} \times AVO_2D$
- **Maximum Oxygen Consumption ($\dot{V}O_{2max}$)** maximum rate at which a person can take in and utilize oxygen to create usable energy
  - defined as plateau of consumption rate increase
  - often estimated with $\dot{V}O_{2peak}$
- **Myocardial Oxygen Consumption** $\dot{V}O_2$ of the heart muscle (myocardium)
  - "estimated" by RPP: HR X SBP

Definitions

- **Systolic Blood Pressure (SBP)** pressure measured in brachial artery during systole (ventricular emptying and ventricular contraction period)
- **Diastolic Blood Pressure (DBP)** pressure measured in brachial artery during diastole (ventricular filling and ventricular relaxation)
- **Mean Arterial Pressure (MAP)** "average" pressure throughout the cardiac cycle against the walls of the proximal systemic arteries (aorta)
  - estimated as: .33(SBP - DBP) + DBP
- **Total Peripheral Resistance (TPR)** - the sum of all forces that oppose blood flow

  - Length of vasculature (**L**)
  - Blood viscosity (**V**)
  - Vessel radius (**r**)

$$TPR = \frac{(8)(V)(L)}{(\pi)(r^4)}$$

Cardiovascular Hemodynamic Basics

$$\text{Flow } (\dot{Q}) = \frac{\text{Pressure (MAP)}}{\text{Resistance (TPR)}} = \frac{P_{\text{aorta}} - P_{\text{vena cava}}}{\frac{(8)\,(V)\,(L)}{(\pi)\,(r^4)}}$$

$$\text{Flow } (\dot{Q}) = \frac{(\pi)\;(P_a - P_v)\;(r^4)}{(8)\,(V)\,(L)}$$

Normally Resting $\dot{Q}$ is about 5 - 6 liters / minute

V = viscosity of fluid (blood) flowing through the pipe
L = length of pipe (blood vessel)
r = radius of the pipe (blood vessel)
P_a = aortic pressure
P_v = venous pressure

Respiratory Physiology - Definitions

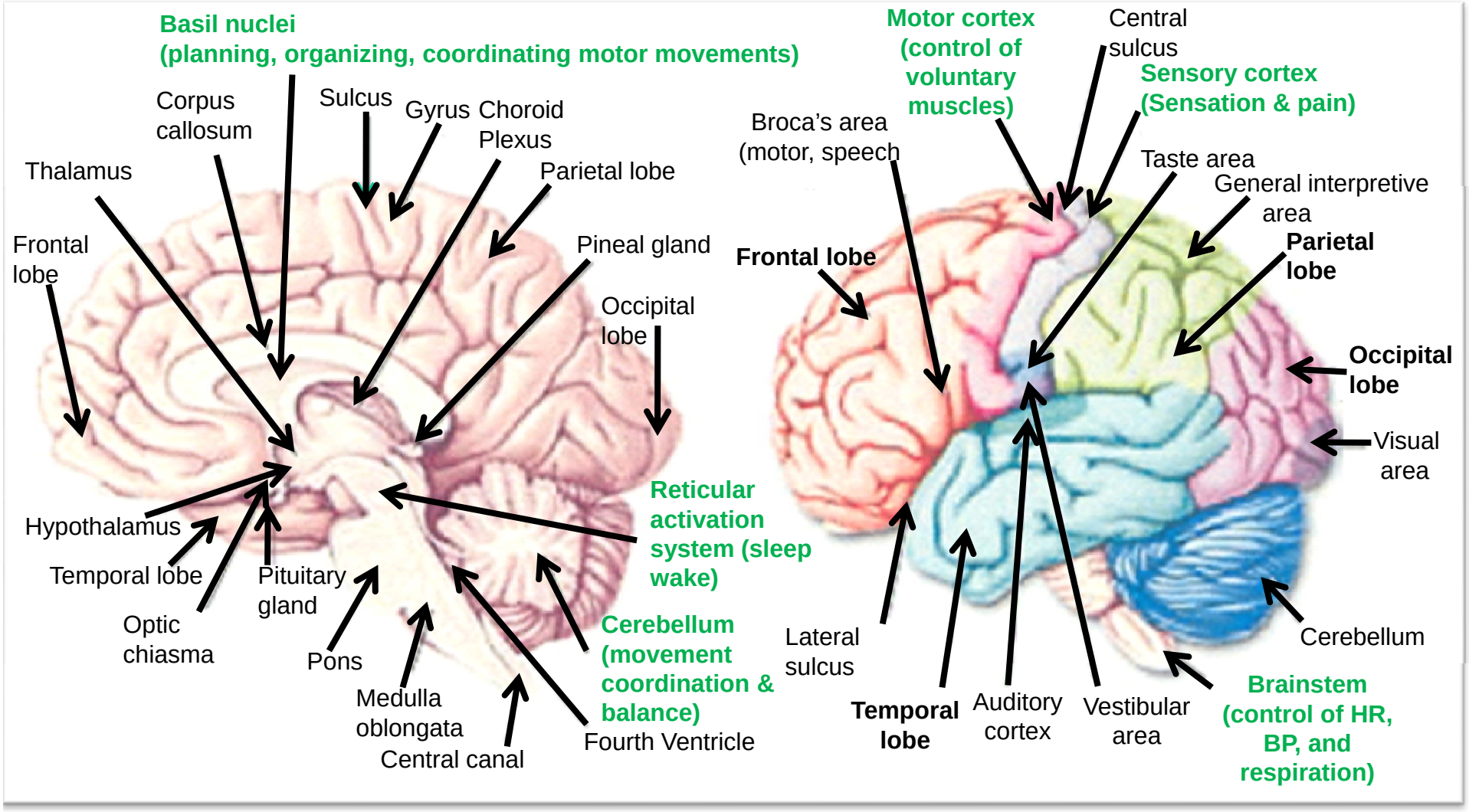
- **Minute Ventilation ($\dot{V}_E$)** – amount of air passing through the lungs in one minute
- **Dyspnea** - breathing difficulty
- **Respiratory Exchange Ratio** - amount of CO_2 expired by the lungs divided by the amount of O_2 extracted from the air in the lungs (VCO_2 / VO_2).

RER = .7	➡	100% fat	0% carb
RER = .85	➡	50% fat	50% carb
RER = 1.0	➡	0% fat	100% carb

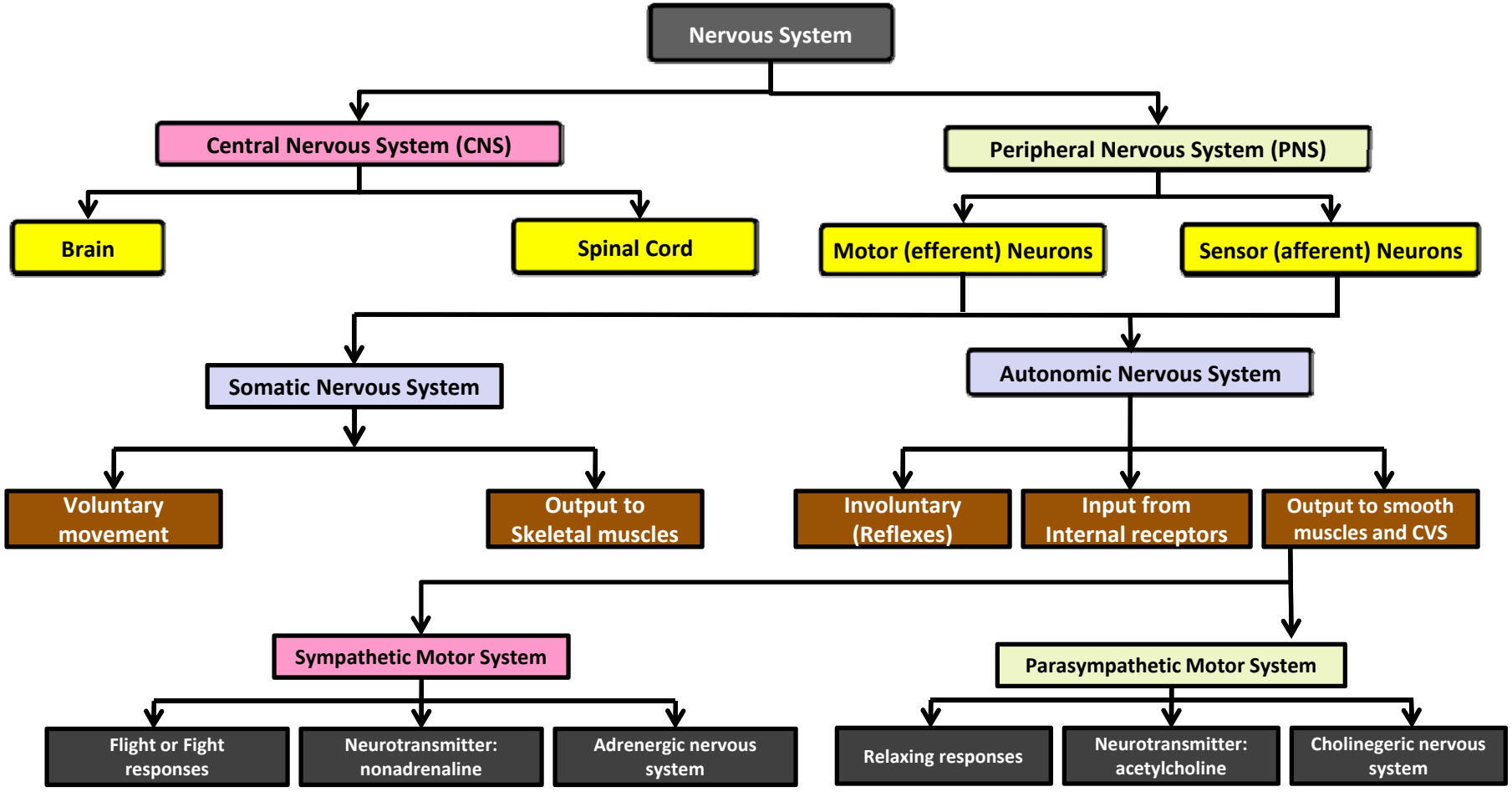
Neurophysiology - Definitions

- **Afferent** - sensory nerves - going toward spinal column
- **Efferent** - effector nerves - going away from spinal column

Essential Knowledge of the Areas of the Brain in Green



Organization of the Nervous System



Adrenergic Receptors & Associated Responses

Agonist – body molecule or drug "stimulator"

Antagonist - body molecule or drug "in-activator"

α_1 stimulation:

- Constriction of blood vessels
  - Vascular smooth muscle activation
- Constriction of lung bronchioles
- Constriction of bladder muscles
- ↑ myocardial cardiac contractility
- Relaxation of GI tract

α_2 stimulation:

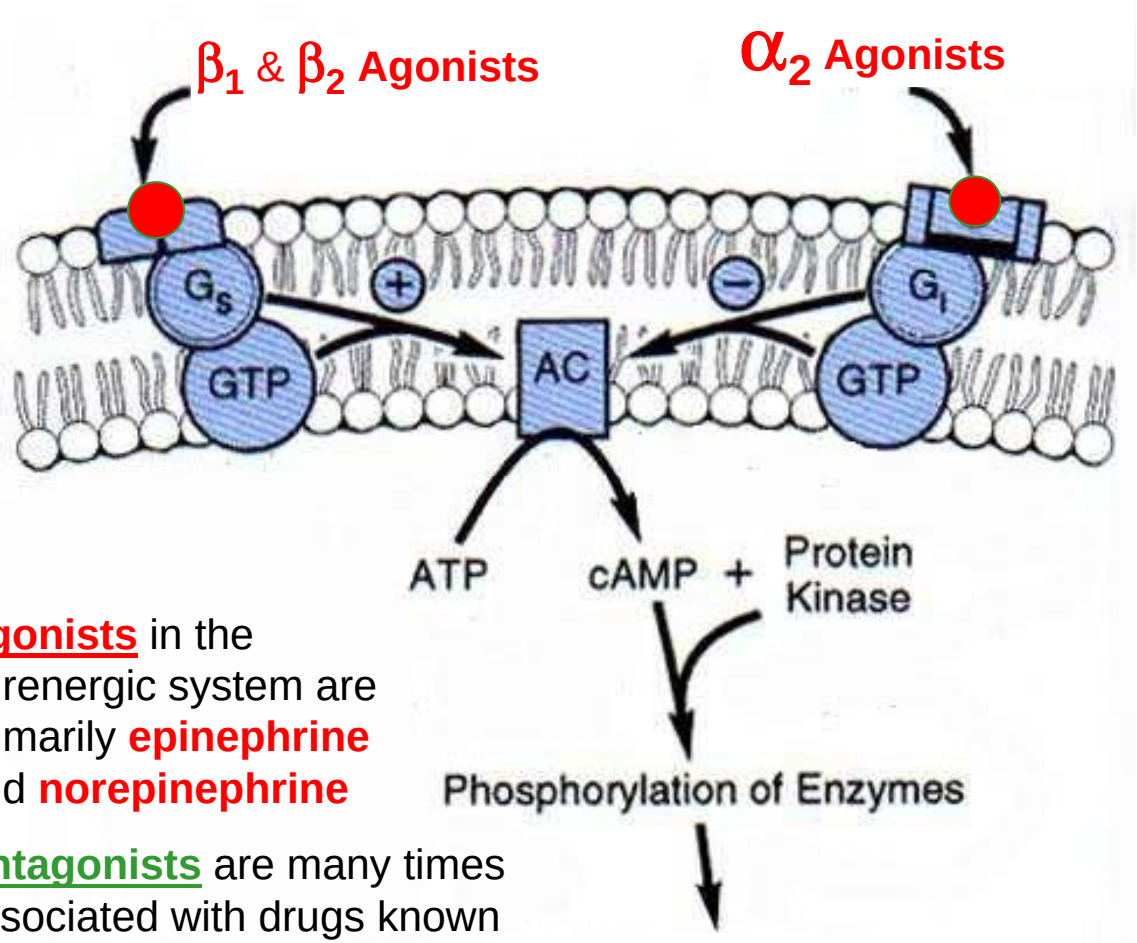
- ↑ central sympathetic outflow
  - ↑ release of E & NE
  - α1 & β1 receptor activation
- Constriction of lung bronchioles

β_1 stimulation:

- ↑ in HR
- ↑ in myocardial contractility
- ↑ in Renin secretion
  - ↑ fluid retention

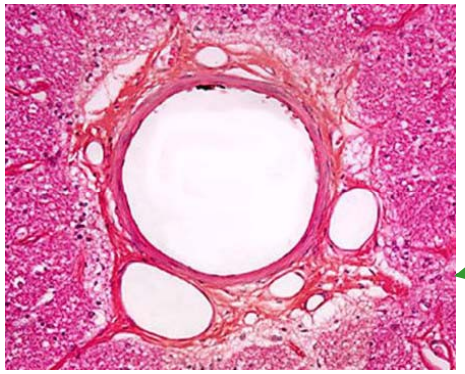
β_2 stimulation:

- Dilation of lung bronchioles
- Dilation of blood vessels

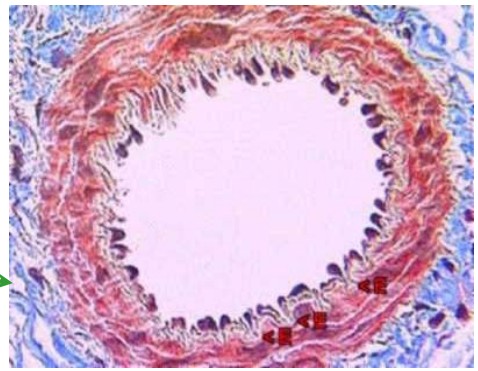


Agonists in the adrenergic system are primarily **epinephrine** and **norepinephrine**

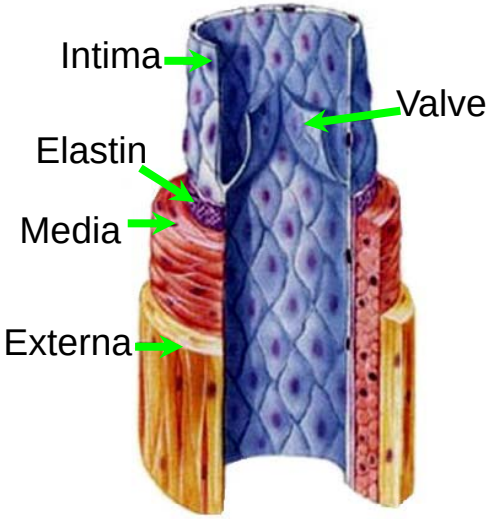
Antagonists are many times associated with drugs known as "blockers" i.e. **"β-blocker"** or **"α-blocker"**

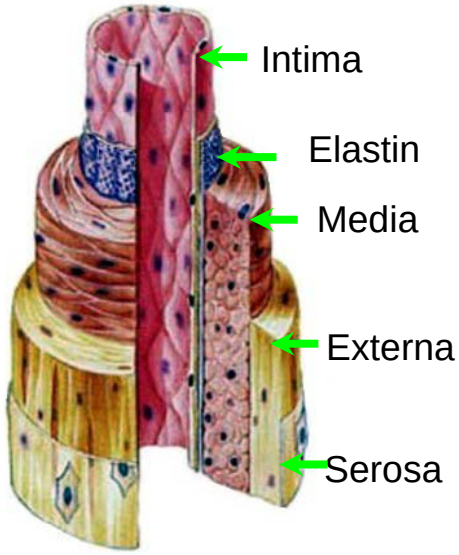


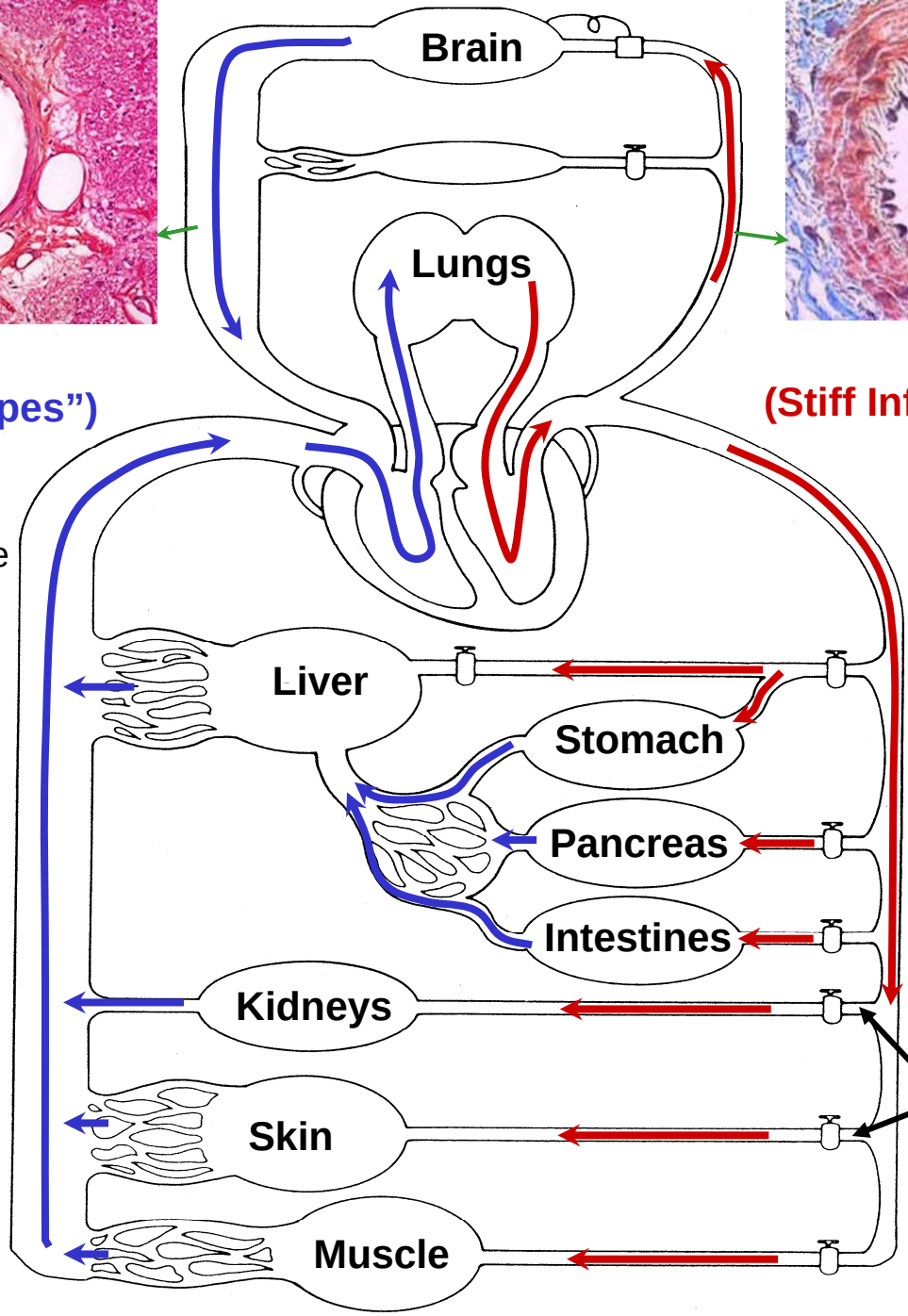
Veins
(Flexible Compliant "Pipes")



Arteries
(Stiff Inflexible "Pipes")

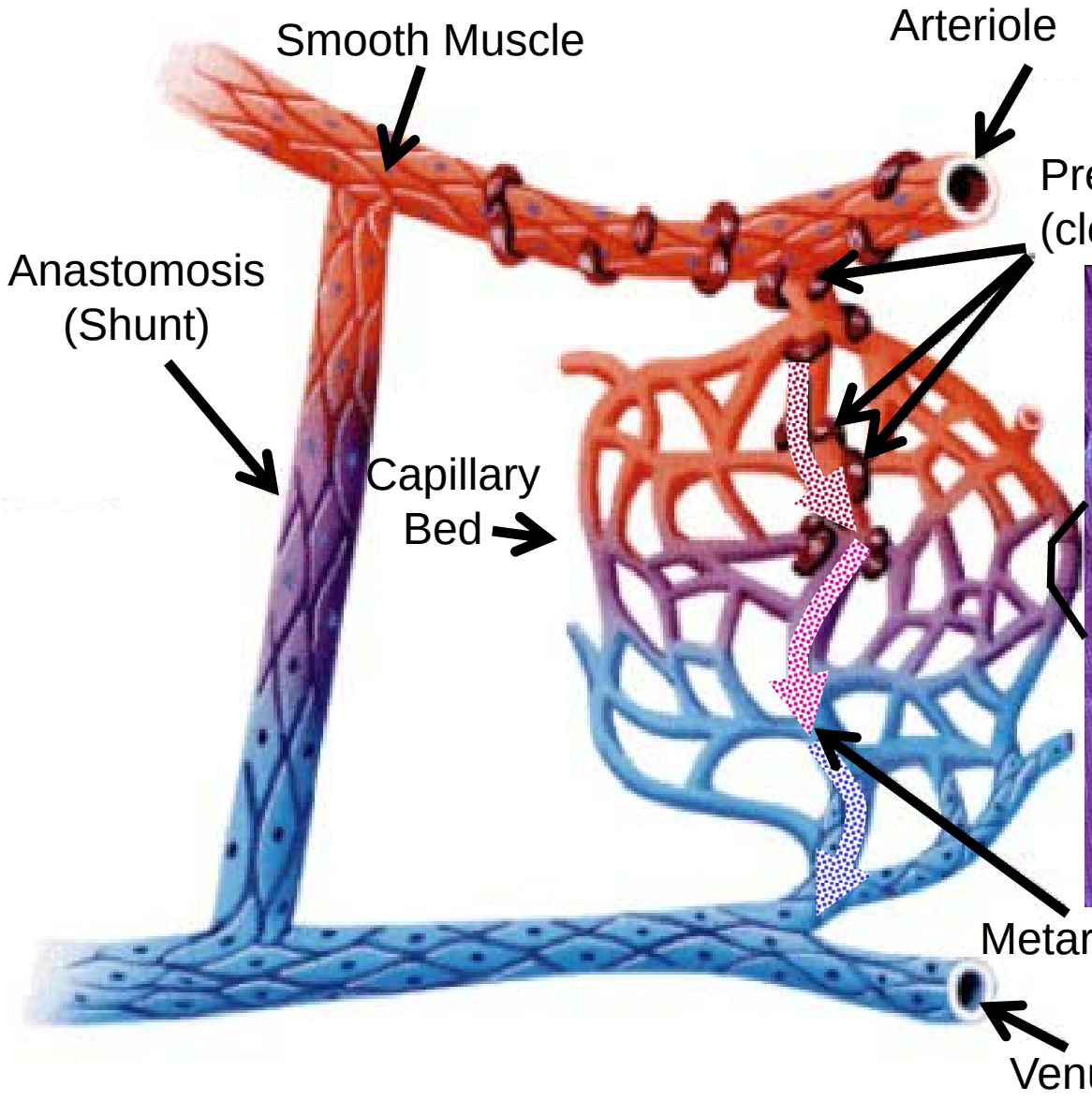


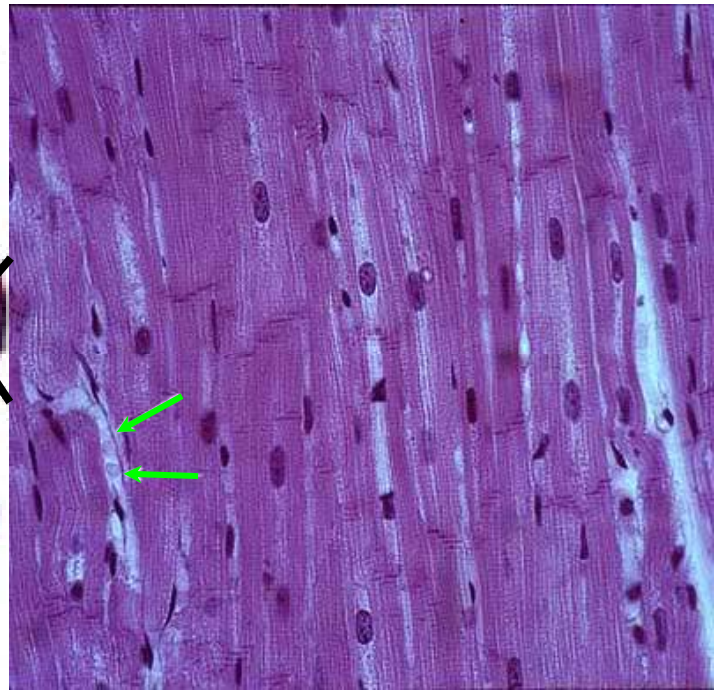




The Systemic Circulation

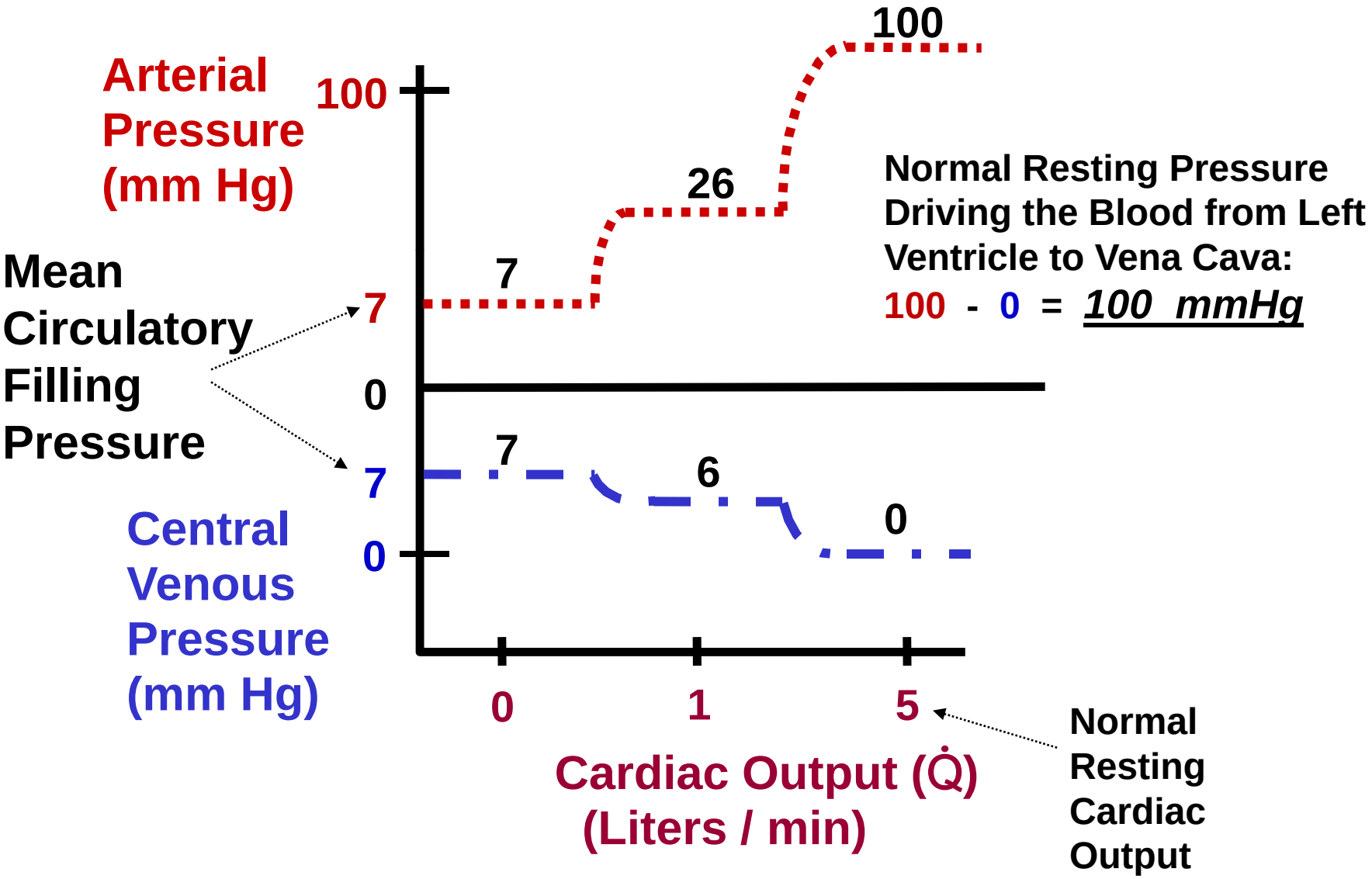
Microcirculatory Anatomy – a Capillary Bed





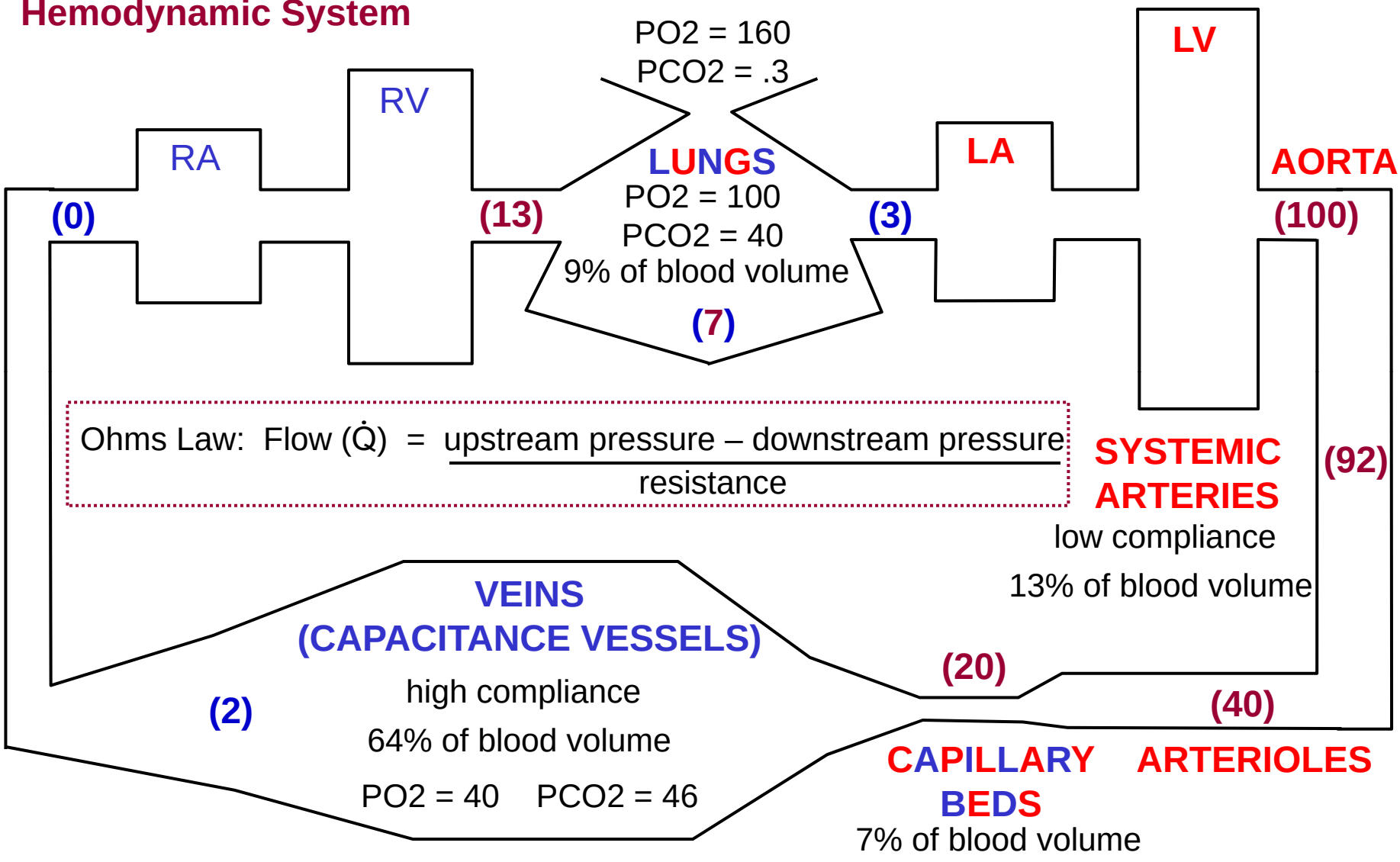
Capillary in Cardiac Muscle (arrows)

Development of the Driving Pressure in the Human Cardiovascular System



The "Closed" Cardiovascular Hemodynamic System

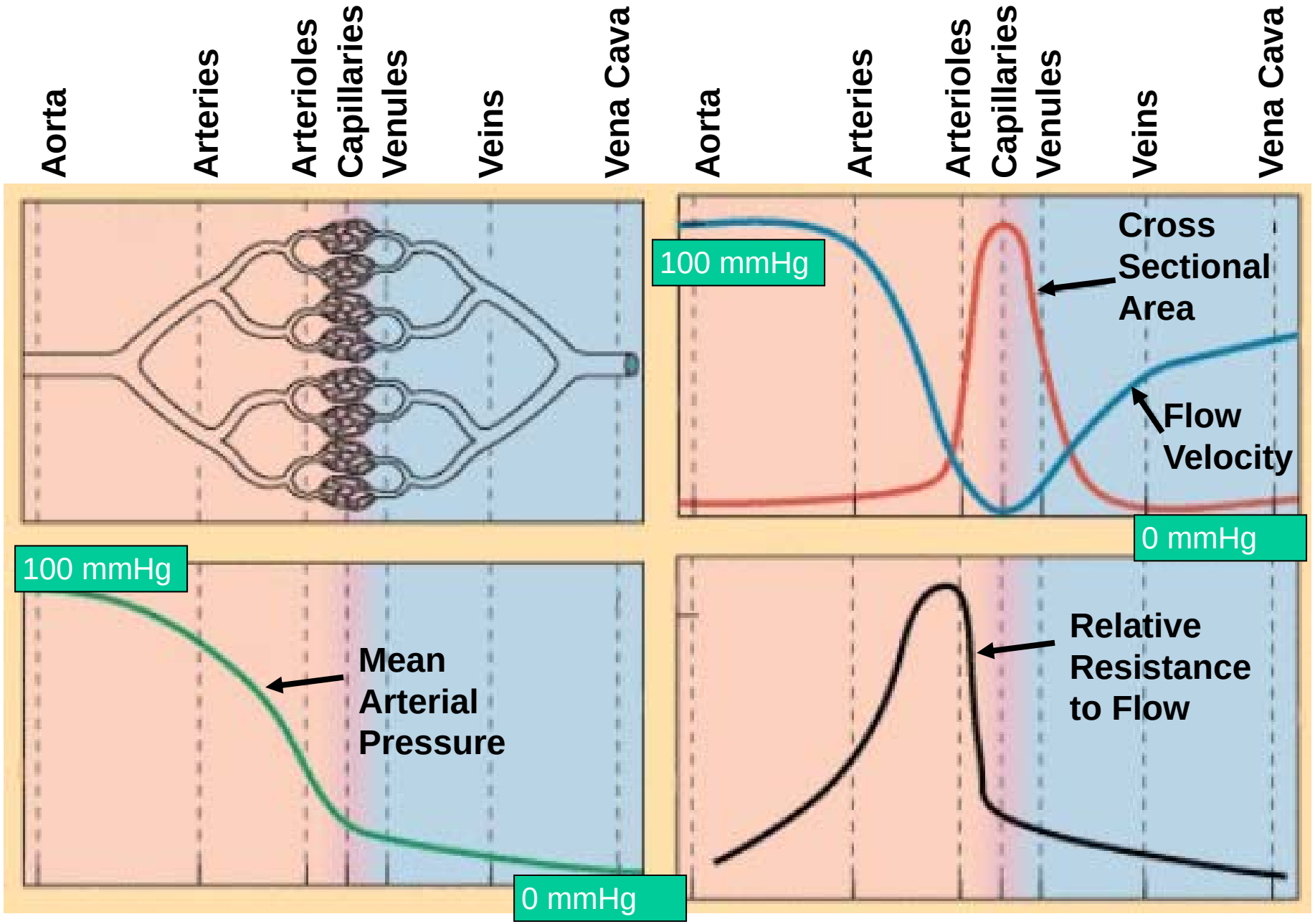
Arterial Pressures in Maroon
Venous Pressures in Blue



Ohms Law: Flow ($\dot{Q}$) = $\frac{\text{upstream pressure} - \text{downstream pressure}}{\text{resistance}}$

Systemic Circulation Flow ($\dot{Q}$) = $\frac{100 \text{ mmHg} - 0 \text{ mmHg}}{1 \text{ mmHg sec / ml}}$ = 100 ml / sec = 6 liters / min

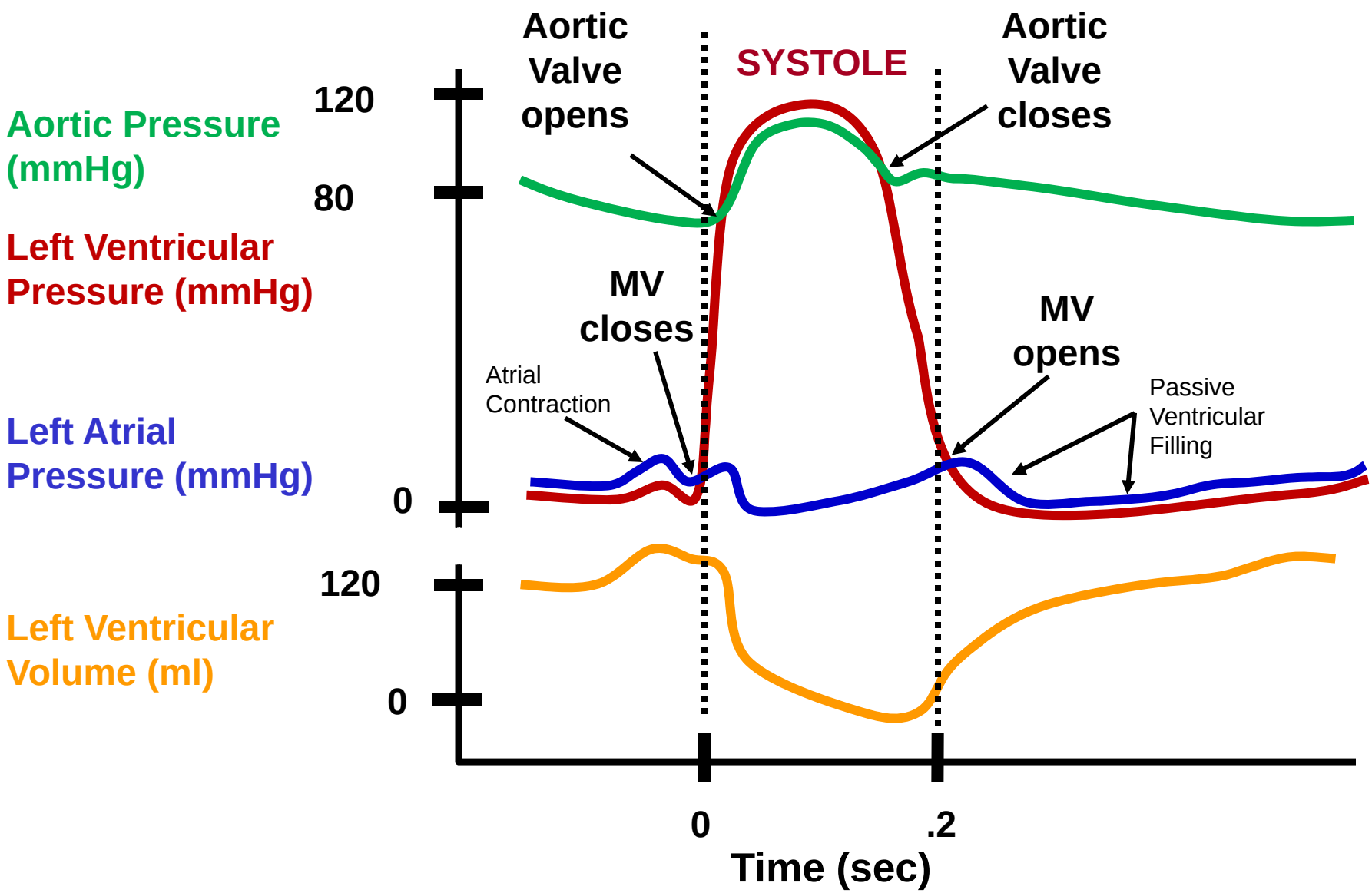
Pressure, Flow, and Resistance by Vascular Cross Sectional Area



MV is an Atrio-Ventricular Valve (AV) and is bi-cuspid

Aortic valve is a semilunar valve

The Cardiac Cycle

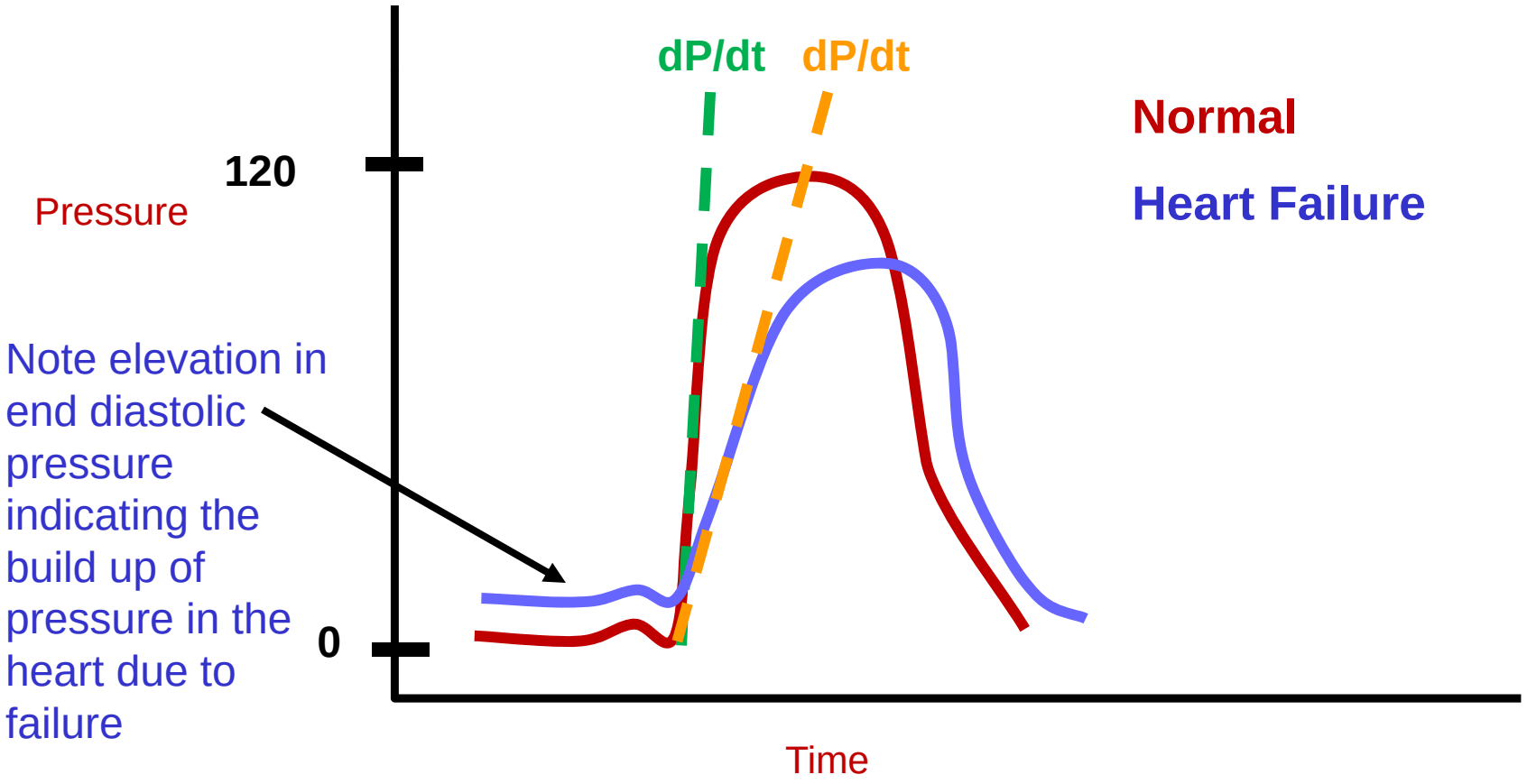


http://www.youtube.com/watch?v=yGlFBzaTuoI&feature=related

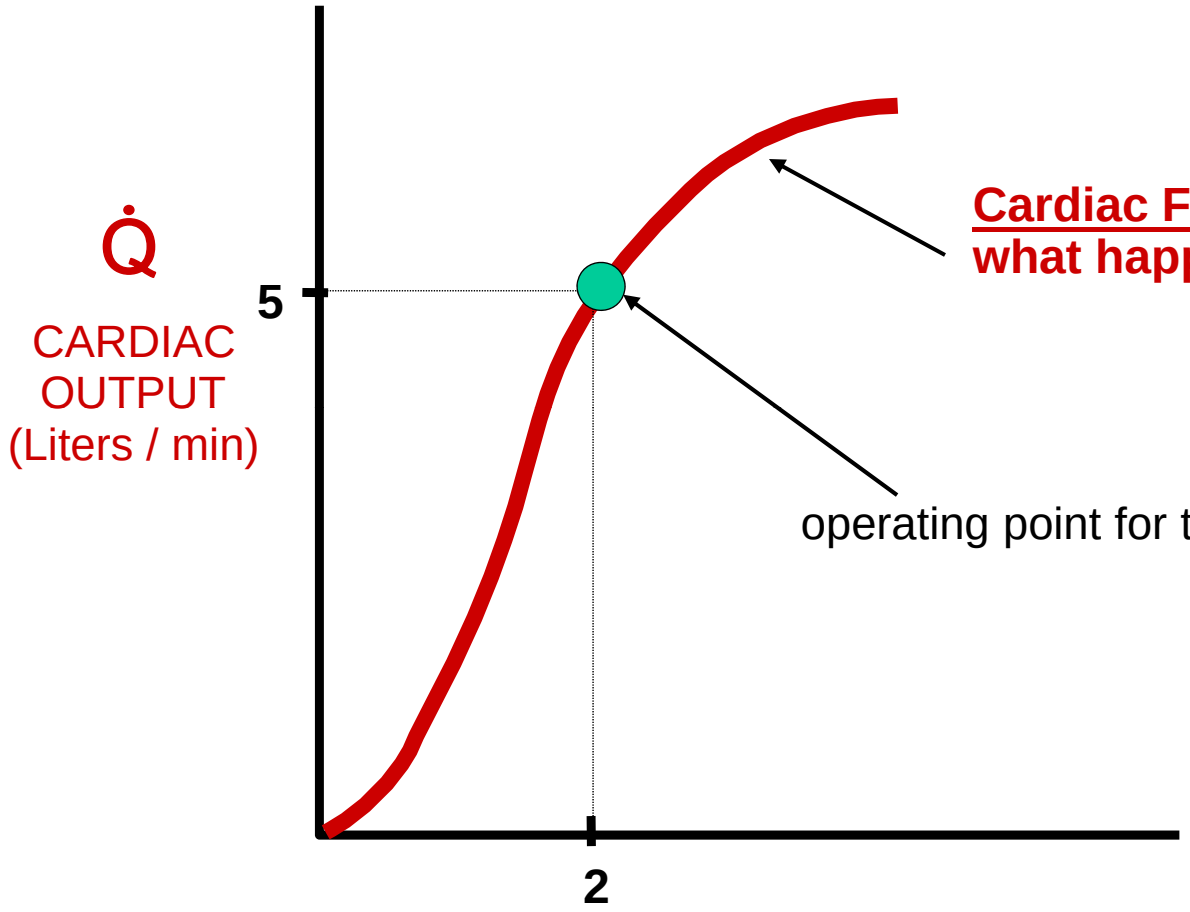
http://www.youtube.com/watch?v=dYgYcH7R29I&NR=1

Using Ventricular Pressure Curves as Indices of Contractility & Cardiac Function

dP/dt = change in pressure per unit of time



Cardiac & Vascular Function Curves

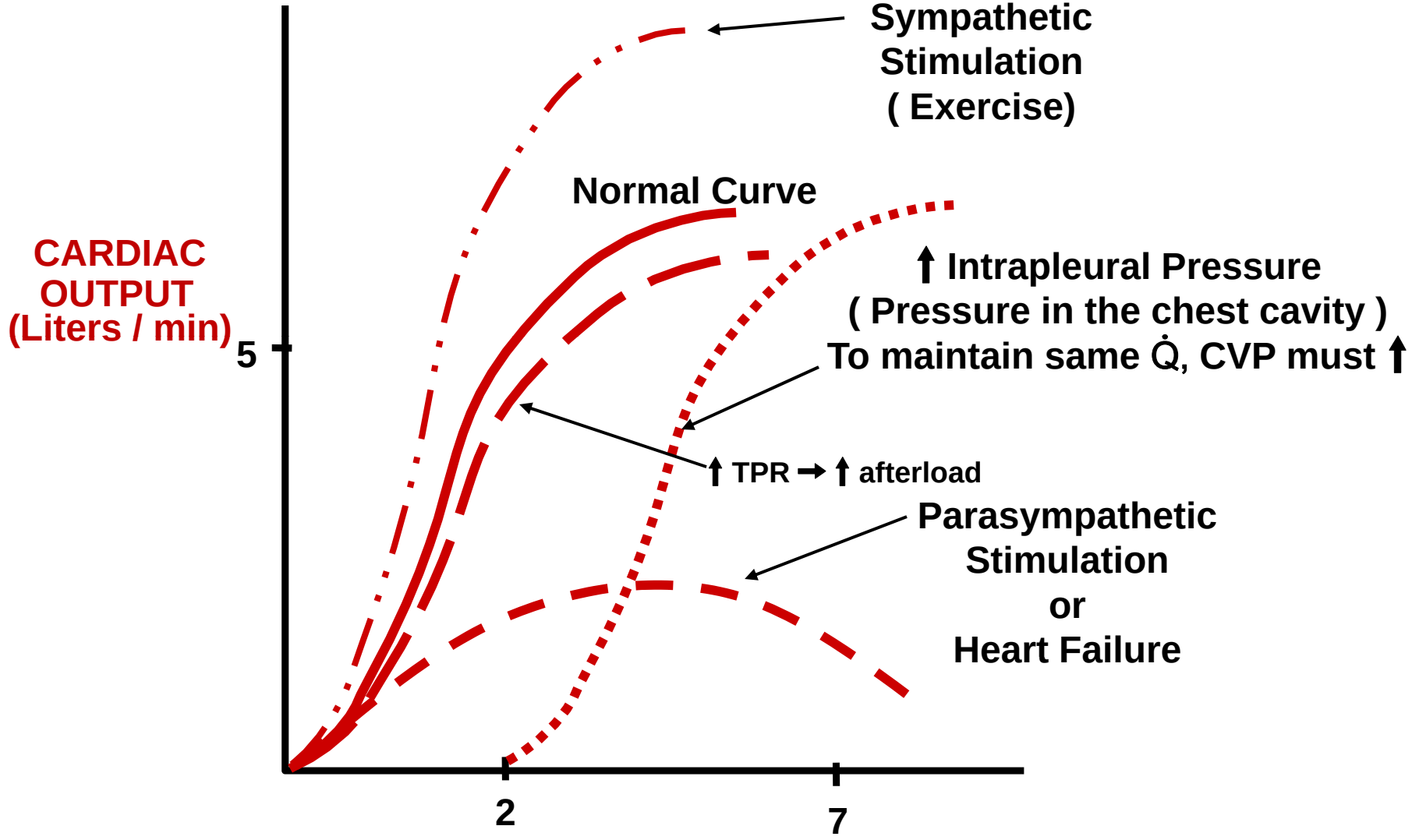


Cardiac Function Curve- illustrates what happens to Q̇ when Pv changes.

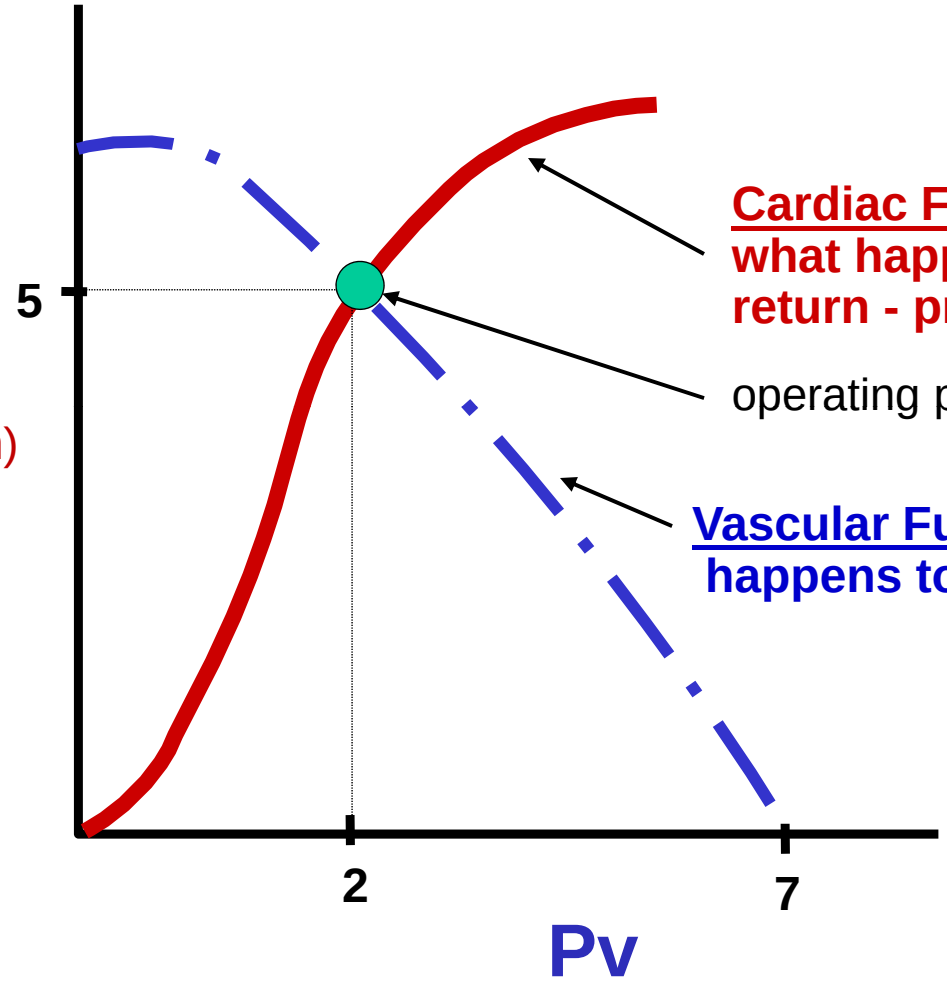
operating point for the cardiovascular system

CENTRAL VENOUS PRESSURE (mmHg)
CARDIAC PRELOAD (mmHg)
RIGHT ATRIAL PRESSURE (mmHg)

Cardiac Function Curve - $\dot{Q}$ is the dependent variable (in effect, $\dot{Q}$ is controlled by venous return & TPR)



Cardiac & Vascular Function Curves

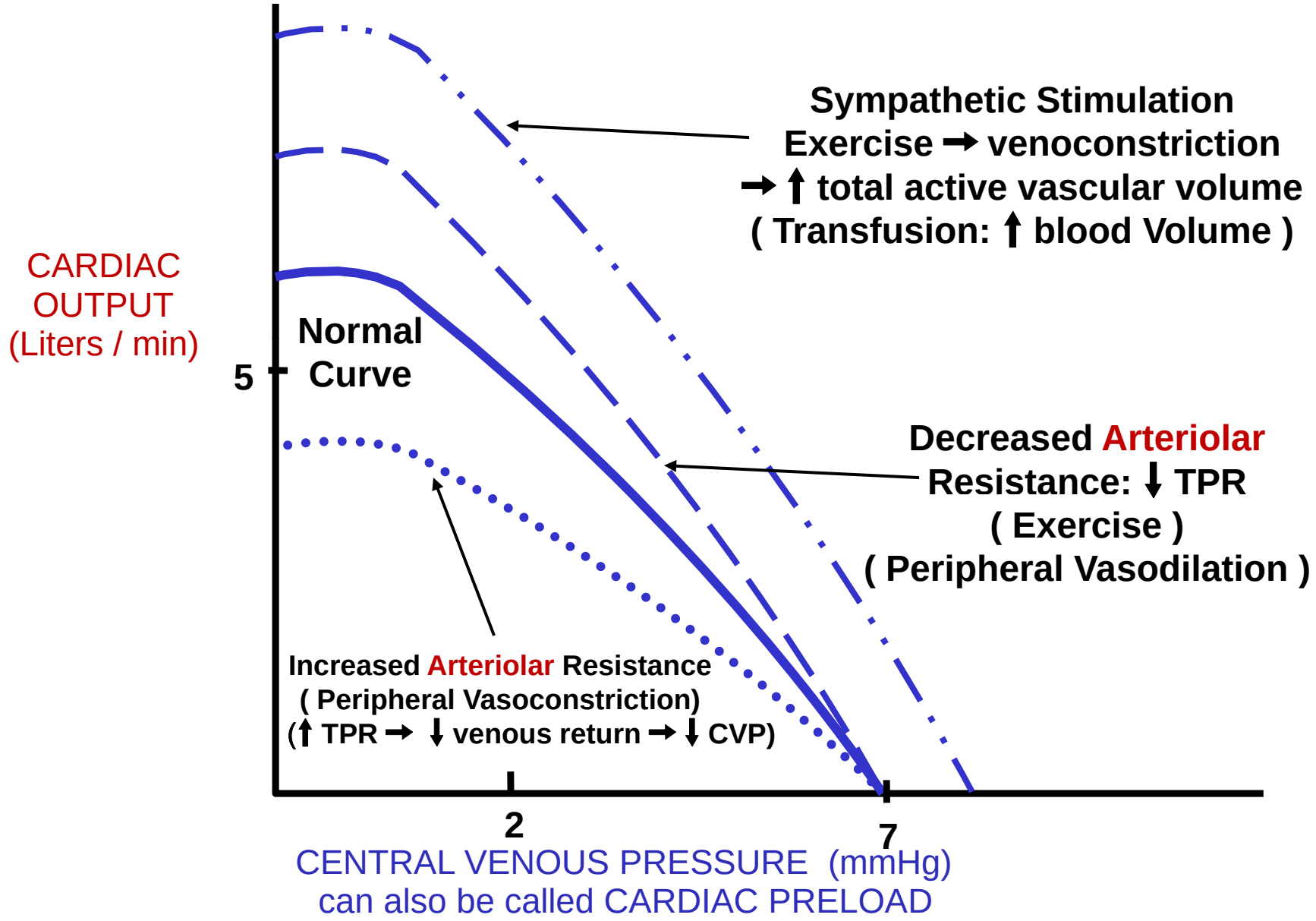


Cardiac Function Curve- illustrates what happens to Q̇ when Pv (venous return - preload) changes.

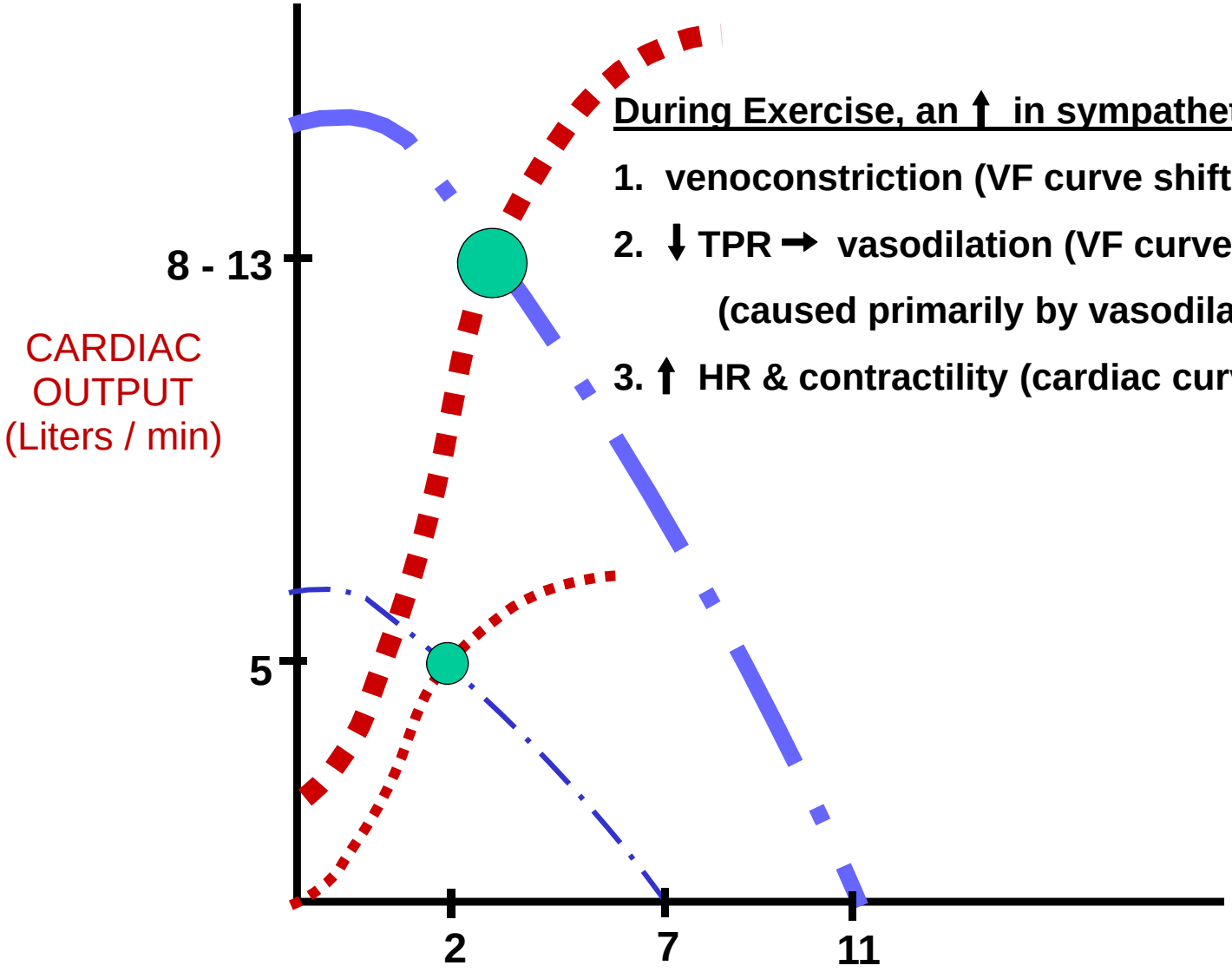
operating point for the cardiovascular system

Vascular Function Curve - illustrates what happens to Pv when Q̇ changes.

Vascular Function Curve - central venous pressure is dependent variable (in effect, CVP and venous return are controlled by $\dot{Q}$)



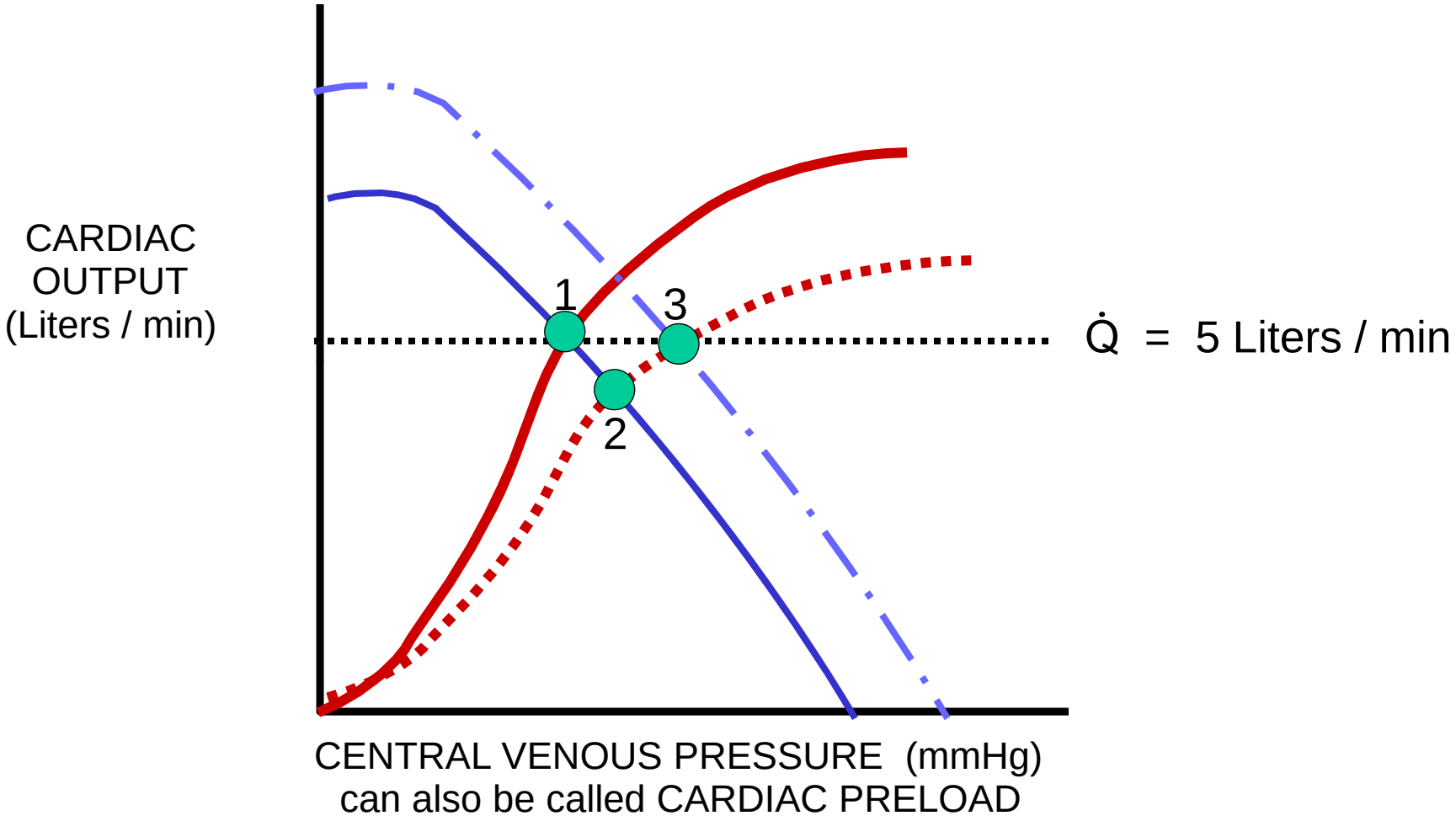
Changes in Cardiac & Vascular Function Curves with Exercise



<u>During Exercise, an ↑ in sympathetic output will cause</u>:

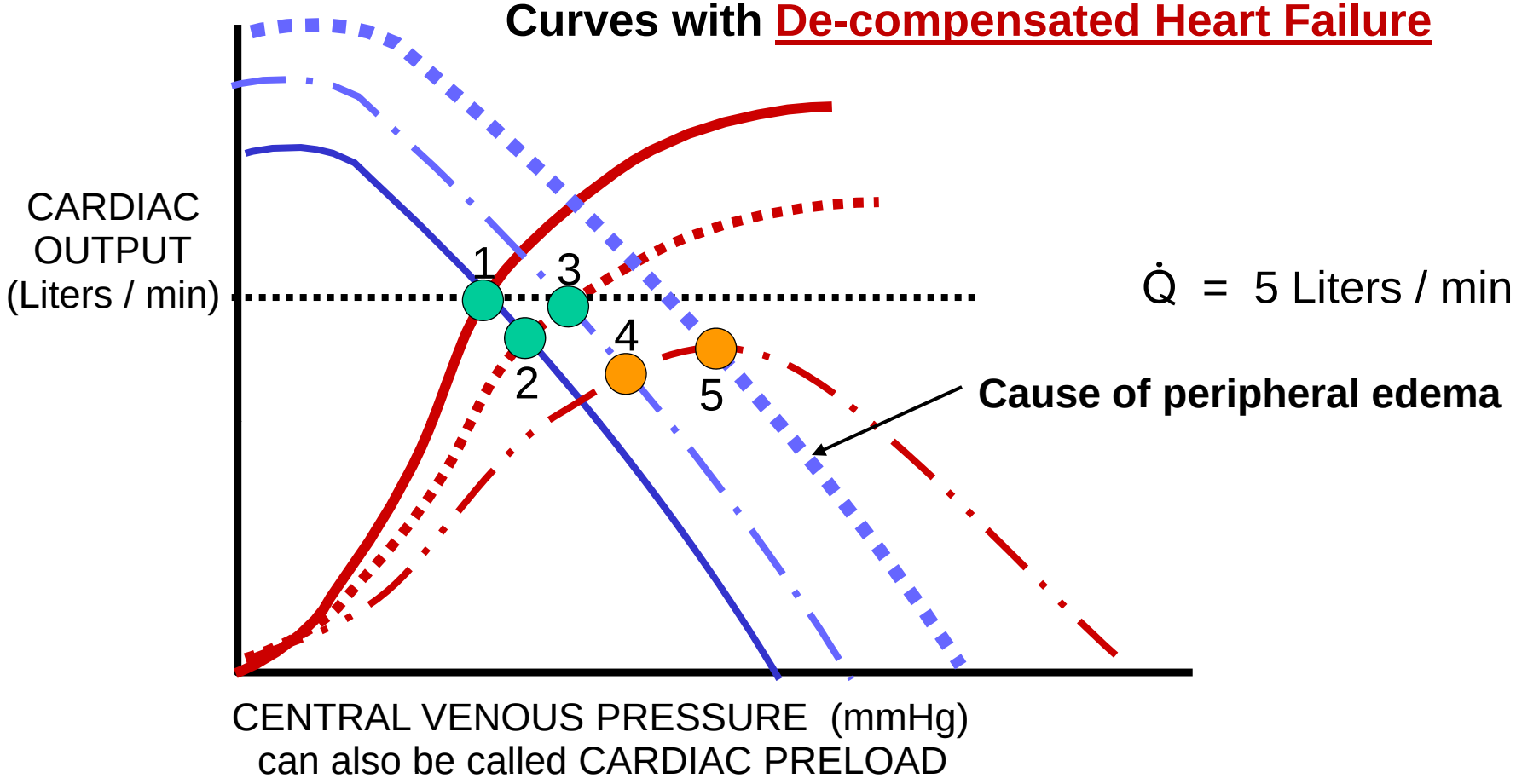
1. **venoconstriction (VF curve shifted rightward)**
2. **↓ TPR → vasodilation (VF curve rotated upward)**
   (caused primarily by vasodilator metabolites)
3. **↑ HR & contractility (cardiac curve shifted upward)**

Changes in Cardiac & Vascular Function Curves with Acute Compensated Heart Failure



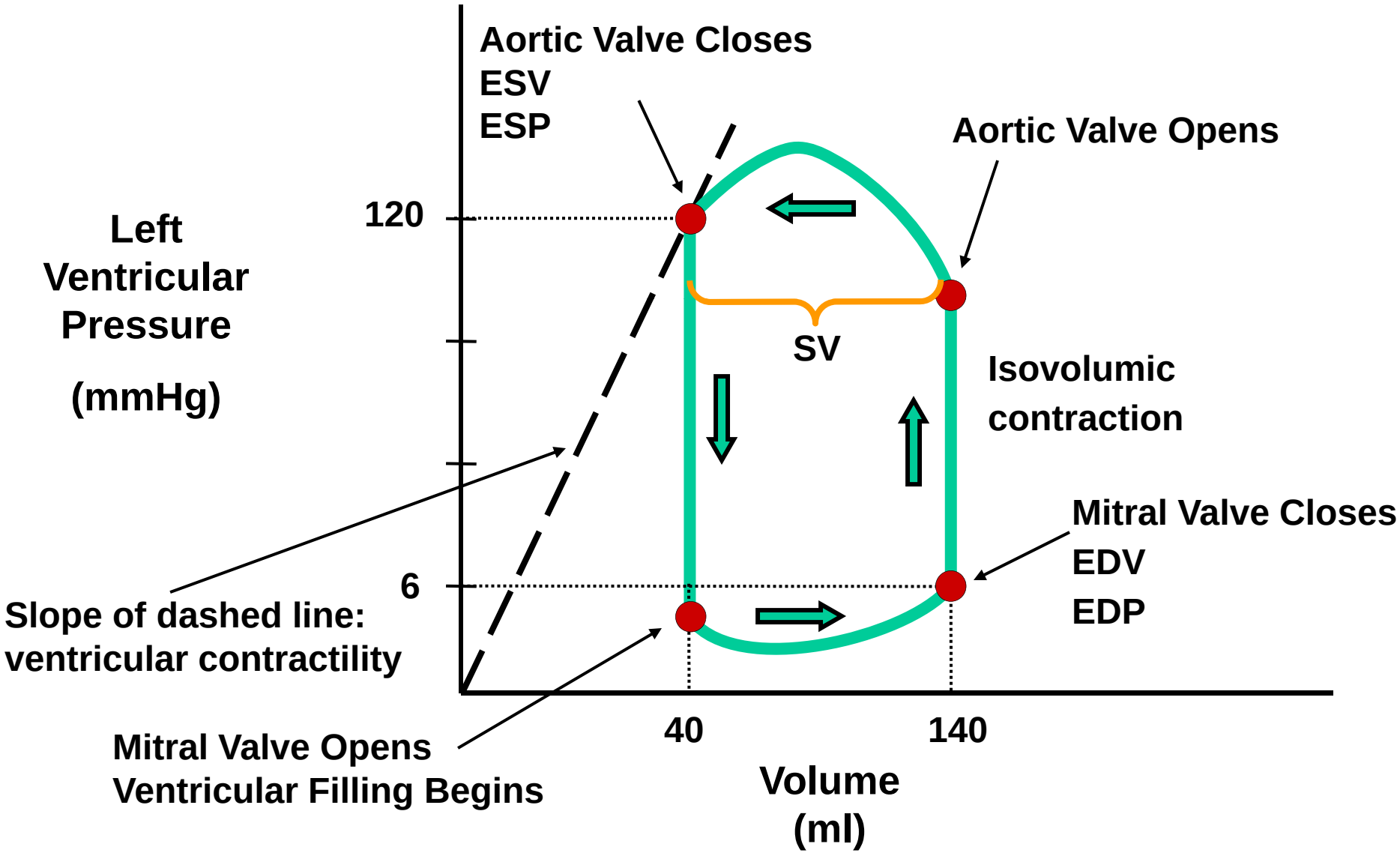
1. Normal point of operating system & normal cardiac output
2 Pump begins to fail → Q̇ falls below normal resting levels
3. Renin-angiotensin system activated → fluid retained → ↑ MCFP → ↑ Q̇

Changes in Cardiac & Vascular Function Curves with De-compensated Heart Failure

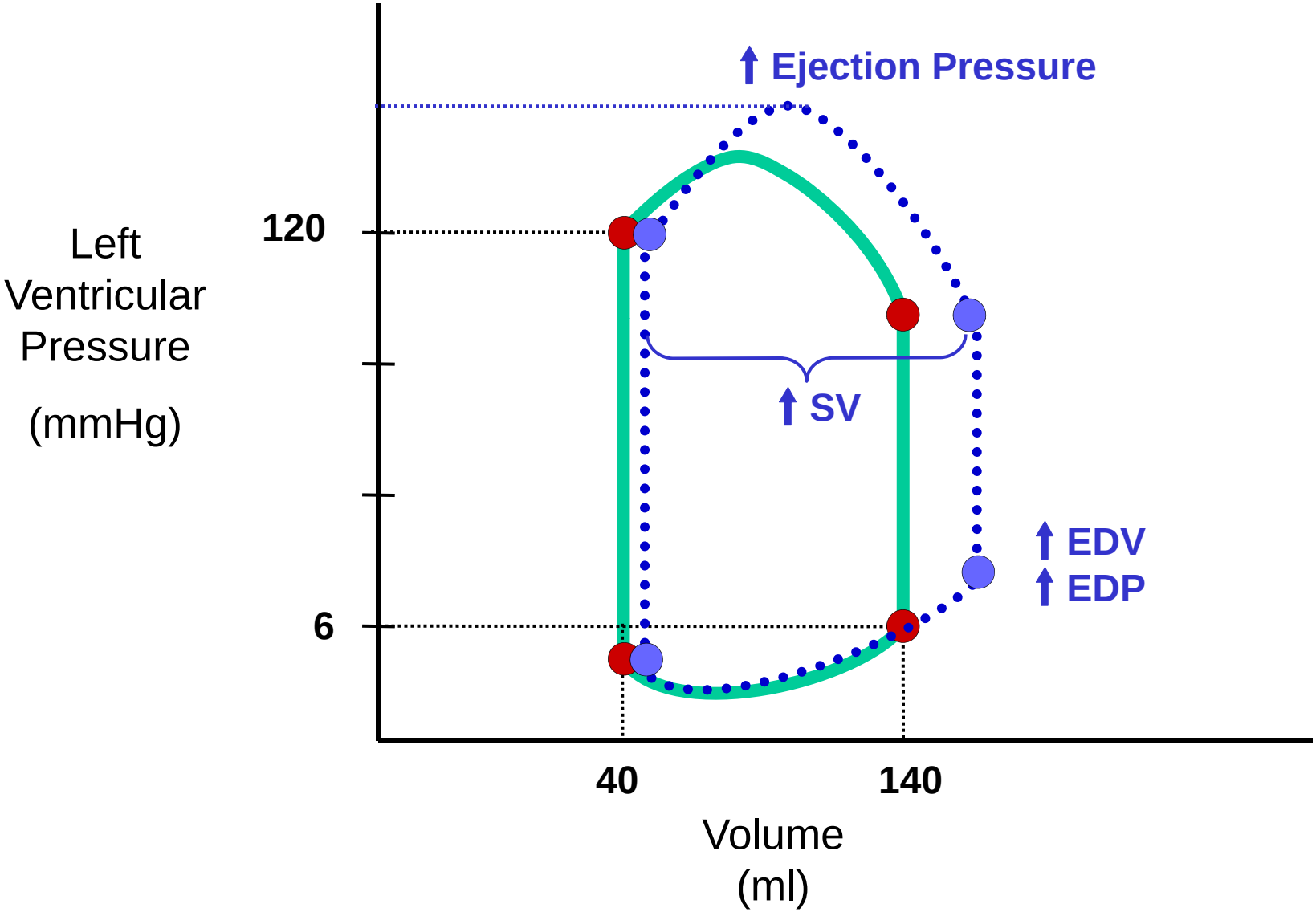


1. Normal point of operating system & normal cardiac output
2. Pump begins to fail → Q̇ falls below normal resting levels
3. Renin-angiotensin system activated → fluid retained → ↑ MCFP → ↑ Q̇
4. Pump decline continues and Q̇ falls once again
5. More fluid is retained to try and compensate, but now Q̇ is below a level where normal fluid balances can be maintained → → pattern continues

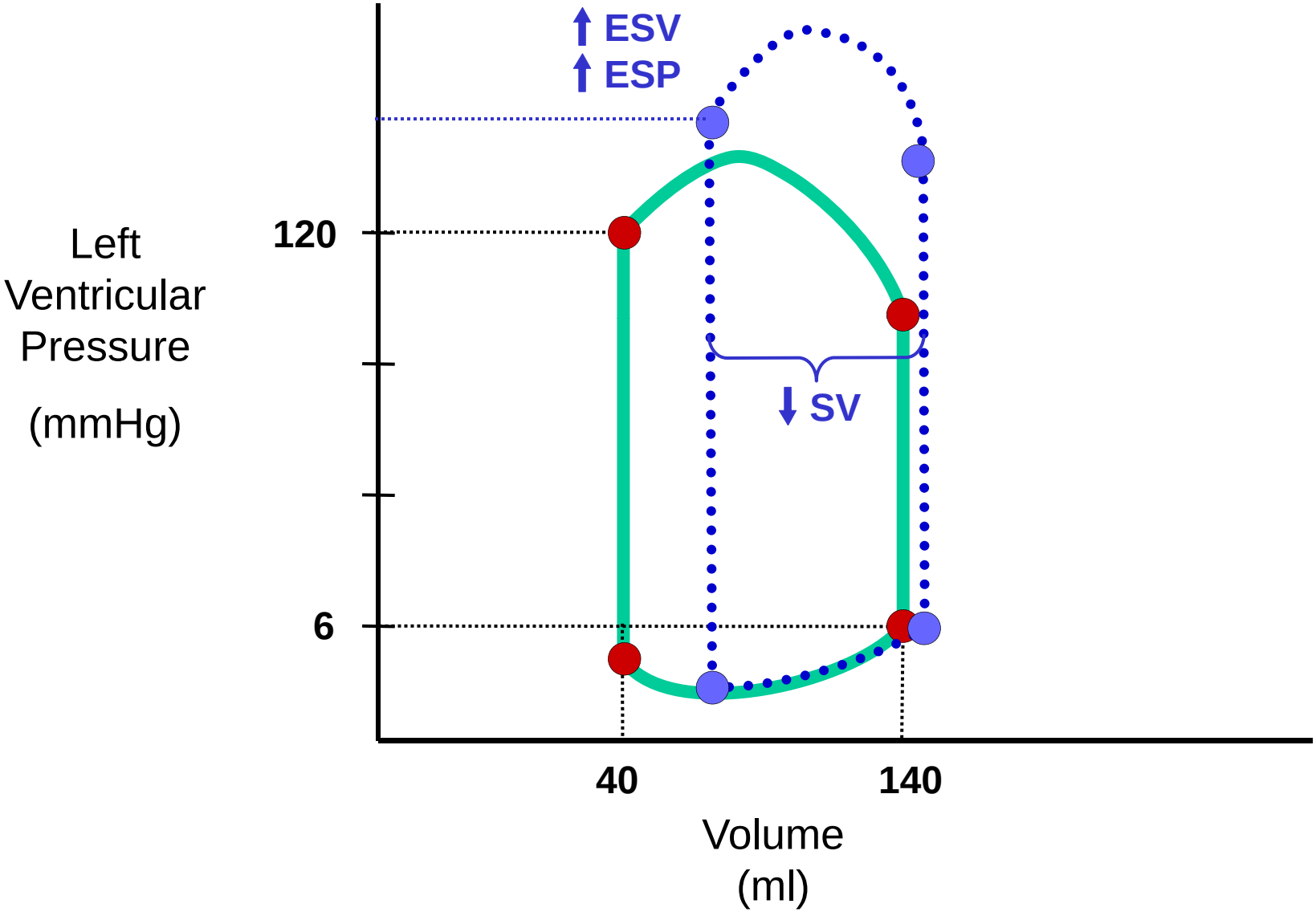
Left Ventricular Pressure Volume Loop



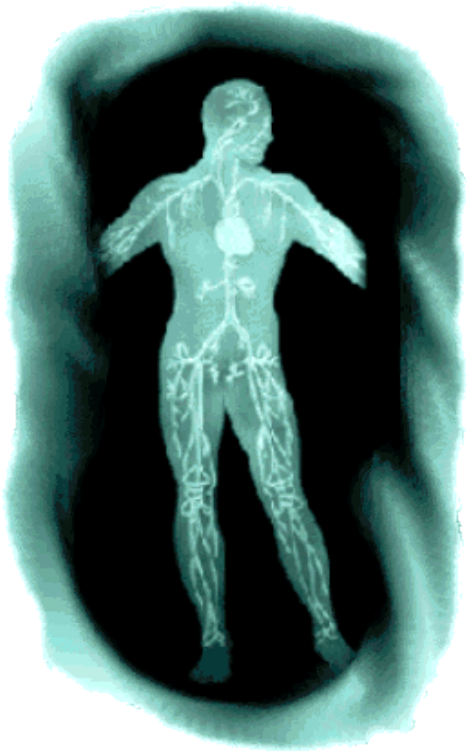
Effects of an Increase in Preload on Left Ventricular Pressure Volume Loop



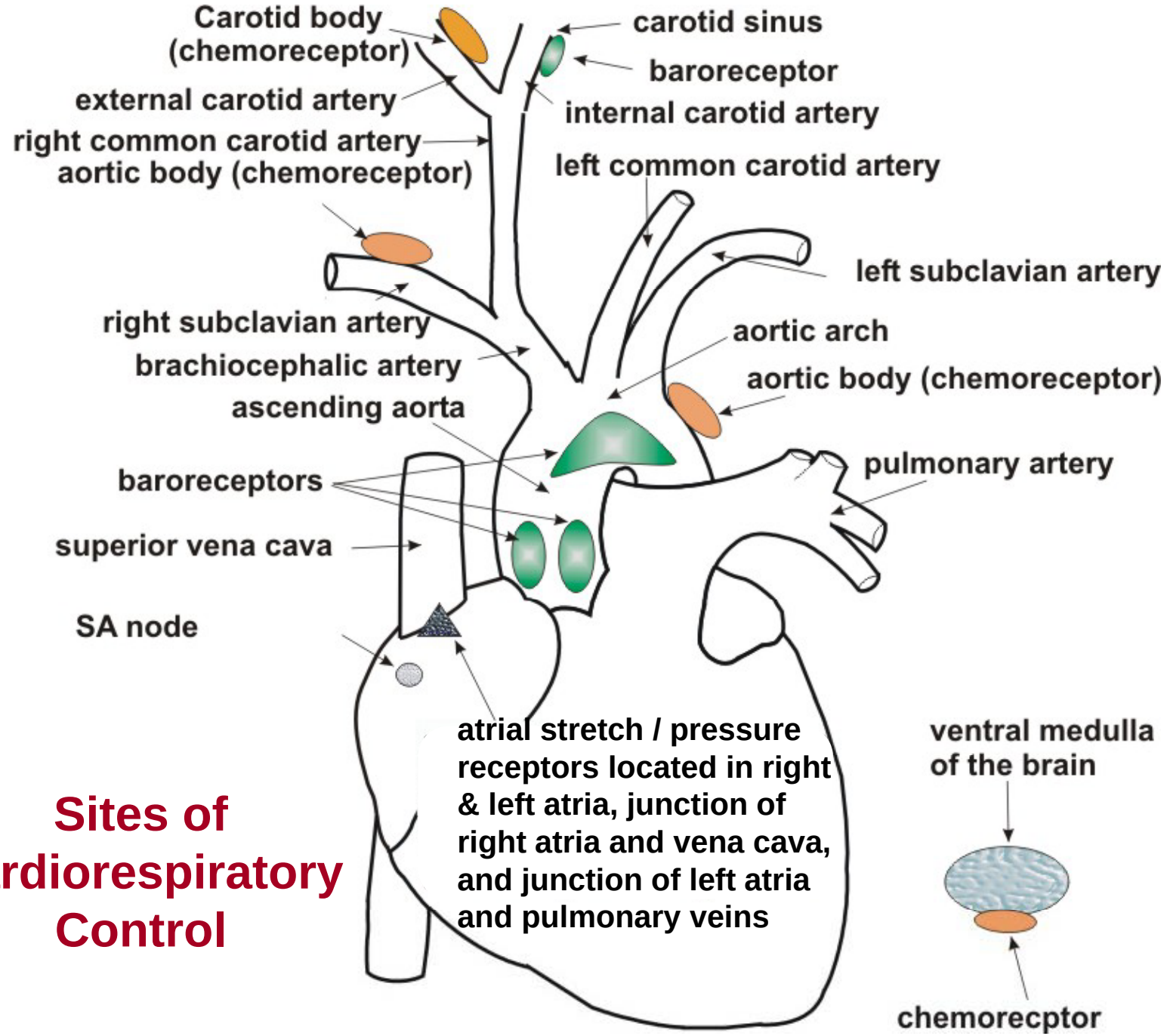
Effects of an Increase in Afterload on Left Ventricular Pressure Volume Loop



Mechanism of Control of Cardiovascular and Respiratory Systems



Sites of Cardiorespiratory Control

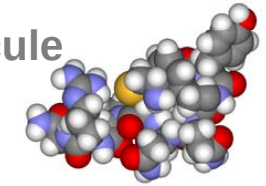


Cardiorespiratory Control

- **Heart Rate** – **Neurohormone (neurotransmitter) and CNS (medulla) regulation**

- **Parasympathetic vagus control** (Neurotransmitter: **Acetylcholine**)
  - Vagal control is dominant at rest – influence is withdrawn when exercise begins
- **Sympathetic cardioacceleration** (Neurotransmitters: **EPINEPHRINE & NOREPINEPHRINE**)
- **Baroreceptor influences**
  - Sympathetic discharge indirectly proportional to firing rate
  - Parasympathetic discharge is directly proportional to firing rate
  - ↓ pressure → ↓ receptor firing → ↑ sympathetics → ↑ HR → ↑ pressure
  - ↑ pressure → ↑ receptor firing → ↑ parasympathetics → ↓ HR → ↓ pressure

ADH Molecule



- **Atrial Stretch receptors:** ↑ receptor stretch → ↑ **ANP** → ↑ Na^+excretion → ↑ urine output
  ↓ receptor stretch → ↑ **ADH** → ↓ Na^+excretion → ↓ urine output
  - **A**trial **N**atriuretic **P**eptide released by myocytes in the atria → ↑ urine flow → ↓ BP
  - **A**niti-**D**iuretic-**H**ormone (vasopressin) released by pituitary → ↓ urine flow → ↑ BP
- **Chemoreceptor influences**
  - Main function: protect brain from poor perfusion
  - ↑ O_2 or ↓ CO_2 → ↑ parasympathetic discharge → ↓ HR
  - ↓ O_2 or ↑ CO_2 → ↓ pH → pressor area stimulation in medulla → ↑ HR

Cardiorespiratory Control

Stroke Volume (SV) – **regulated by Frank Starling mechanism**

- ↑ venous return → ↑ EDV → ↑ stroke volume

Cardiac Output ($\dot{Q}$) – main determinant: body O_2 needs

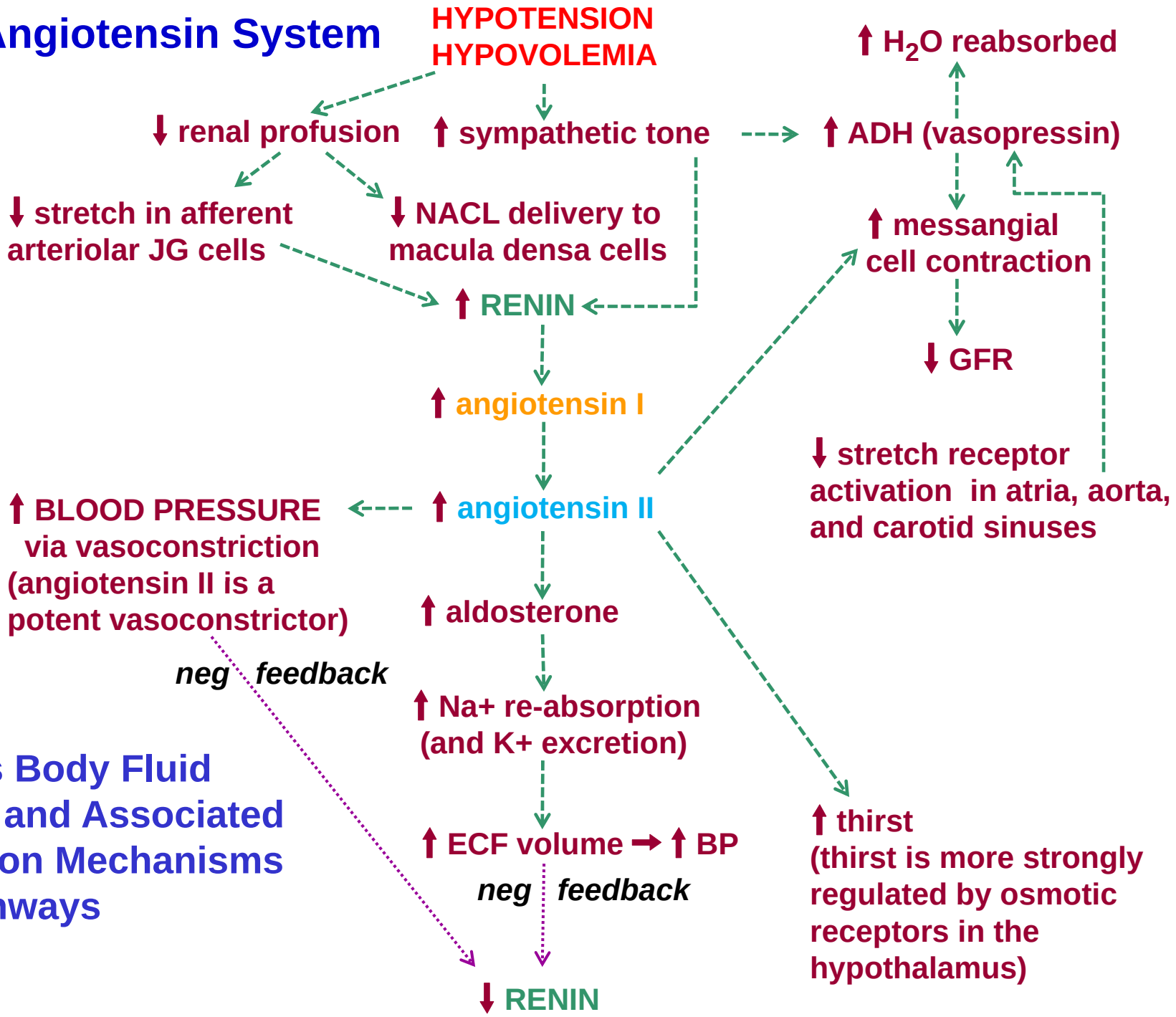
- Autoregulated by two distinct mechanisms
  - **Intrinsic changes in preload, afterload, and SV**
    - ↑ afterload → initial ↓ in $\dot{Q}$ → ↑ EDV (preload) → ↑ SV back to normal
  - **Extrinsic hormonal influences**
    - Norepinephrine release → ↑ HR and SV

Cardiorespiratory Control

Blood Pressure – influenced by 4 major factors (some interrelated)

- **Total peripheral resistance**
  - Baroreceptor (BR) and CNS Influences
    - ↑ BP → ↑ BR firing rate → vasodilation → ↓ BP
    - ↓ BP → ↓ BR firing rate → ↑ sympathetics → ↑ BP
- **Chemoreceptor influences**
  - ↓O_2, ↑ CO_2, ↓ pH → CNS stim. → vasoconstriction
  - Circulating catecholamine influences
    - E and NE have varying effects on TPR
    - E and NE usually activate α receptors → ↑ TPR
  - Fight or flight response
- **$\dot{Q}$**
- **Blood Volume**
  - Renin – Angiotensin System

Renin - Angiotensin System



Controls Body Fluid Balance and Associated Regulation Mechanisms and Pathways

Dehydration

- **Dehydration: the loss of body water and associated electrolytes**
- **Causes:**
  - Gastroenteritis (viral / bacterial infection → vomiting & diarrhea) - most common
  - Diseases: yellow fever, cholera,
  - **Excessive alcohol consumption**
    - The excess fluid is flushed out by the kidneys: ↑ water usage → **dehydration**
    - Most liquors have **congeners** which are toxic to body → removal necessary
      - The clearer & better quality your liquor (vodka & gin) the less congeners
        - more distillation cycles → better quality
    - When you drink, head vessels dilate….constriction next morning → headache
    - Congener removal done by liver: ↓ liver glucose → hypoglycemia & lethargy
  - Prolonged exercise without fluid replacement (heat exhaustion & heat stroke risk)
  - Diabetes: hyperglycemia → ↑ glucose excretion → ↑ water loss → dehydration
  - Shock: blood loss due to some hypotensive state caused by injury or disease
    - Gastrointestinal blood loss: bleeding from ulcers or colorectal cancer

Dehydration

- **Signs & Symptoms of dehydration:**
  - Dry mouth, dry swollen tongue, rapid heart rate (possible chest palpitations)
  - Lethargy (sluggishness), confusion
  - Poor skin turgor (a pinch of skin does not spring back into position)
    - Good test for ailing elderly folks
  - Elevated BUN (renal function test): NH_4 metabolized in liver & excreted by kidneys
  - Elevated creatinine → ↓ GFR (kidney clearance of waste products)
  - Increased blood viscosity
  - Headache
  - Fluid loss → low blood pressure → dizziness upon standing up
  - A high urinary specific gravity (comparison of density to water: 1 gram / cm^2)
- **Treating Dehydration**
  - Sip small amounts of water
  - Drink carbohydrate / electrolyte solutions: Gatorade, Pedialyte, etc.
  - If core body temperature > 104^0 + ↓ BP or ↑ HR → consider IV fluid replacement

Cardiorespiratory Control

Skeletal Muscle Blood Flow – autoregulated – 2 mechanisms

- **Mechanism 1: Vasodilator Metabolites**
  - Usually overrides adrenergic neurohormone control
  - Mediated by vasodilator metabolite (VDM) buildup & removal
    - Adenosine (ATP by-product), CO_2, H^+, prostaglandins
    - **Exercise Example** – **(negative feedback control)**
      - Muscle exercises → VDM's released → ↑ vasodilation
      - ↑ vasodilation ↑ blood flow → VDM's removed → vasoconstriction

- **Mechanism 2: Myogenic response**
  - Involves stretch activated Ca^{++} channels **(negative feedback control)**
    - ↑ blood flow → vessel stretch → Ca^{++} channel activation
    - ↑ [Ca^{++}] in smooth muscle → vasoconstriction → ↓ flow

Cardiorespiratory Control

Systemic Blood Flow During Exercise: Autonomic influences

- **Sympathetic outflow & circulating catecholamines**
  - α activation → vasoconstriction in non - exercising tissue

- **Redistribution of blood flow during maximal exercise**

- NC in brain blood flow	- 500 ml/min ↑ to heart
- 11,300 ml/min ↑ to muscle	- 400 ml/min ↑ to skin
- 500 ml/min ↓ to kidneys	- 800 ml/min ↓ to viscera

- 200 ml/min ↓ to various other parts of the body

Cardiorespiratory Control

Respiration: Minute Ventilation ($\dot{V}_E$) = Tidal Volume X Respiratory Rate

- **Generally Controlled via central chemoreceptors in the medulla-pons respiratory center**

- Peripheral chemoreceptors
  - ↑ blood CO_2 content → receptor activation → ↑ $\dot{V}_E$
  - ↓ blood O_2 content → receptor activation → ↑ $\dot{V}_E$

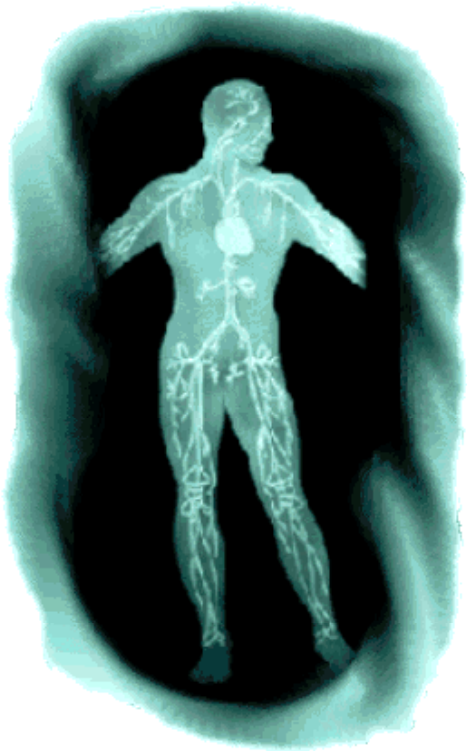
- Central chemoreceptors in the medulla respiratory center – **Dominant Influence**
  - ↑ blood CO_2 & lactate → receptor activation → ↑ $\dot{V}_E$
  - P_aCO_2 → ↑ HCO_3^- + H^+ → H^+ activates receptor → ↑ $\dot{V}_E$

- Respiratory control during exercise – no consensus but research suggests:
  - Muscle spindle & proprioceptor activation → ↑ $\dot{V}_E$ at early onset of an exercise bout
  - Respiratory centers (medulla) sends afferent signals to expiratory muscles during exercise
  - ↑ venous return → atrial receptor activation → ↑ $\dot{V}_E$??
  - Intrapulmonary receptor activation → ↑ $\dot{V}_E$??
  - Peripheral chemoreceptors may play a role in steady state & high intensity exercise $\dot{V}_E$??

- Minute ventilation mechanistic changes during an ↑ in exercise intensity
  - Low exercise intensity: $\dot{V}_E$ ↑ by both ↑ TV and ↑ RR
  - High exercise intensity: $\dot{V}_E$ ↑ by ↑ RR only

- Notes:
- O_2 cost of breathing during exercise: 4.5% $\dot{V}O_2$ (low int.) up to 12.5%$\dot{V}O_2$ (high int.)

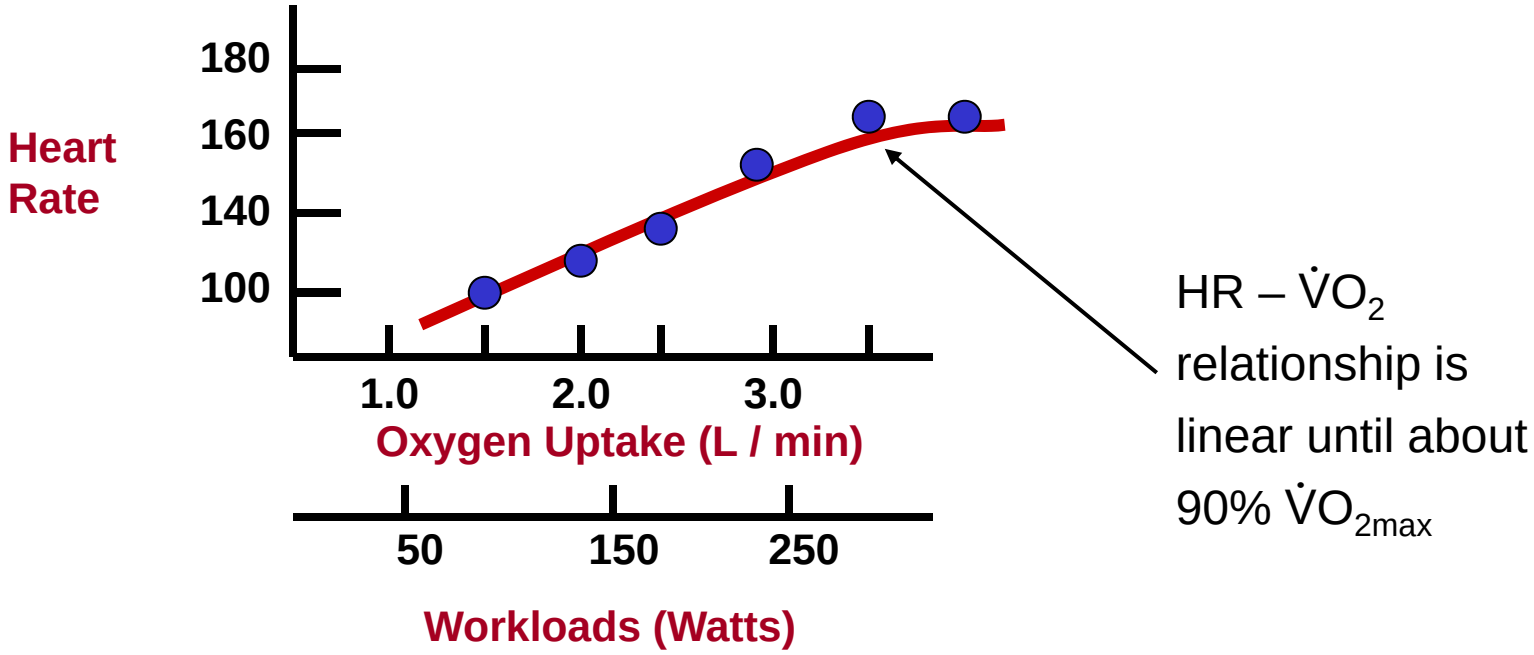
Acute Cardiorespiratory Responses to Endurance Exercise



Acute Responses to Aerobic Exercise

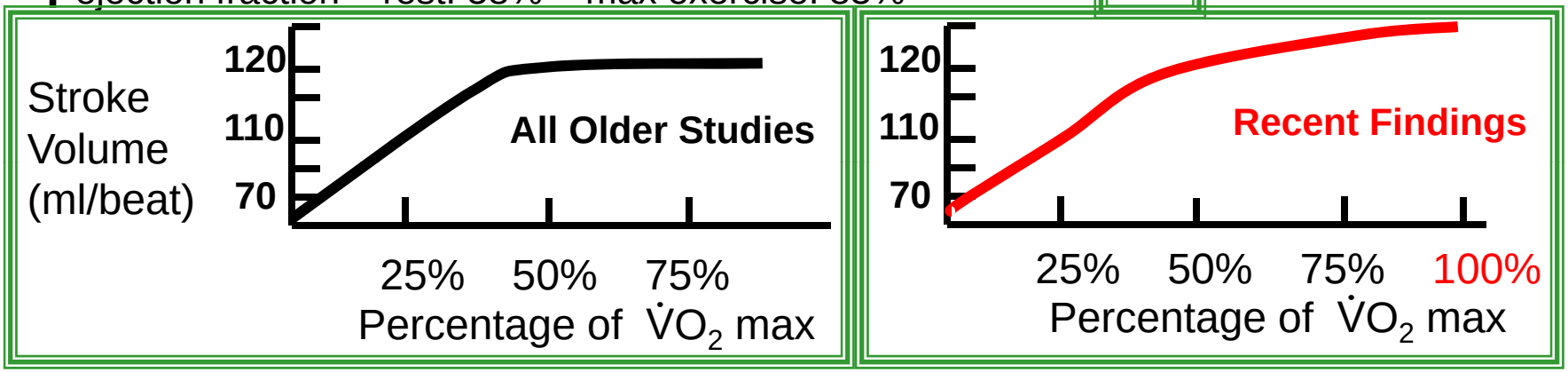
- **Oxygen Consumption ($\dot{V}O_2$)**
  - **↑ VO_2 in direct proportion to ↑ workload (power requirement of exercise)**
  - Expressed in both relative and absolute terms
    - Relative: ml O_2/kg/min Absolute: ml/min or L/min
    - Average VO_{2max} for 40 year old male 37 ml/kg/min
  - Oxygen consumption linked to caloric expenditure (1 liter of O_2 consumed = 5 kcal)

- **Heart Rate**
  - ↑ up to 3 times resting value at peak exercise (mainly due to ↓ time spent in diastole)



Acute Responses to Aerobic Exercise

- **Stroke Volume**
  - ↑ up to 1.5 resting value at peak exercise
    - Increase levels off at 40% - 50% $\dot{V}O_2$ max **??**
  - ↑ in venous return → ↑ EDV (Starling mechanism)
  - ↓ ESV eluding to an ↑ in myocardial contractility
  - ↑ ejection fraction rest: 58% max exercise: 83%



- **Cardiac Output ($\dot{Q}$)**
  - ↑ up to 4 times resting value at peak exercise (↑ is rapid at onset, then levels off)
  - ↑ $\dot{Q}$ → ↑ venous return
    - Venous return mediated by and related to:
      - Sympathetic venoconstriction
      - Muscle pump
      - ↑ inspiration → ↓ thoracic pressure
        - Blood flows to an area of reduced pressure
      - ↑ inspiration → ↑ abdominal pressure
        - Contraction of abdominal muscles → squeezing of abdominal veins

Acute Responses to Aerobic Exercise

- **Arteriovenous oxygen difference**
  - Difference in [O_2] between arterial and mixed venous blood
  - Illustrated by the oxyhemoglobin desaturation curve
  - ↑ approximately 3 fold from rest to max exercise
  - At rest, about 25% of arterial O_2 is extracted
  - At peak exercise about 75% - 85% of arterial O_2 is extracted
- **Blood Pressures and Resistance to Flow**
  - SBP: ↑ - failure to ↑ signifies heart failure
  - DBP: slight ↑ or slight ↓ or NC
  - MAP: slight ↑
  - TPR: ↓ - mainly due to vasodilation in exercising muscle
- **Coronary (Myocardial) Blood Flow**
  - 4.5% of $\dot{Q}$ goes to myocardium at rest and at peak exercise
    - This increase is due to ↑ MAP and CA vasodilation
- **Blood Flow to the Skin**
  - ↑ as exercise duration ↑ to allow for heat dissipation
  - ↓ at max exercise to meet exercising muscle demands
  - ↑ during exercise recovery, again for heat dissipation

Acute Responses to Aerobic Exercise

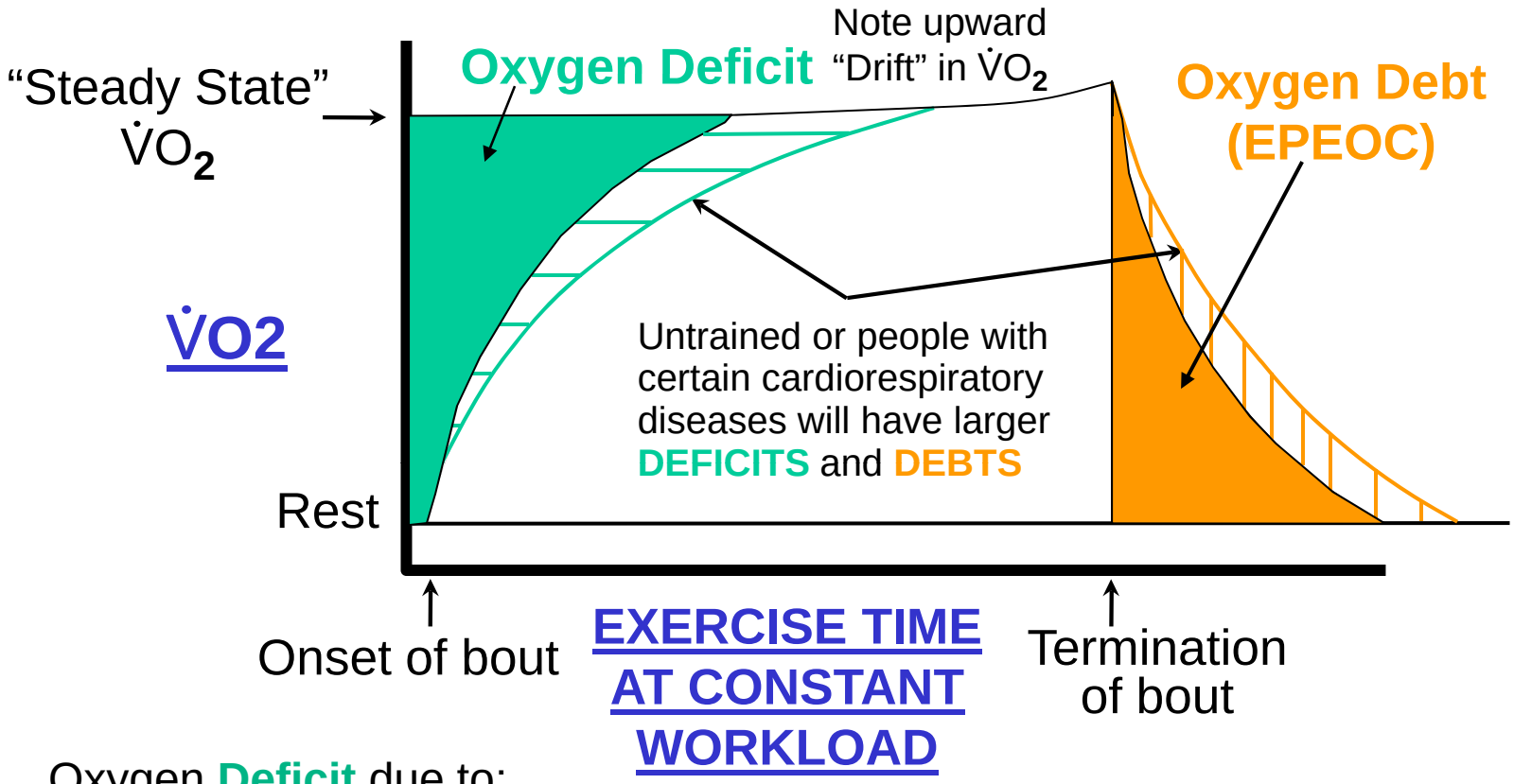
- **Minute Ventilation**
  - Resting average: 6 Liters/min
  - Peak exercise average: 175 Liters/min (29 fold increase from rest to max)
  - Respiratory rate: resting 12-18 peak exercise: 45-60
  - Tidal volume: resting .5 liters peak exercise: 2.25 Liters

- **Plasma Volume**
  - Blood plasma ↑ in the interstitium of exercising muscle
  - Fluid shift results in a 5% ↓ in plasma volume
    - This is termed “Hemoconcentration”
  - Blood viscosity increases

Acute Responses to Aerobic Exercise

- **Immune system**
  - During moderate / vigorous exercise, the following changes occur in immune activity
    - Transient ↑ in the re-circulation of neutrophils, NKC's, and immunoglobulins
      - More pathogens detected and killed
    - Transient ↓ in stress hormones (cortisol) and inflammatory mediators (cytokines)
      - Cortisol and cytokines suppress the immune system
  - Immune function returns to normal in a few hours, but exercise improves "surveillance"
  - 25% - 50% reduction in sick days with upper respiratory infections (colds, flu, etc.)
  - Opposite effect occurs with prolonged heavy exercise
    - Example: after marathon, immune function ↓ 2 - 6 fold depending on time of year

Oxygen Debt and Deficit

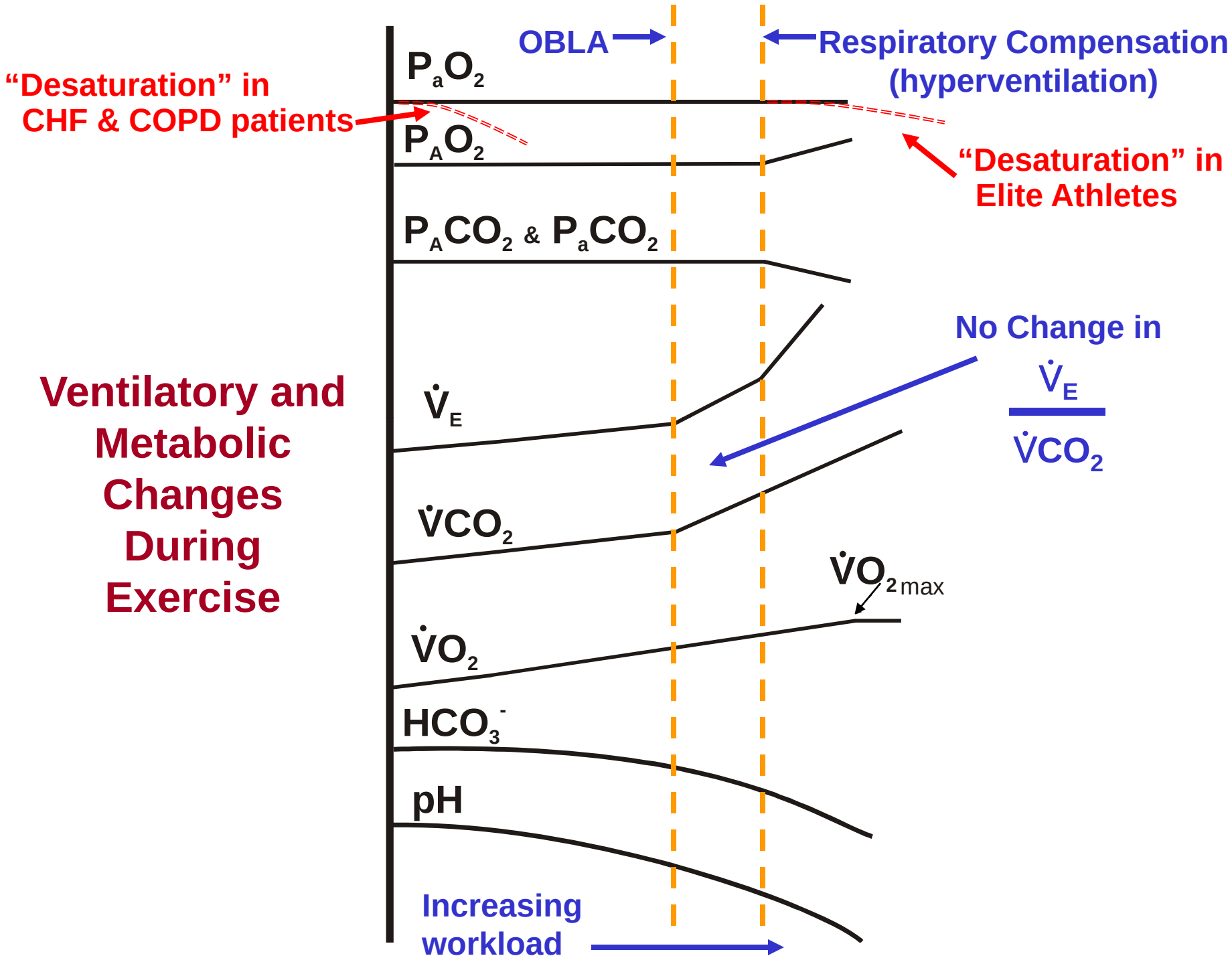


Oxygen **Deficit** due to:

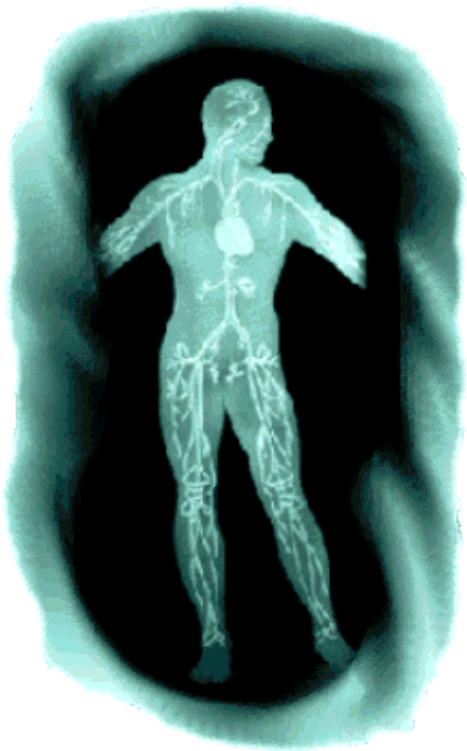
- Delay in time for aerobic ATP production to supply energy

Oxygen **Debt** due to:

- Resynthesis of high energy phoshosphates (CP, ATP)
- Replace oxygen stores
- Lactate conversion to glucose (gluconeogenesis)
- ↑ HR, respiration, catecholamines, body temperature


Ventilatory and Metabolic Changes During Exercise
"Desaturation" in CHF & COPD patients
OBLA
Respiratory Compensation (hyperventilation)
"Desaturation" in Elite Athletes
P_aO_2
P_AO_2
P_ACO_2 & P_aCO_2
No Change in $\frac{\dot{V}_E}{\dot{V}CO_2}$
$\dot{V}_E$
$\dot{V}CO_2$
$\dot{V}O_{2\,max}$
$\dot{V}O_2$
HCO_3^-
pH
Increasing workload

Training Adaptations to Chronic Endurance Exercise



Effects of Exercise Training on the Components of the Fick Relationship

- **Resting**

NC ↓ ↑ NC

$\dot{V}O_2$ = HR x SV x AVO_2diff

due to:
- ↑ time in diastole

due to:
- ↑ preload
- ↓ afterload (small)
- ↑ ventricle size
- ↑ blood volume

- **Submax Workload (measured at same pre-training workload)**

NC ↓ ↑ NC

$\dot{V}O_2$ = HR x SV x AVO_2diff

note: a slight ↓ in afterload (mentioned above) accompanied by a ↓ in HR translates into a reduction myocardial $\dot{V}O_2$ at rest or at any submaximal workload

- **Max Workload (measured at peak exercise)**

↑ NC ↑ ↑

$\dot{V}O_2$ = HR x SV x AVO_2diff

some studies show a slight decrease

Training Adaptations

- **Mean Arterial Pressure**
  - Small ↓ at rest or during exercise
- **Systolic and Diastolic Blood Pressure**
  - Small ↓(6 – 10 mmHg) at rest
  - Larger ↓ (10 – 12 mmHg) at submaximal workload
    - Exercise: first line of therapy for borderline hypertensives
    - Some studies report a mean ↓ of about 9 mmHg
- **Total Peripheral Resistance and Afterload**
  - ↑ capillarization (more parallel circuits) → ↓ Transit time for blood
  - ↓ TPR → ↓ Afterload
- **Respiratory Variables**
  - Respiratory Rate
    - Rest: NC
    - Submax exercise: ↓ slightly
      - Air remains in lungs longer
      - More O_2 extracted (about 2%)
    - Max exercise: ↑
  - Tidal Volume
    - Rest: NC
    - Submax exercise: ↑↑ significantly
    - Max exercise: ↑

↑ $\dot{V}_E$ during submax & max exercise

↓ $\dot{V}_E$ / $\dot{V}O_2$ during submax exercise

- **Anaerobic Threshold or OBLA or Ventilatory Threshold**
  - **Occurs at a higher percentage of $\dot{V}O_2$ max**
  - **Pre-training: 50% VO_2max Post-training: 80% VO_2max**

Training Adaptations

- **Mitochondria**
  - ↑ number, size and membrane surface area
- **Aerobic Enzymes in Exercising Muscle**
  - ↑ Krebs cycle enzymes (succinate dehydrogenase)
  - ↑ β oxidation enzymes (carnitine acyltransferase)
  - ↑ electron transport enzymes (cytochrome oxydase)
- **Fatty Acid & Glycogen Utilization**
  - ↑ utilization of β oxidative pathways to produce ATP
  - Called the "glycogen sparring effect"
  - ↓ RER for any given submaximal workload
  - ↑ muscle glycogen stores (with high carbohydrate diet)
- **No Appreciable Change in Resting Metabolic Rate**

  Exception: training induced ↑ in lean muscle mass

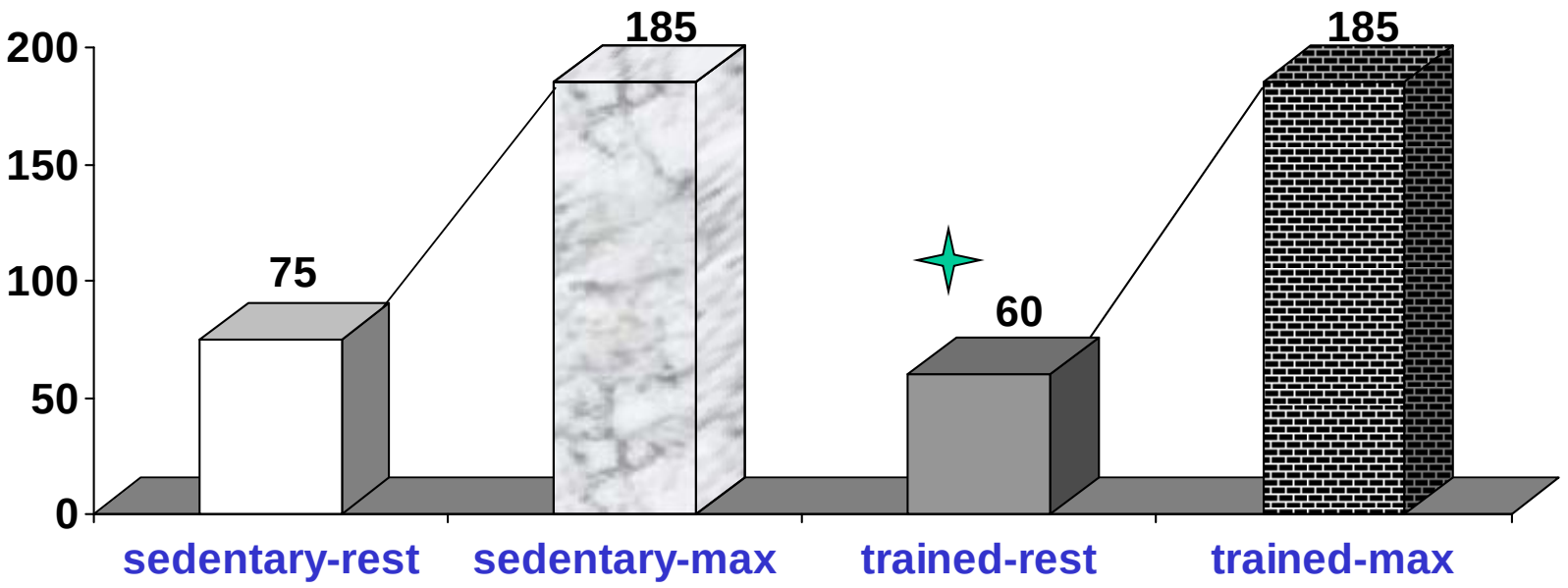
Death from **all causes** increases significantly when VO2max falls below 7.9 METS (27.65 ml/kg/min)

Kodama's Meta Analysis, JAMA, 301, 19, 2009.

- **↓ Platelet Aggregation**
- **↑ Fibrinolytic Activity**
- **↓ Circulating Catecholamines**
  - ↑ vagal tone → ↓ risk of arrhythmia
- **↑ Resistance to Pathological Events**
  - Smaller infarct size and quicker recovery
  - Less of a ↓ in ventricular function during ischemia

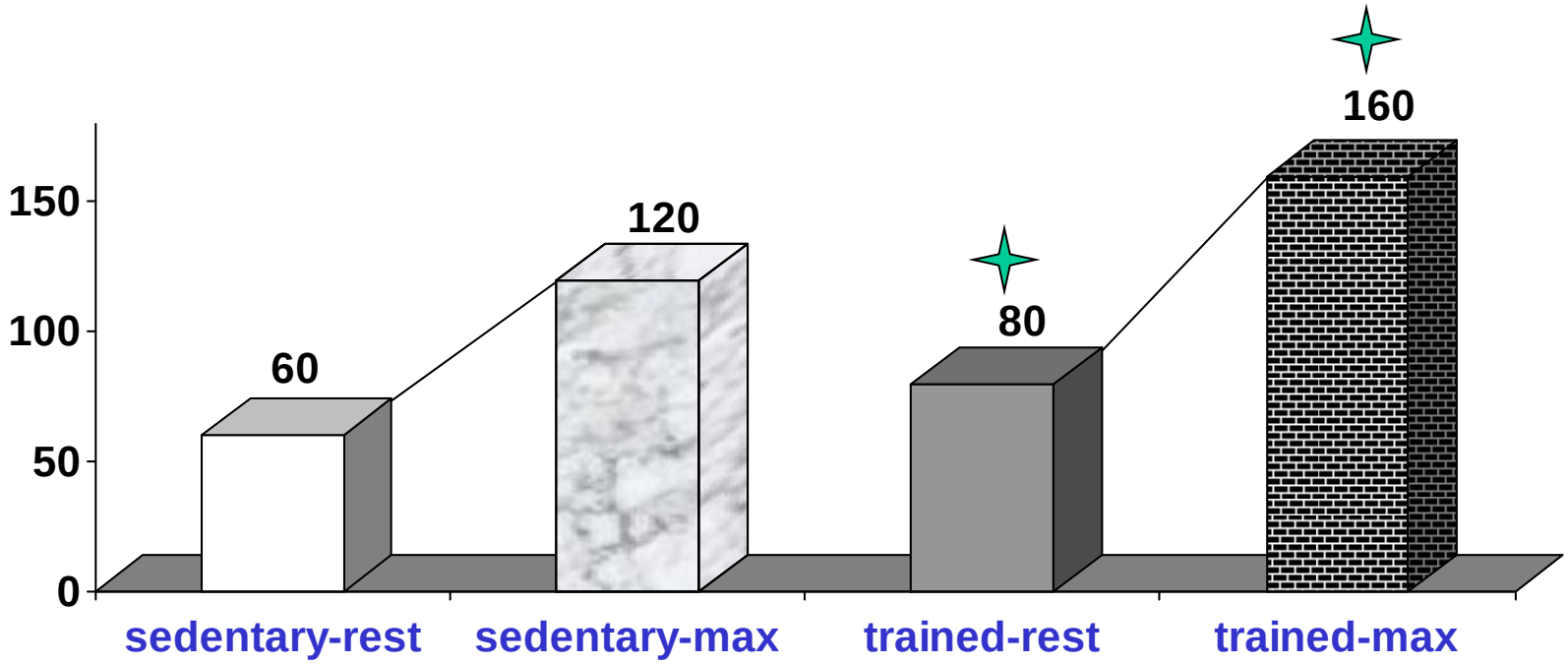
"Average" Values for Sedentary and Trained Individuals

Heart Rate
(beats / minute)



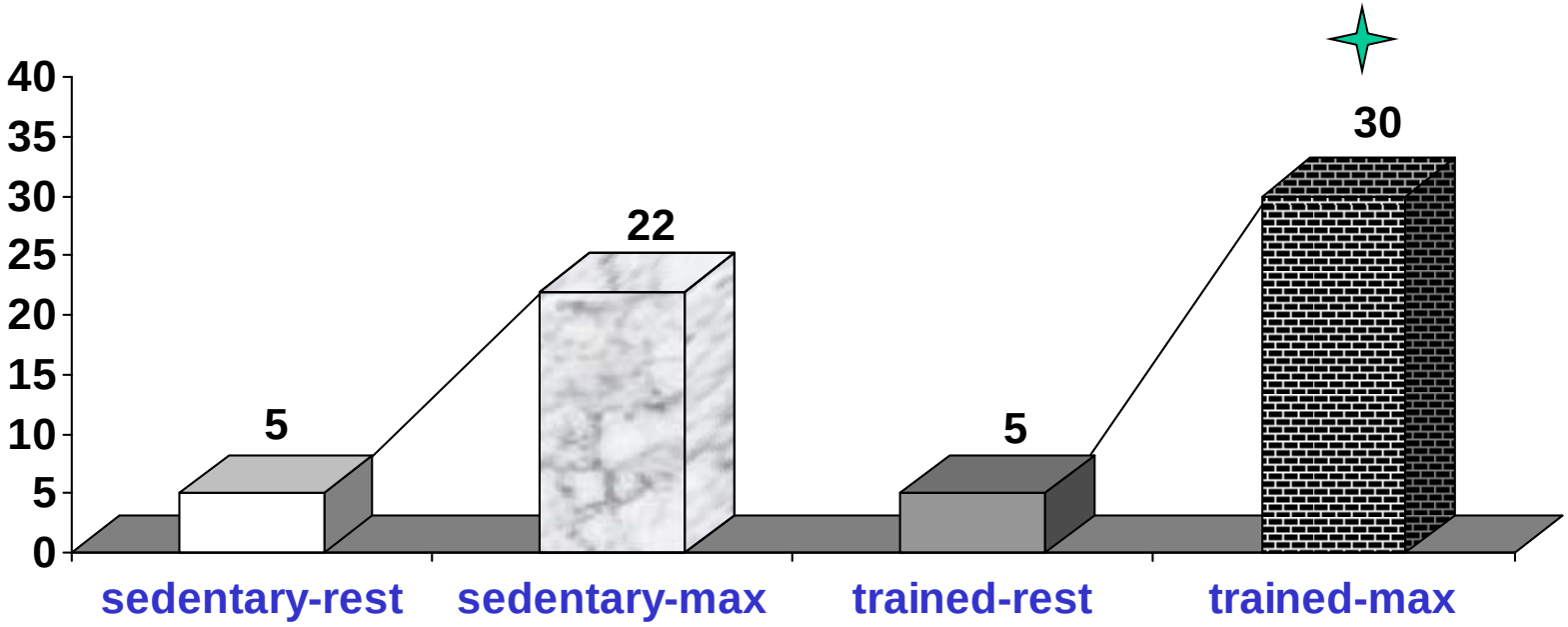
"Average" Values for Sedentary and Trained Individuals

Stroke Volume
(ml / beat)

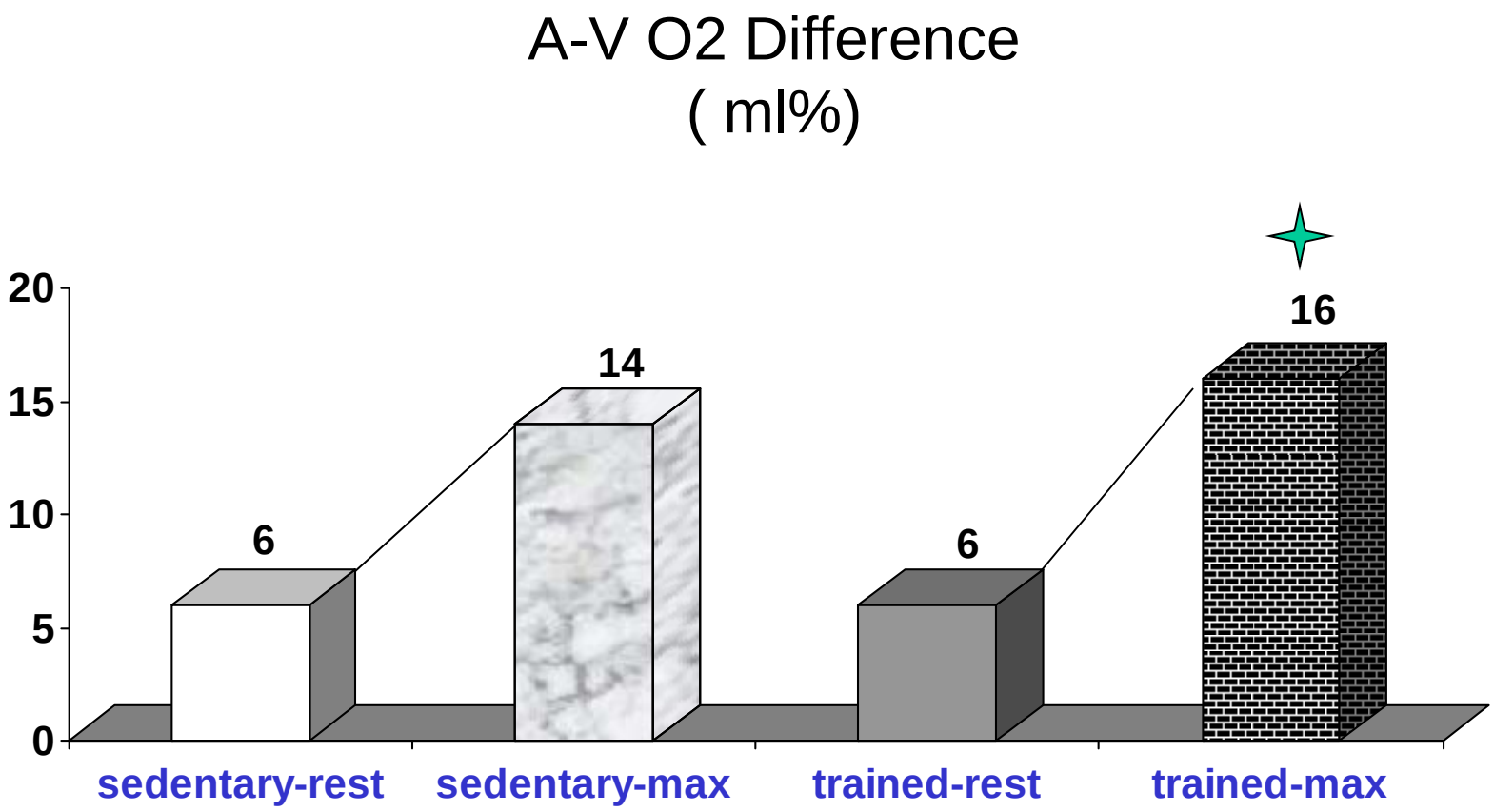


"Average" Values for Sedentary and Trained Individuals

Cardiac Output
(liters / minute)

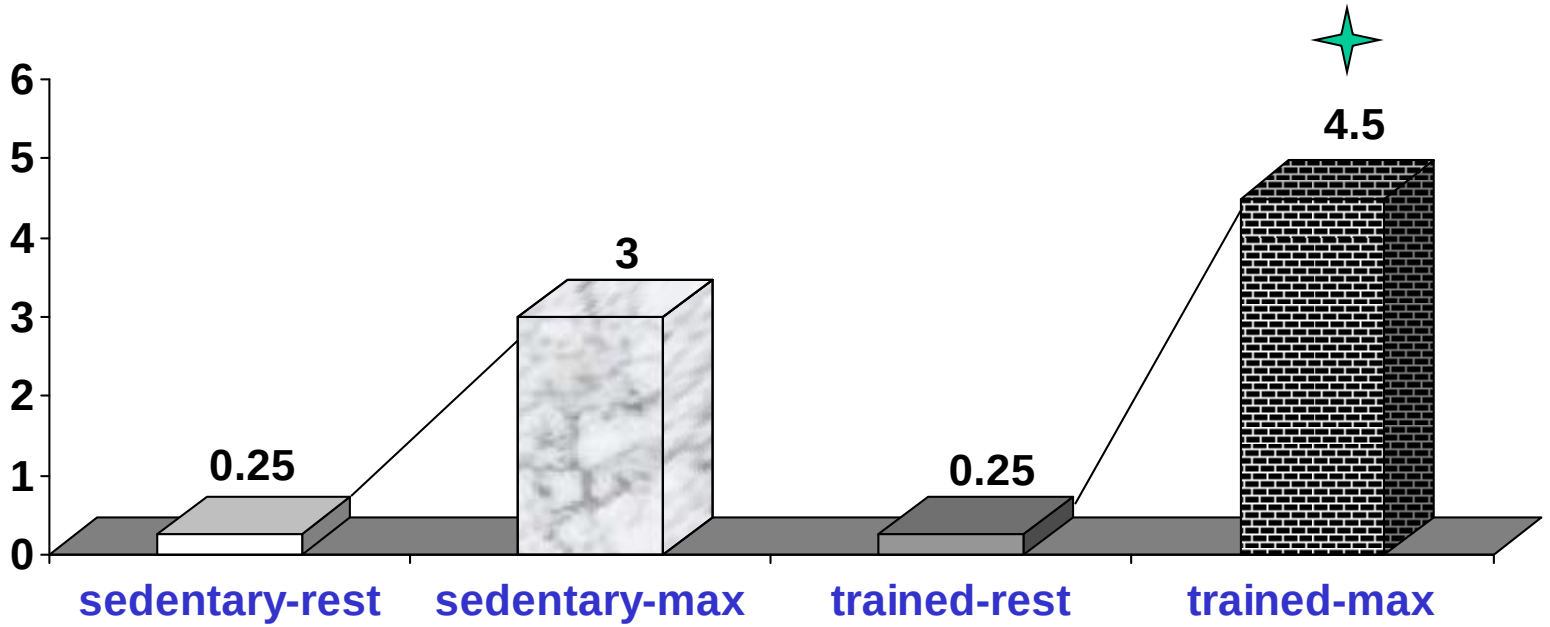


"Average" Values for Sedentary and Trained Individuals



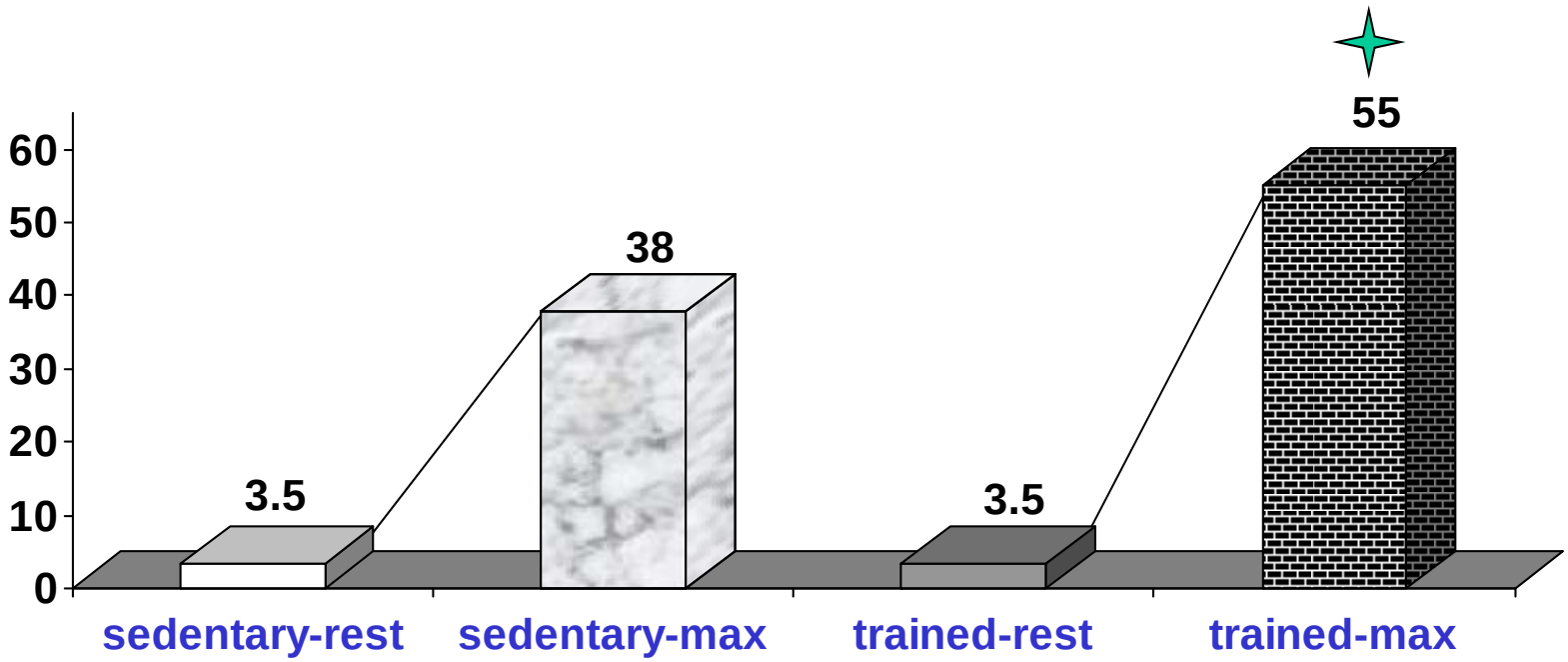
"Average" Values for Sedentary and Trained Individuals

Oxygen Consumption
(liters / minute)



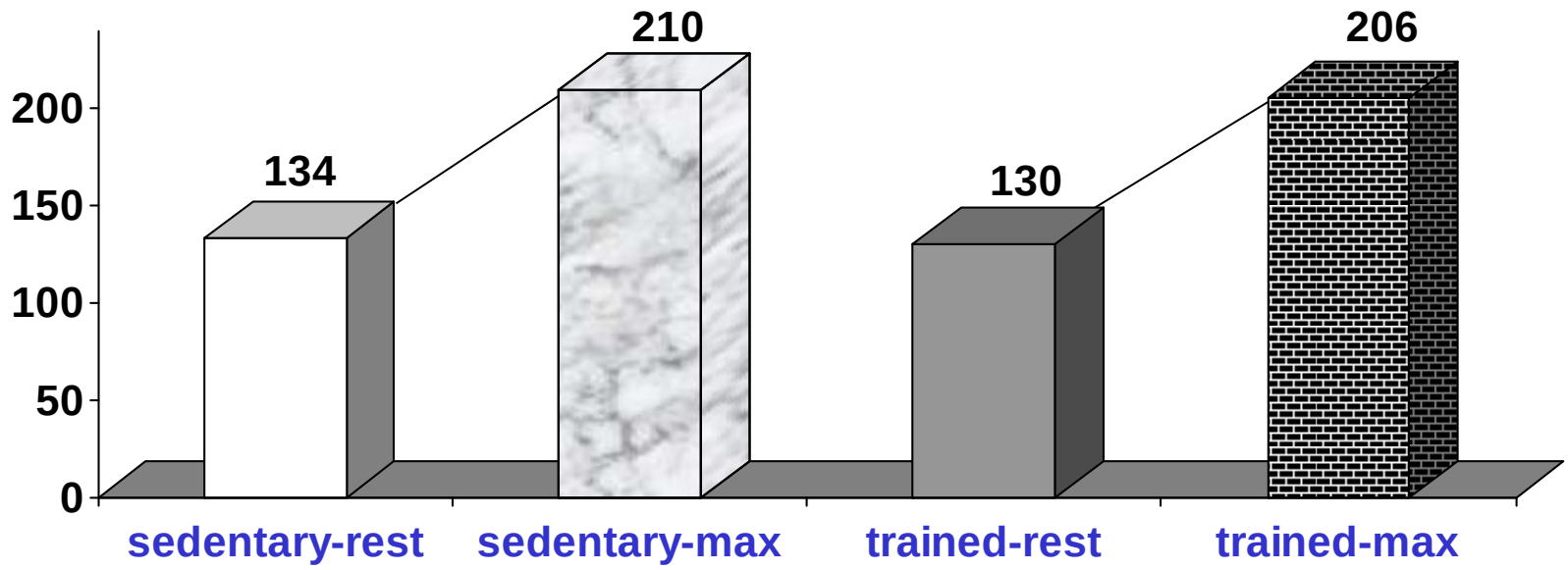
"Average" Values for Sedentary and Trained Individuals

Oxygen Consumption
(ml / kg / minute)



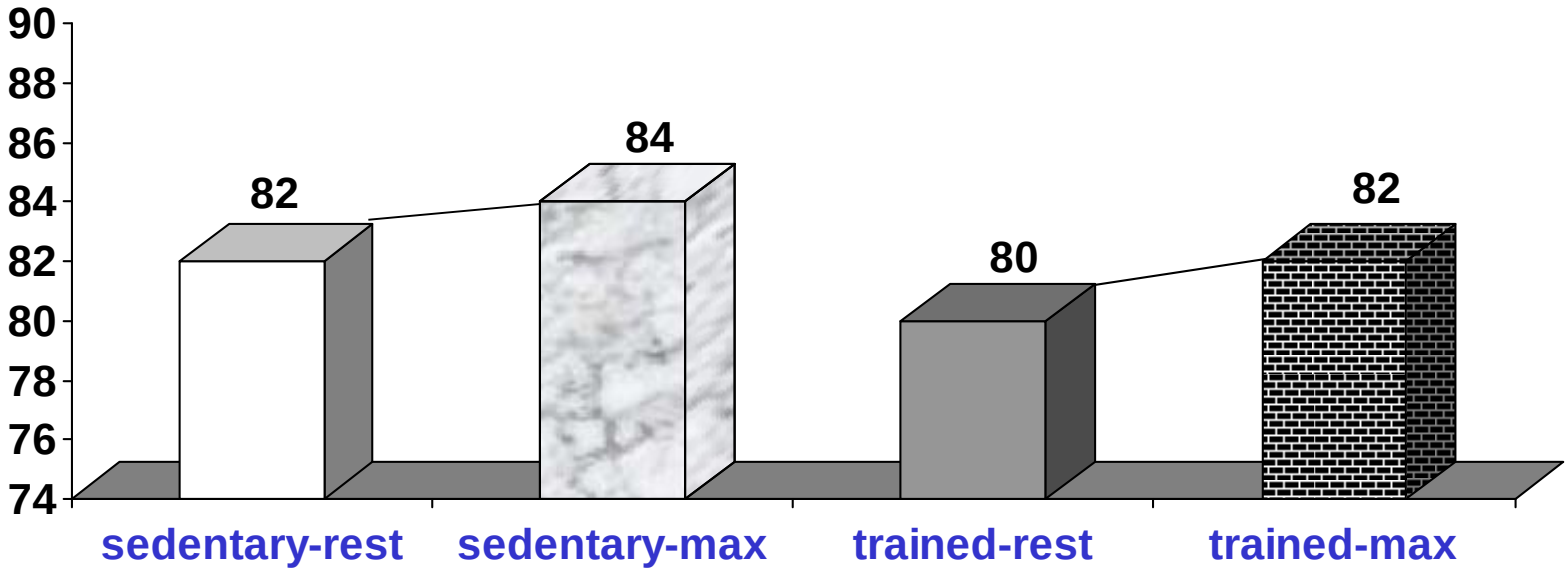
"Average" Values for Sedentary and Trained Individuals

Systolic Blood Pressure
(mm Hg)



"Average" Values for Sedentary and Trained Individuals

Diastolic Blood Pressure
(mm Hg)



"Average" Values for Sedentary and Trained Individuals