Lec 12 Circulatory Systems III

Animal Physiology

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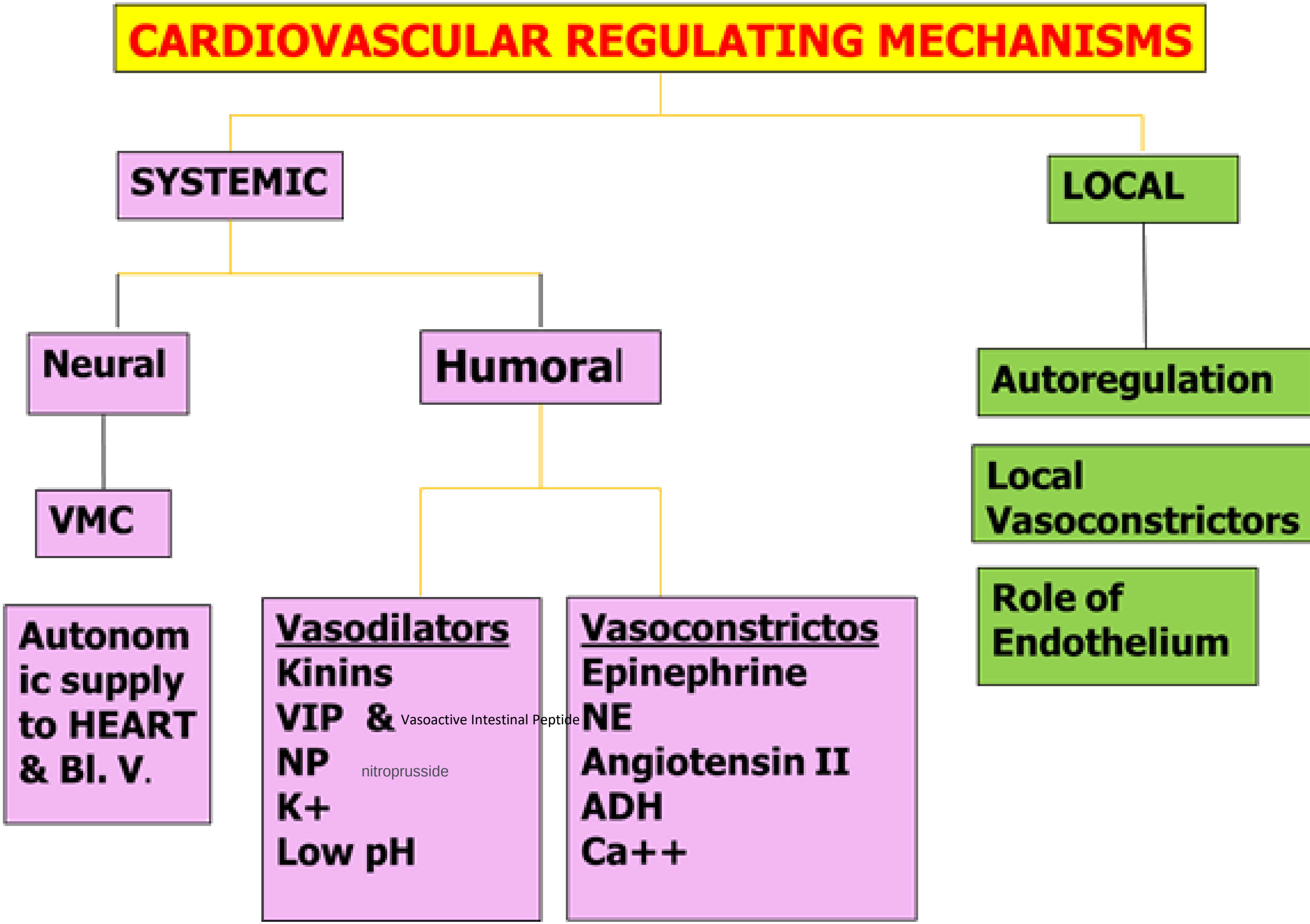
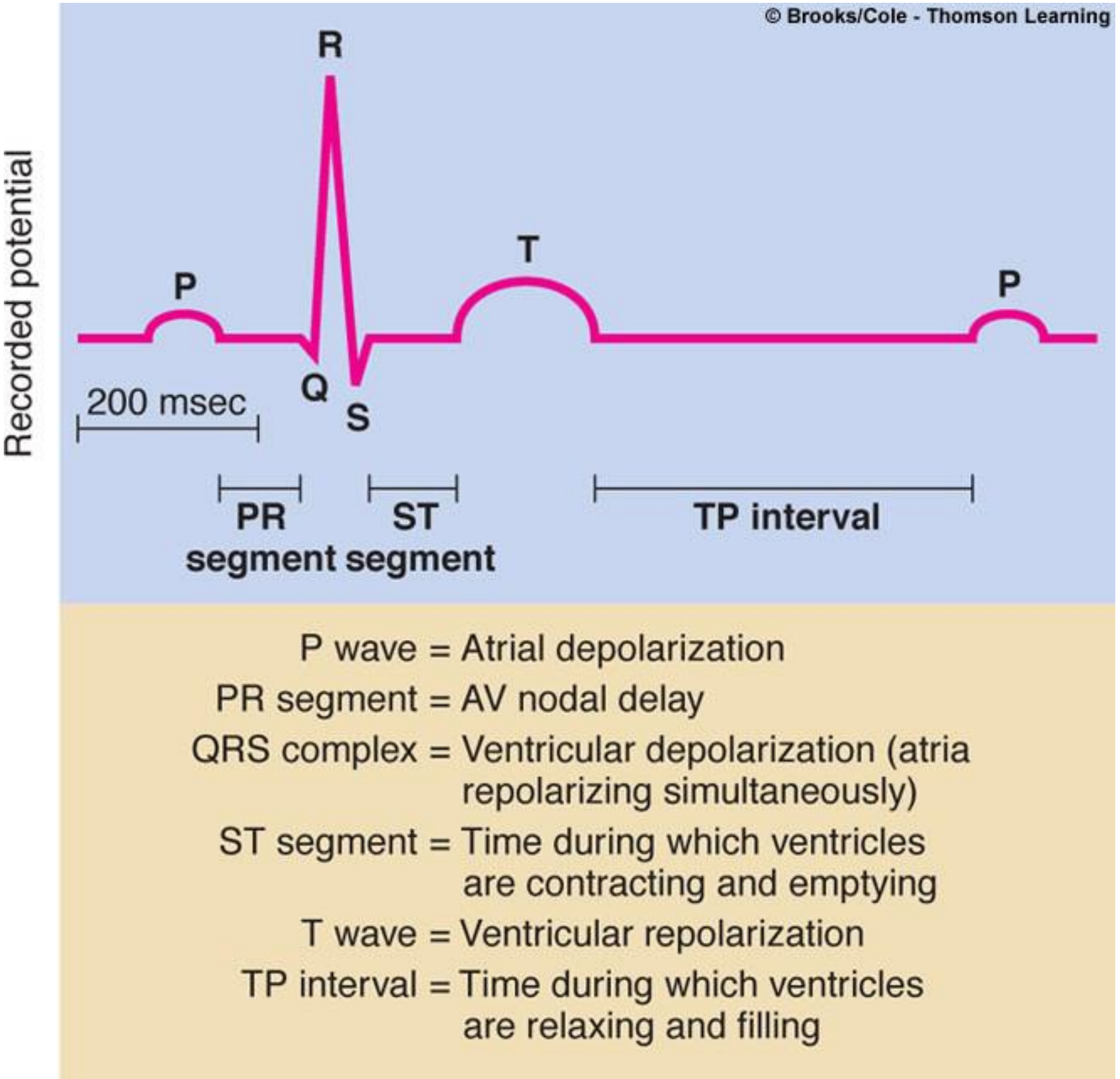
3rd Biology

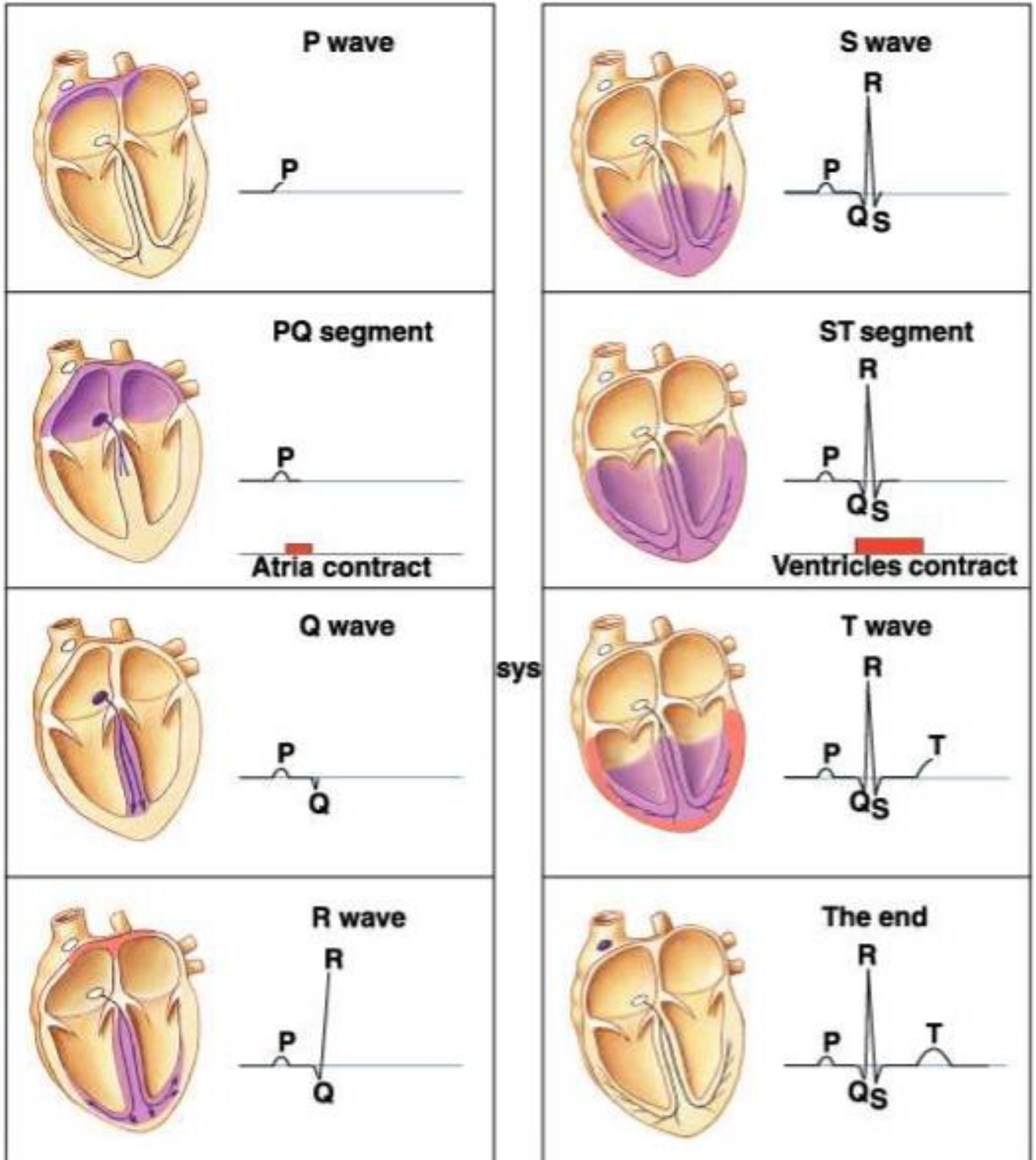

CARDIOVASCULAR REGULATING MECHANISMS
SYSTEMIC
LOCAL
Neural
Humoral
Autoregulation
VMC
Local Vasoconstrictors
Role of Endothelium
Autonomic supply to HEART & Bl. V.
Vasodilators
Kinins
VIP &
Vasoactive Intestinal Peptide
NP
nitroprusside
K+
Low pH
Vasoconstrictos
Epinephrine
NE
Angiotensin II
ADH
Ca++

Electrocardiogram (ECG)

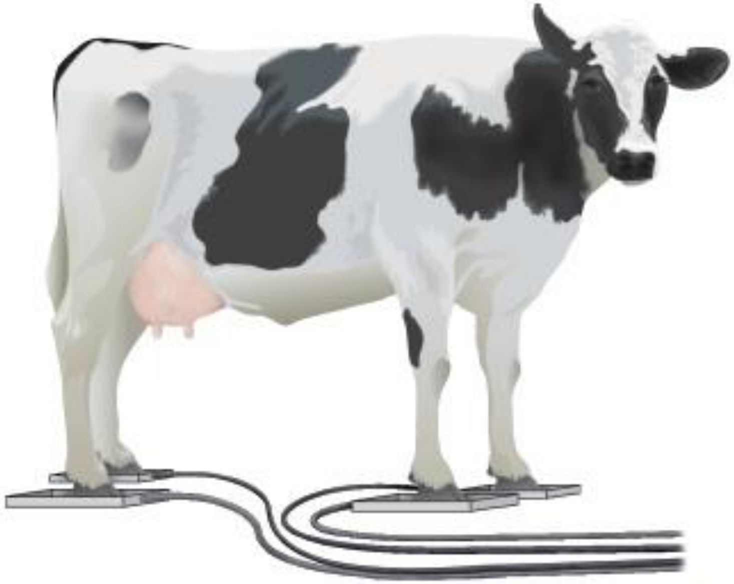
- Different parts of ECG record can be correlated to specific cardiac events

- is a diagnostic tool that is routinely used to **assess** the **electrical** and **muscular** functions of the heart from different **angles** to identify and locate pathology.
- The ECG machine records electrical impulses coming from human body

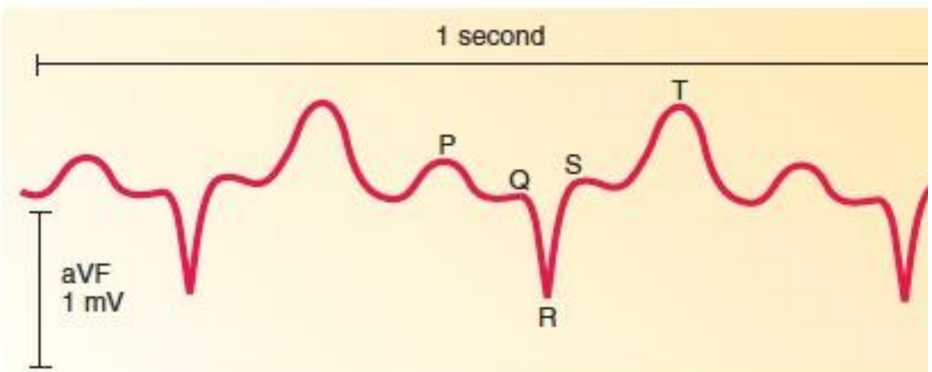




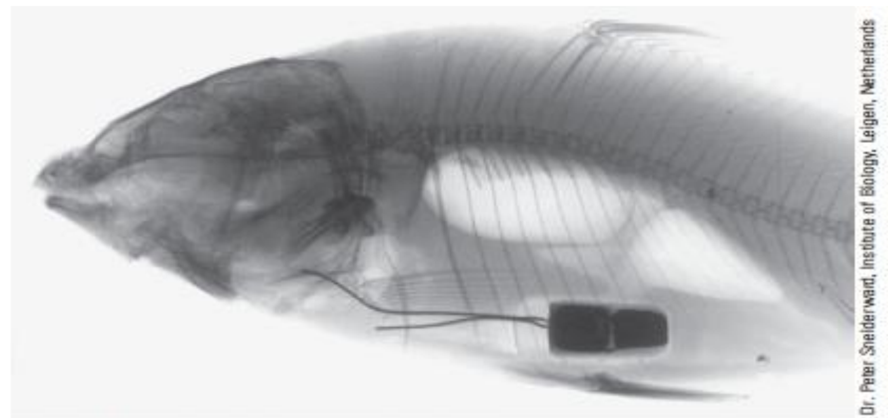
P wave
P
PQ segment
P
Atria contract
Q wave
P
Q
R wave
R
P
Q
S wave
R
P
Q
S
ST segment
R
P
Q
S
Ventricles contract
sys
T wave
R
P
T
Q
S
The end
R
P
T
Q
S



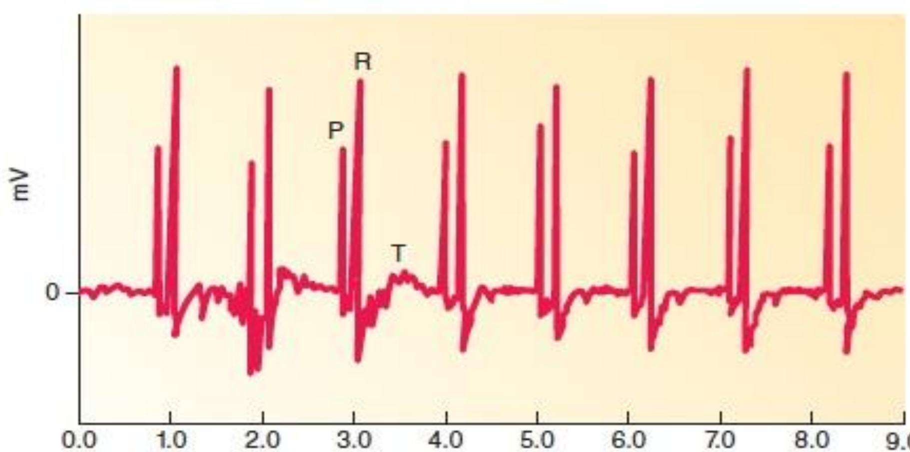
(a)



(b)



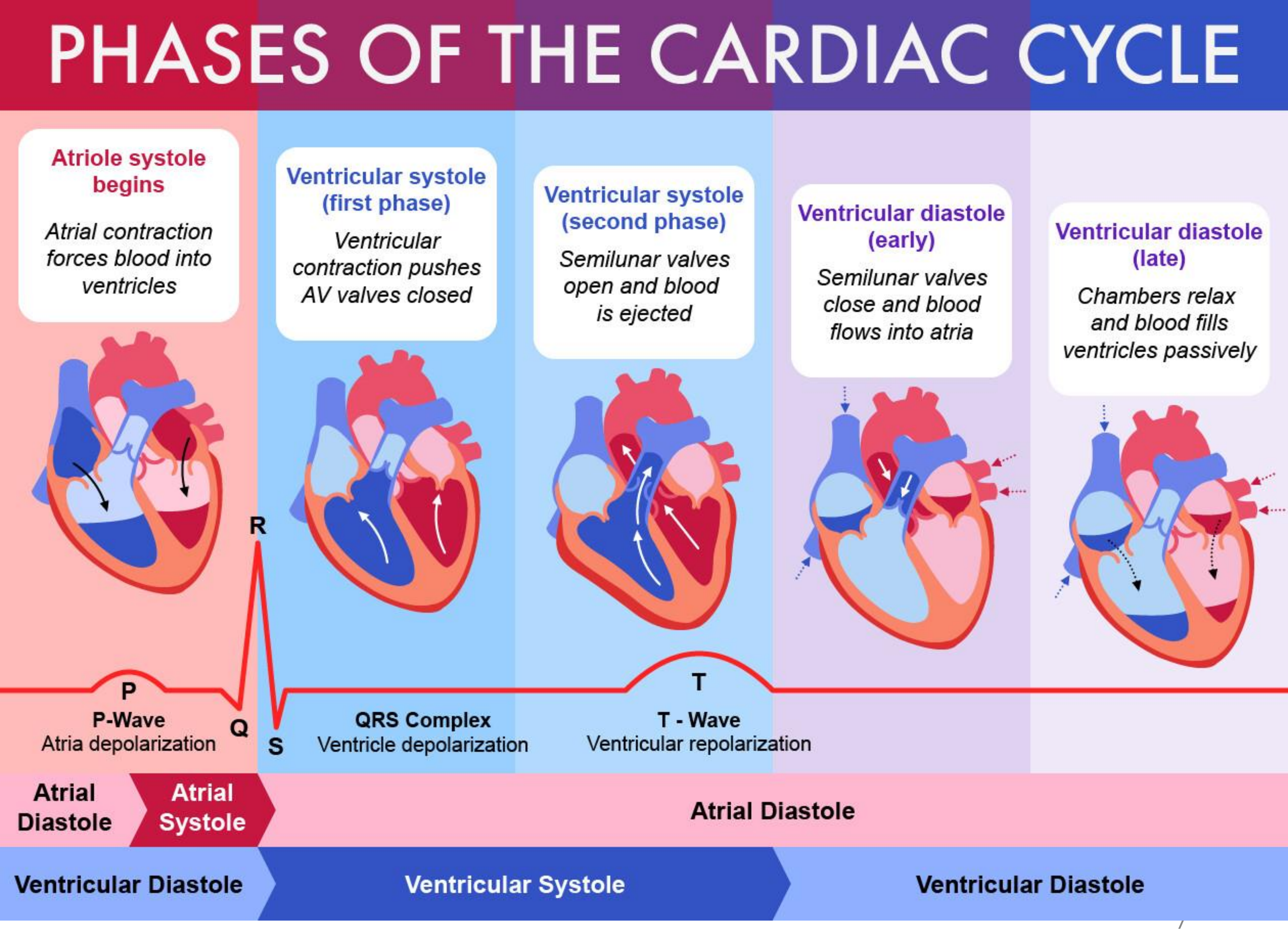
(c) Goldfish with implanted sensor for ECG

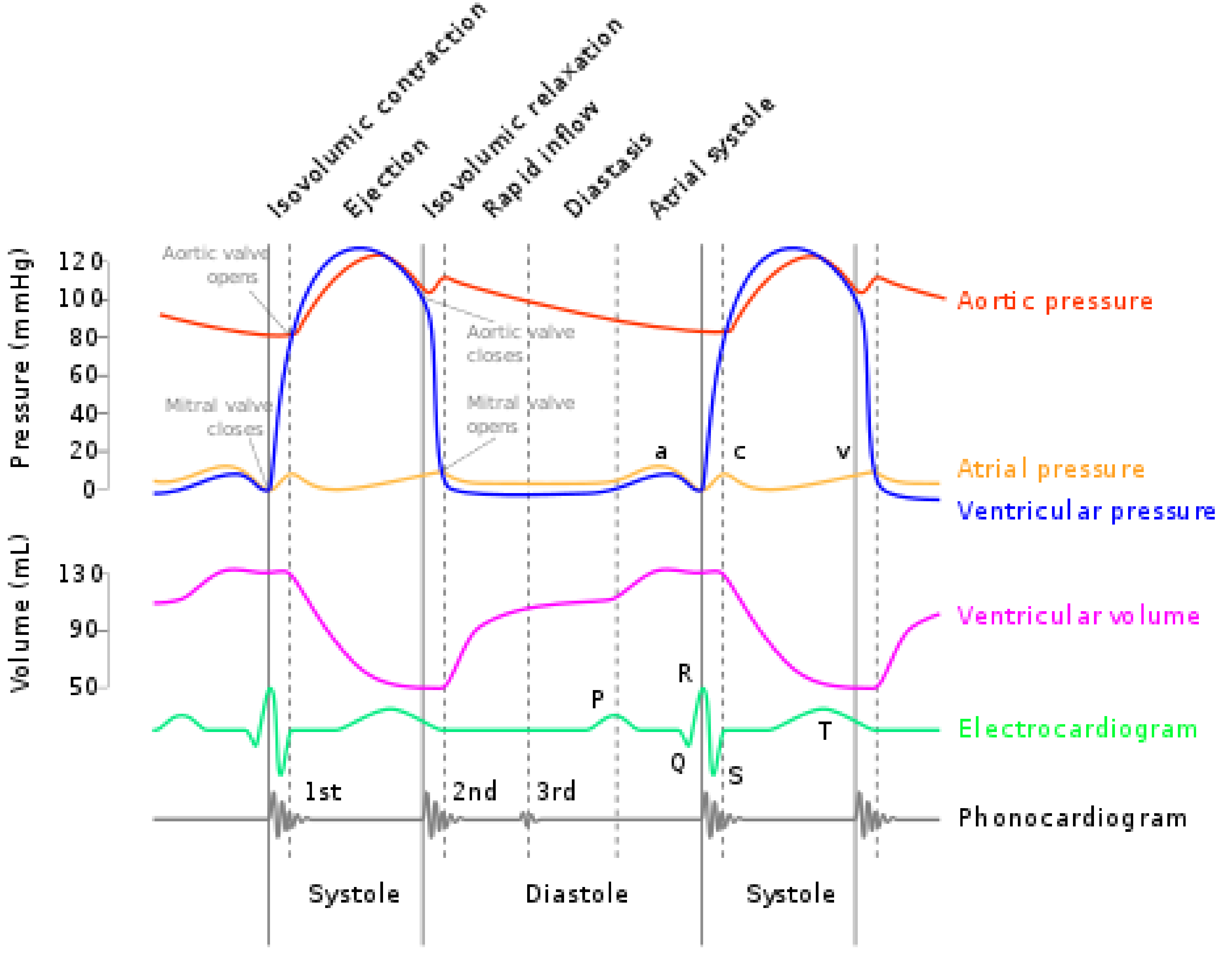


(d) Electrocardiogram of a goldfish

Heart Mechanics and the Cardiac Cycle

- The **cardiac cycle** consists of alternating periods of **systole** and **diastole**
  - **Systole** is the period of **contraction** and **emptying**
  - **Diastole** is the period of **relaxation** and **filling**
  - Events are the **same** on the left and right sides of the heart
  - **Pressures are lower** on the right


PHASES OF THE CARDIAC CYCLE
Atriole systole begins
Atrial contraction forces blood into ventricles
Ventricular systole (first phase)
Ventricular contraction pushes AV valves closed
Ventricular systole (second phase)
Semilunar valves open and blood is ejected
Ventricular diastole (early)
Semilunar valves close and blood flows into atria
Ventricular diastole (late)
Chambers relax and blood fills ventricles passively
R
P
Q
S
T
P-Wave
Atria depolarization
QRS Complex
Ventricle depolarization
T - Wave
Ventricular repolarization
Atrial Diastole
Atrial Systole
Atrial Diastole
Ventricular Diastole
Ventricular Systole
Ventricular Diastole



Isovolumic contraction
Ejection
Isovolumic relaxation
Rapid inflow
Diastasis
Atrial systole
Pressure (mmHg)
120
100
80
60
40
20
0
Aortic valve opens
Aortic valve closes
Mitral valve closes
Mitral valve opens
Aortic pressure
a
c
v
Atrial pressure
Ventricular pressure
Volume (mL)
130
90
50
Ventricular volume
P
R
Q
S
T
Electrocardiogram
1st
2nd
3rd
Phonocardiogram
Systole
Diastole
Systole

Cardiac Output and Its Control

- **Cardiac output** (C.O.) is the volume of blood pumped per minute by a heart to the body.

Cardiac output = heart rate x stroke volume

- Larger animals have slower heart rates, but larger stroke volumes

- Cardiac output **increases** with **warmer body temperature**, **age** during development, and **increased activity level.**

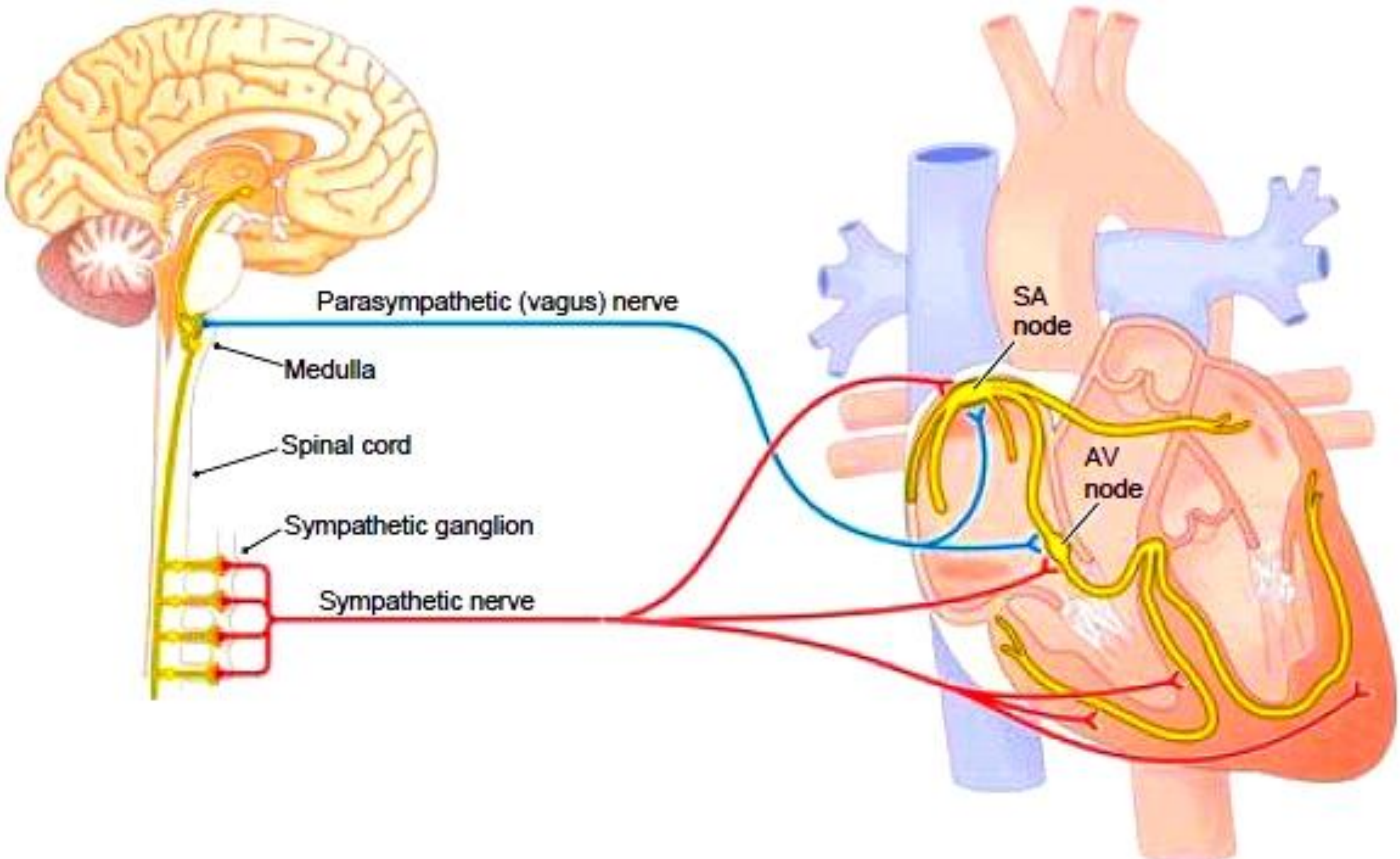
- **Heart rate** is determined by **antagonistic regulation** by the **autonomic nervous system**

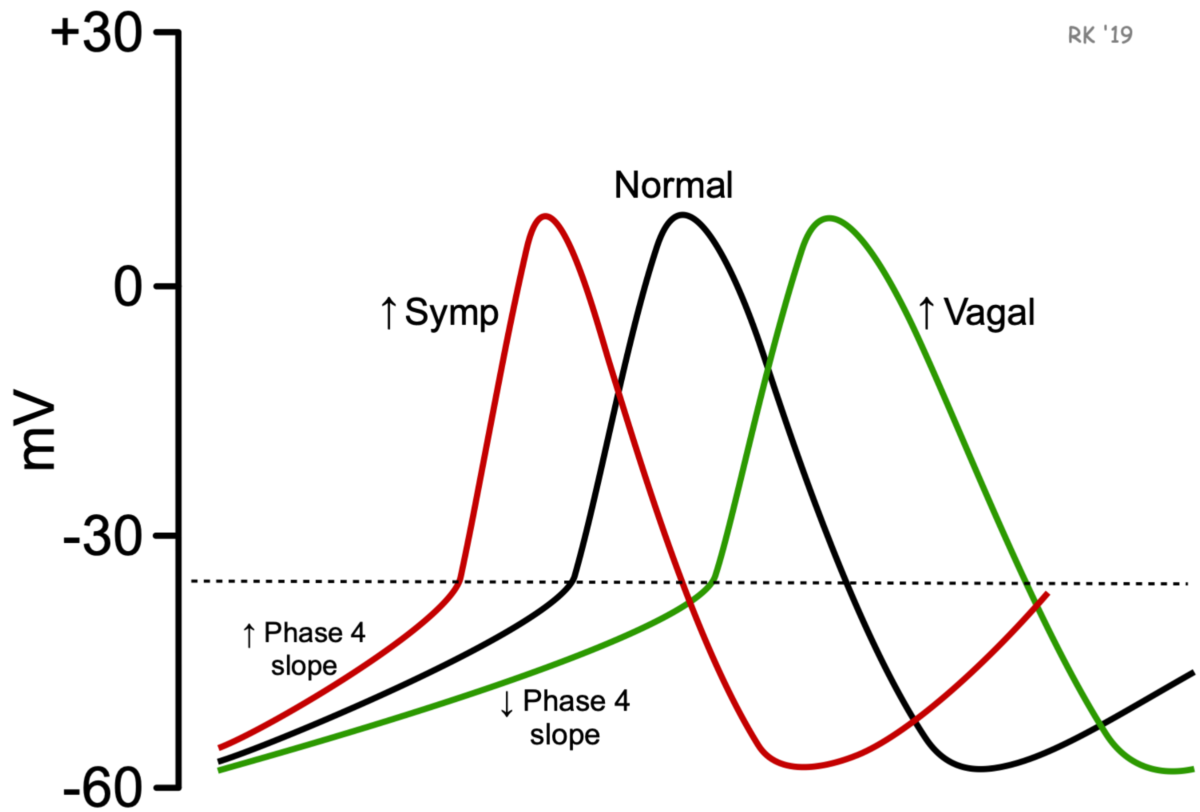
  - Coordinated by the **cardiovascular control center** in the **brain stem**

  - **ACh** from **vagus nerve** binds to **muscarinic receptors**
    - <u>**Decreases**</u> heart rate (SA node)
    - <u>**Decreases**</u> excitability of the AV node
    - <u>**Shortens the plateau**</u> phase of atrial contractile cells

  - **NE** from **sympathetic neurons** and **epinephrine** from the **adrenal medulla** bind to **β_1-adrenergic receptors**
    - <u>**Increases**</u> heart rate
    - <u>**Reduces**</u> AV nodal delay
    - <u>**Speeds the spread**</u> of action potentials through the conduction pathway
    - <u>**Increases**</u> contractile strength of atrial and ventricular cells

Control of Heart Rate - Autonomic Nervous System

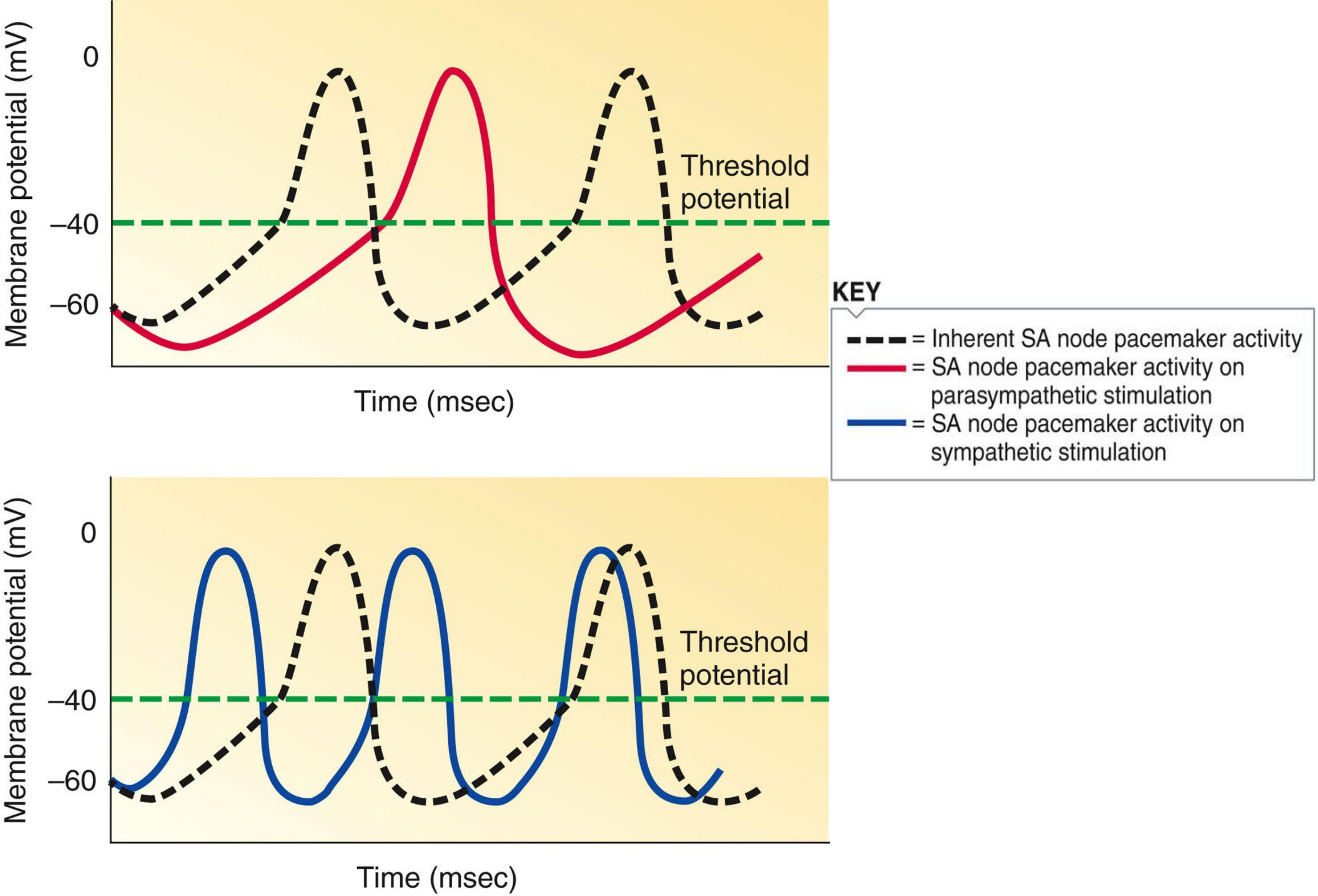




Autonomic nervous system modulates the **<u>frequency</u>** of depolarization of pacemaker

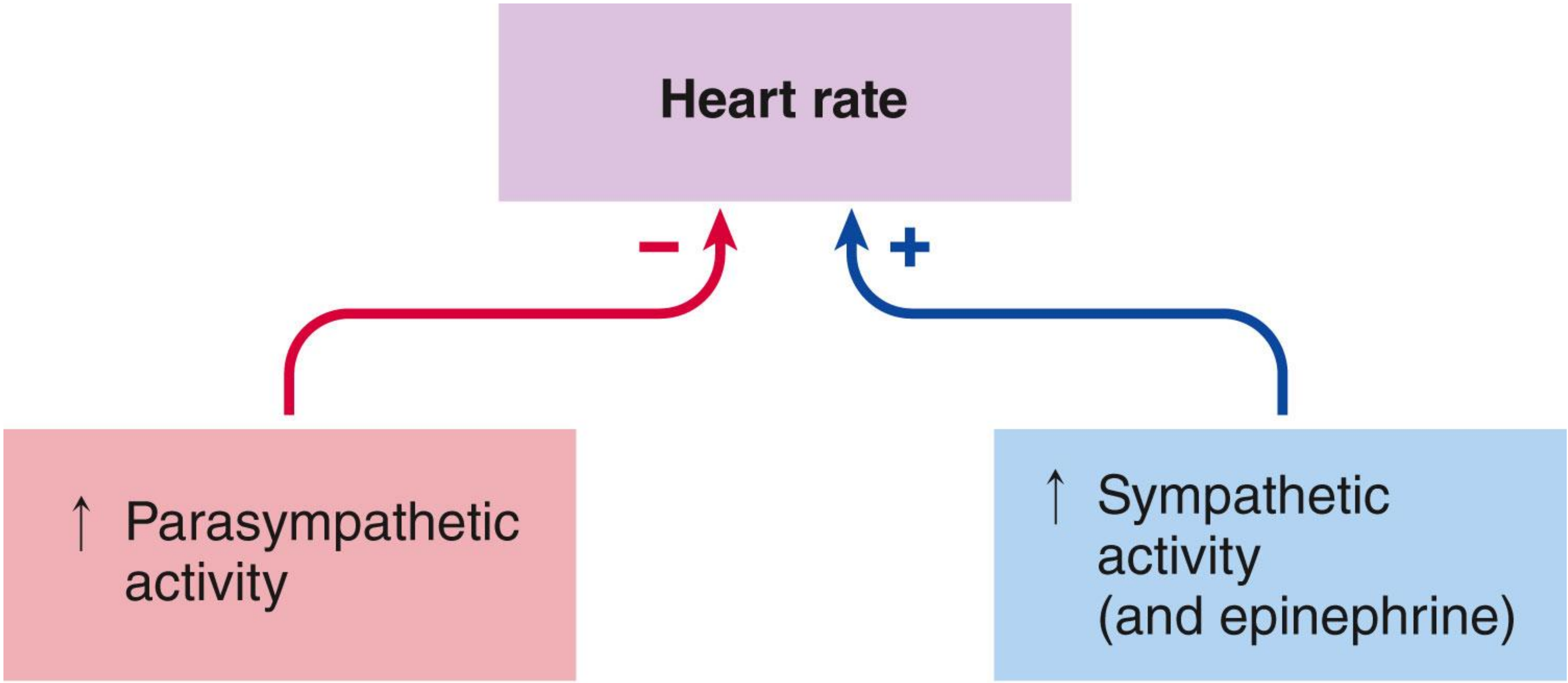
<u>Sympathetic stimulation</u> (neurotransmitter = **<u>adrenaline</u>**); binds to beta1 receptors on the SA nodal membranes

<u>Parasympathetic stimulation</u> (neurotransmitter = **<u>Acetyl choline</u>**); binds to muscarinic receptors on nodal membranes; increases conductivity of K+ and decreases conductivity of Ca2+



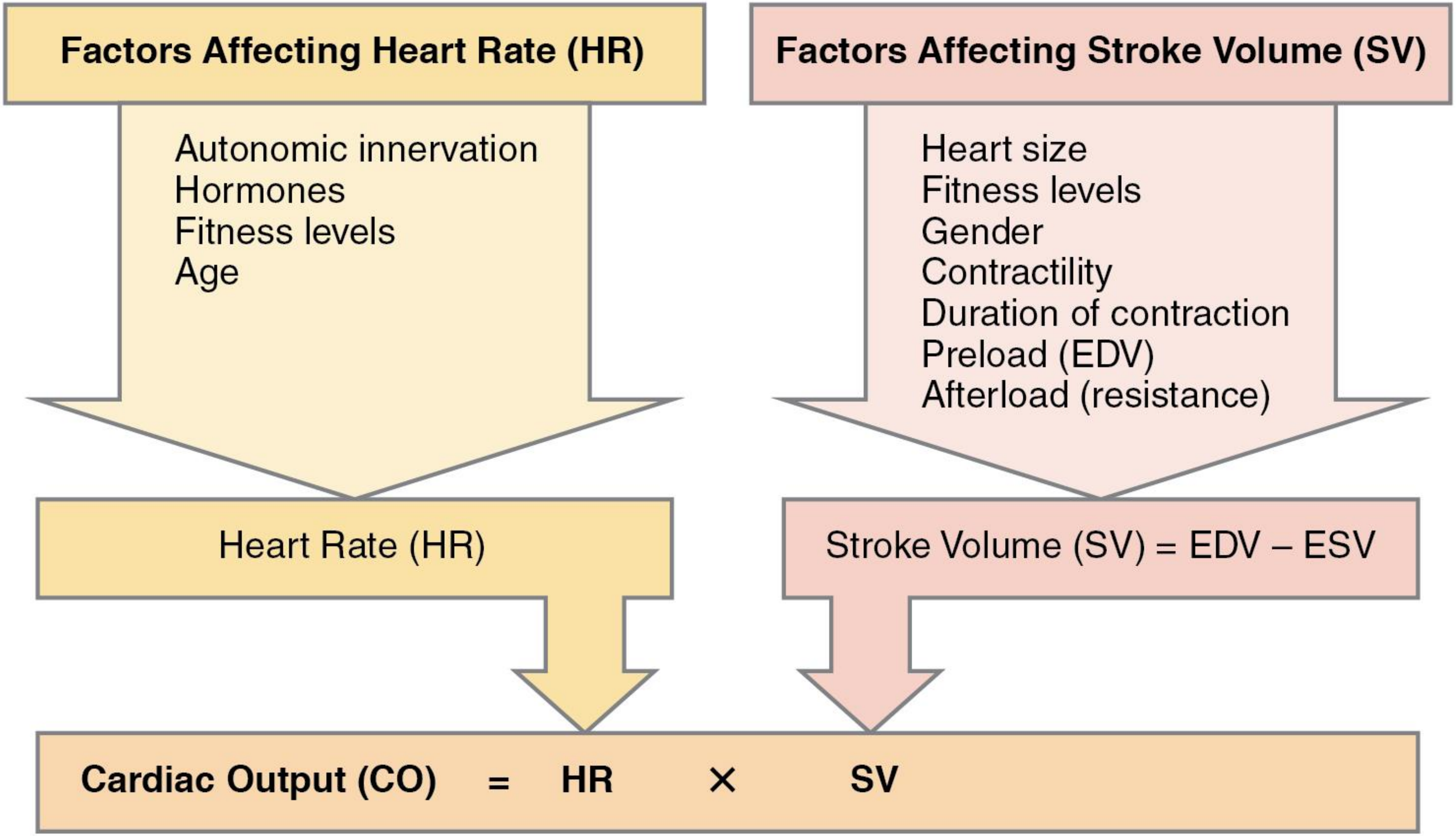
(a) Autonomic influence on SA node potential

Figure 9-28a p416



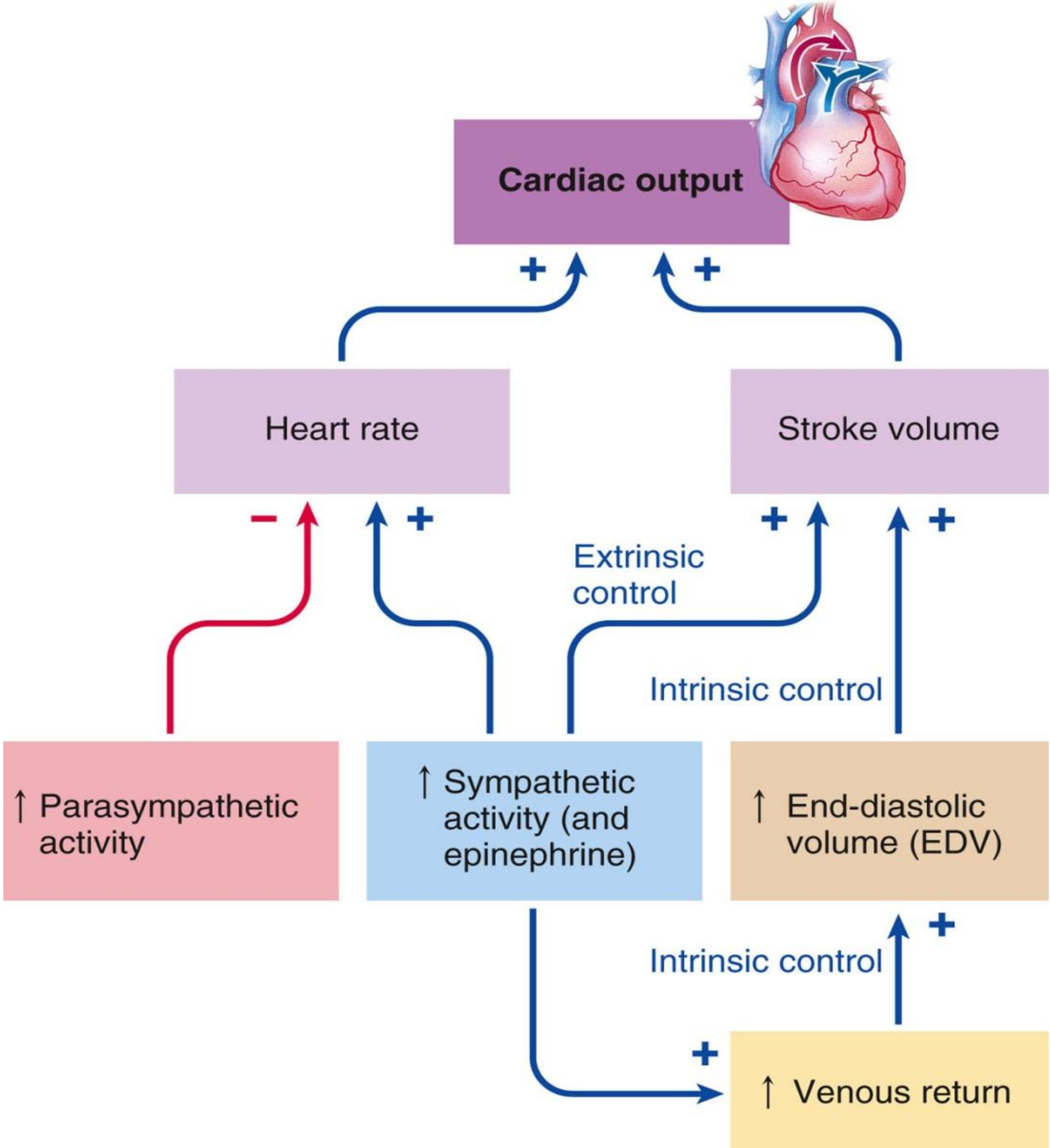
(b) Control of heart rate by autonomic nervous system

Figure 9-28b p416


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

• Control of stroke volume

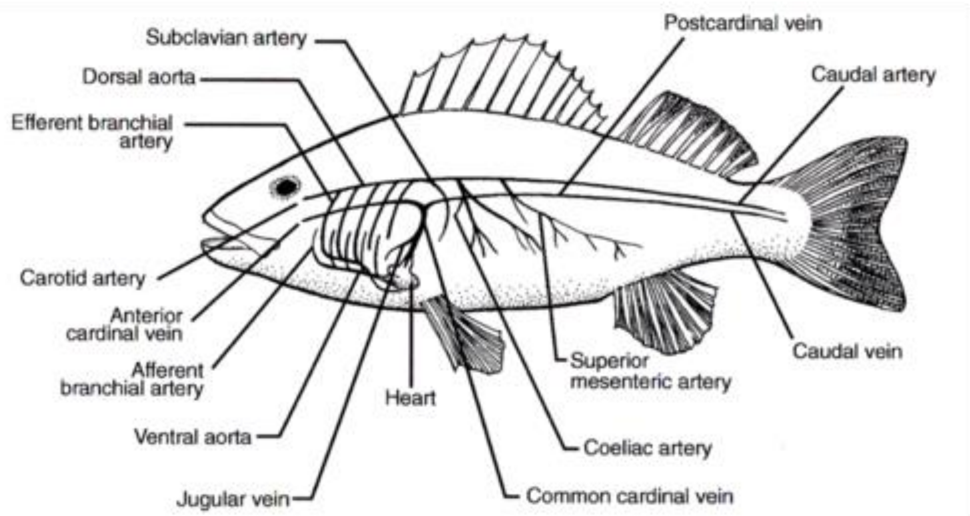
- **Intrinsic control**
  - Direct correlation between **end-diastolic volume (EDV)** and **stroke volume (SV)**
    - Depends on the **length-tension relationship** of cardiac muscle
    - The greater the volume of blood entering the heart, the greater the volume ejected (**Frank-Starling law of the heart**)
- **Extrinsic control**
  - **Sympathetic stimulation** enhances contractility of the heart
  - **Sympathetic stimulation** constricts veins, enhancing venous return and increasing stroke volume


Cardiac output
+
+
Heart rate
Stroke volume
–
+
+
+
Extrinsic control
Intrinsic control
↑ Parasympathetic activity
↑ Sympathetic activity (and epinephrine)
↑ End-diastolic volume (EDV)
+
Intrinsic control
+
↑ Venous return

- The heart receives its blood supply through the **coronary circulation**

  - Heart muscle **cannot** extract **oxygen** or nutrients from blood within its **chambers**

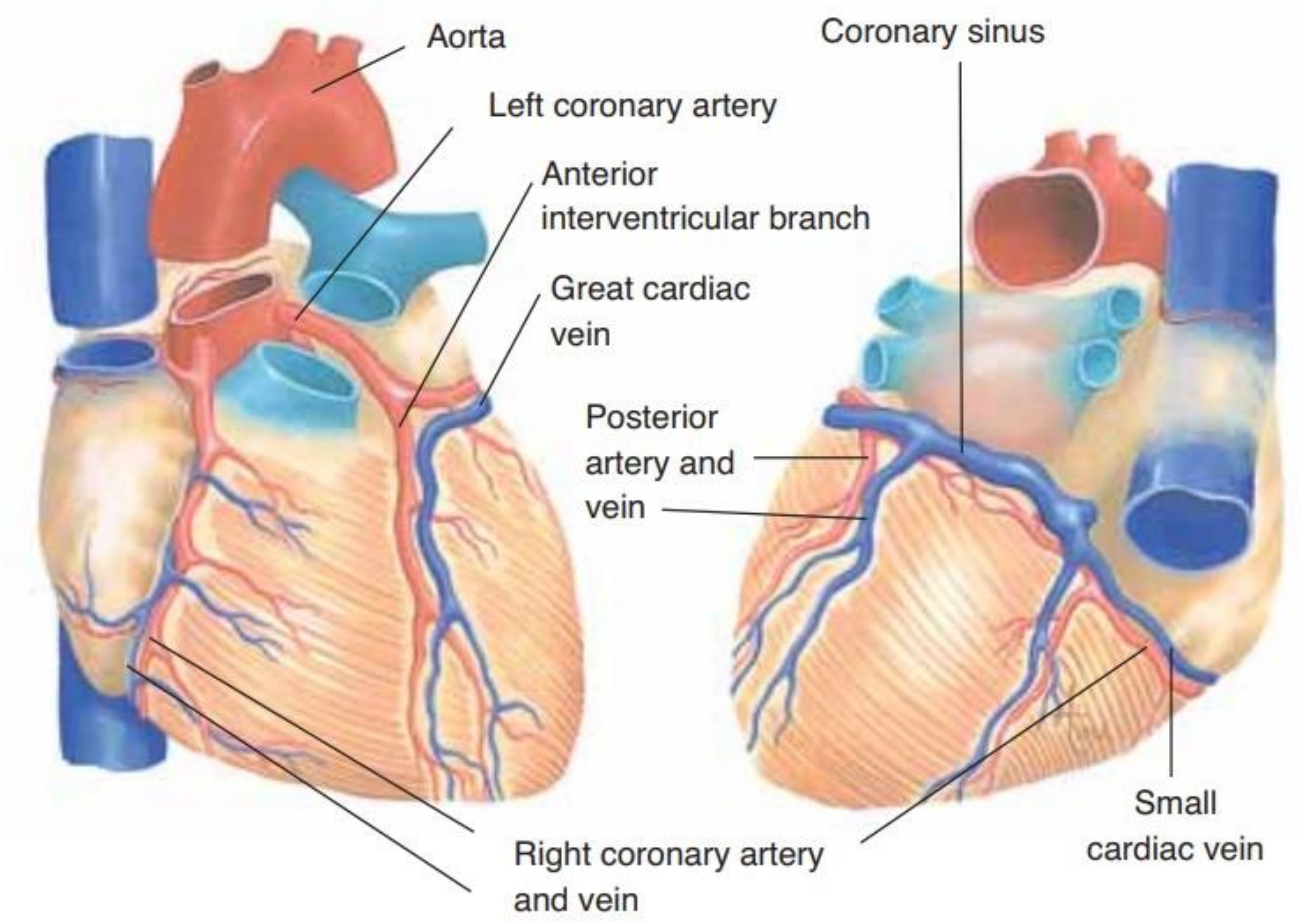
- **Coronary arteries** first evolved in active fishes
  - Branch off of brachial arteries leaving the gills
  - Coronary arteries branch off of the aorta in mammals



  - Coronary **blood flow increases** during activity
    - **Dilation** of coronary vessels is induced by **adenosine**
      - Adenosine is formed from ATP when oxygen supplies are low or cardiac activity is increased

  - Obstruction **of coronary arteries** is a leading cause of death in humans

Coronary arteries



<u>**Arterioles**</u> are the **major resistance vessels**

Radii of arterioles are small enough to offer considerable **resistance to flow**

Large **drop in blood pressure** through the arterioles
- Mean arterial pressure of 93 mmHg drops to 37 mmHg where blood enters the capillaries

<u>Eliminates pulsatile pressure swings</u>

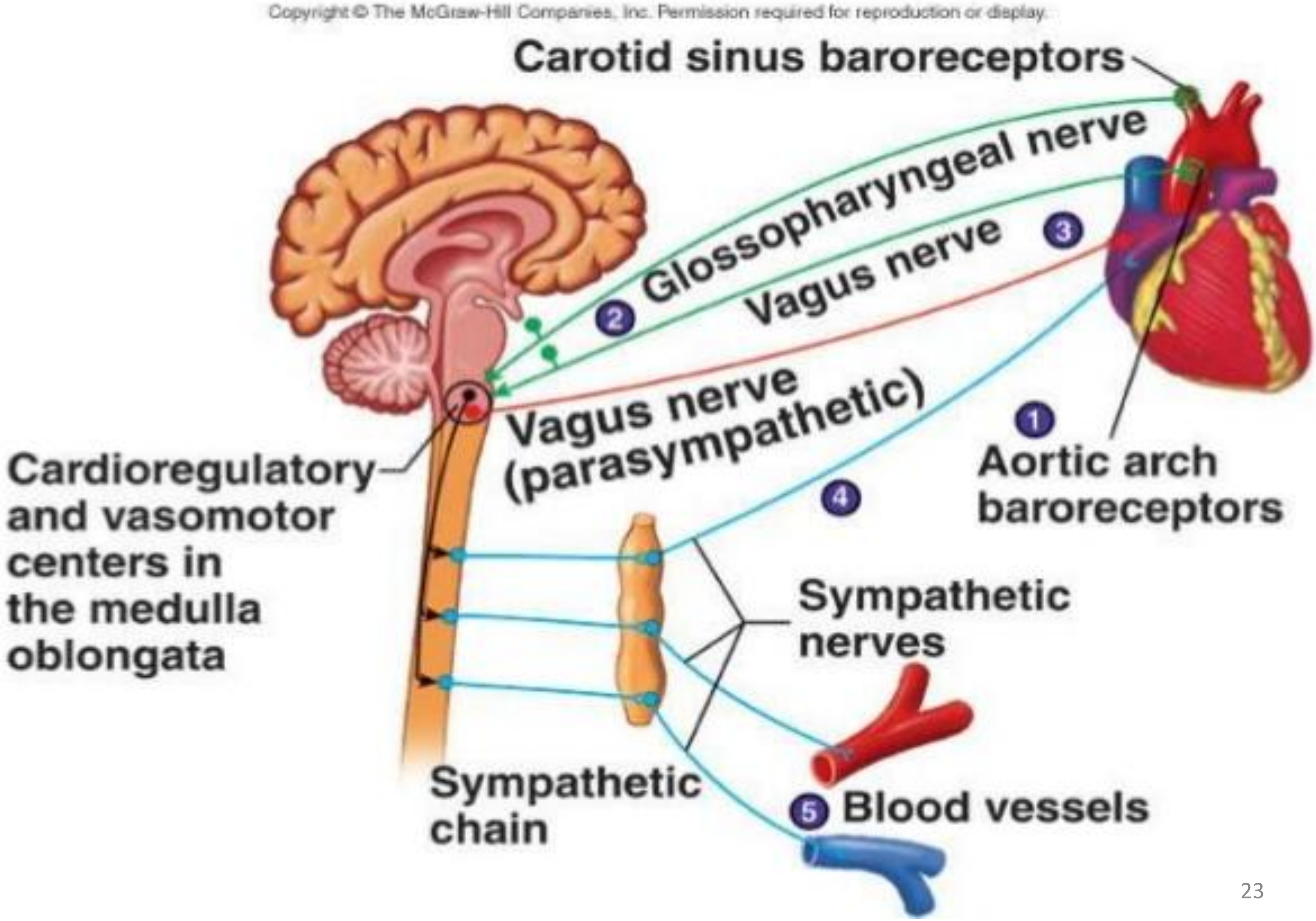
Thick layer of **smooth muscle** is innervated by **sympathetic** nerve fibers
- **Vasoconstriction** results from smooth muscle contraction —-> decreased radius, increased resistance
- **Vasodilation** results from smooth muscle relaxation —-> increased radius, decreased resistance

- Blood pressure is the force exerted by the blood against the wall of blood vessel
- **Systolic blood pressure** : is force exerted by arterial walls during systole. It is the maximum pressure during ventricle contraction
- **Diastolic blood pressure** : is the force exerted by blood against arterial wall during diastole. It is the maximum pressure when the ventricles are relaxed
- Unit of measuring blood pressure =**mmHg**
- Normal blood pressure is **120/80** mm of Hg
- **Pulse pressure** is the difference between systolic & diastolic pressure
- Normally, The pulse pressure is 40 mmHg

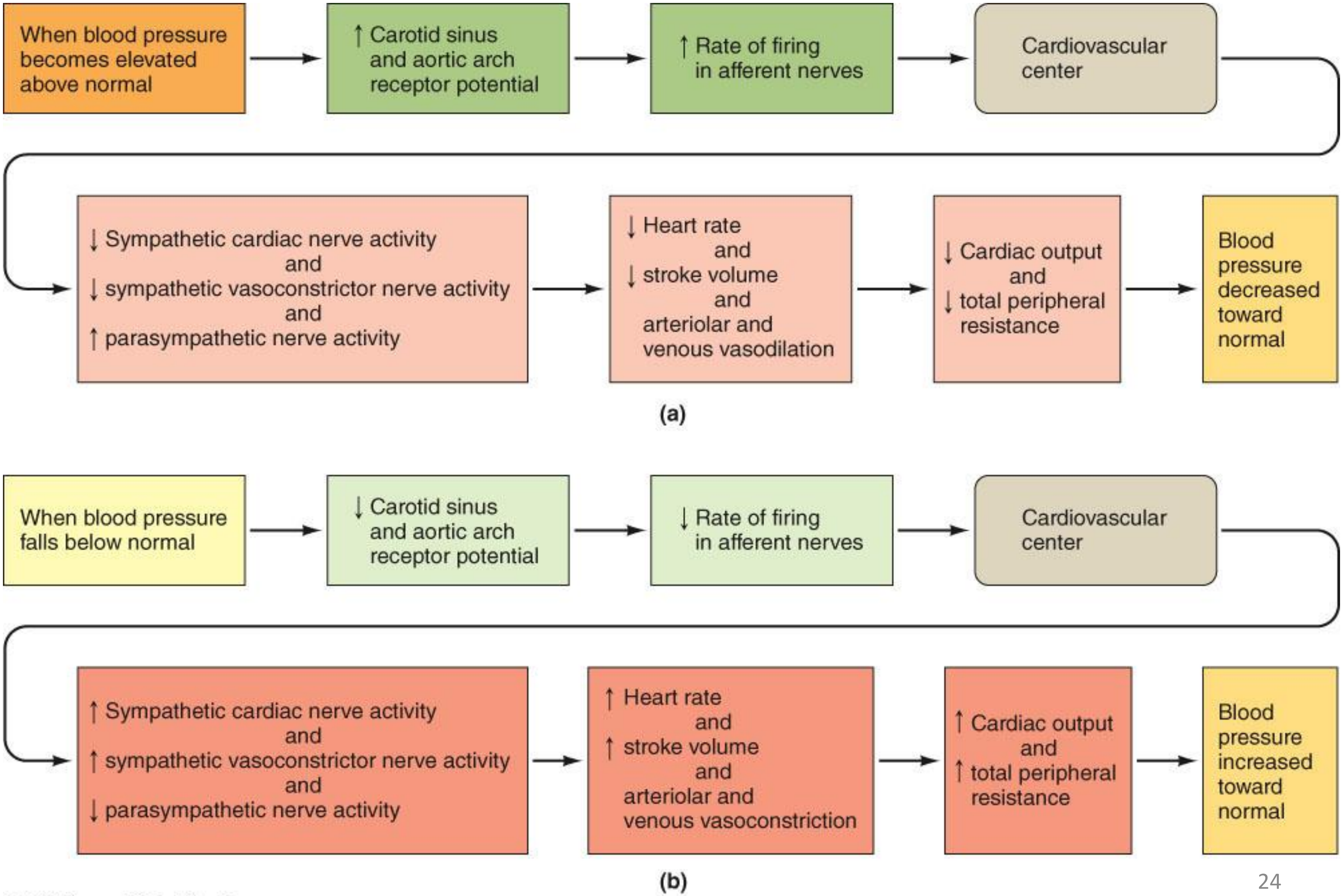
Factors affecting the blood pressure

- Disease
- Age
- Heredity
- Blood Volume
- Weight
- Diet
- Hormones
- Salt
- Caffeine
- Environmental factors
- Psychological factors
- Stress/Anxiety
- Gravity
- Drugs
- Alcohol
- Time of day

Baroreceptors

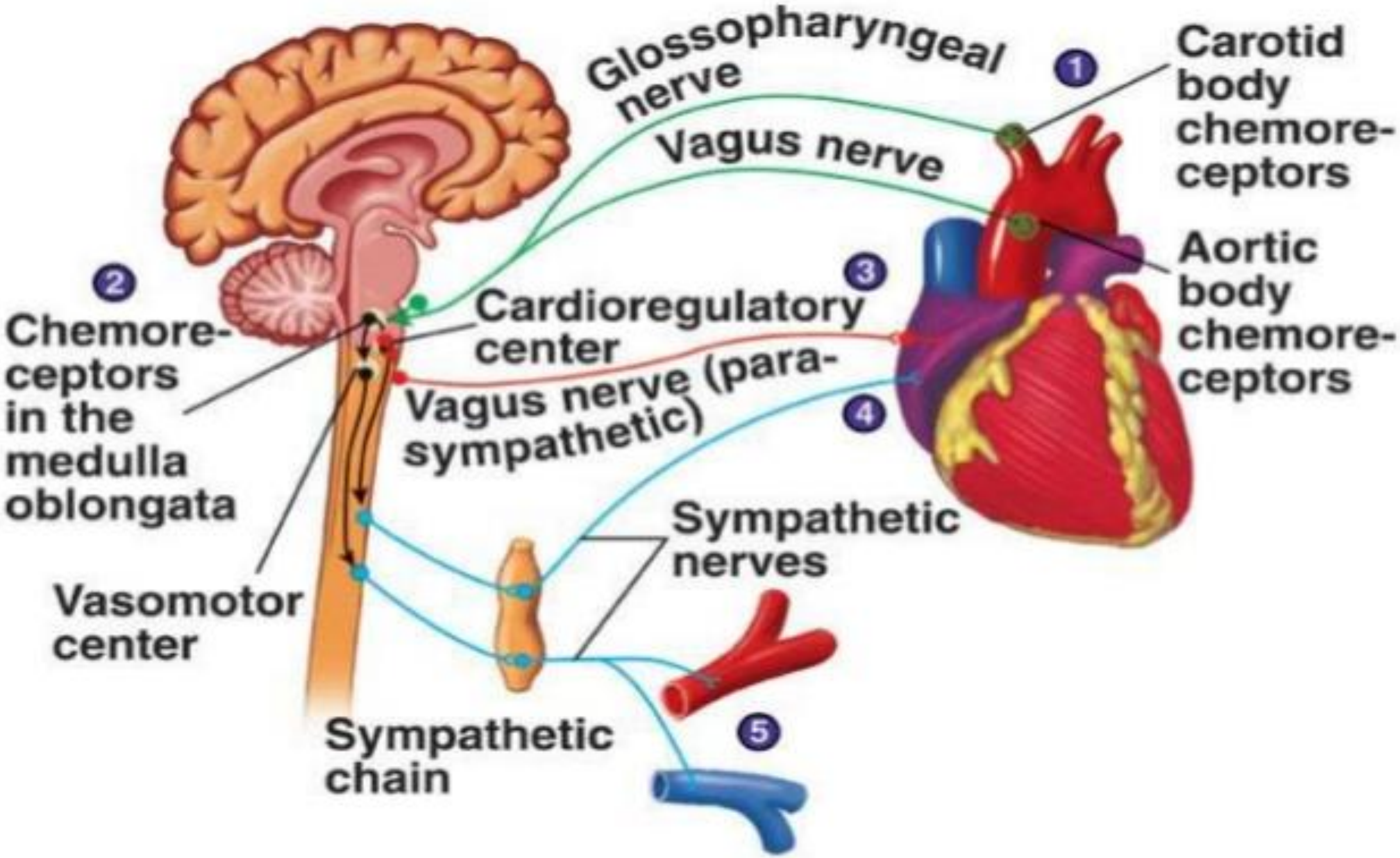


Baroreceptor Reflex



Regulation of blood pressure

II. Chemoreceptor

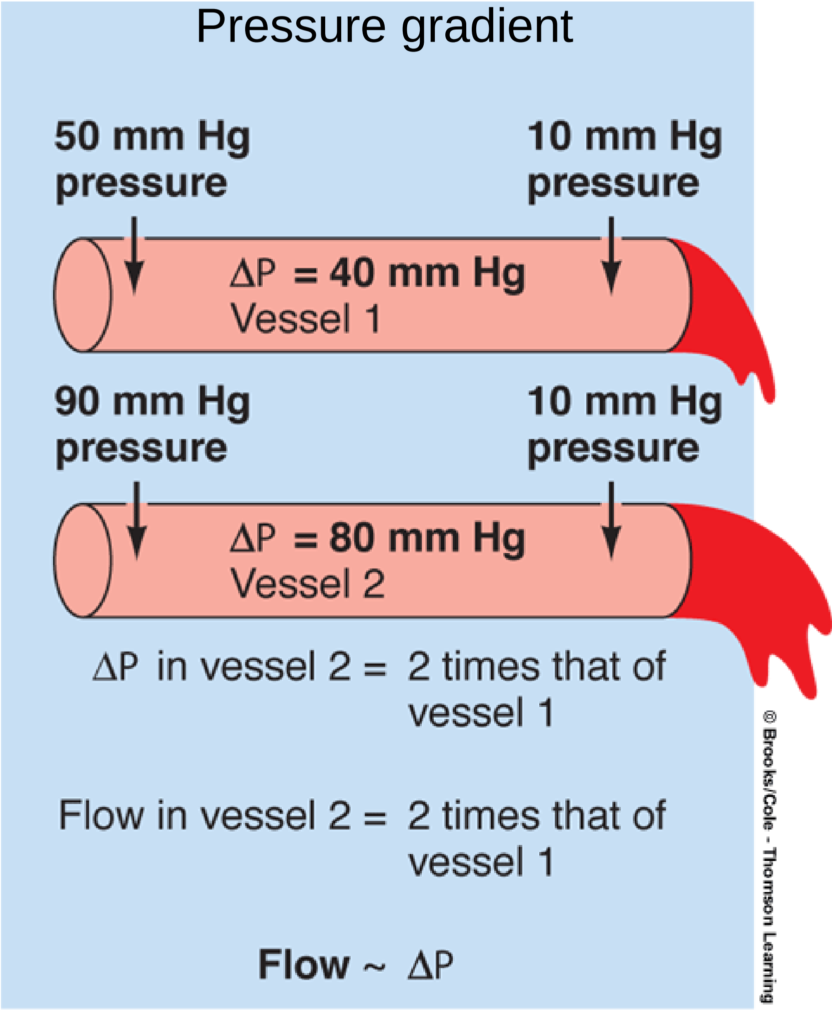


Blood Flow

- Flow rate – volume of blood per unit time

$$F = \Delta P/R$$

- Depends on:
  - **Pressure gradient** – difference in pressure between the beginning and ending of a vessel
  - Vascular resistance – prevention or opposition to blood flow through a vessel

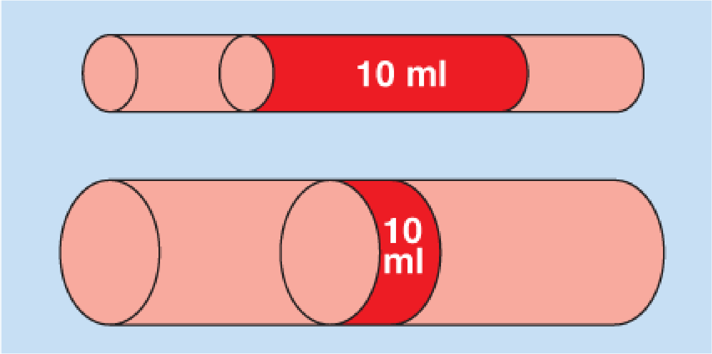


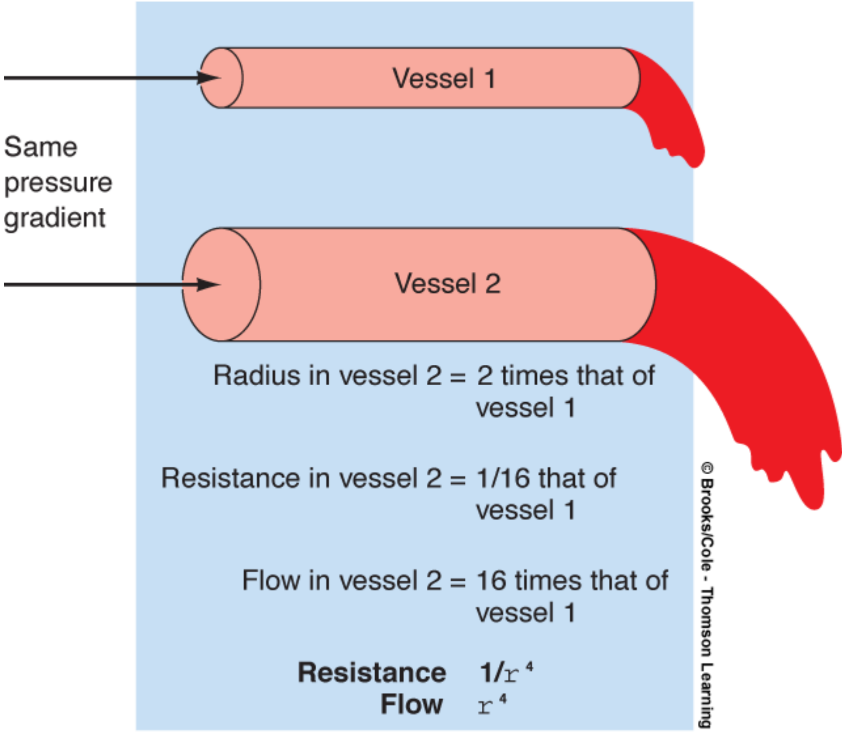
Blood Flow

- **Resistance**
  - As resistance increases flow rate decreases
  - Factors affecting resistance
    - **Viscosity** – friction between molecules increases resistance (no. of circulating RBC)
    - **Length** – increased surface area increases resistance (remains constant in body)
    - **Elasticity**
    - **Peripheral** resistance

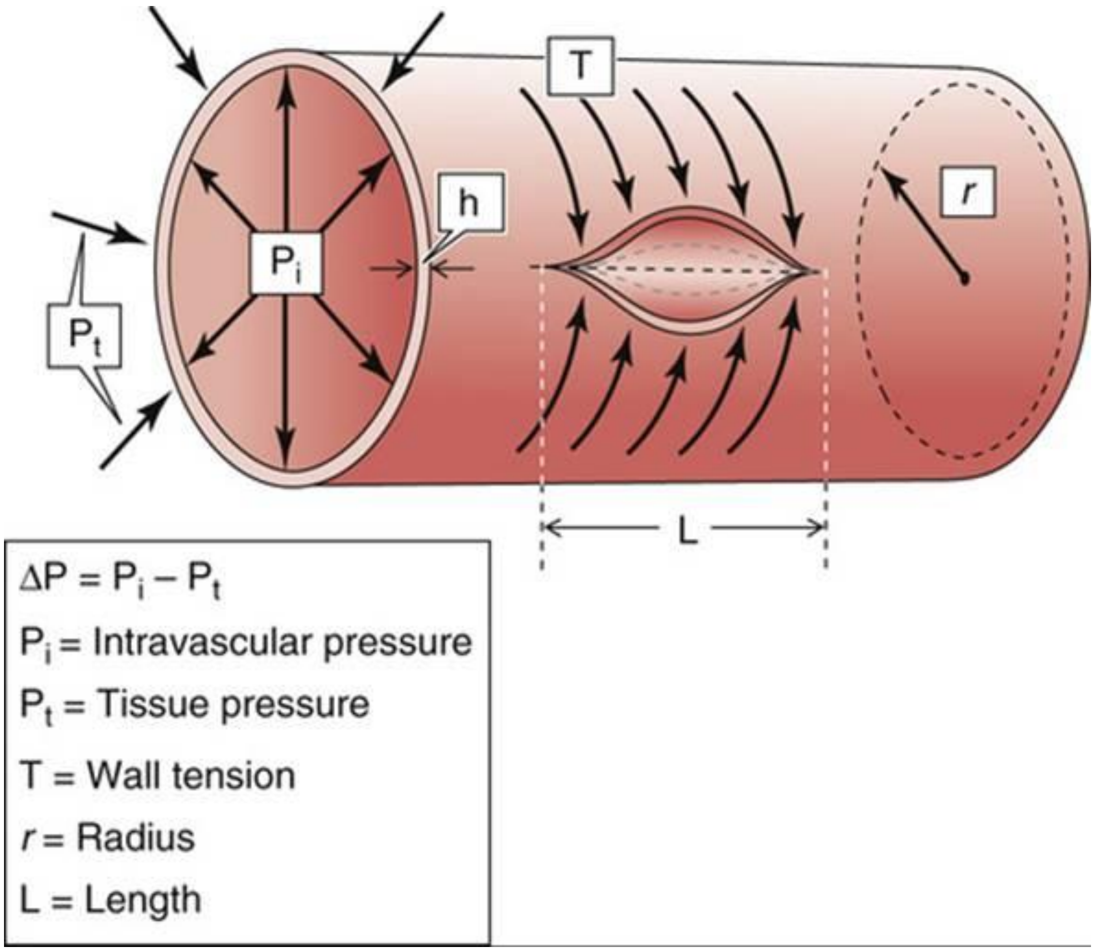
Resistance

- **Peripheral resistance** – friction between the blood and the vessel wall
  - **Radius** the main determinant of resistance
  - Increased **surface area** exposed to blood increases resistance
  - **Flow** is faster in larger vessels than smaller





<u>Laplace law</u> – mechanical stress of blood vessel walls is directly proportional to the **<u>pressure</u>** and **vessel radius**



- Vessels are "built to withstand the wall tensions they normally "see"
- If **<u>intravascular</u>** pressure increases will increase **<u>vessel wall tension</u>** (T)
- In response, vascular smooth muscle **<u>contracts</u>** and T returns to normal

Law of LaPlace

$$T = (\Delta P * r) / \mu m$$

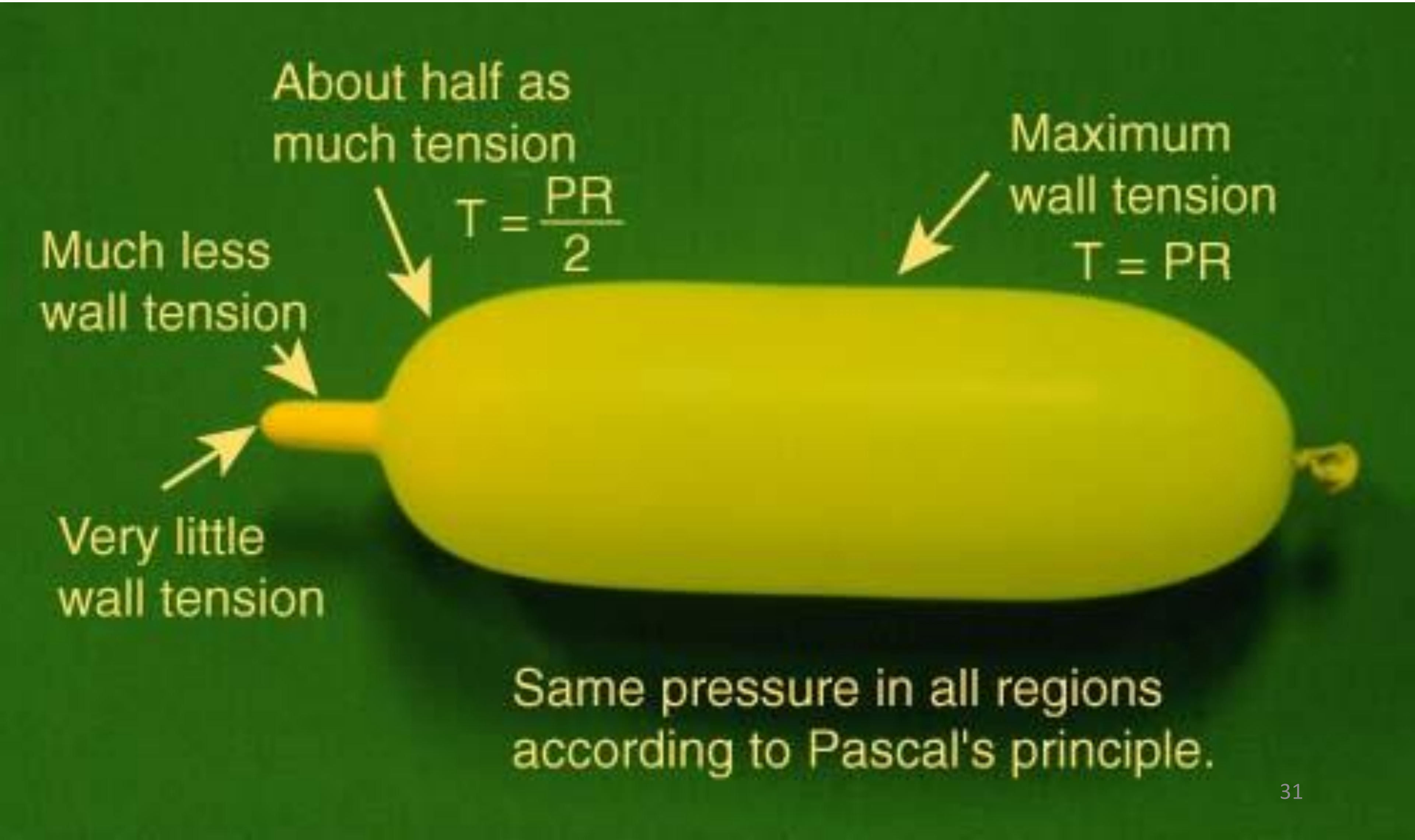
Where T = tension in the vessel wall

ΔP = Transmural pressure

r = radius of the vessel

μm = wall thickness

Law of Laplace



Blood Pressure: Generated by Ventricular Contraction

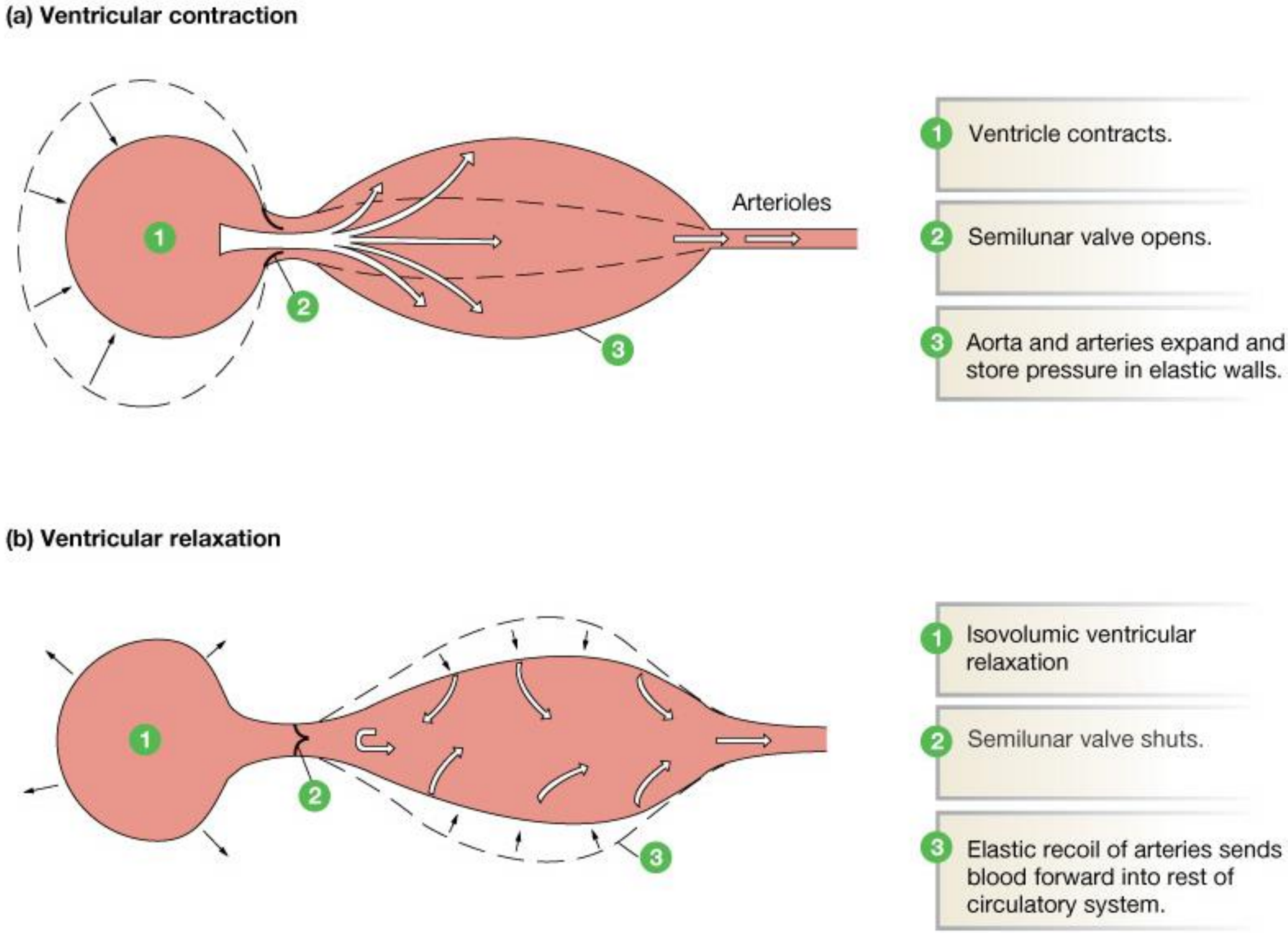
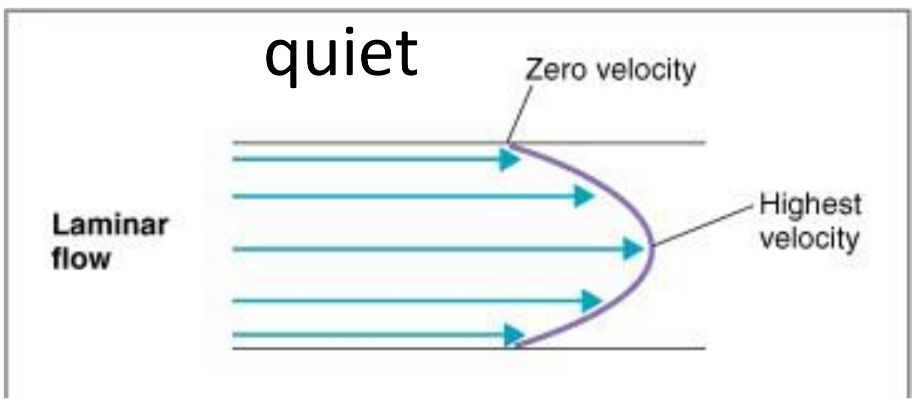


Figure 15-4: Elastic recoil in the arteries

Laminar flow vs Turbulent flow

<u>Laminar</u> flow is parabolic, highest velocity in center (least resistance), lowest adjacent to vessel walls

<u>Turbulent</u> flow is disoriented, no longer parabolic, energy wasted, thus more pressure required to drive blood flow.



noisy