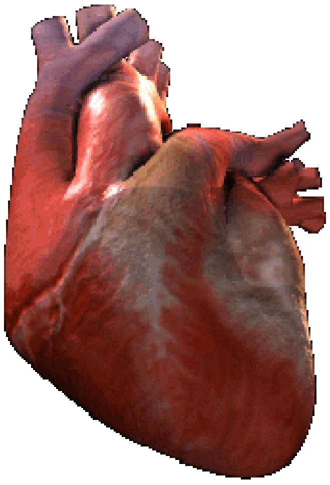
Chapter 4.

Circulation System

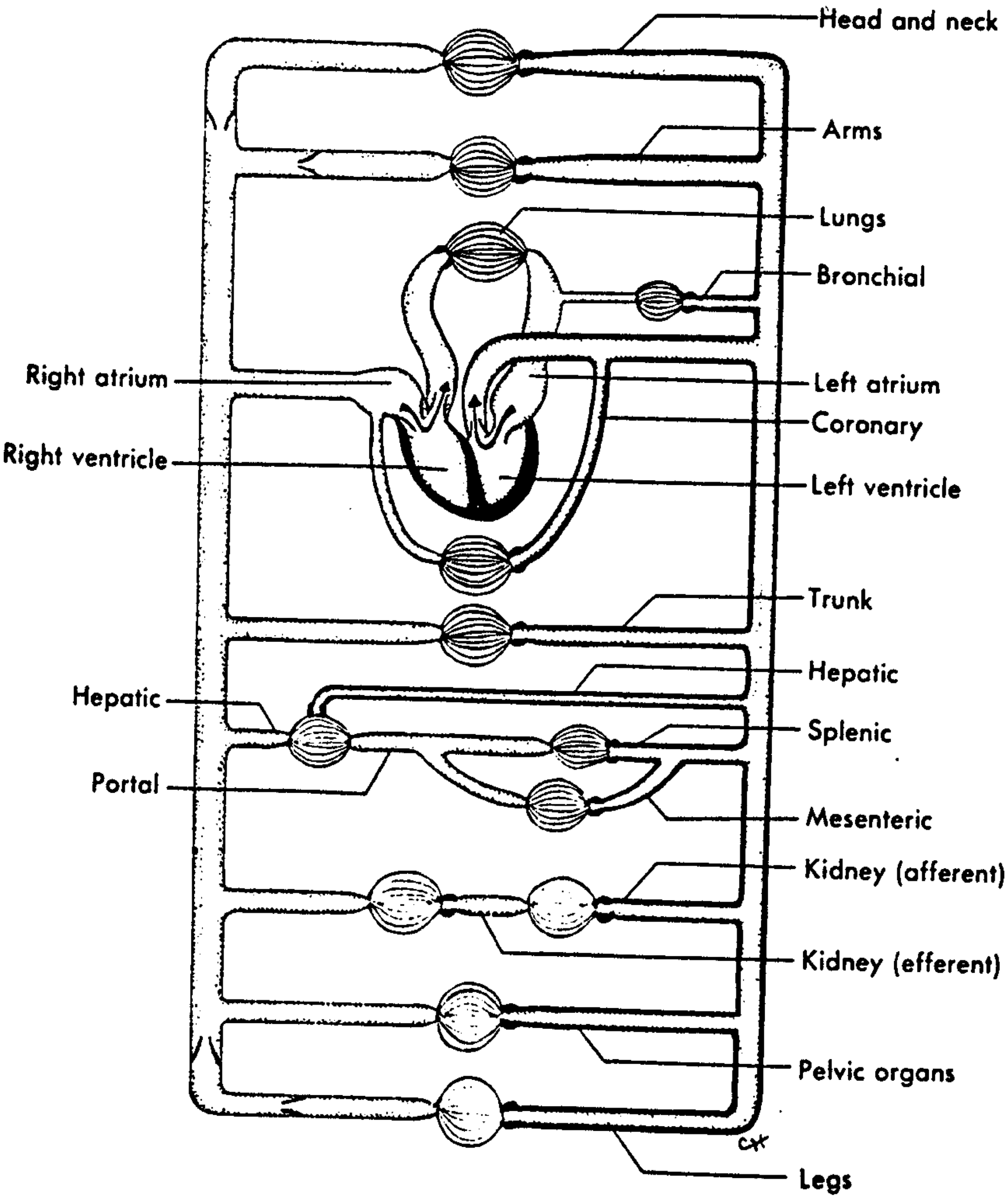
Dong Jing
Physiology department of medical college of Qingdao university
Email: jingdong8@yahoo.com.cn

Section 2.
The Heart as a Pump



The heart as a pump

- **Cardiac Cycle**
- **Regulation OF Cardiac Output**
  - Heart Rate via sympathetic & parasympathetic nerves
  - Stroke Volume
    - **Frank-Starling “Law of the Heart”**
    - **Changes in Contractility**
- **Myocardial Cells (Fibers)**
  - **Regulation of Contractility**
  - **Length-Tension and Volume-Pressure Curves**



PULMONARY CIRCULATION

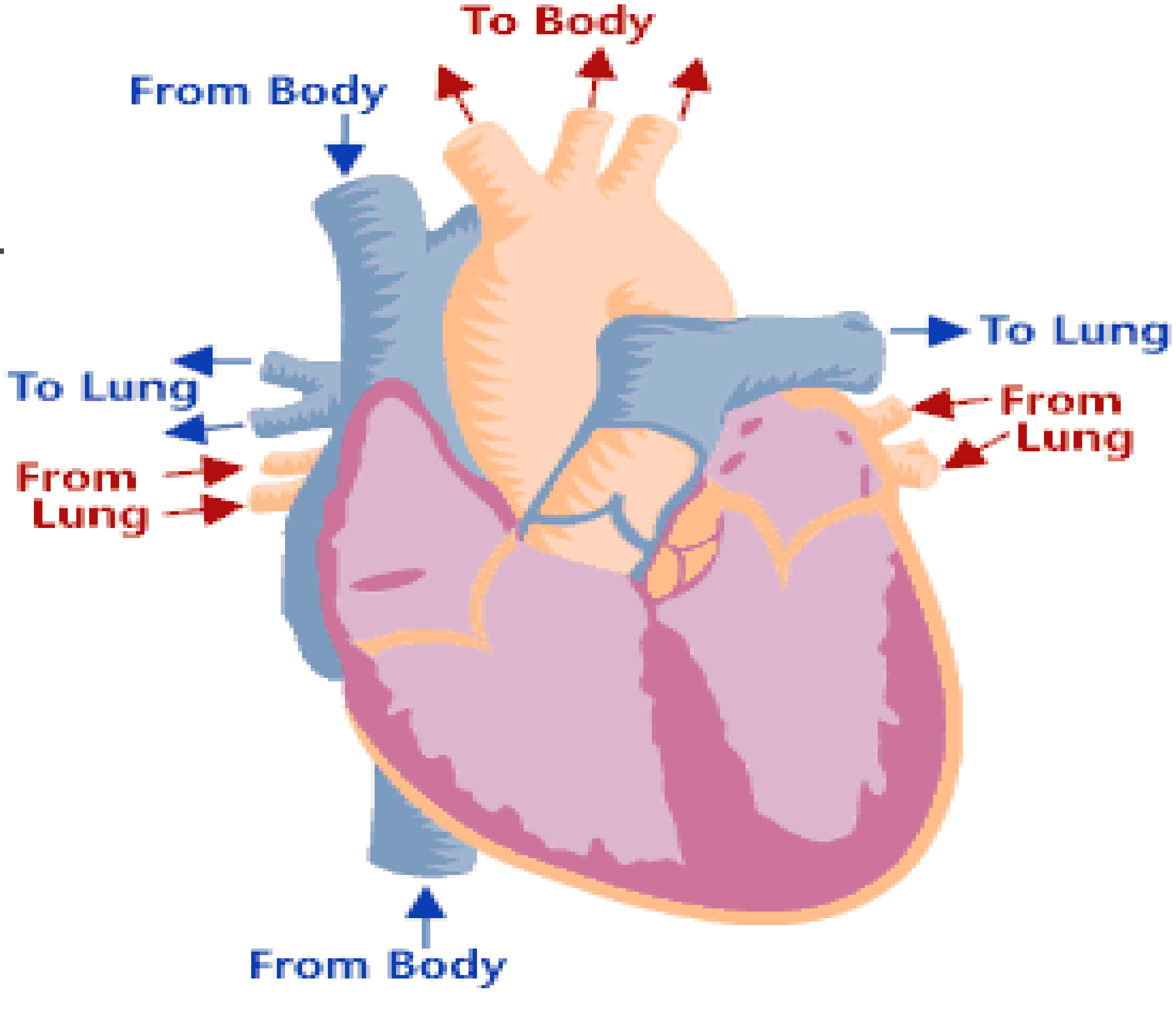
1. LOW RESISTANCE
2. LOW PRESSURE (25/10 mmHg)

SYSTEMIC CIRCULATION

1. HIGH RESISTANCE
2. HIGH PRESSURE (120/80 mmHg)

PARALLEL SUBCIRCUITS

UNIDIRECTIONAL FLOW


To Body
From Body
To Lung
From Lung
To Lung
From Lung
From Body

CARDIAC MUSCLE

- Functional Syncytium
- Automaticity
- modulated by autonomic nervous system

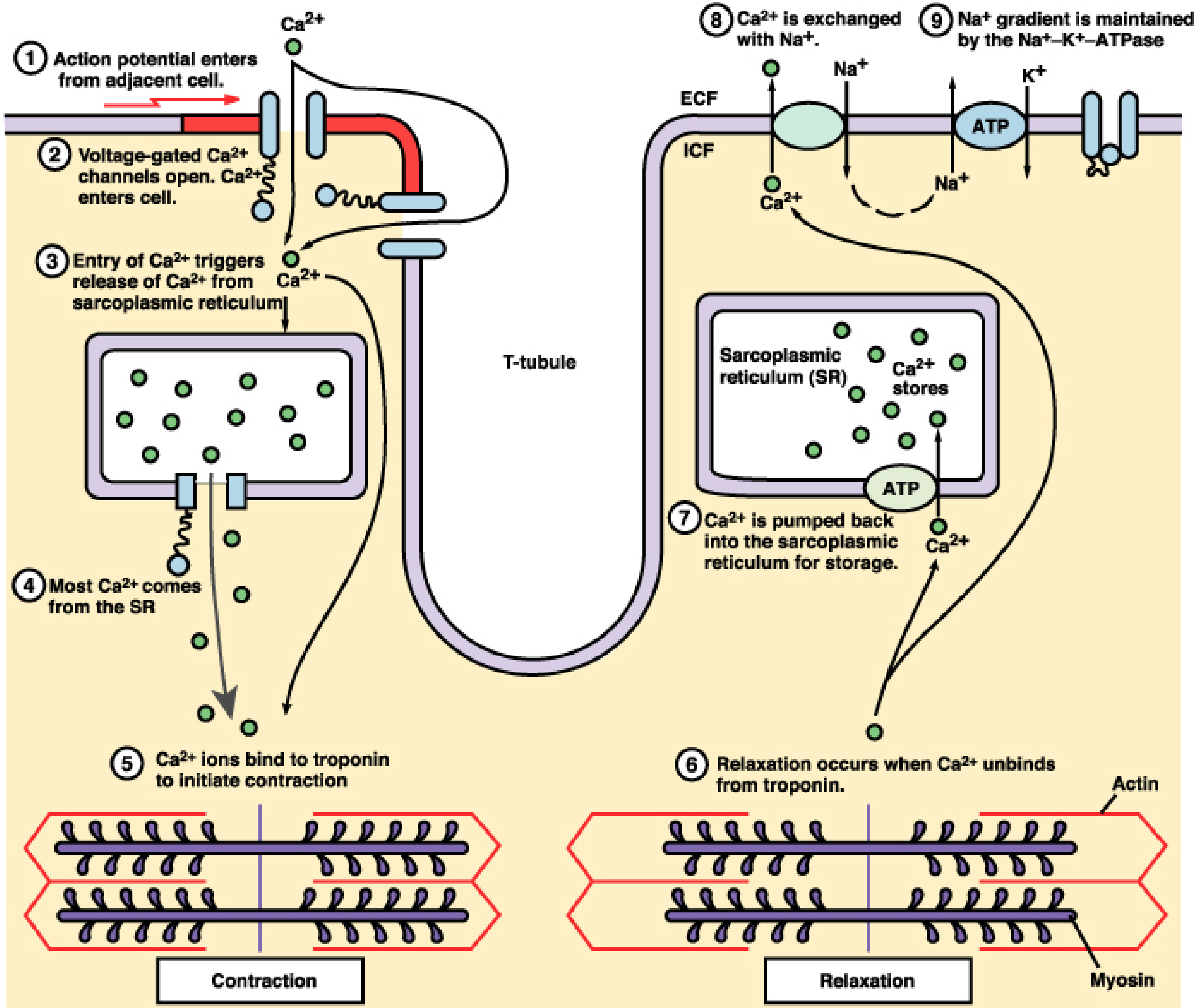
STRIATED MUSCLE

SKELETAL MUSCLE

- Motor Units
- Stimulated by Motor Nerves

Ca^{2+}-induced Ca^{2+} release

- In cardiac M.,Ca^{2+} entry through L-type Ca^{2+} channel is essential for raising $[Ca^{2+}]_i$ in the vicinity of Ca^{2+} -release channels(CRCs): ryanodine R on SR, this trigger activates an adjacent cluster of CRCs, causing them to release Ca^{2+} into cytoplasm.



1 Action potential enters from adjacent cell.
2 Voltage-gated Ca2+ channels open. Ca2+ enters cell.
3 Entry of Ca2+ triggers release of Ca2+ from sarcoplasmic reticulum
4 Most Ca2+ comes from the SR
5 Ca2+ ions bind to troponin to initiate contraction
6 Relaxation occurs when Ca2+ unbinds from troponin.
7 Ca2+ is pumped back into the sarcoplasmic reticulum for storage.
8 Ca2+ is exchanged with Na+.
9 Na+ gradient is maintained by the Na+–K+–ATPase
Ca2+
Na+
K+
ECF
ICF
ATP
T-tubule
Sarcoplasmic reticulum (SR)
Ca2+ stores
Actin
Myosin
Contraction
Relaxation

The cardiac cycle

- The sequence of mechanical and electrical events that repeat with every heartbeat is called the cardiac cycle.

What are the phases of the cardiac contraction?

- ✓ Diastole - Ventricles are relaxed.
- ✓ Systole - Ventricles contract

The duration of the cardiac cycle is the reciprocal of heart rate:

Cardiac cycle= 60 sec ⁄ 75

= 0.8 sec

Diastole(0.5s)

Phase 1- Isovolumetric Relaxation

Phase 2- Rapid Filling

Phase 3 - Reduced Filling

Phase 4 - Atrial Contraction

} Inflow phase

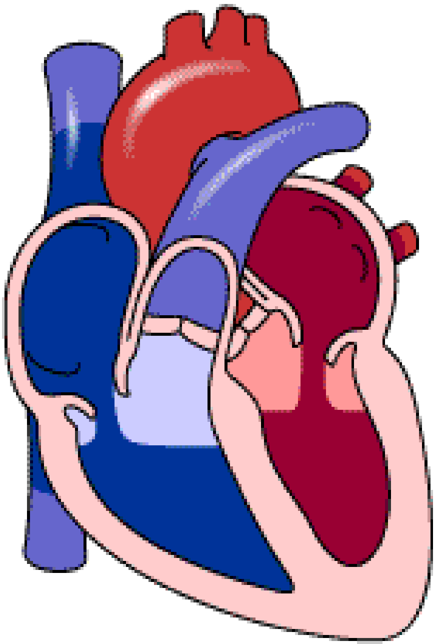
Systole(0.3s)

Phase 5 - Isovolumetric Contraction

Phase 6- Rapid Ejection

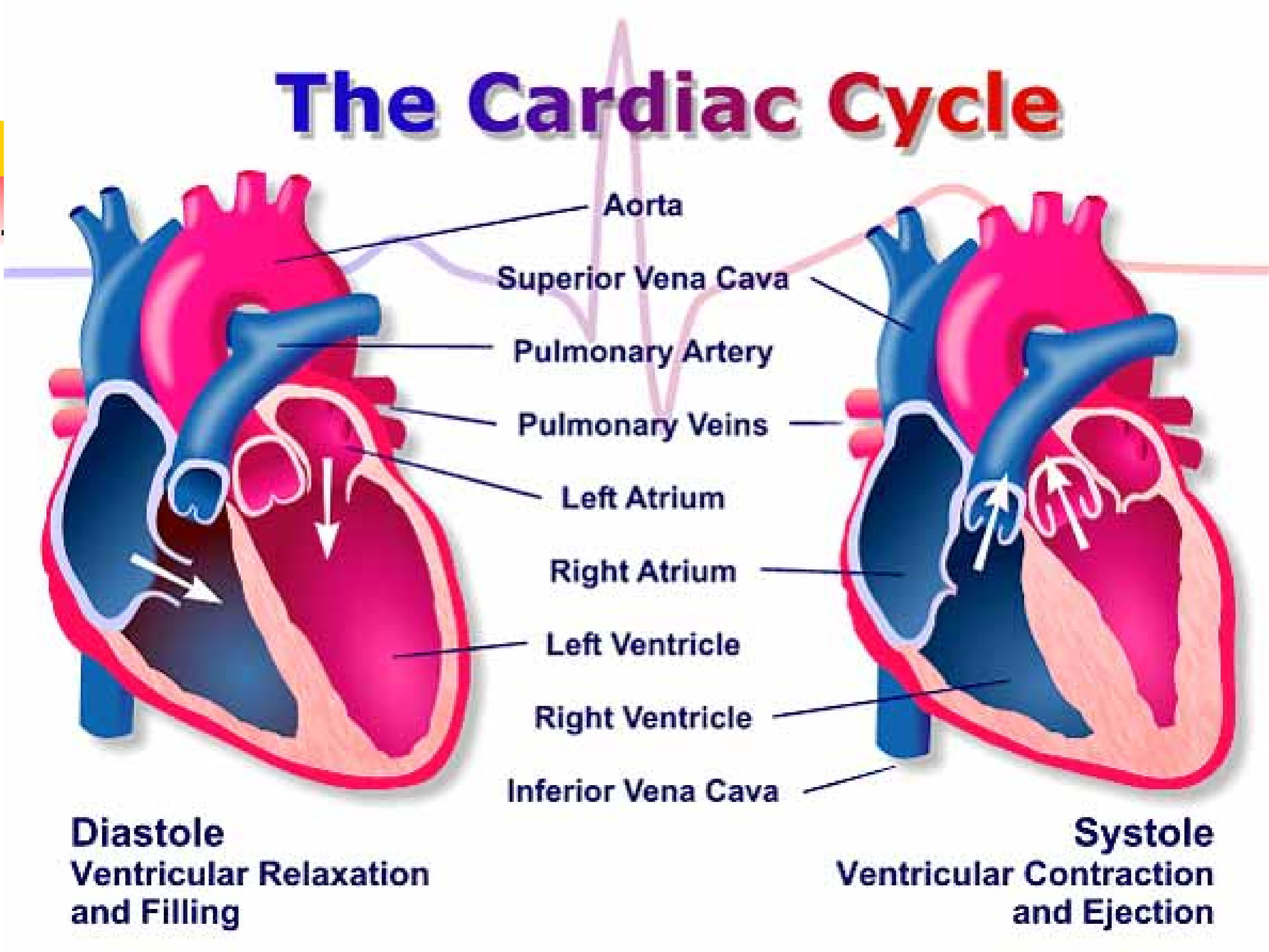
Phase 7- Reduced Ejection

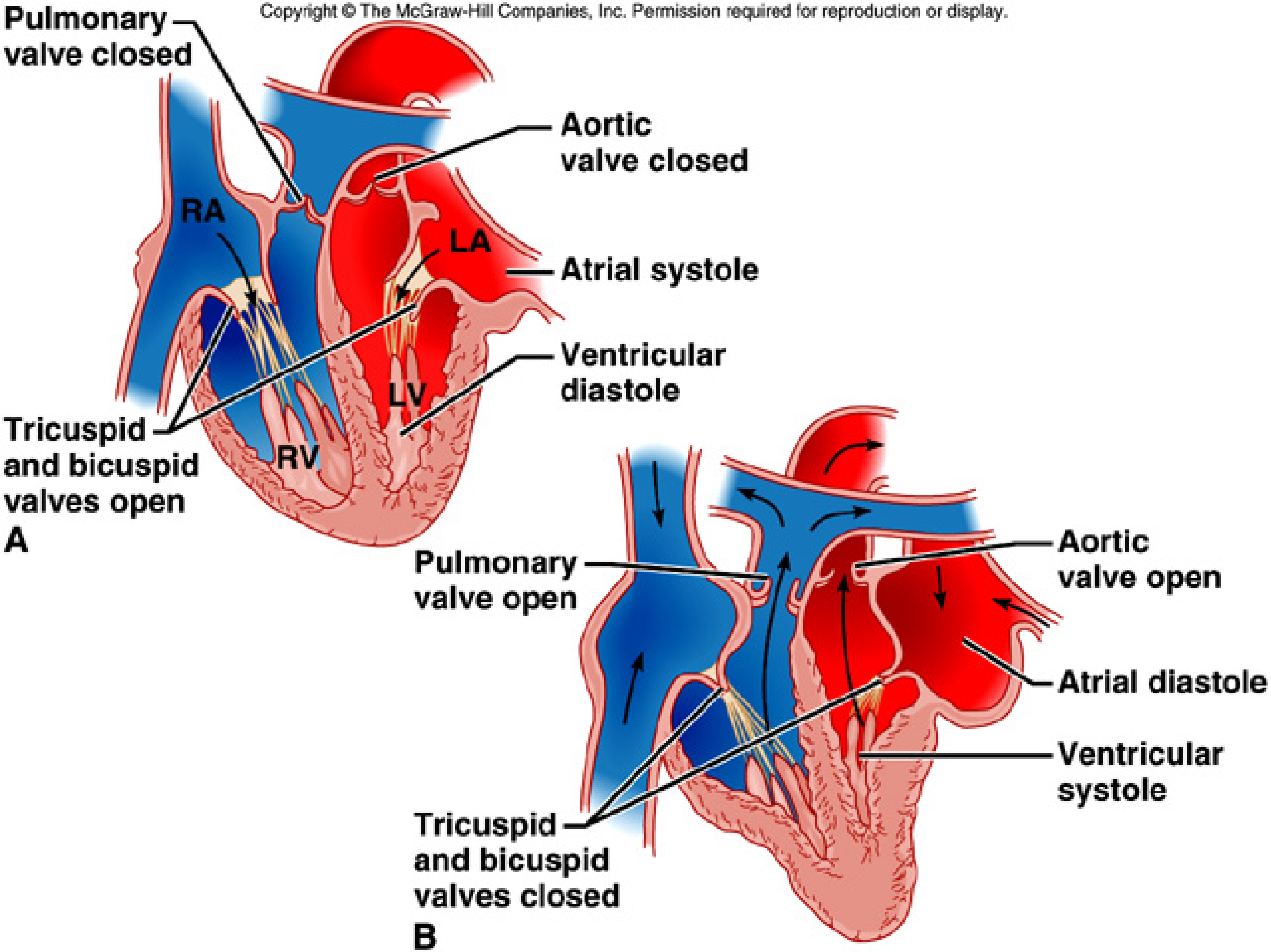
} Outflow phase



What happens during each cardiac cycle?

- An impulse arising from the SA node → Contraction/relaxation → changes in pressure → valves (open/close， heart sounds) → blood flow → changes in volume


The Cardiac Cycle
Aorta
Superior Vena Cava
Pulmonary Artery
Pulmonary Veins
Left Atrium
Right Atrium
Left Ventricle
Right Ventricle
Inferior Vena Cava
Diastole
Ventricular Relaxation and Filling
Systole
Ventricular Contraction and Ejection

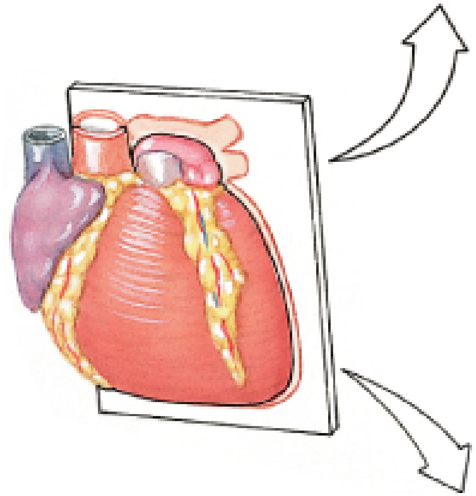


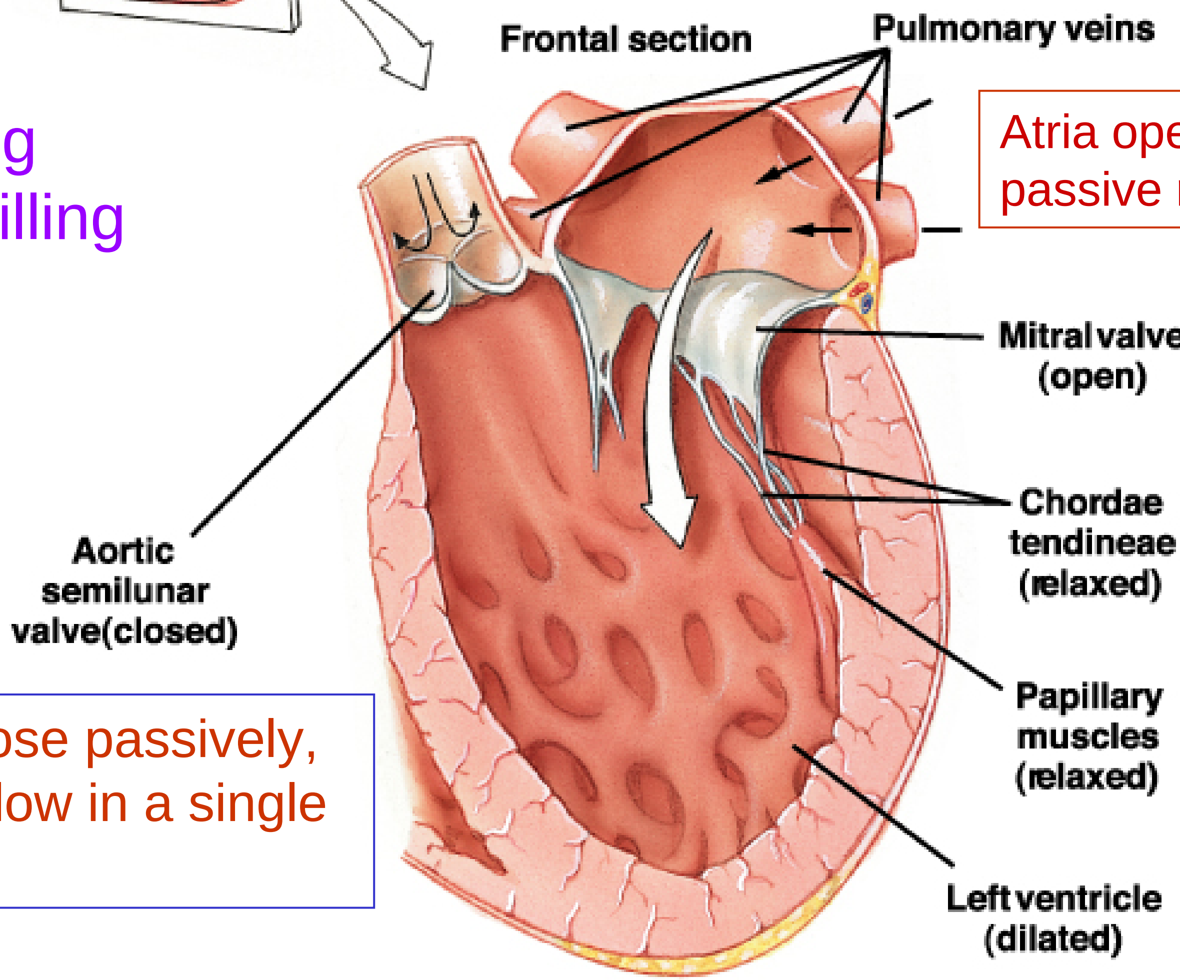
Pulmonary valve closed
Aortic valve closed
RA
LA
Atrial systole
Ventricular diastole
LV
Tricuspid and bicuspid valves open
RV
A
Pulmonary valve open
Aortic valve open
Atrial diastole
Ventricular systole
Tricuspid and bicuspid valves closed
B

Ventricle Filling

①Rapid Filling
②Reduced Filling
= diastasis
③Atrial Contraction

Atria operate as passive reservoirs





Valves open/close passively, allow blood to flow in a single direction

Diastasis, Atrial contraction, isovolmetric contraction

- Atrial P. is only slightly higher above the ventricular P., P wave of ECG occurs at the end of this phase.
- Atrial contraction immediately following the ECG's P wave
- Left ventricle contracts with both mitral and aortic valves closed, causes the P. to rise rapidly,eventually exceeding the P. in the aorta

Ejection

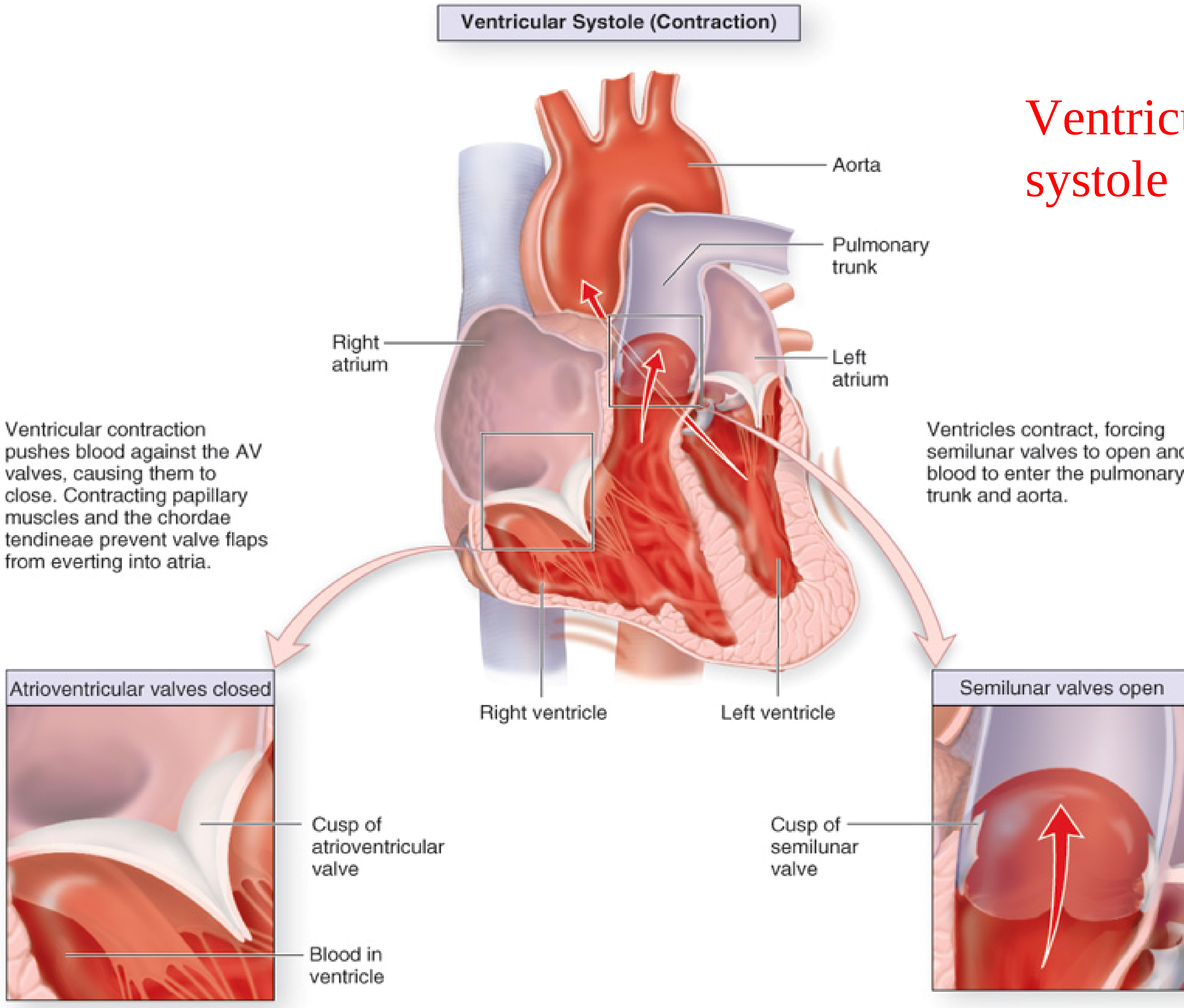
- Aortic valve opens→ventricular and aortic P. continues to rise → aortic P.eventually exceeds ventricular P., aortic valve do not immediately snap shut because of the inertia of blood flow →decreased ejection.

- Stroke volume: 70 ml, ESV: 50ml

Isovolumetric relaxation, rapid ventricular filling

- Late in the ejection phase,blood flow across aortic valve reverses direction →aortic valve closes →dicrotic notch or incisura →P. falls rapidly
- P. in the left vetricle falls below that in the left atrium →mitral valve opens → rapid filling

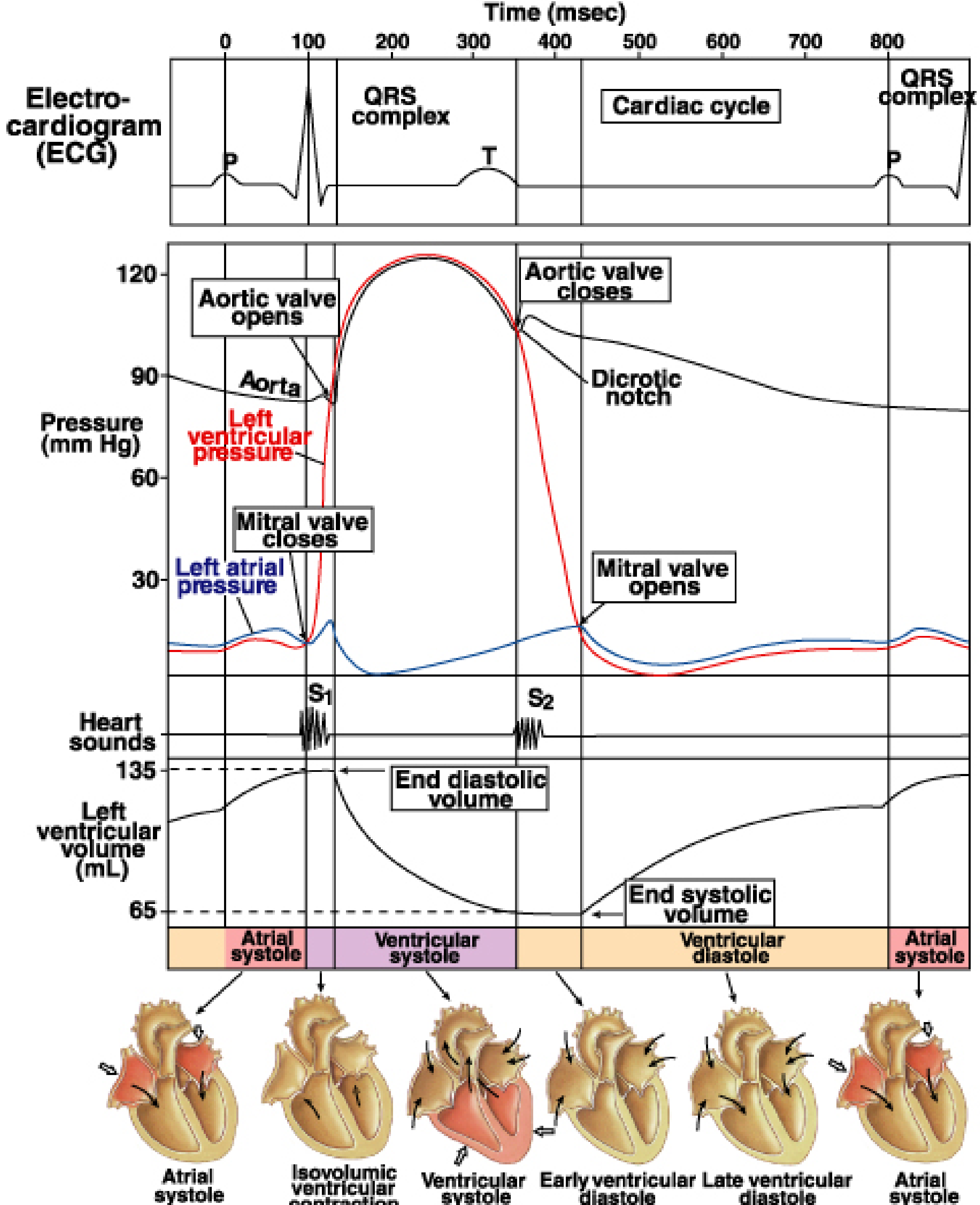
Ventricular systole



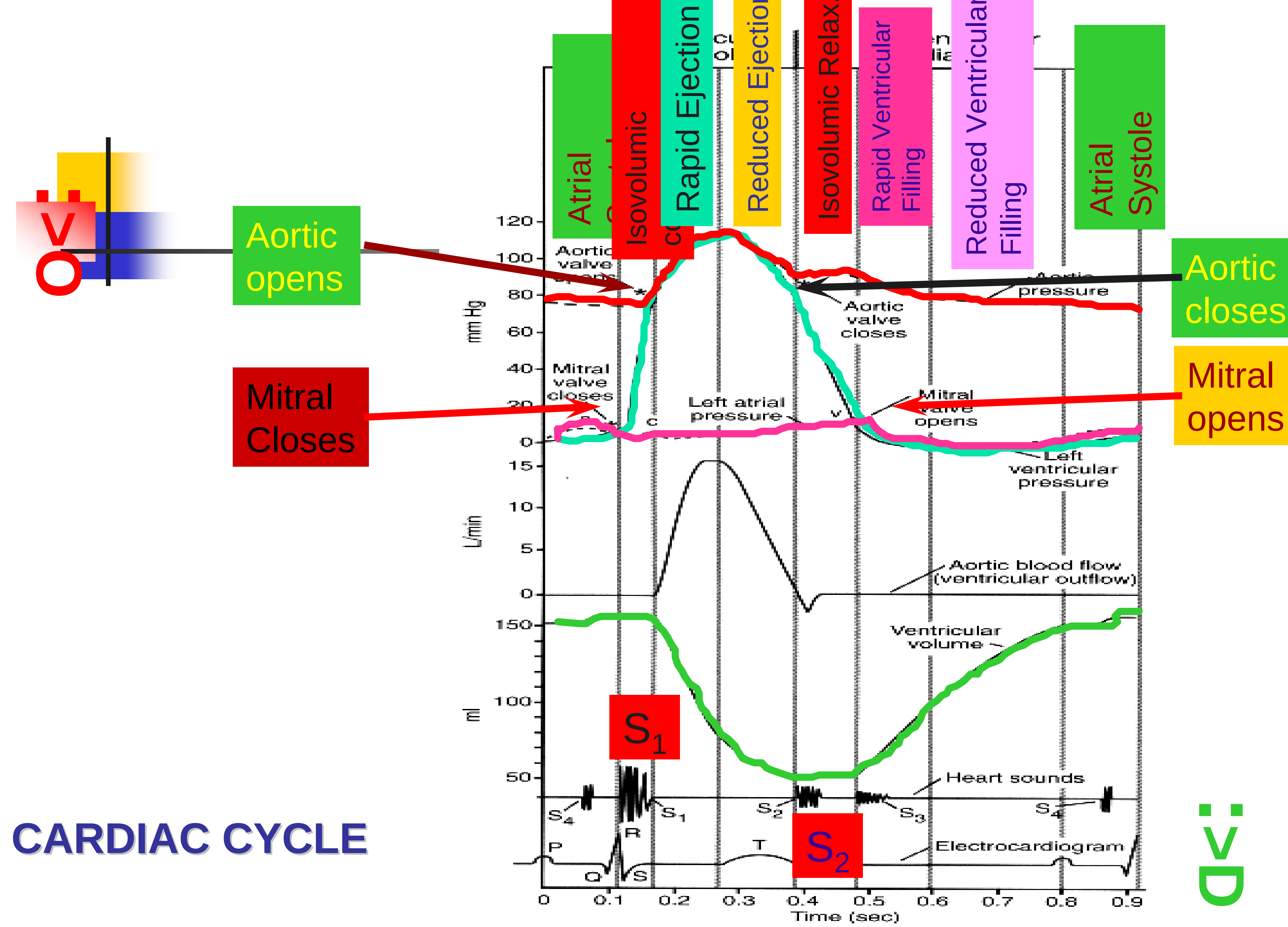
Events in the cardiac cycle

Valvular Events	Cardiac chamber Events	Phase
Opening of AV valves	**①Rapid(0.11s)／②(0.22s) reduced ventricular filling, ③Atrial contraction**	**Diastole, inflow phase**
Closing of AV valves	④ Isovolumetric ventricular contraction(0.05s)	Systole
Opening of semilunar valves	**⑤Rapid(0.1s)／⑥reduced ejection(0.15s)**	**Systole Outflow phase**
Closing of semilunar valves	⑦Isovolumetric ventricular relaxation (0.06-0.08s)	Diastole
Opening of AV valves	2nd cardiac cycle	

What are the key features of the cardiac cycle?



Time (msec)
0
100
200
300
400
500
600
700
800
Electro-cardiogram (ECG)
P
QRS complex
T
Cardiac cycle
P
QRS complex
120
90
60
30
Pressure (mm Hg)
Aortic valve opens
Aorta
Left ventricular pressure
Aortic valve closes
Dicrotic notch
Mitral valve closes
Left atrial pressure
Mitral valve opens
Heart sounds
S1
S2
135
65
Left ventricular volume (mL)
End diastolic volume
End systolic volume
Atrial systole
Ventricular systole
Ventricular diastole
Atrial systole
Atrial systole
Isovolumic ventricular contraction
Ventricular systole
Early ventricular diastole
Late ventricular diastole
Atrial systole



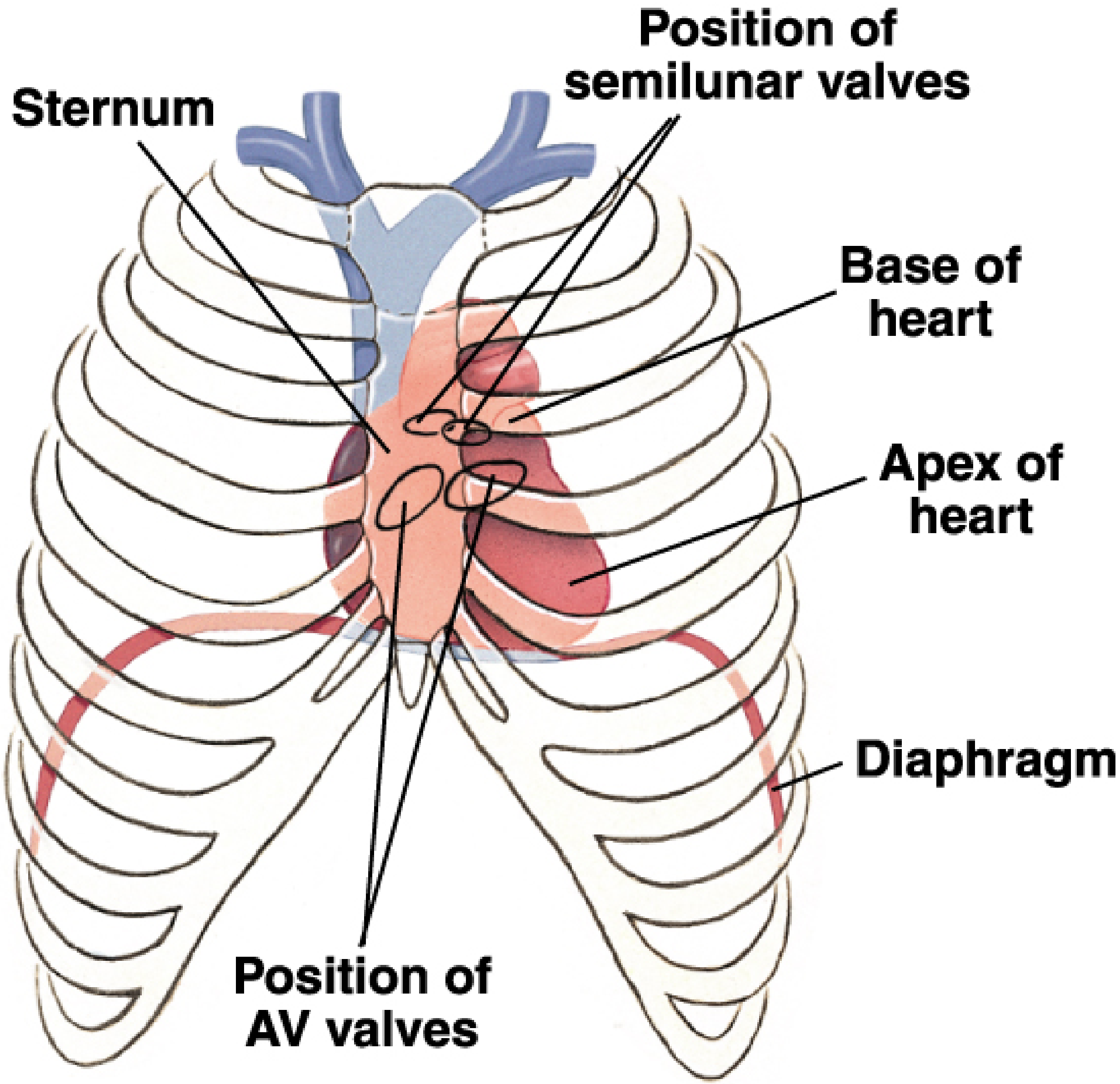
CARDIAC CYCLE

- the duration of systole is much more fixed than that of diastole, and when the heart rate is increased,diastole is shortened to a much greater degree.
- It is during diastole that the heart muscle rests, and coronary blood flow to the subendocardial portions of the left ventricle occurs only during diastole, most of the ventricular filling occurs in diastole.

	Heart Rate 75/min	Heart Rate 200/min	Skeletal Muscle
Duration, each cardiac cycle	0.80	0.30	. . .
Duration of systole	0.27	0.16	. . .
Duration of action potential	0.25	0.15	0.007
Duration of absolute refractory period	0.20	0.13	0.004
Duration of relative refractory period	0.05	0.02	0.003
Duration of diastole	0.53	0.14	. . .

First sound, caused by vibrations set up by the sudden closure of the mitral and tricuspid
valves at the start of ventricular systole, is a low, slightly prolonged “lub”

Second sound, caused by vibrations associated with closure of the aortic and pulmonary valves just after the end of ventricular systole, is a shorter, high-pitched “dup”



Cardiac Output

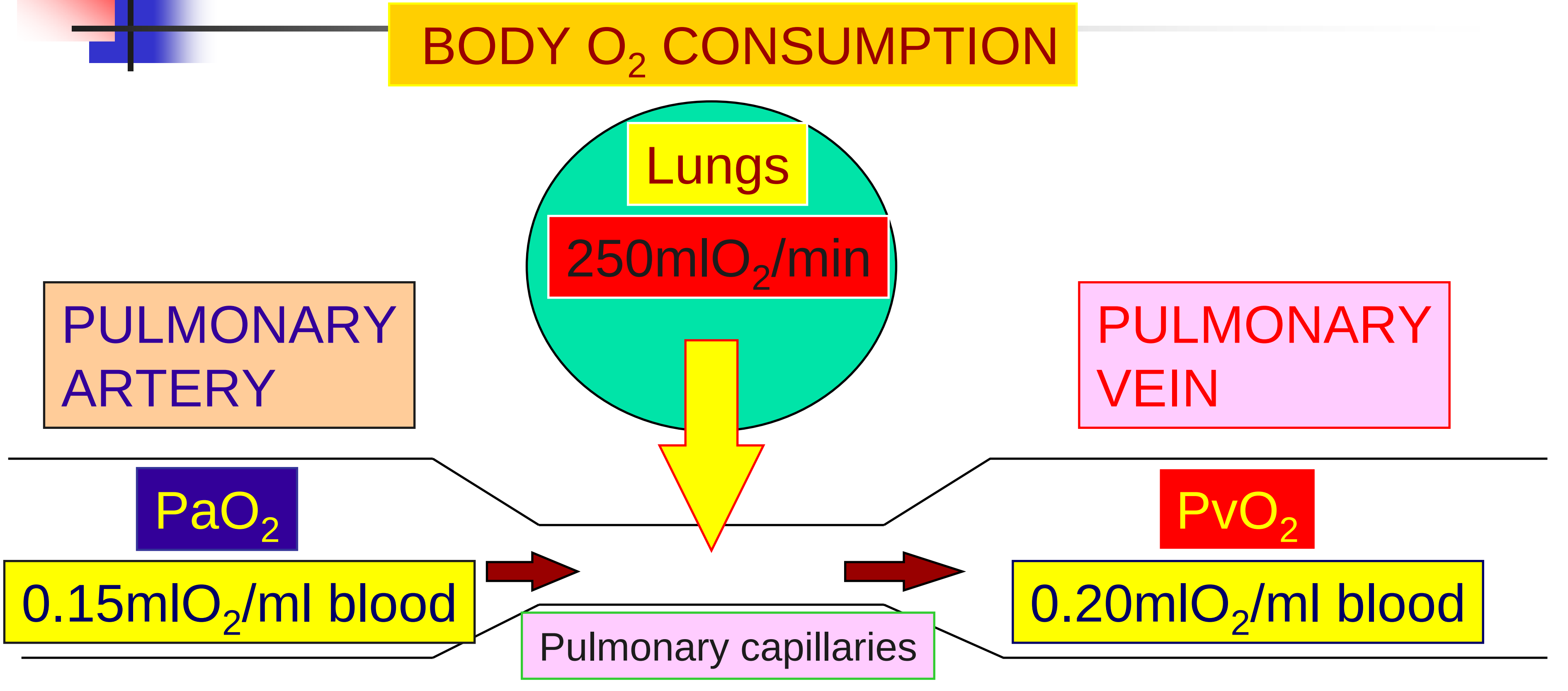
- Stroke volume(SV): the amount of blood pumped out of each ventricle per beat
- The SV in the left ventricle is exactly the same as those in the right ventricle
- Cardiac output :**t**he total volume of blood pumped by each ventricle per min.

  CO=SV• HR

Preload，Afterload

- Preload: is the degree to which the myocardium is stretched before it contracts （EDV）
- Afterload: is the resistance against which blood is expelled （aortic BP ）
- Ventricular contractility
- Heart rate

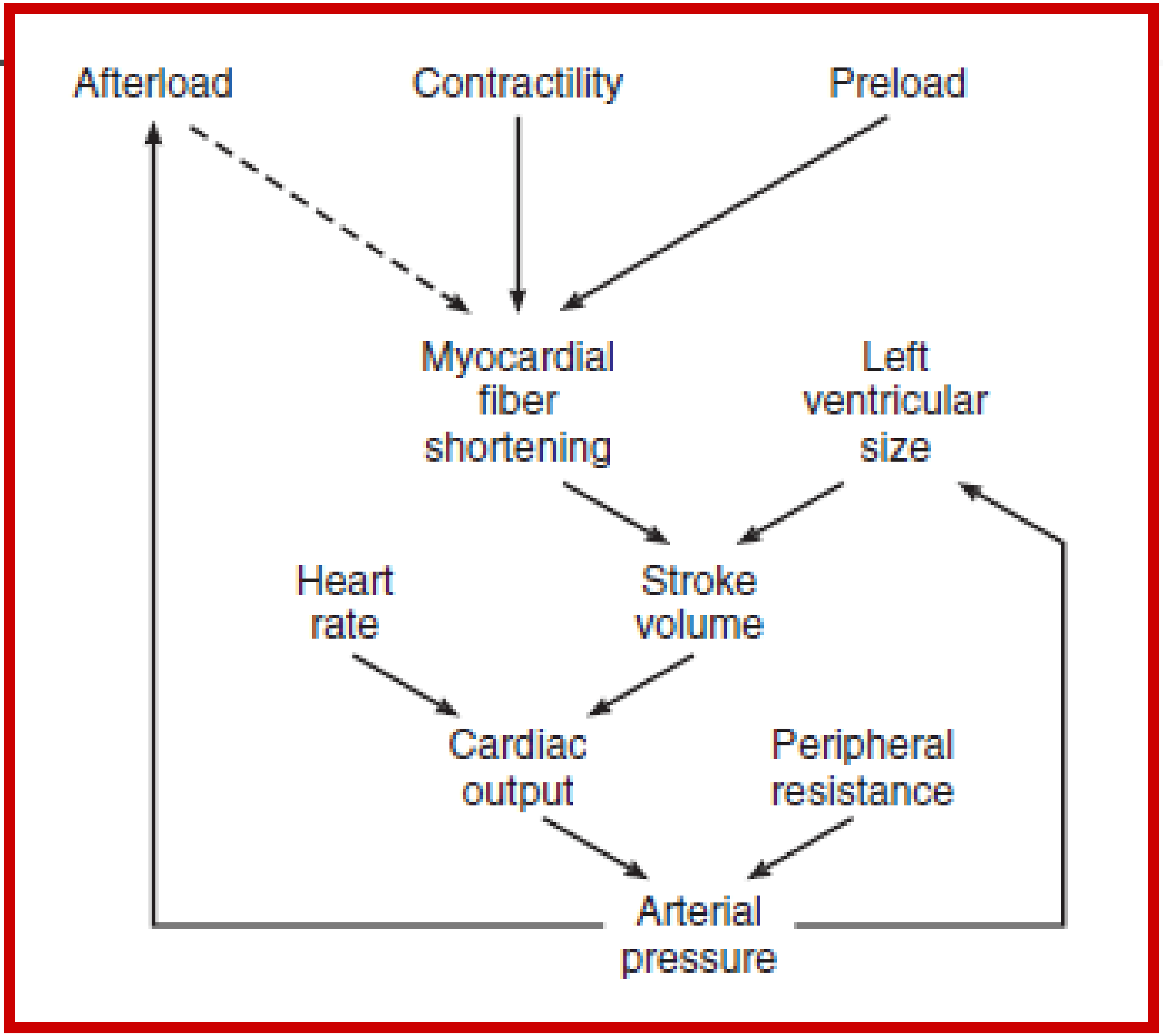
Cardiac output and the fick

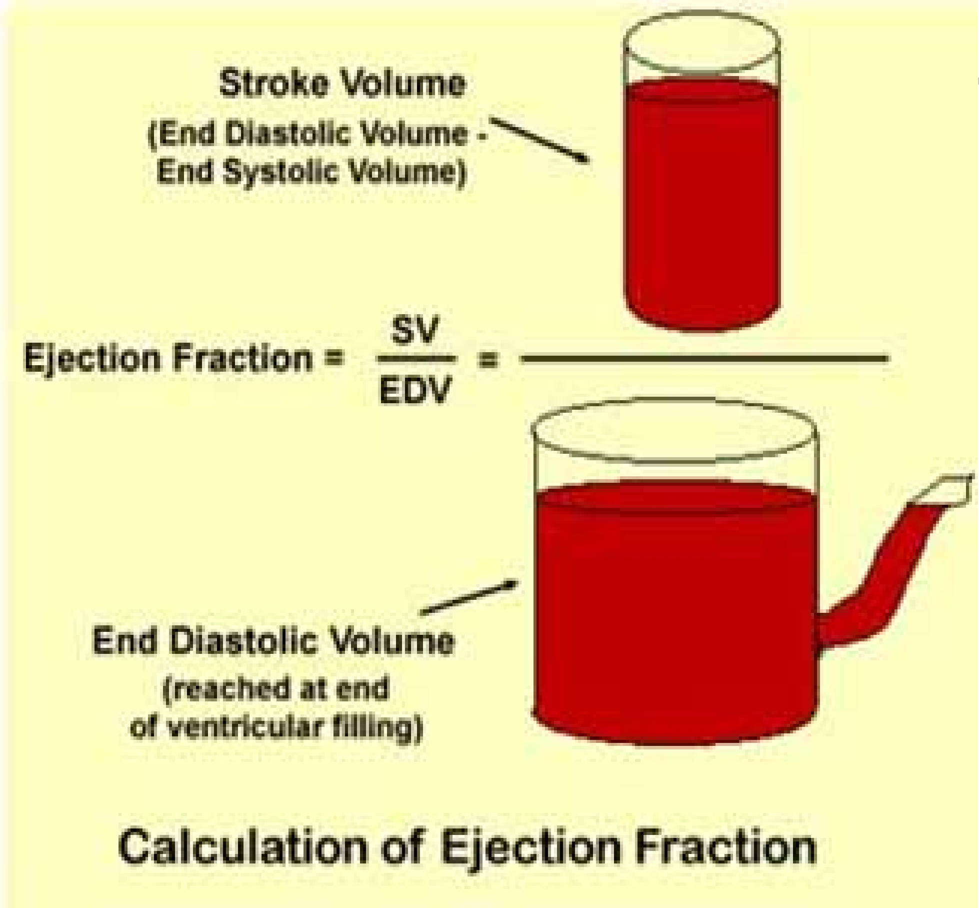


$$\text{CARDIAC OUTPUT} = \frac{O_2 \text{ CONSUMPTION (ml/min)}}{PvO_2 - PaO_2}$$

Parameter	Value
end-diastolic volume (EDV)	135 ml
end-systolic volume (ESV)	65 ml
stroke volume (SV)	70 ml
ejection fraction (E_f)	58%
heart rate (HR)	70 bpm
cardiac output (CO)	5.0 L/m

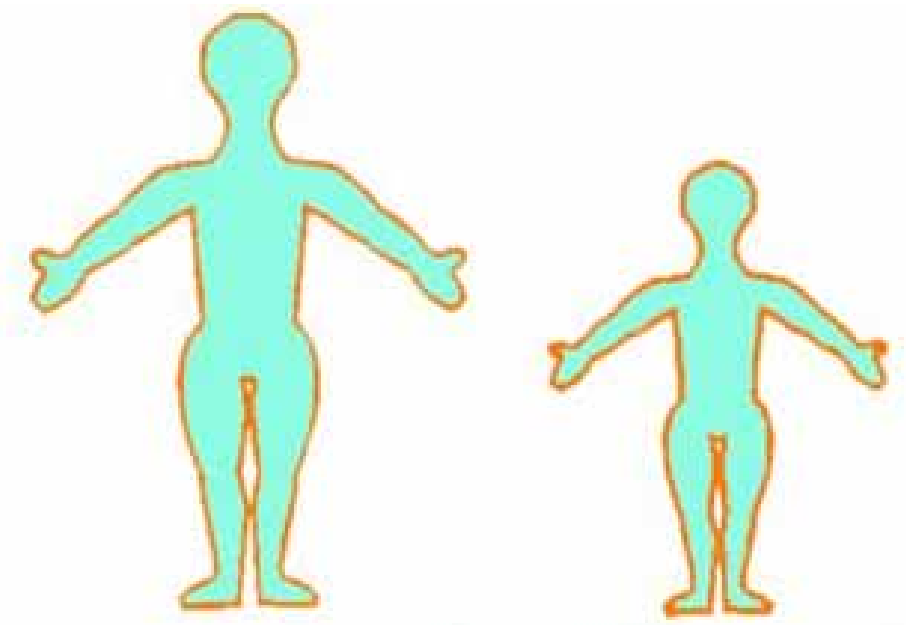
Factors Controlling Cardiac Output





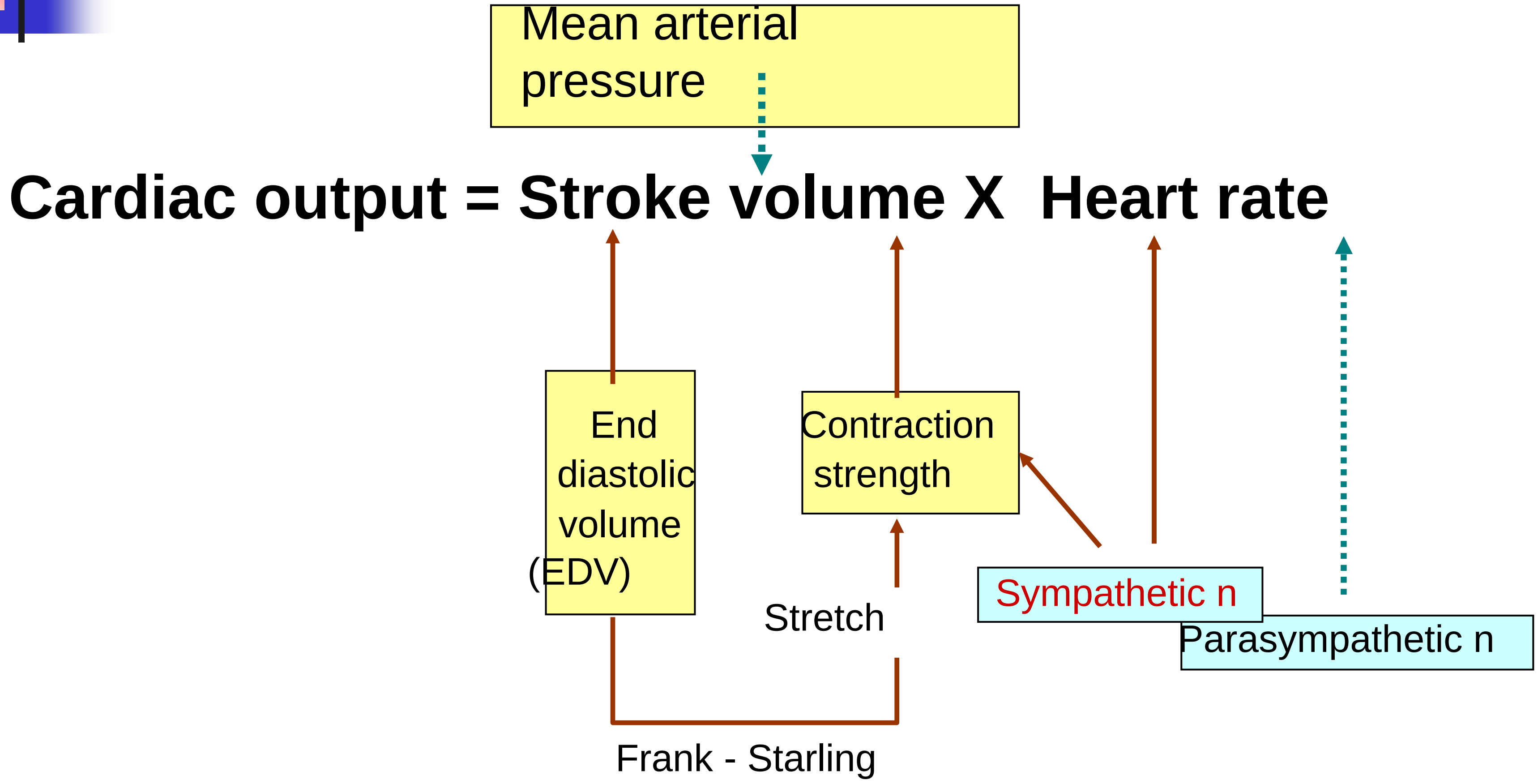
Ejection Fraction= $\frac{SV}{EDV}$ (58%)

Cardiac Index



Cardiac index :The output per minute per square meter of body surface , averages 3.2 L／min ／m^2

Regulation of the CO:



•Relation between ***SV*** and ***EDV*** ***is called*** ***Frank-Starling curve*** ***ie, heterometric regulation (intrinsic control). Energy of contraction is proportional to the initial length of the cardiac muscle fiber.” Ventricle stretch more, it’ll contract with more force.***

Increased venous return

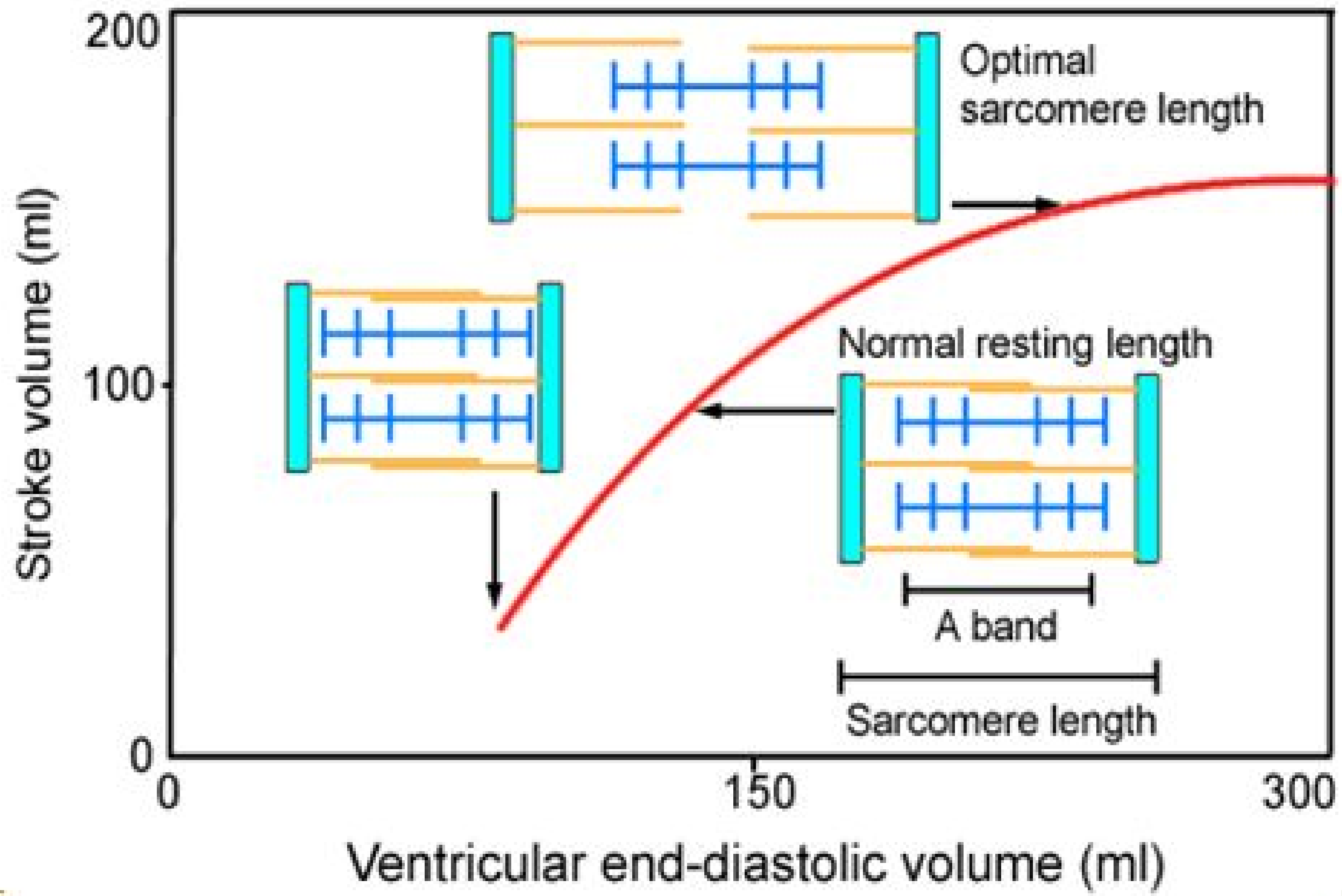
↓

Increased EDV

↓

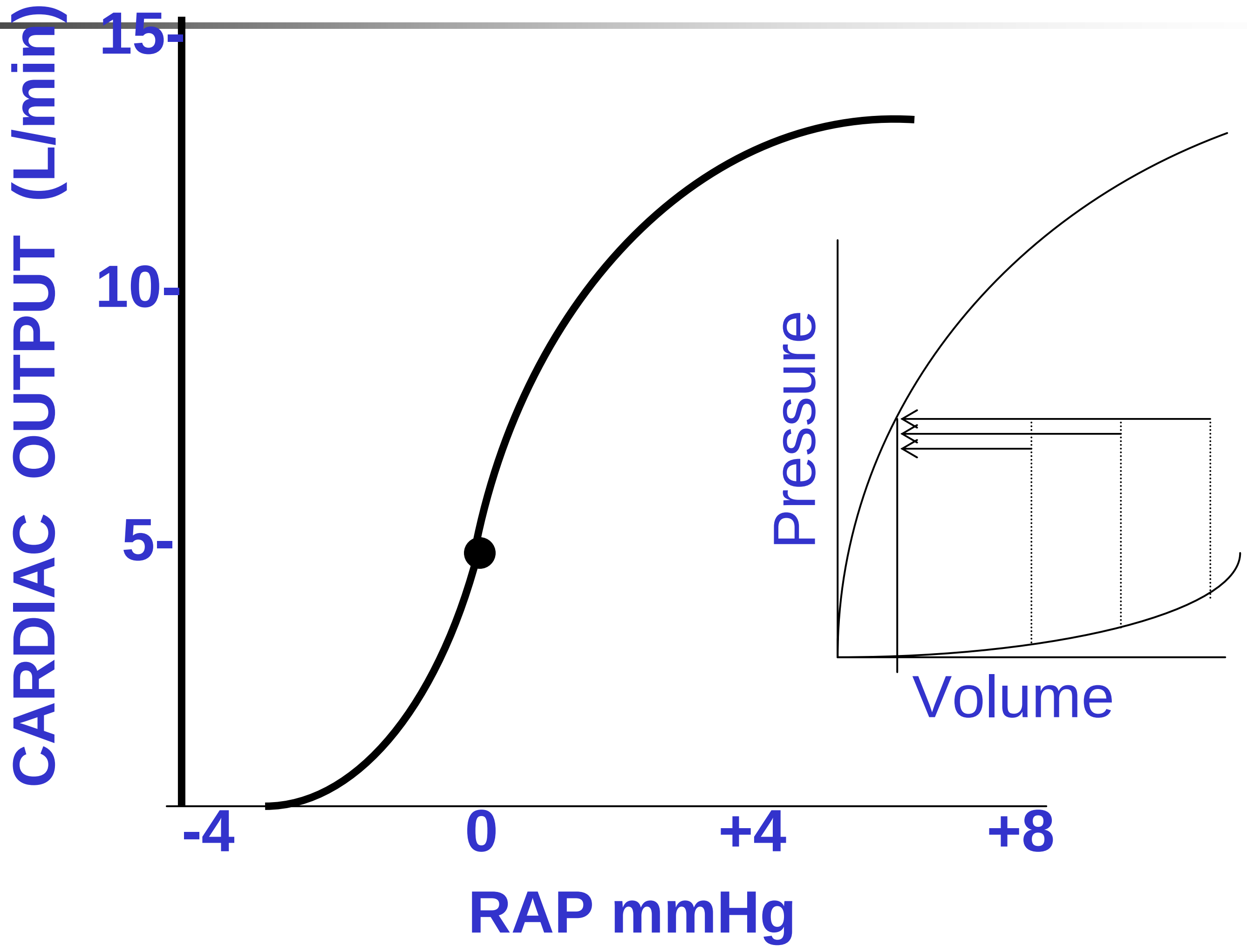
Increased stretch and a greater force of contraction

Ventricular function curve (Frank-Starling curve)



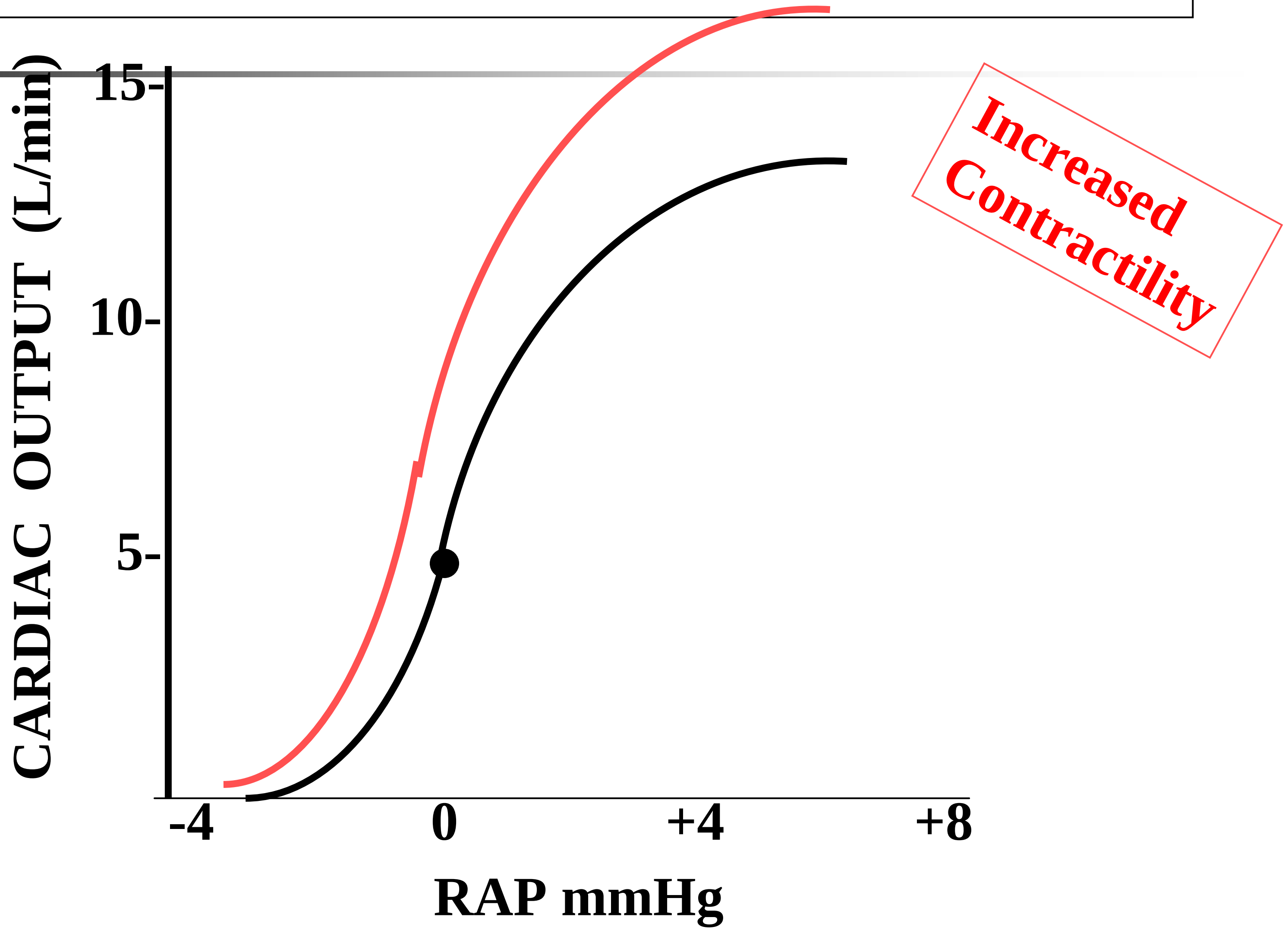
Cardic Function Curve

THE FRANK- STARLING "LAW OF THE HEART"



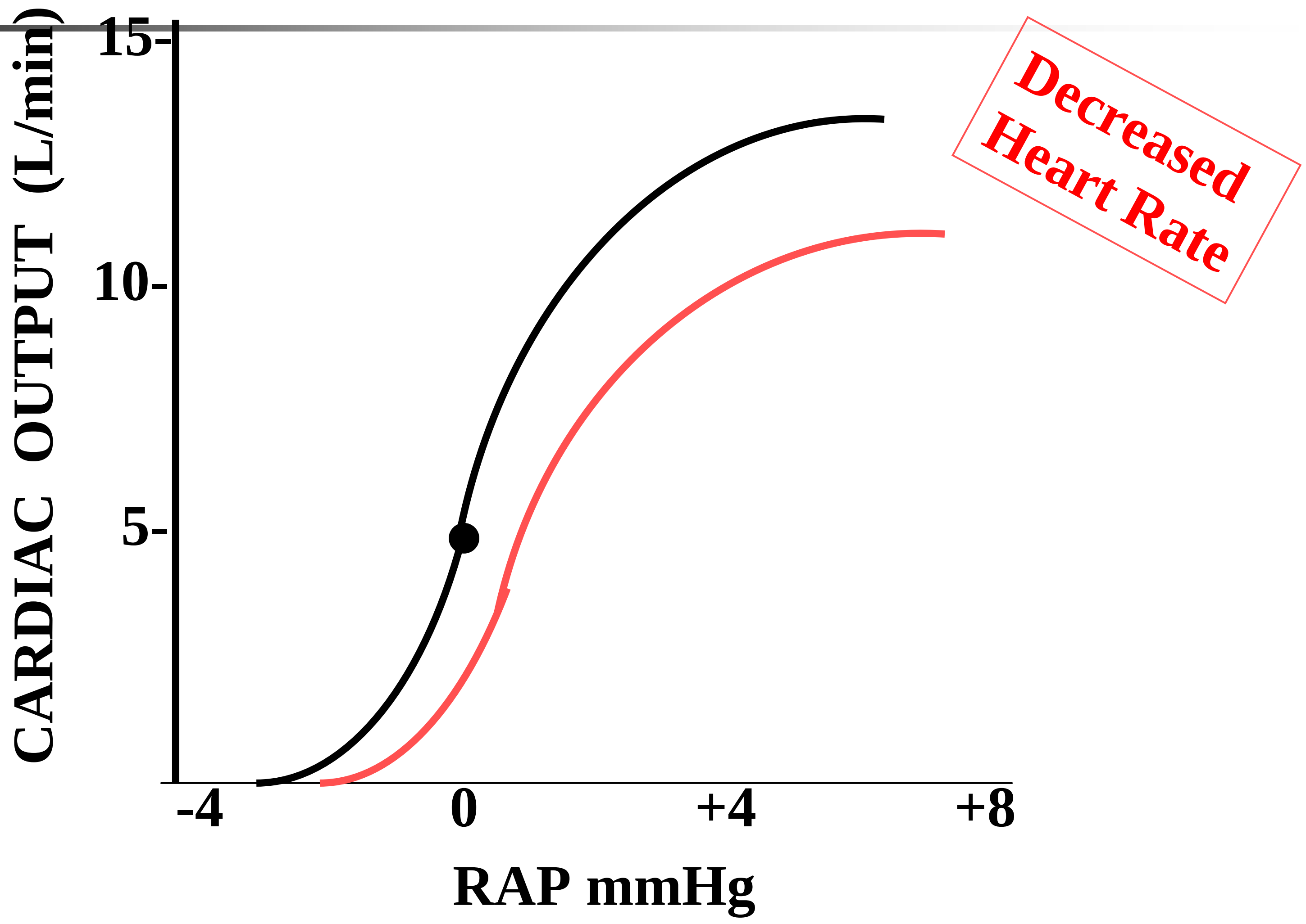
Cardic Function Curve

THE FRANK- STARLING "LAW OF THE HEART"



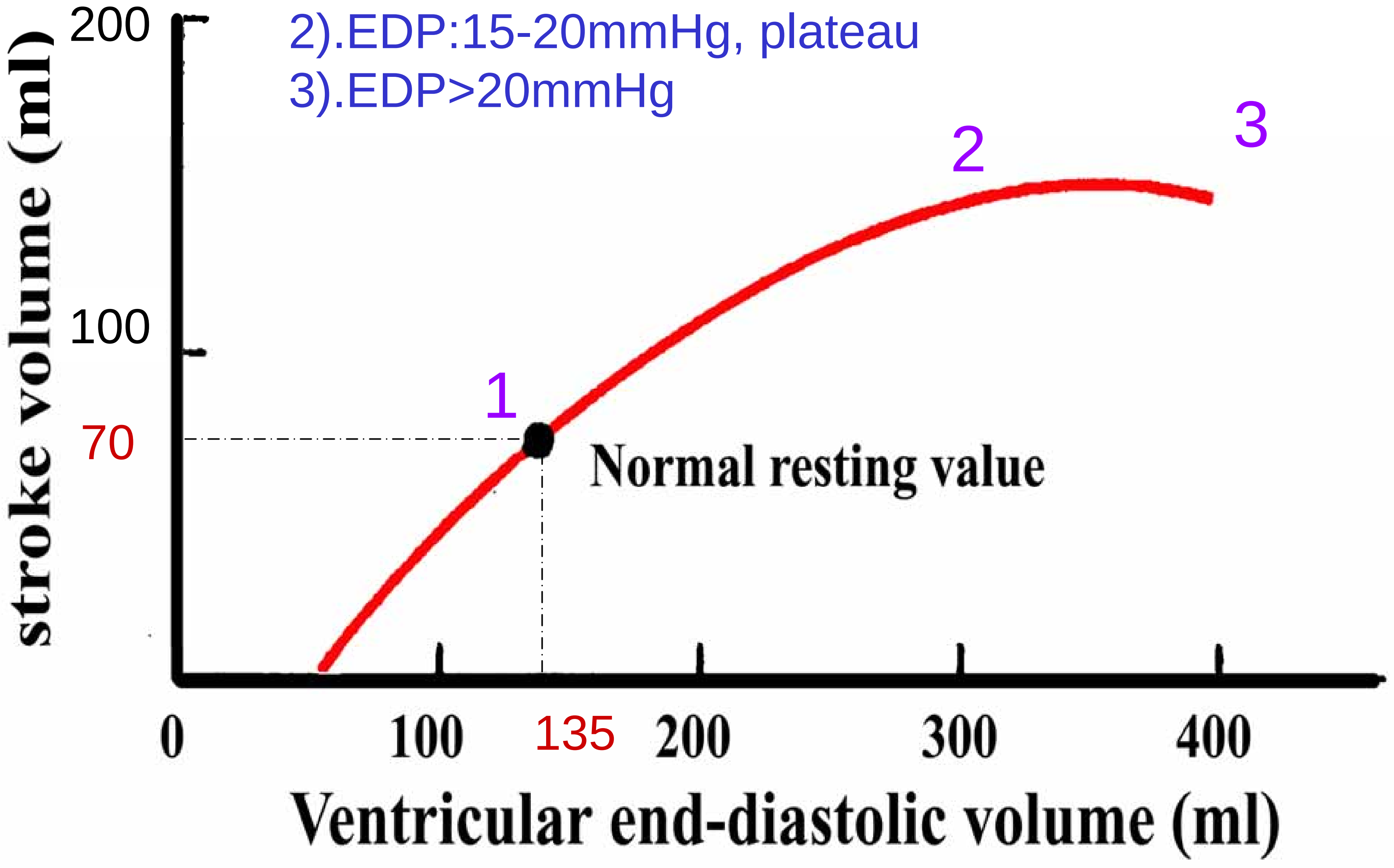
Cardic Function Curve

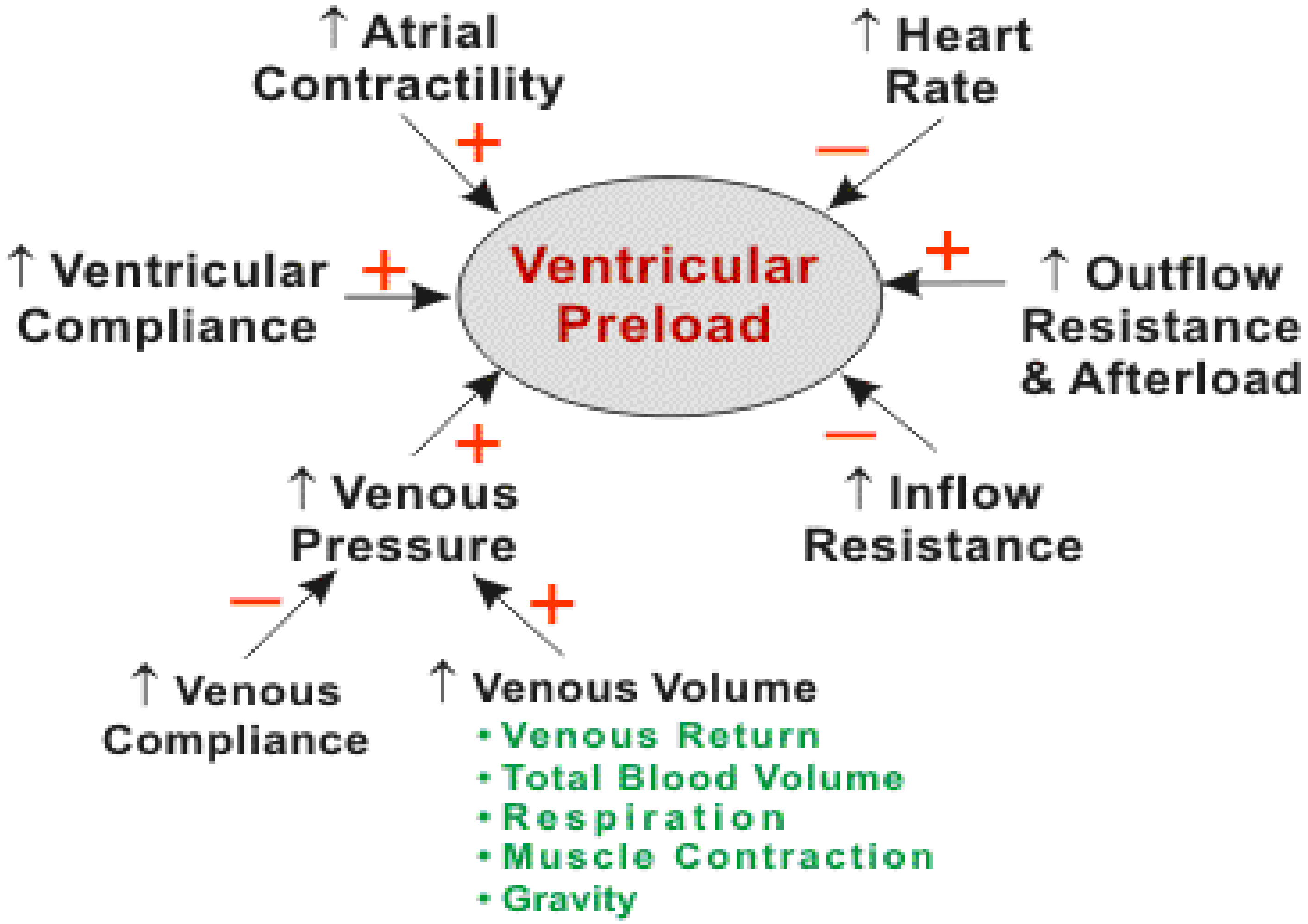
THE FRANK- STARLING "LAW OF THE HEART"



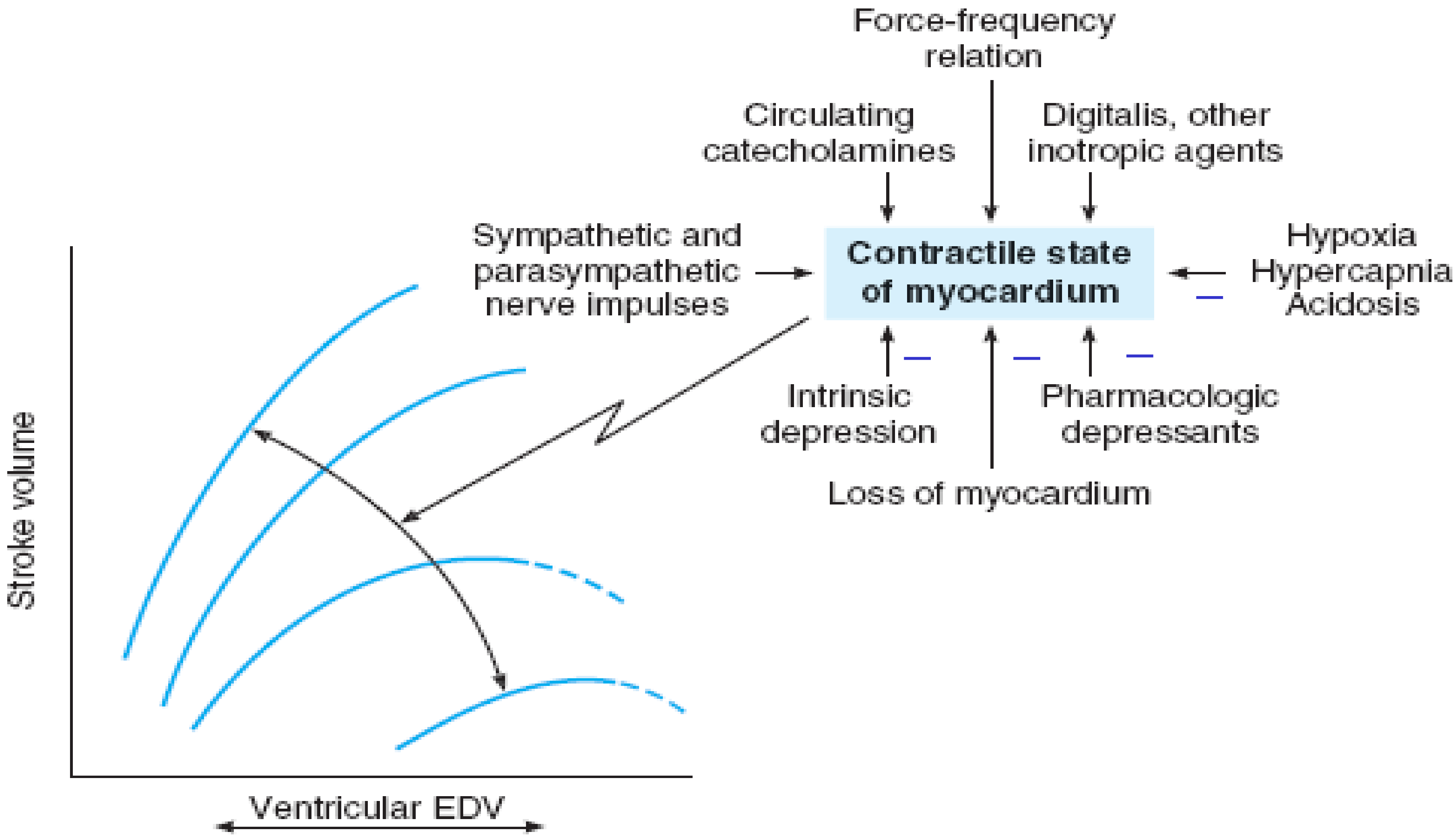
Length-force relationship in the intact heart

1).EDP:5-6 mmHg,
2).EDP:15-20mmHg, plateau
3).EDP>20mmHg



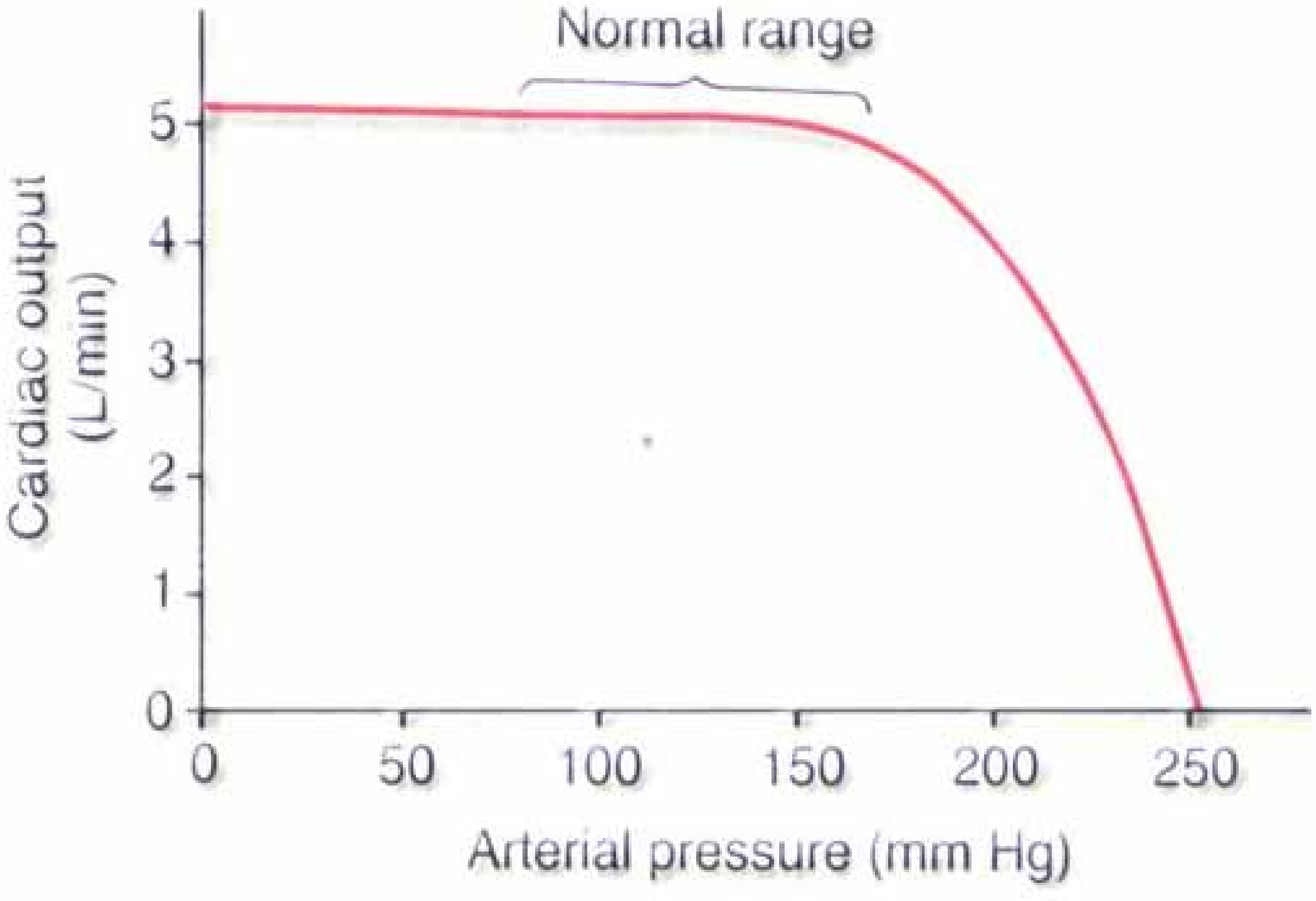


Factors determining ventricular preload. A "+" sign indicates that an increase in this particular variable increases ventricular end-diastolic volume, and therefore preload, while the "-" indicates that the variable decreases preload.



The dashed lines indicate portions of the ventricular function curves where maximum contractility has been exceeded; ie, they identify points on the "descending limb "of the Frank–Starling curve.

After load: arterial pressure



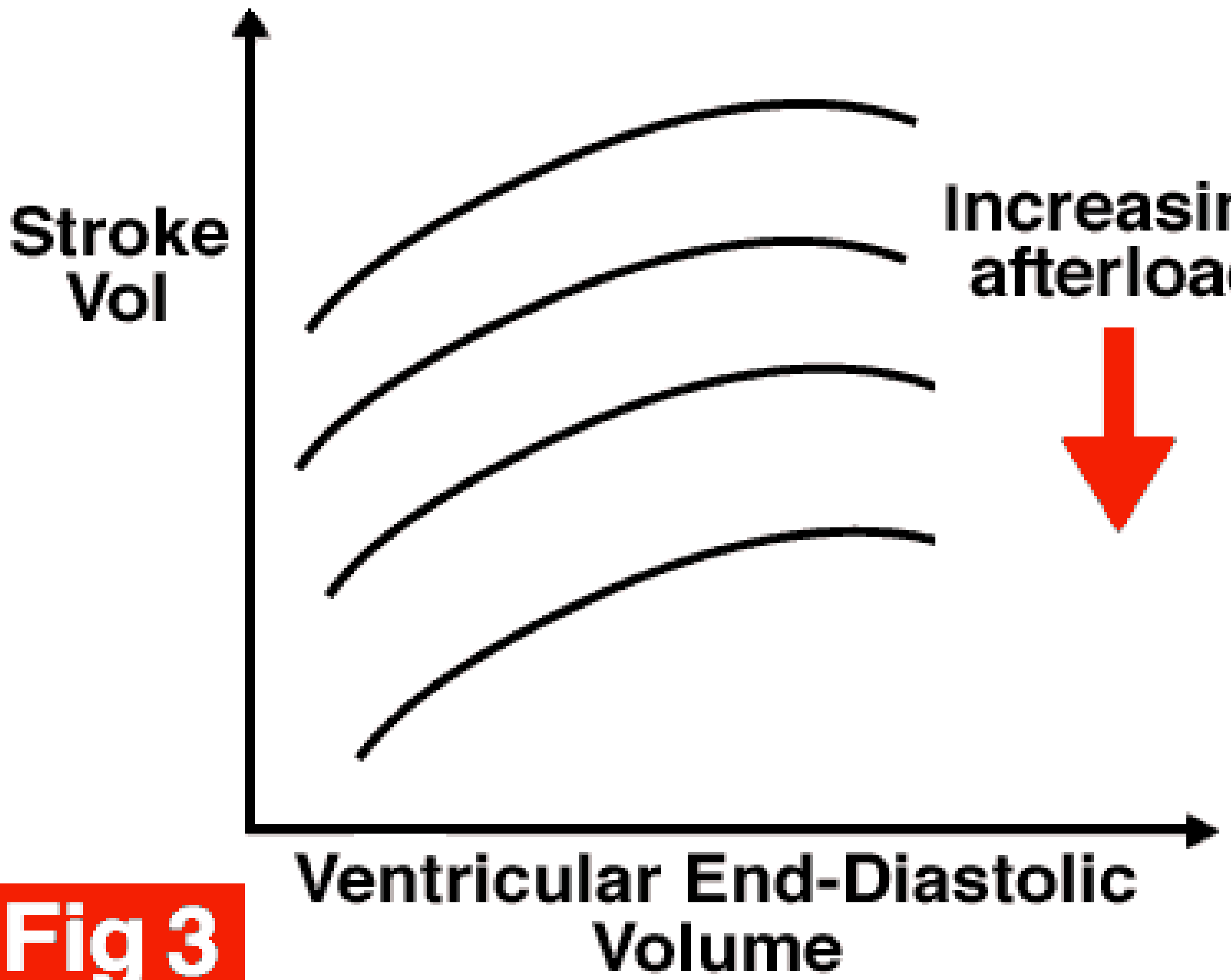

Stroke Vol
Increasing afterload
Ventricular End-Diastolic Volume

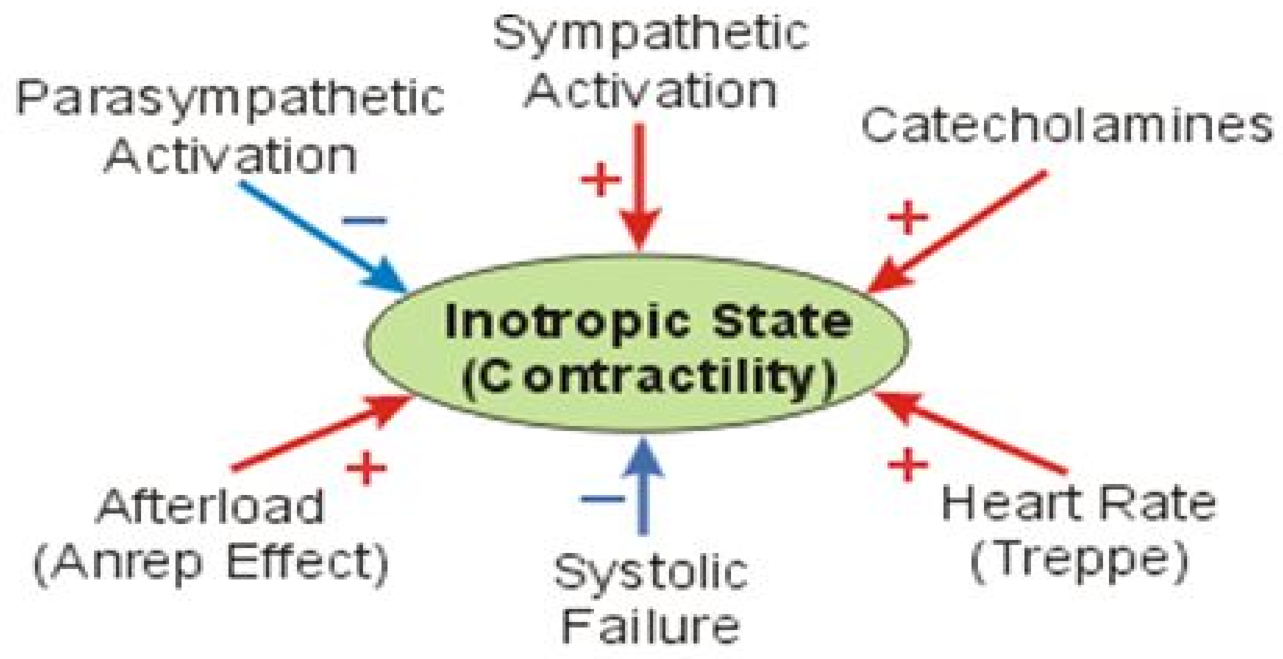
Fig 3

Rrgulation of contrctility

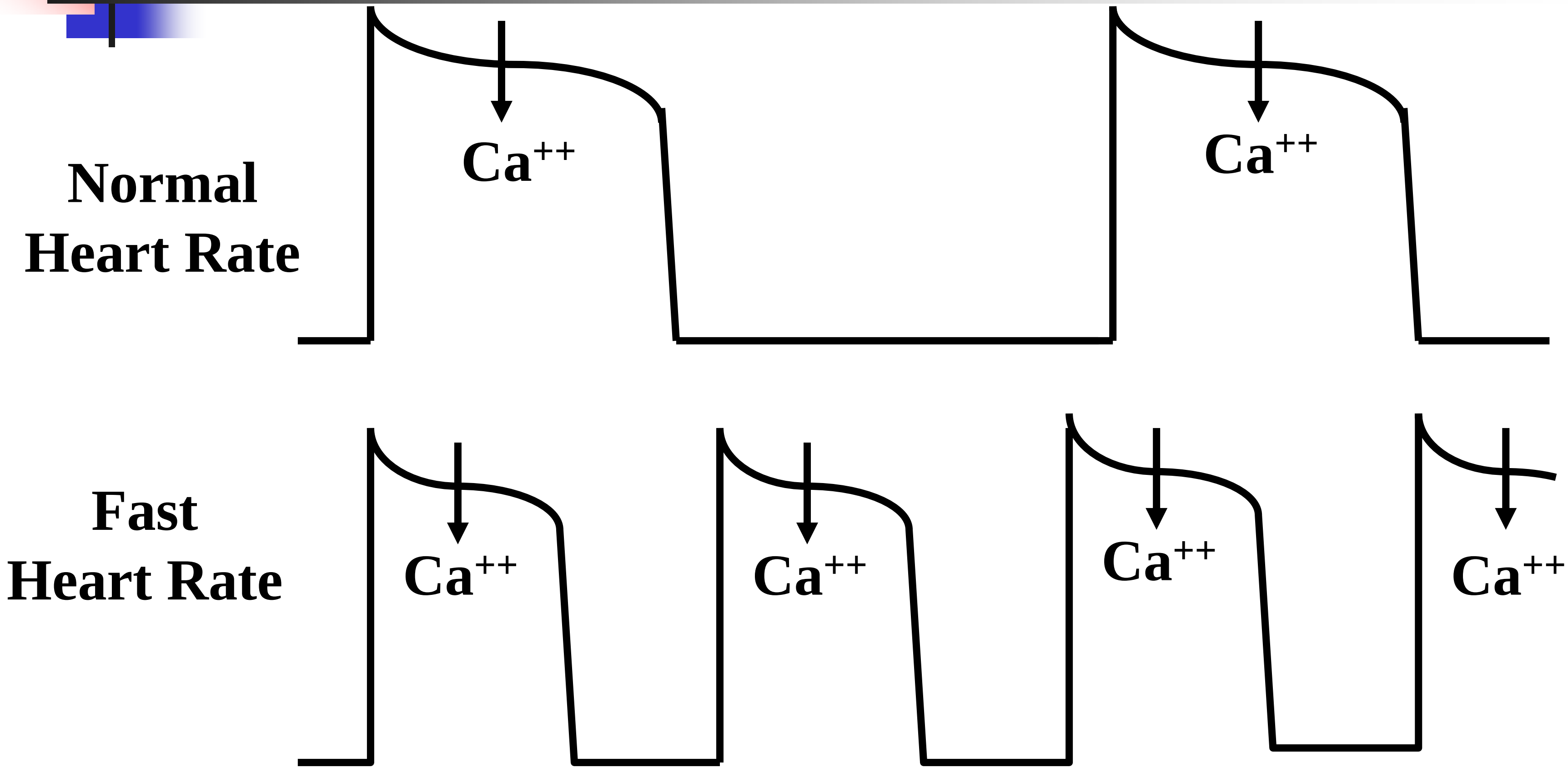
- Recruitment of motor units
- Increase frequency of firing of motor nerves
- Calcium to trigger contraction

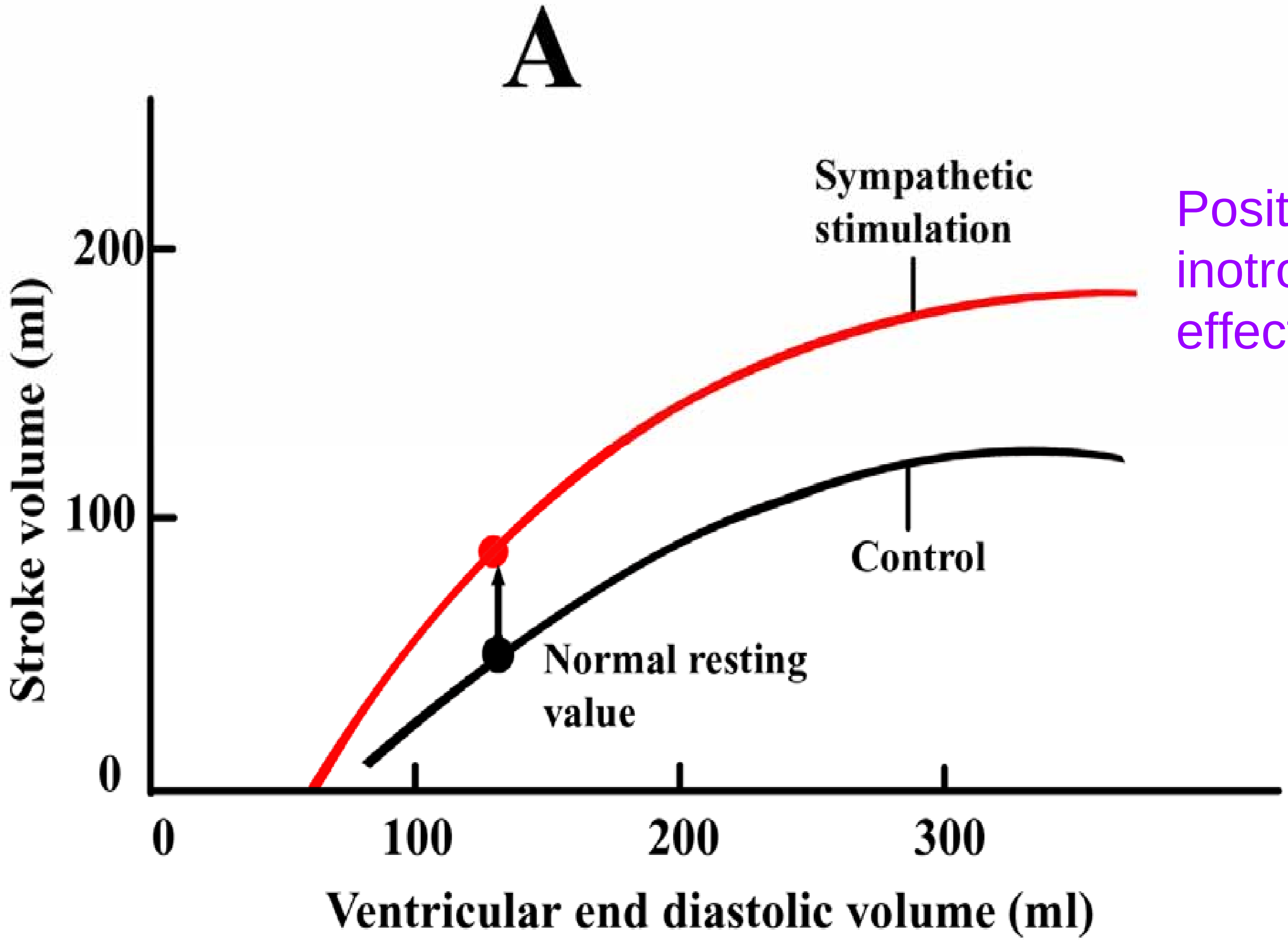
Myocardial contractility (Inotropic state)

Factors regulating contractility



Increasing heart rate increases contractility





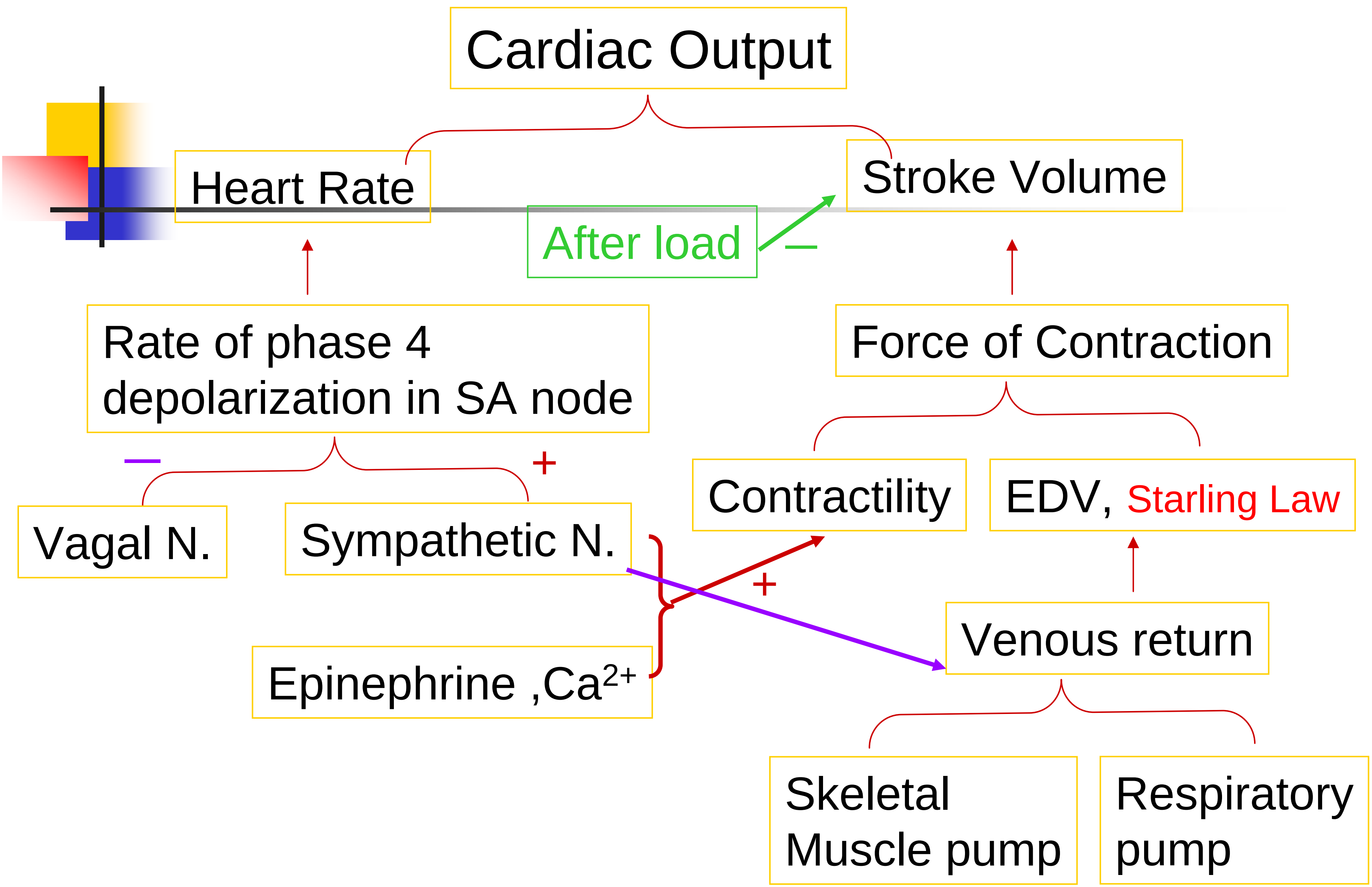
Positive inotropic effect

Regulation of the heart rate

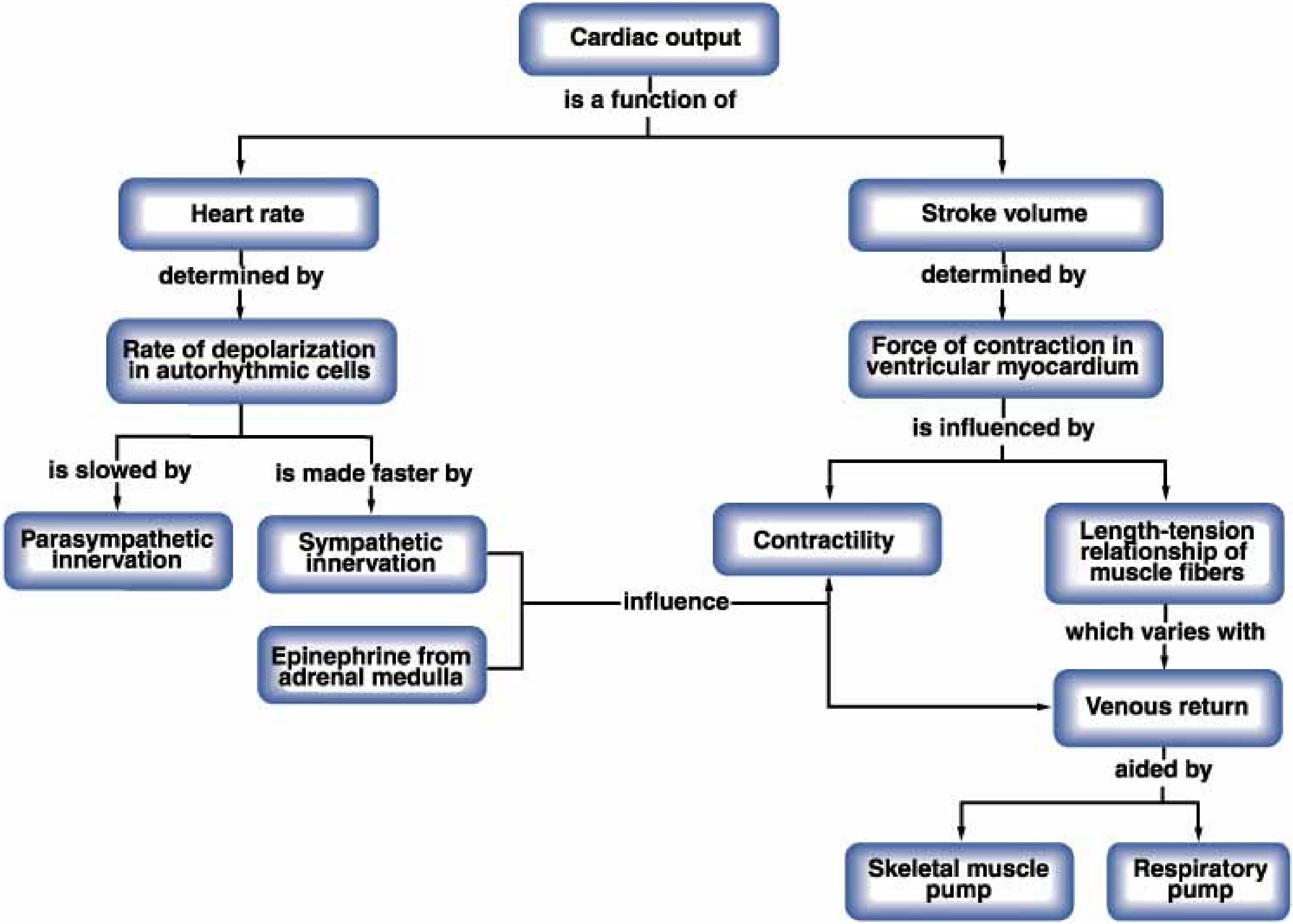
- HR↑ → CO↑
- HR↑ → contractility↑ (Treppe effect)
- HR↑ → diastolic filling time↓
- HR>180／min, or<40／min, CO↓
- HR: 40~180／min, HR↑ → CO↑

Pumping Work

- The product of the force applied to an object and the distance the object move: W=force·distance
- W= Pa(EDV-ESV)=Pa·SV
- Pumping work of right ventricle :as one fifth as large.



Cardiac Output
Heart Rate
Stroke Volume
After load
—
Rate of phase 4 depolarization in SA node
Force of Contraction
—
+
Vagal N.
Sympathetic N.
Contractility
EDV, Starling Law
+
Epinephrine ,Ca2+
Venous return
Skeletal Muscle pump
Respiratory pump


Cardiac output
is a function of
Heart rate
Stroke volume
determined by
determined by
Rate of depolarization in autorhythmic cells
Force of contraction in ventricular myocardium
is influenced by
is slowed by
is made faster by
Parasympathetic innervation
Sympathetic innervation
Contractility
Length-tension relationship of muscle fibers
influence
which varies with
Epinephrine from adrenal medulla
Venous return
aided by
Skeletal muscle pump
Respiratory pump