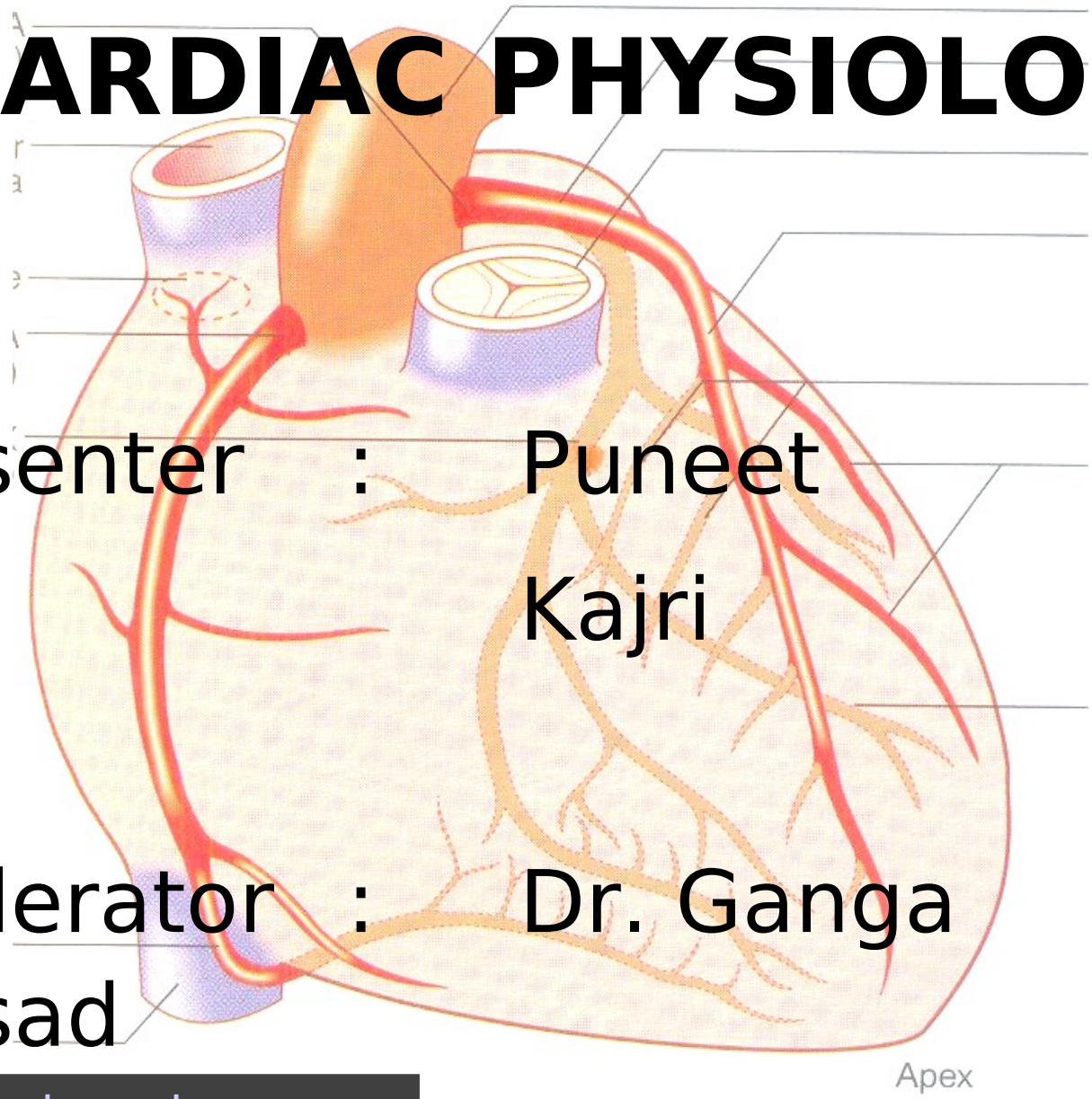
CARDIAC PHYSIOLOGY



Presenter : Puneet Kajri

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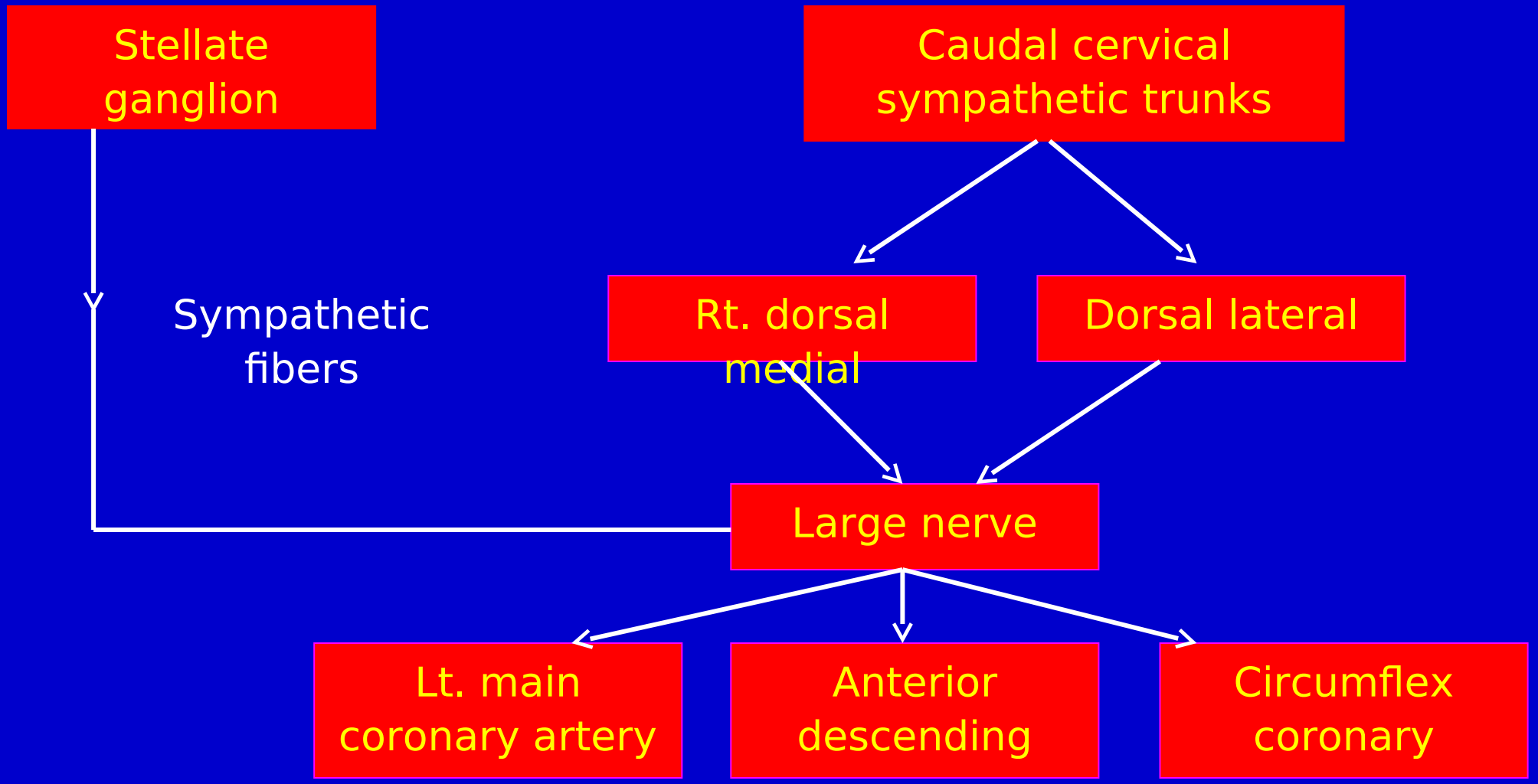
CONTENTS

- Nerve supply of the heart
- Neural control of heart rate
- Contractility – systolic and diastolic function
- Baroreceptor reflex
- Chemoreceptor reflex
- Bain bridge atrial reflex
- Von Bezold-Jarisch reflex
- Valsalva maneuver
- Cushing reflex
- Occulo cardiac reflex

NERVE SUPPLY OF THE HEART

- Nerves to the heart originate from sympathetic neurons of thoracolumbar region and parasympathetic nerves originate from the cervical region.

SYMPATHETIC SYSTEM

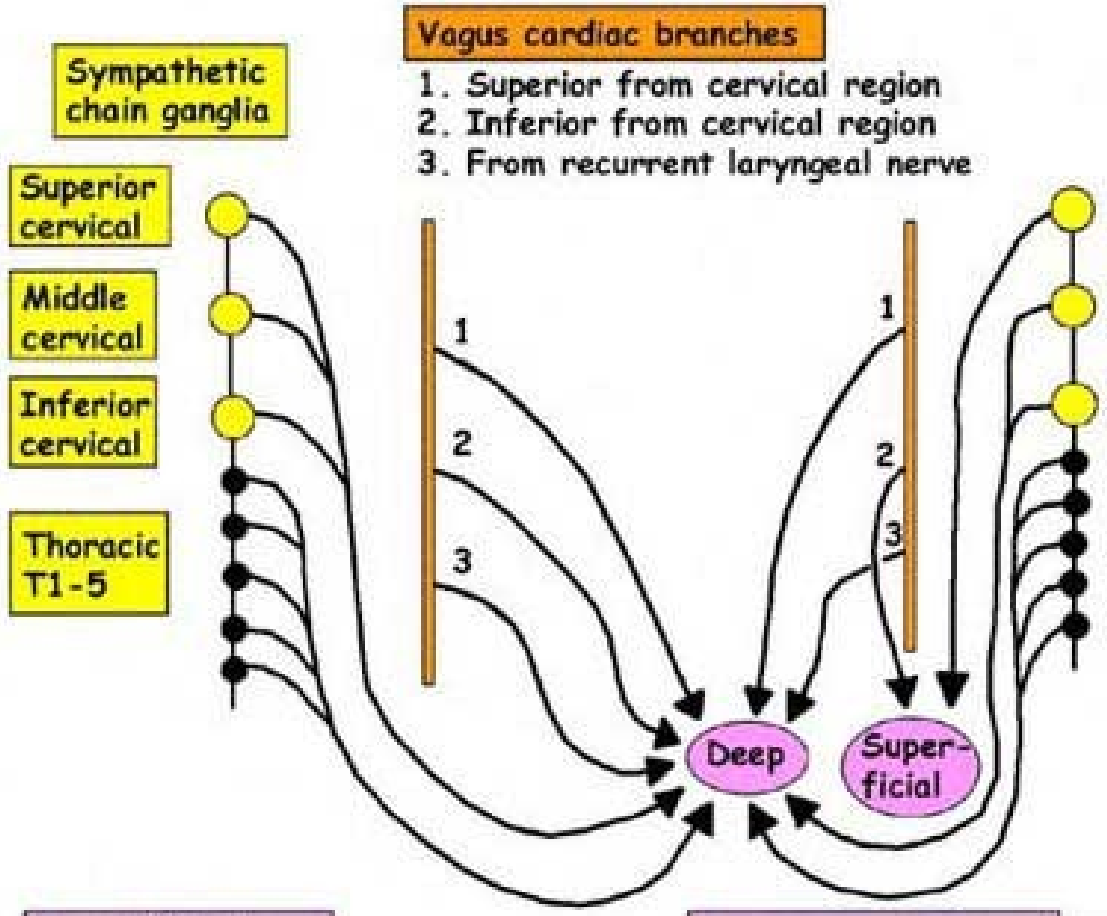


PARASYMPATHETIC SYSTEM

- Parasympathetic: Preganglionic neurons arise in the medulla oblongata in the dorsal vagal nucleus ambiguous
- Fibres enter the thorax as branches from the recurrent laryngeal and thoracic vagus nerves
- Parasympathetic nerves are particularly abundant near the coronary sinus and SVC
- Ganglia occur within the heart usually close to the structures innervated by short post ganglionic neurons

- Afferent nerves from the heart ascend vial 10th cranial nerve and spinal cord to the nucleus tractus solitarius and the dorsal vagal nucleus and nucleus ambiguous to form parasympathetic motor efferent system of medullary vasomotor center
- Sympathetic activation of heart originates in rostral ventrolateral medulla, ventromedial rostral medulla and parvocellular region of paraventricular nucleus

AUTONOMIC NERVE SUPPLY TO HEART



Superficial plexus

Lies on aortic arch between phrenic and vagus nerves

Deep plexus

Lies to right of ligamentum arteriosum, inferior & medial to aortic arch

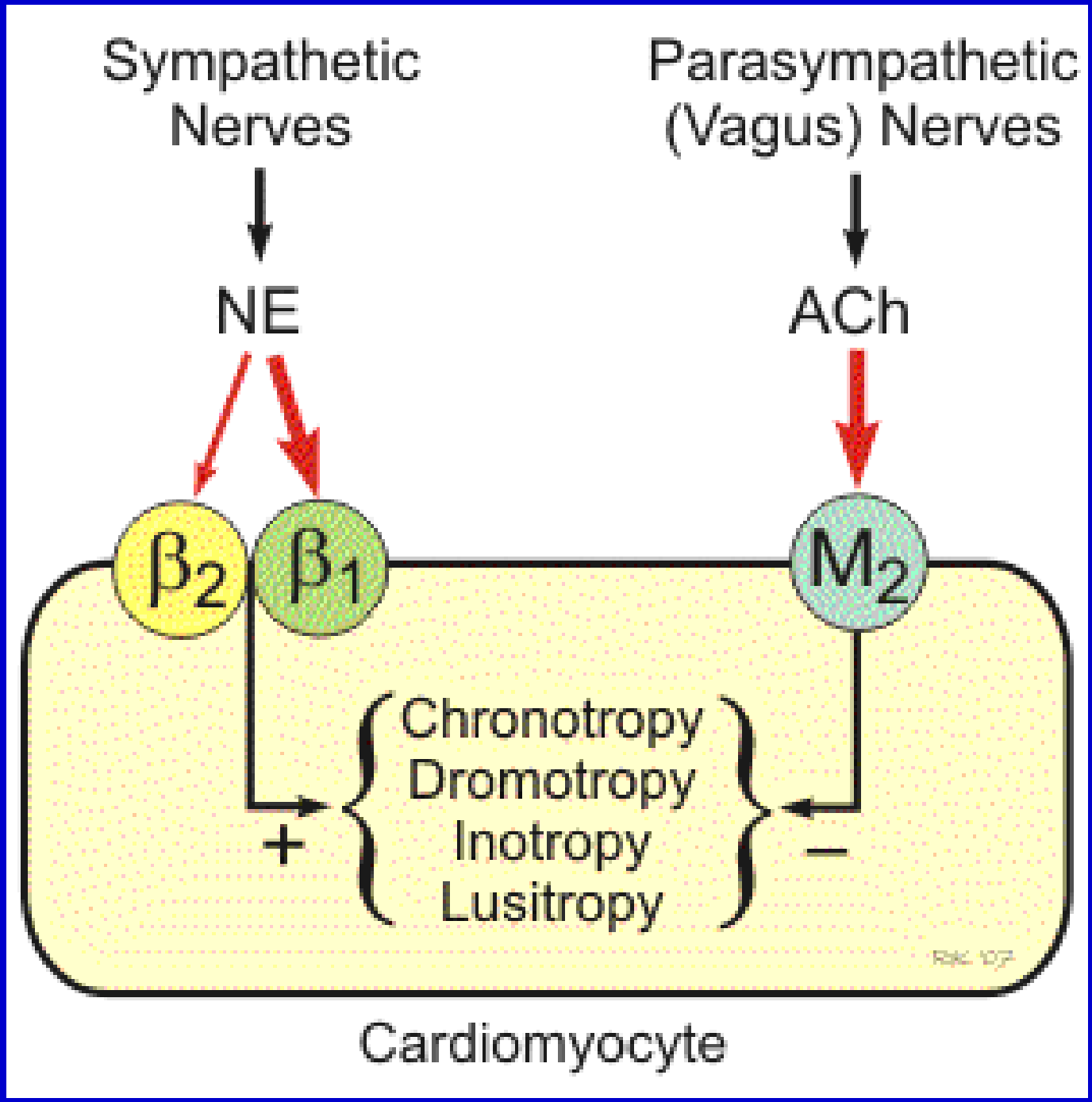
NERVE SUPPLY OF THE HEART

- Both the parasympathetic and sympathetic nerves form the superficial and deep cardiac plexuses
- The superficial cardiac plexus is situated below the arch of aorta in front of the right pulmonary artery, it is formed by –
  - The superior cervical cardiac branch of the left sympathetic chain
  - The inferior cervical cardiac branch of left vagus
- It gives branches to
  - The deep cardiac plexus
  - The right coronary artery
  - The left anterior pulmonary plexus

- The deep cardiac plexus is situated in front of the bifurcation of trachea and behind the arch of aorta, it is formed by -
  - All the cardiac branches derived from the cervical and upper thoracic ganglia of sympathetic chain
  - Cardiac branches of vagus and recurrent laryngeal nerves except those which form the superficial plexus
- Right and left halves of plexus distribute branches to the corresponding coronary and pulmonary plexus. Separate branches are given to atria

CARDIAC RECEPTORS

- Most important adrenoreceptor is heart is B1
- B2 adrenoreceptor in heart has similar cardiac effect B1
- Prejunctional α2 adrenoreceptor inhibit NE release
- Prejunctional β2 adrenoreceptor facilitate NE release
- Prejunctional M2 adrenoreceptor inhibit NE release
- Right atrium – 74% β1 and 26% β2
- Ventricles – 86% β1 and 14% β2


Sympathetic Nerves
Parasympathetic (Vagus) Nerves
NE
ACh
β2
β1
M2
Chronotropy
Dromotropy
Inotropy
Lusitropy
+
–
Cardiomyocyte

NEURAL CONTROL OF HEART RATE

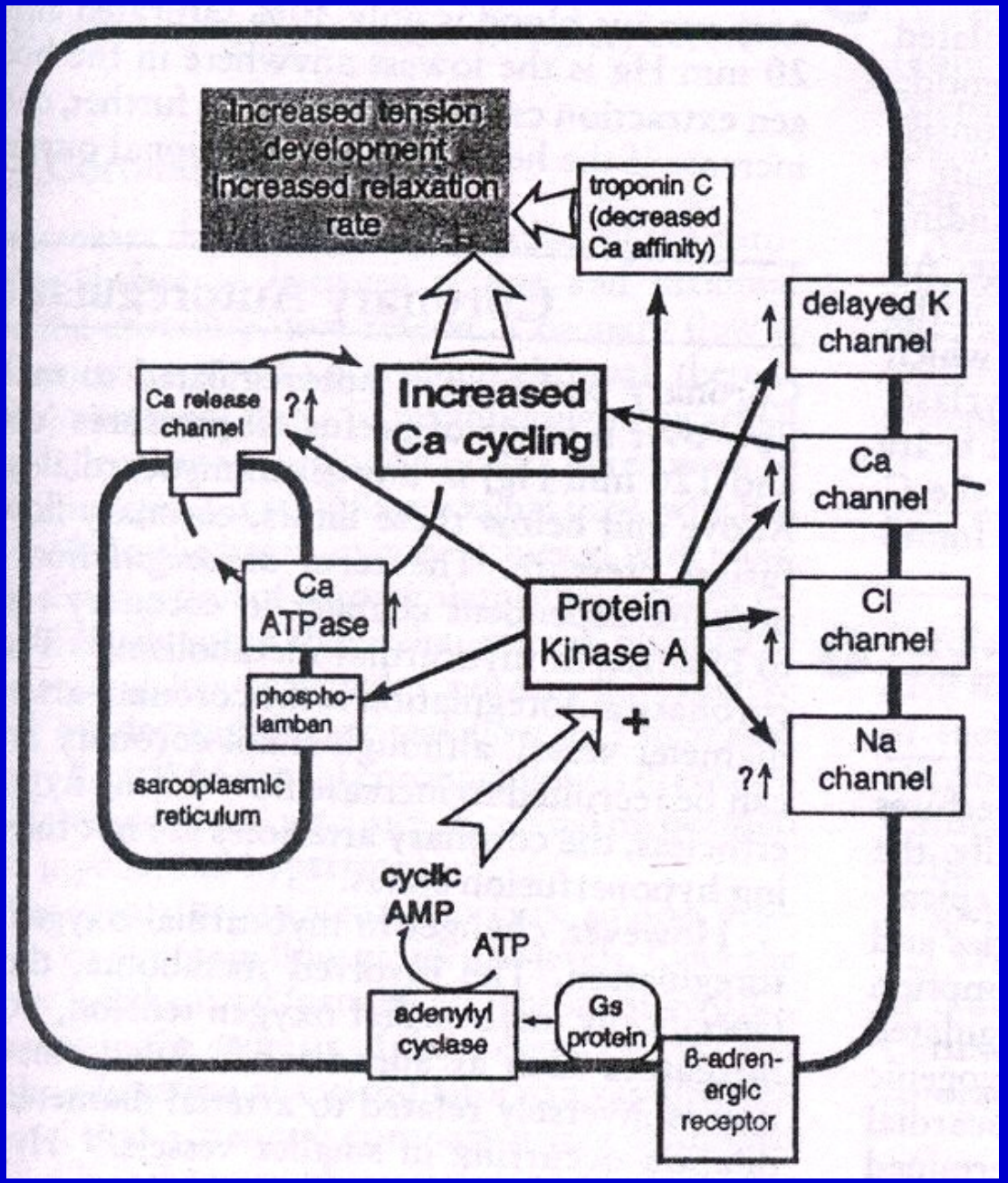
- Dominance of sympathetic or parasympathetic system
  - Age
  - Physical condition
- Inhibitory parasympathetic system usually predominates
- The right stellate ganglion has effect on heart rate, the left has as more effect on contractility
- Abnormalities of sympathetic cardiac nerve tone occur in long QT interval syndromes

Atrial receptors

- Parasympathetic receptors type A, type B and receptors innervate by group C fibers reflexly alter intravascular volume or heart rate
- Type A receptors respond to heart rate rather than atrial pressure
- Type B receptors response to the atrial volume

Ventricular receptor

- Baroreceptor/mechanoreceptor/sympathetic mechenosensitive or chemosensitive receptor
- Postcardiotomy hypertension results from a cardiogenic reflex transmitted through the sympathetic afferent fibers of the stellate ganglion
- K type, opiate receptors found in vagus, cardiac, and sympathetic ganglia, may mediate dysrhythmias, particularly during ischemia and reperfusion



Adrenergic stimulation

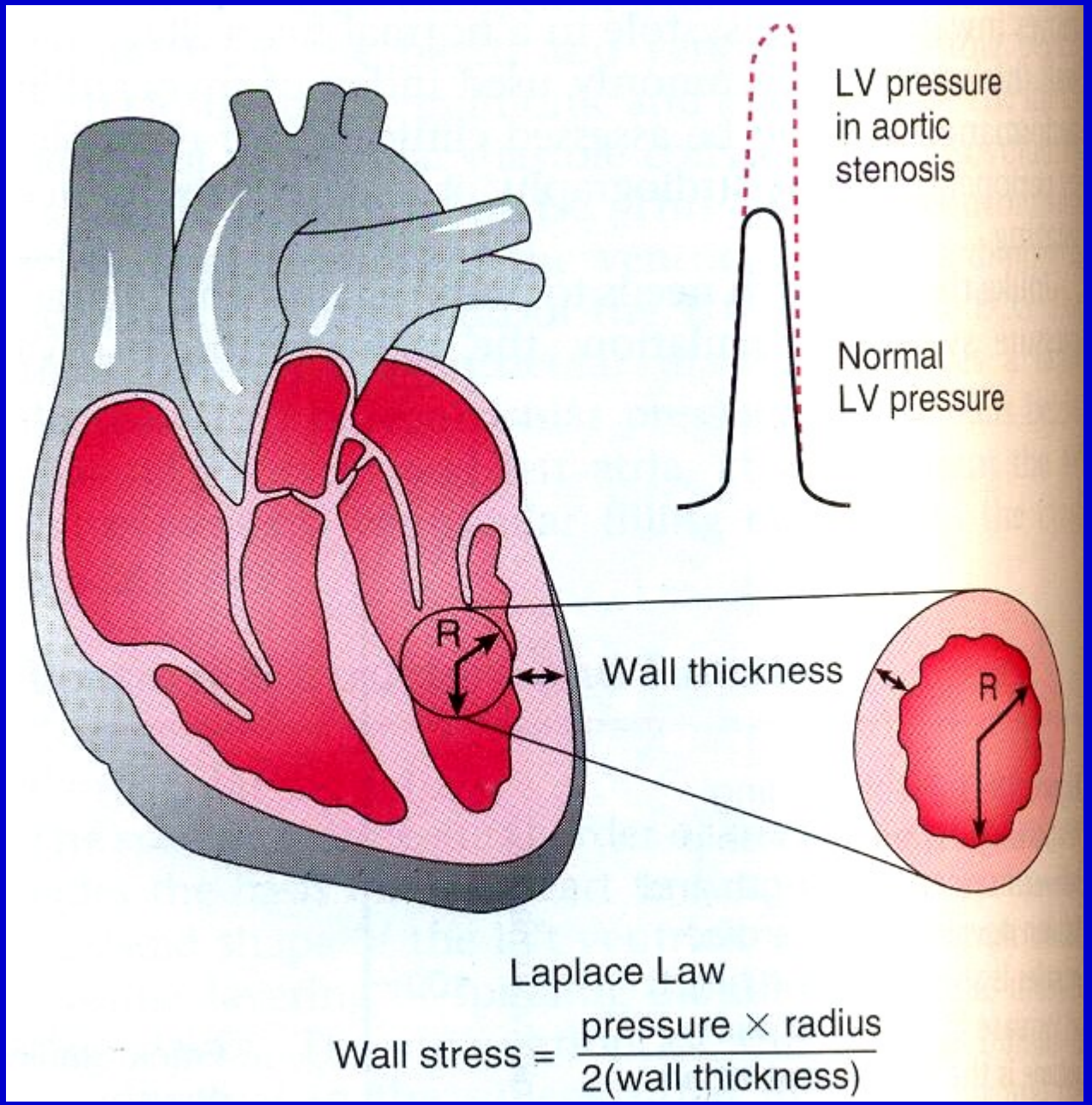
SYSTOLIC FUNCTION

- Traditional description of systolic function is limited to left ventricle
- Systolic performance
  - Loading condition
  - Contractility
- Preload and afterload – extrinsic independent factors
- Preload – ventricular load at end of diastole, before contaction has started
- Afterload – systolic load on the left ventricle after contraction has begun

- Aortic compliance is an additional determine of after load
- Preload and after load can be considered as wall stress that is present at end of diastole and LV ejection

Law of laplace 6 = P X R/2H

- 6 = wall stress
- P = pressure
- R = radius
- H = wall thickness
- Aortic stenosis
- Heart failure



Laplace Law

$$\text{Wall stress} = \frac{\text{pressure} \times \text{radius}}{2(\text{wall thickness})}$$

Contractility

- Factors known to modify contractility are:
- Max velocity of contraction (V Max) – Max velocity of ejection at zero load
- V max is obtained by plotting velocity of muscle shortening in isolated papillary muscle at varying degree of force
- V max cannot be measured in intact heart

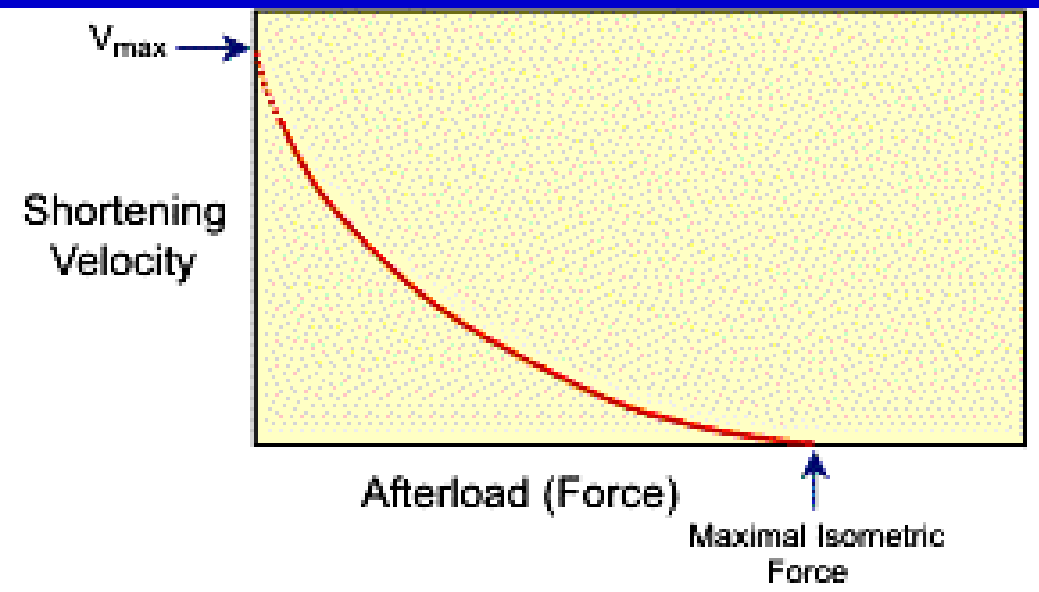


Figure 1. Force-Velocity relationship.

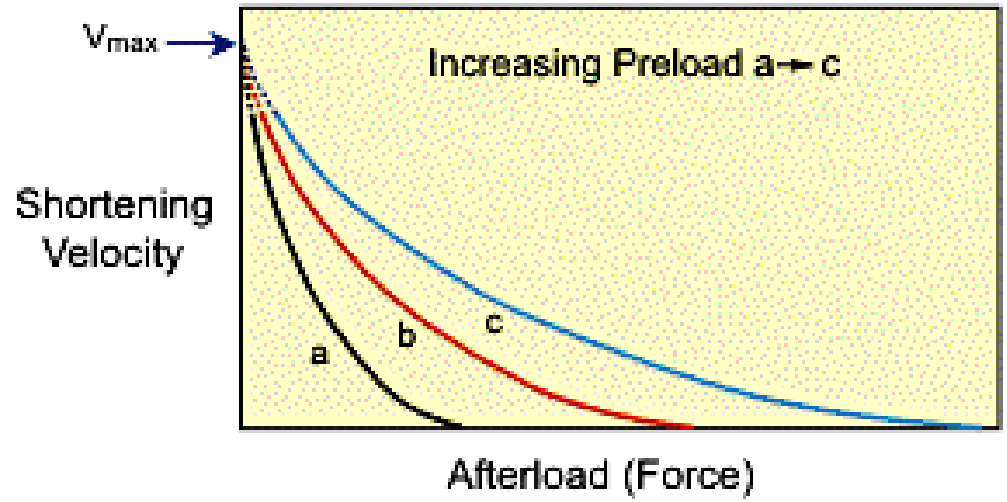


Figure 2. Effects of increasing preload (shift from curve a to c) on the force-velocity relationship.

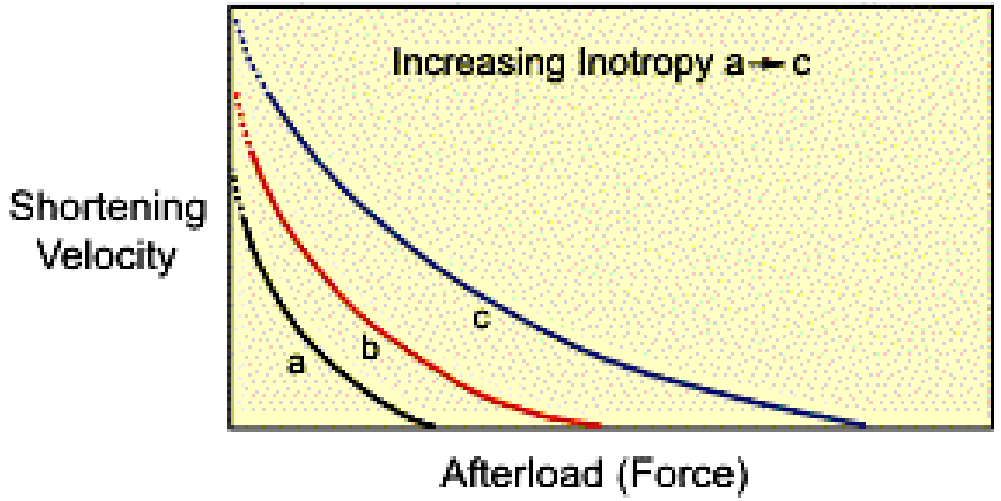


Figure 3. Effects of increasing inotropy (shift from curve a to c) on the force-velocity relationship.

- To measure intrinsic contractile activity of intact heart
- Best method: pressure volume loops, requiring catheterization of left side of heart
- Most common: non invasive index – ejection fraction assessed by echocardiography angiography or radionucleotide ventriculography

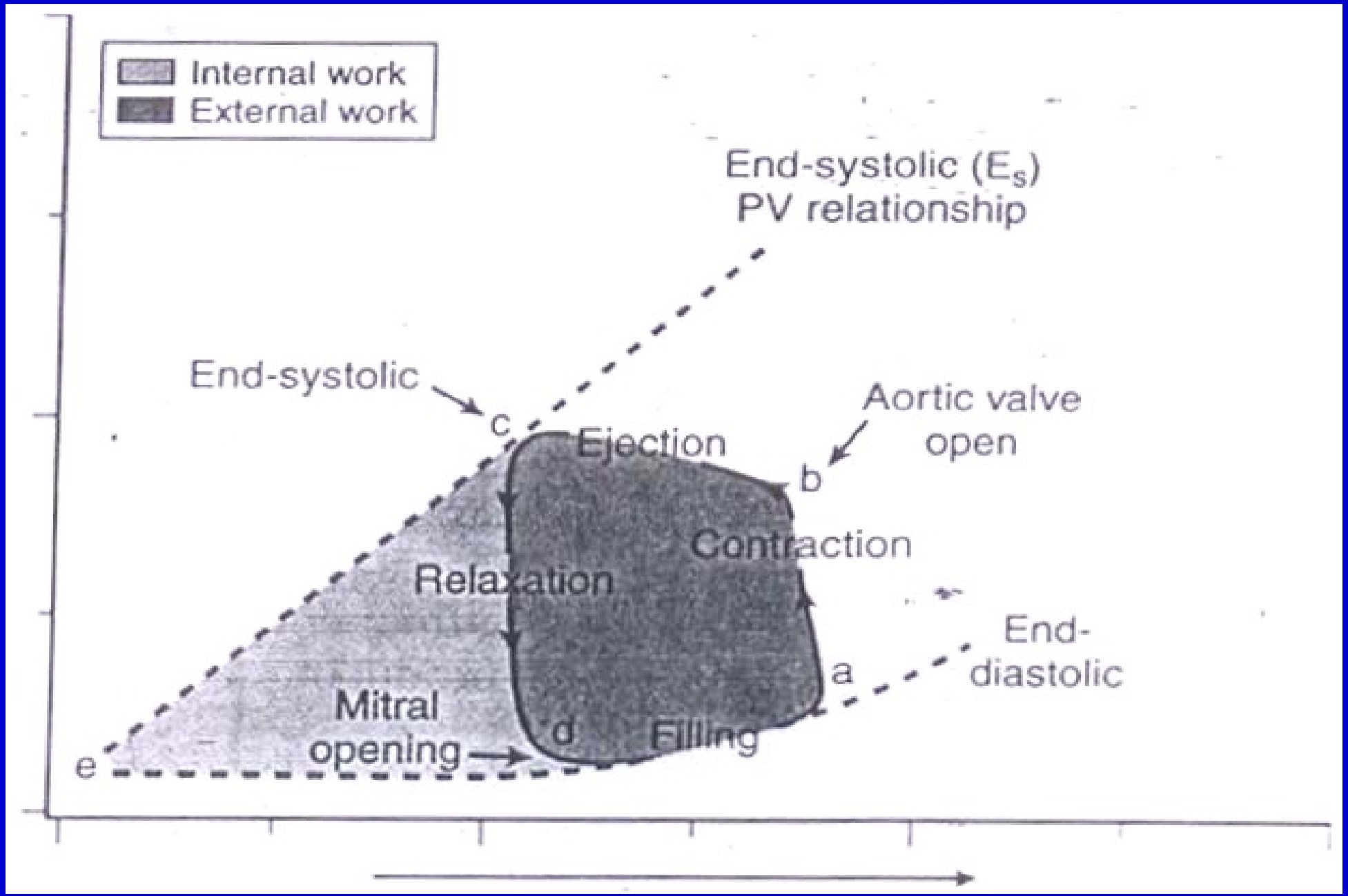
  EF = (LVED – LVESV)/LVEDV

Cardiac work

- External work (Stroke work): expended to eject blood under pressure
- Internal work: expended within ventricle to change the shape of heart and prepare heart for ejection
- Wall stress is directly proportional to internal work of heart
- External work = Stroke volume x pressure

  SV x P (LVEDV – LVESV) x P

- Clinical significance – In case of poorly drained LV external work is provided by roller pump during bypass – myocardial ischaemia still occurs because of poor drainage of LV
- Efficiency of cardiac contraction is estimated by cardiac efficiency – external work/energy equivalent of O_2 consumption
- Heart failure
- Ventricular dilatation reduce cardiac efficiency because it increase wall stress


Internal work
External work
End-systolic (E_s)
PV relationship
End-systolic
c
Ejection
Aortic valve
open
b
Contraction
Relaxation
End-
diastolic
a
Mitral
opening
d
Filling
e
Ventricular volume

HEART RATE AND FORCE FREQUENCY RELATIONSHIP

- TREPPE or staircase phenomenon or force frequency
- In isolated cardiac muscle an increase in the frequency of stimulation induces an increase in the force of contraction
- Increased frequency of stimulation – incrementaly increases contraction
- Lower frequency of stimulation – decreases contractile force
- Extremely rapid frequency of stimulation – force of contraction decrease

Clinical significance

* Pacing induced positive inotropic effects may have effective only upto a certain heart rate
* In failing heart this relation may be less effective in producing a positive inotropic effect

DISATOLIC FUNCTION

- Diastole is ventricular relaxation and it occurs in four distinct phases
  - Isovolumetric relaxation
  - Rapid filling phase
  - Slow filling or diastasis
  - Final filling during atrial systole
- Phase
  - 1: does at contribute to ventricular filling
  - 2: maximum contribution
  - 3: 5%
  - 4: 15%

INDICES TO ASSESS DIASTOLIC FUNCTION

- Index used for examining isovolumic relaxation phase is peak instantanecus rate of LV pressure decline (-dp/dt) or time constant of isovolumic LV pressure decline
- Estimation of diastolic dysfunction during auxotonic relaxation – Echocardiography
- Ventricular compliance can be evaluated by pressure volume relationship

- Many different factors influence diastolic function
- Diastolic dysfunction is decreased ability of the heart to fill
- Diastolic dysfunction is now being recognized as predominant cause of CHF
- In diastolic ventricular interaction dilatation of either LV or RV will have an impact on affective filling of contractive ventricle

BARO RECEPTOR REFLEX

- Stretch receptors in walls of heart and blood vessels
- Responsible for maintenance of blood pressure

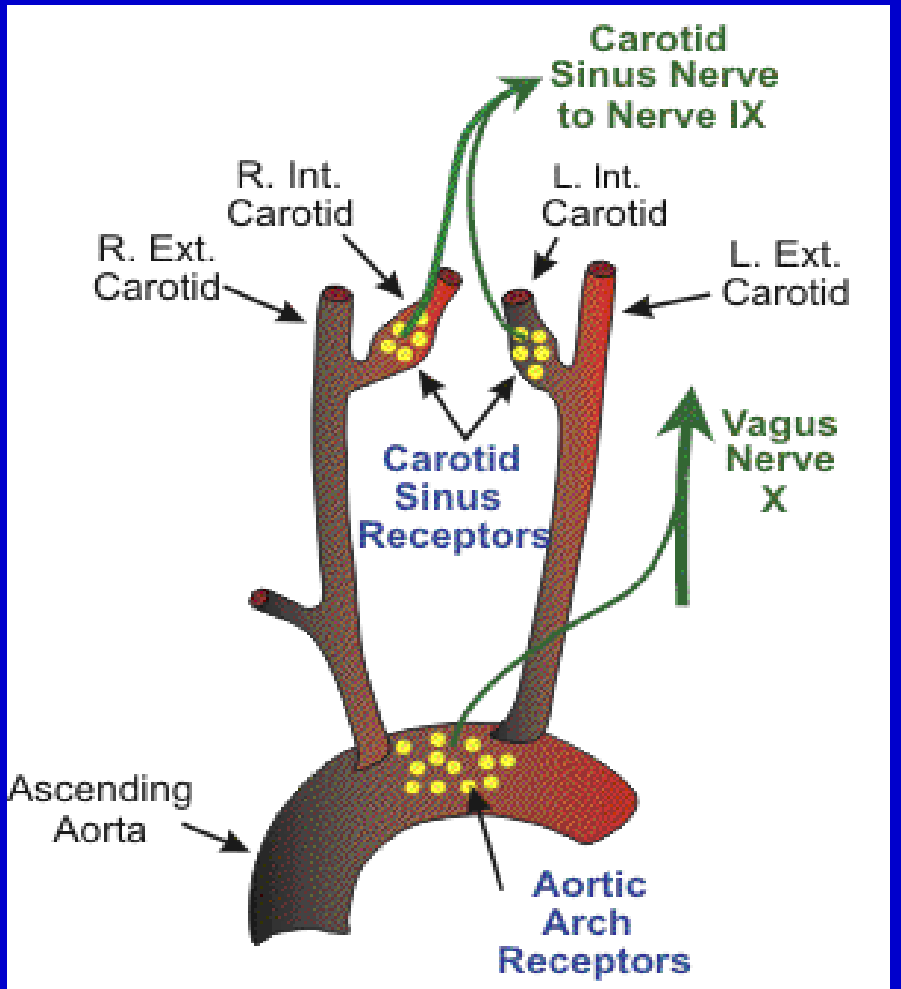


Figure 1. Location and innervation of arterial baroreceptors.

GRADING OF DIASTOLIC DYSFUNCTION

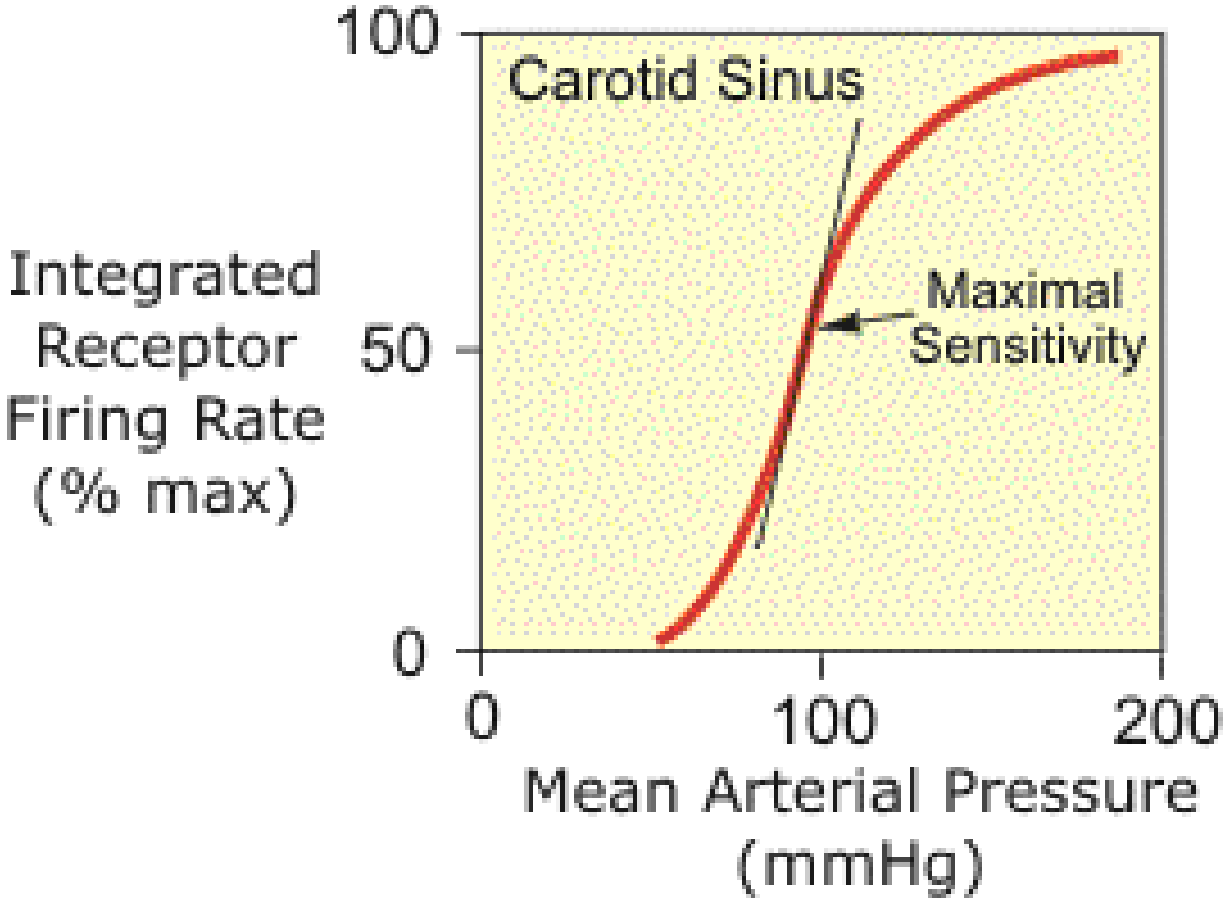
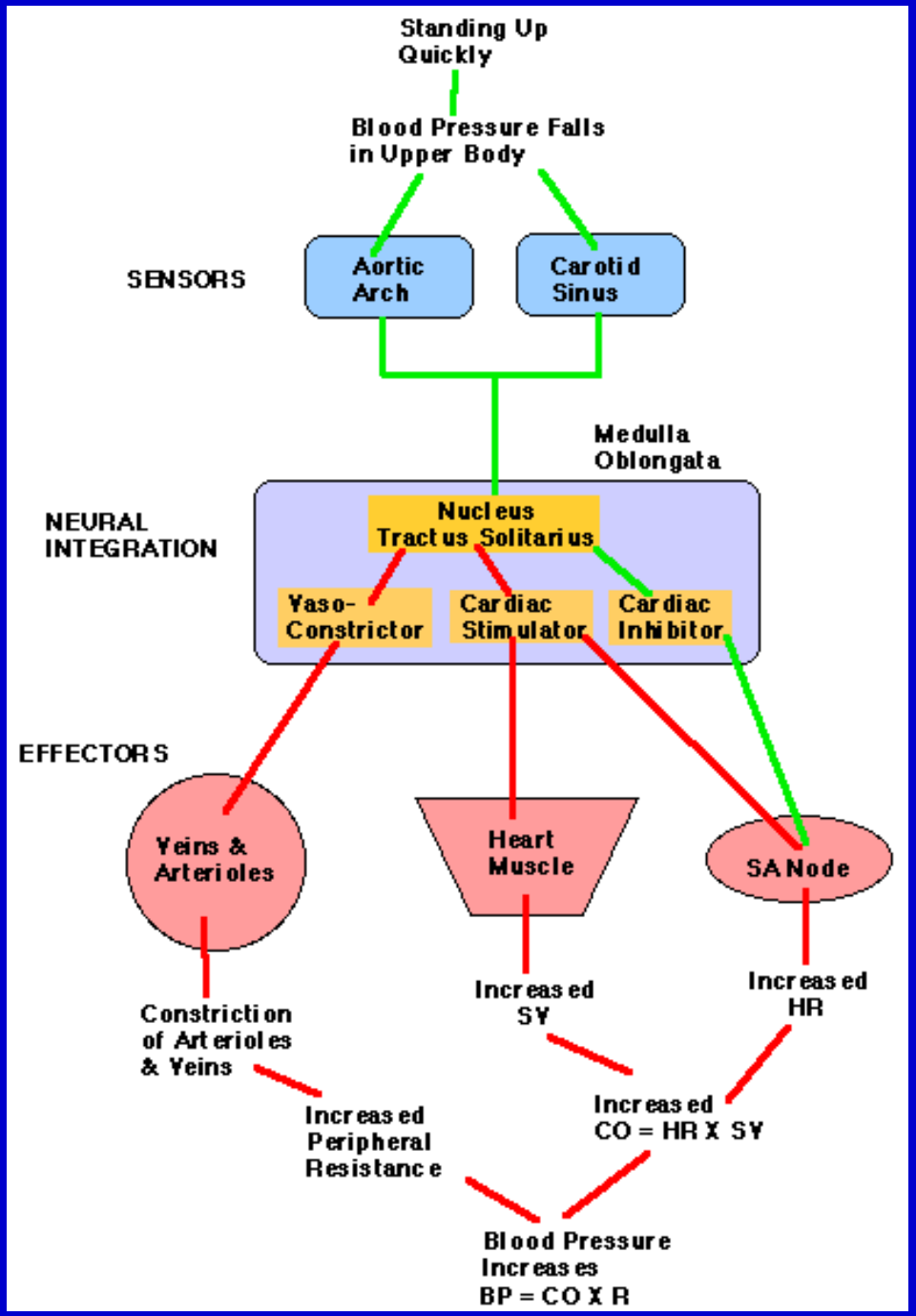
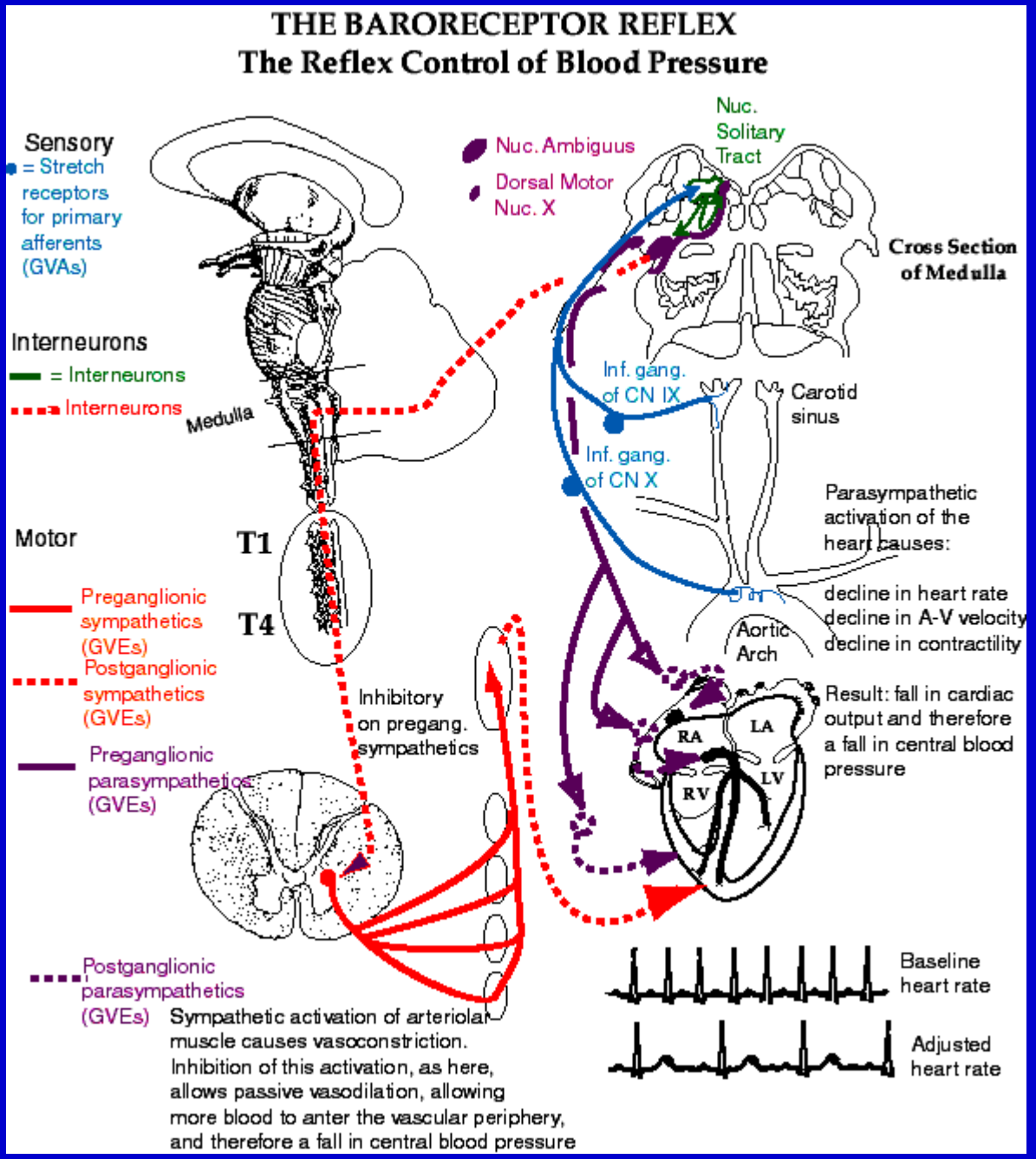
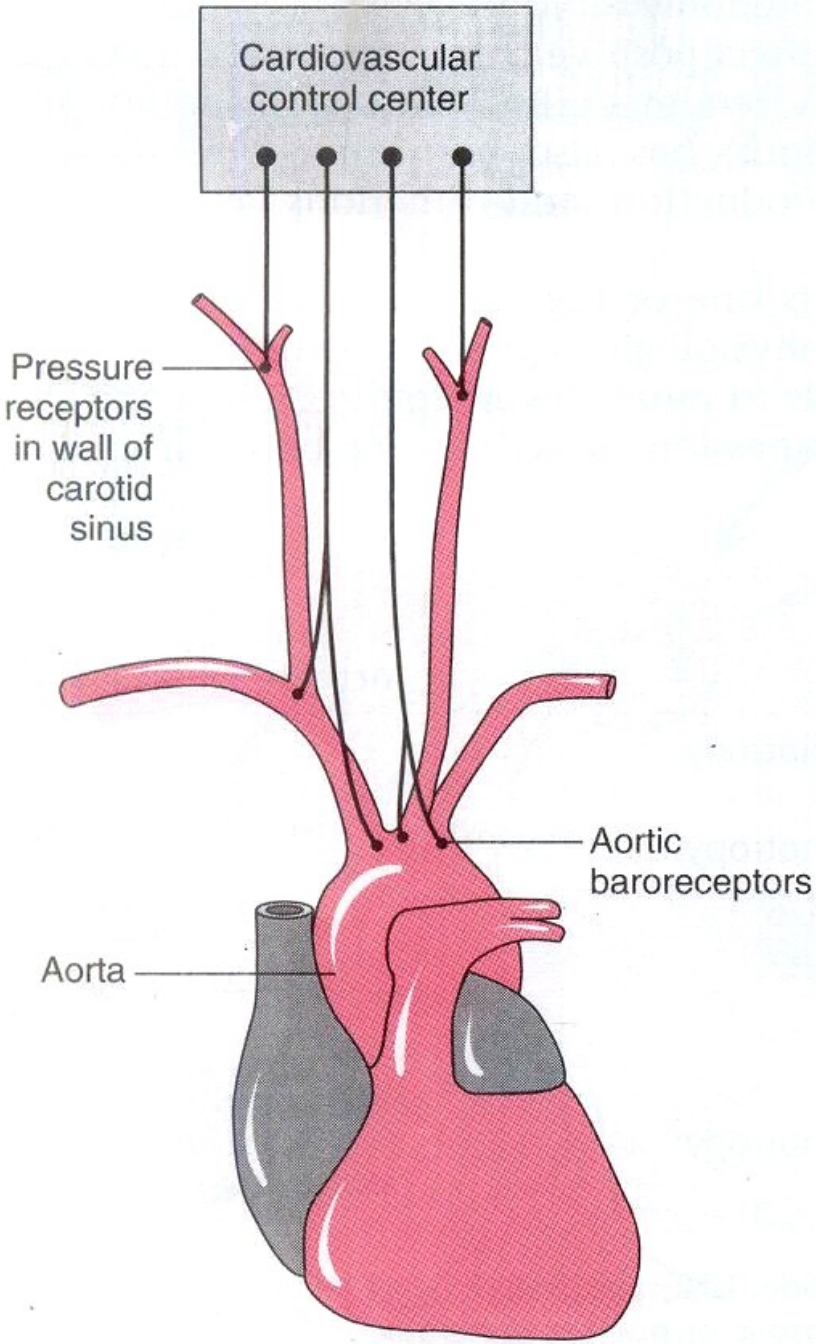


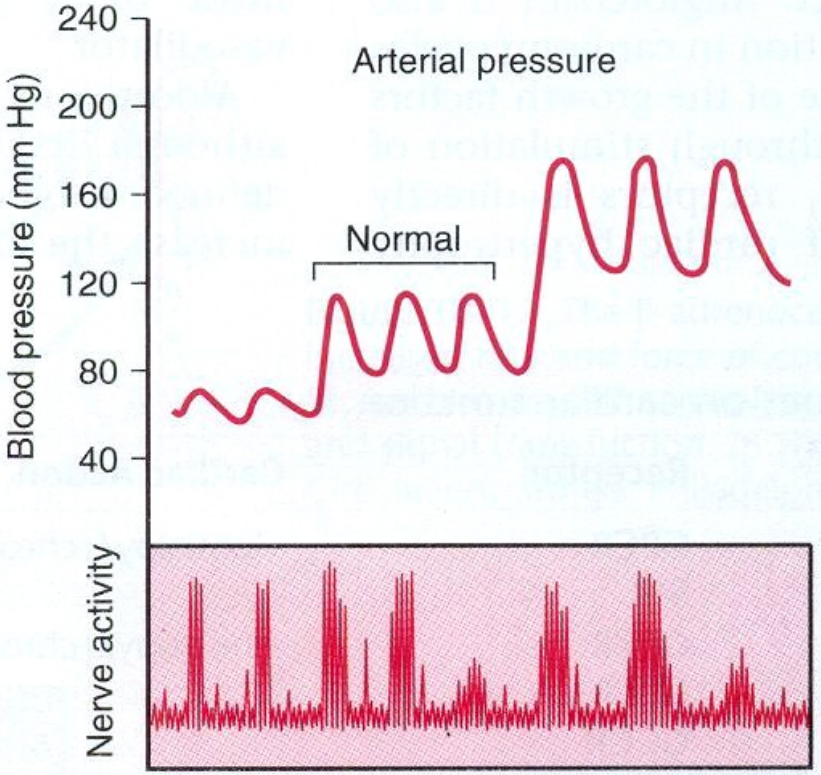
Figure 2. Effects of arterial pressure on carotid sinus firing rate. Increasing arterial pressure increases carotid sinus firing rate. Maximal baroreceptor sensitivity occurs near normal mean arterial pressure.


Standing Up Quickly
Blood Pressure Falls in Upper Body
SENSORS
Aortic Arch
Carotid Sinus
Medulla Oblongata
NEURAL INTEGRATION
Nucleus Tractus Solitarius
Vaso-Constrictor
Cardiac Stimulator
Cardiac Inhibitor
EFFECTORS
Veins & Arterioles
Heart Muscle
SA Node
Constriction of Arterioles & Veins
Increased SV
Increased HR
Increased Peripheral Resistance
Increased CO = HR X SV
Blood Pressure Increases
BP = CO X R

THE BARORECEPTOR REFLEX
The Reflex Control of Blood Pressure




Cardiovascular control center
Pressure receptors in wall of carotid sinus
Aortic baroreceptors
Aorta


Arterial pressure
Blood pressure (mm Hg)
240
200
160
120
80
40
Normal
Nerve activity

Anatomy

- Carotid-afferent nerve of Hering (glossopharyngeal)
- Aortic-vagus
- Cardiovascular centres in medulla

Stimulus

- Increased blood pressure

Response

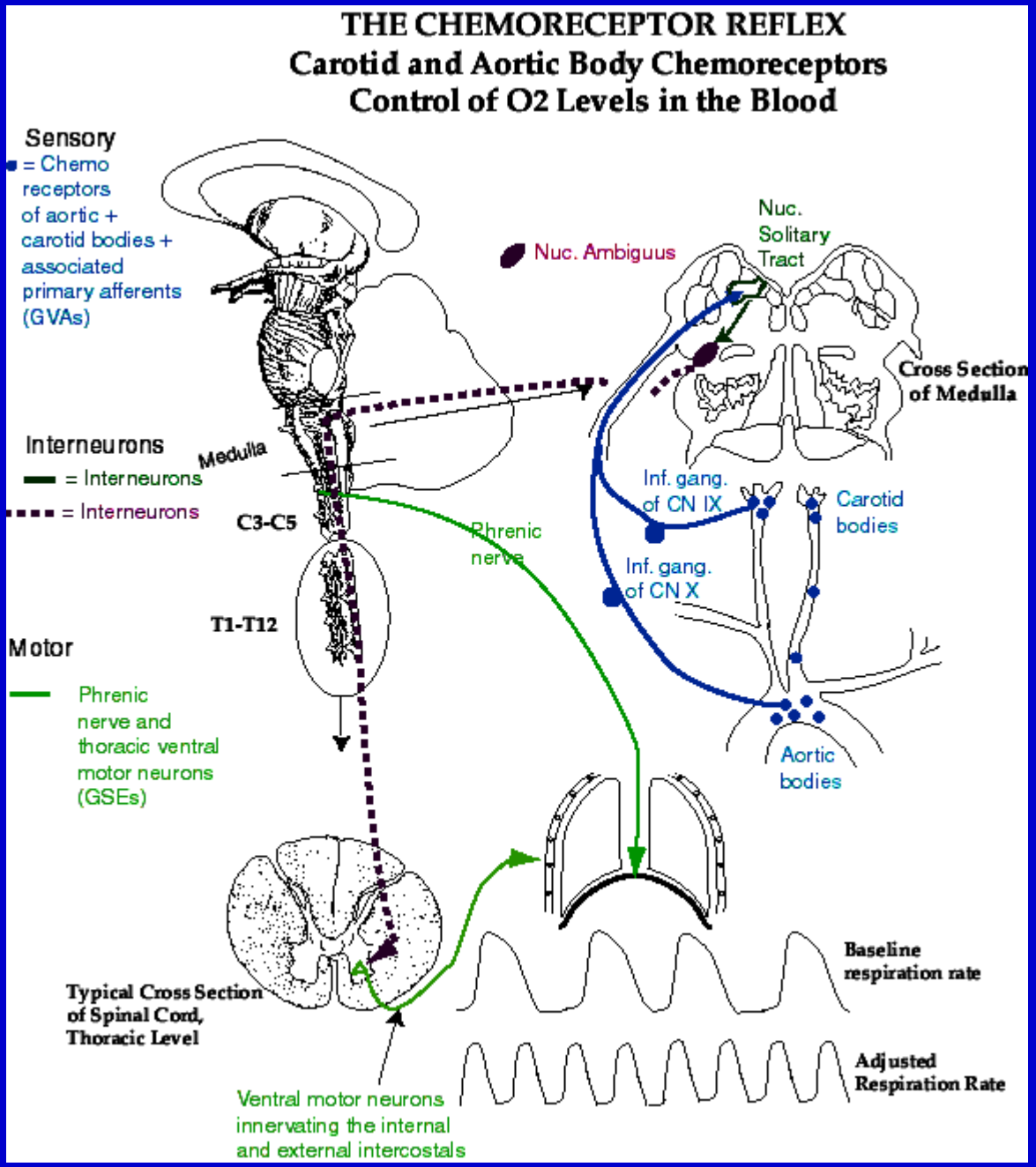
- Inhibition of sympathetic and increase in parasympathetic activity, causing decreased cardiac contractility heart rate

Clinical significance

- Others – Stretch receptors are activated if systemic blood pressure is greater than 170 mm of Hg
- Important role during blood loss and shock, however reflex arch loses it's functional capacity at blood pressure < 50
- Volatile anesthetics (in particular halothane) inhibit the heart rate component of this reflex
- Concomitant use of CCB, ACE inhibitor or PDE inhibitor will ↓ the CVS response of ↑ BP through baroreceptor
- Patients with chronic hypertension often exhibit perioperative circulatory instability as a result of ↓ in baroreceptor reflex response

CHEMORECEPTOR REFLEX

- **Anatomy:** Carotid and aortic bodies chemoreceptors whose nerve pass through the nerve of Herring and the vagus nerve to the medullary vasomotor centres
- **Stimulus:** Decreasing oxygen tension or increased hydrogen ion concentrations
- **Response:** Increased pulmonary ventilation and blood pressure with decreased heart rate – stimulation of aortic bodies causes tachycardia
- **Other:** Peripheral chemoreceptors are minimally active


THE CHEMORECEPTOR REFLEX
Carotid and Aortic Body Chemoreceptors
Control of O2 Levels in the Blood
Sensory
= Chemo receptors of aortic + carotid bodies + associated primary afferents (GVAs)
Nuc. Ambiguus
Nuc. Solitary Tract
Cross Section of Medulla
Interneurons
= Interneurons
= Interneurons
Medulla
C3-C5
T1-T12
Phrenic nerve
Inf. gang. of CN IX
Carotid bodies
Inf. gang. of CN X
Aortic bodies
Motor
Phrenic nerve and thoracic ventral motor neurons (GSEs)
Typical Cross Section of Spinal Cord, Thoracic Level
Baseline respiration rate
Adjusted Respiration Rate
Ventral motor neurons innervating the internal and external intercostals

CHEMORECEPTION IN CAROTID BODY CELLS

- Glomus cells have Na^{+}, Ca^{++}, K^{+} channels and can fire action potentials repetitively
- Voltage gated K^{+} channel is influenced by PO_2 level K^{+} current is selectively inhibited by low PO_2

BAIN BRIDGE ATRIAL REFLEX

Anatomy

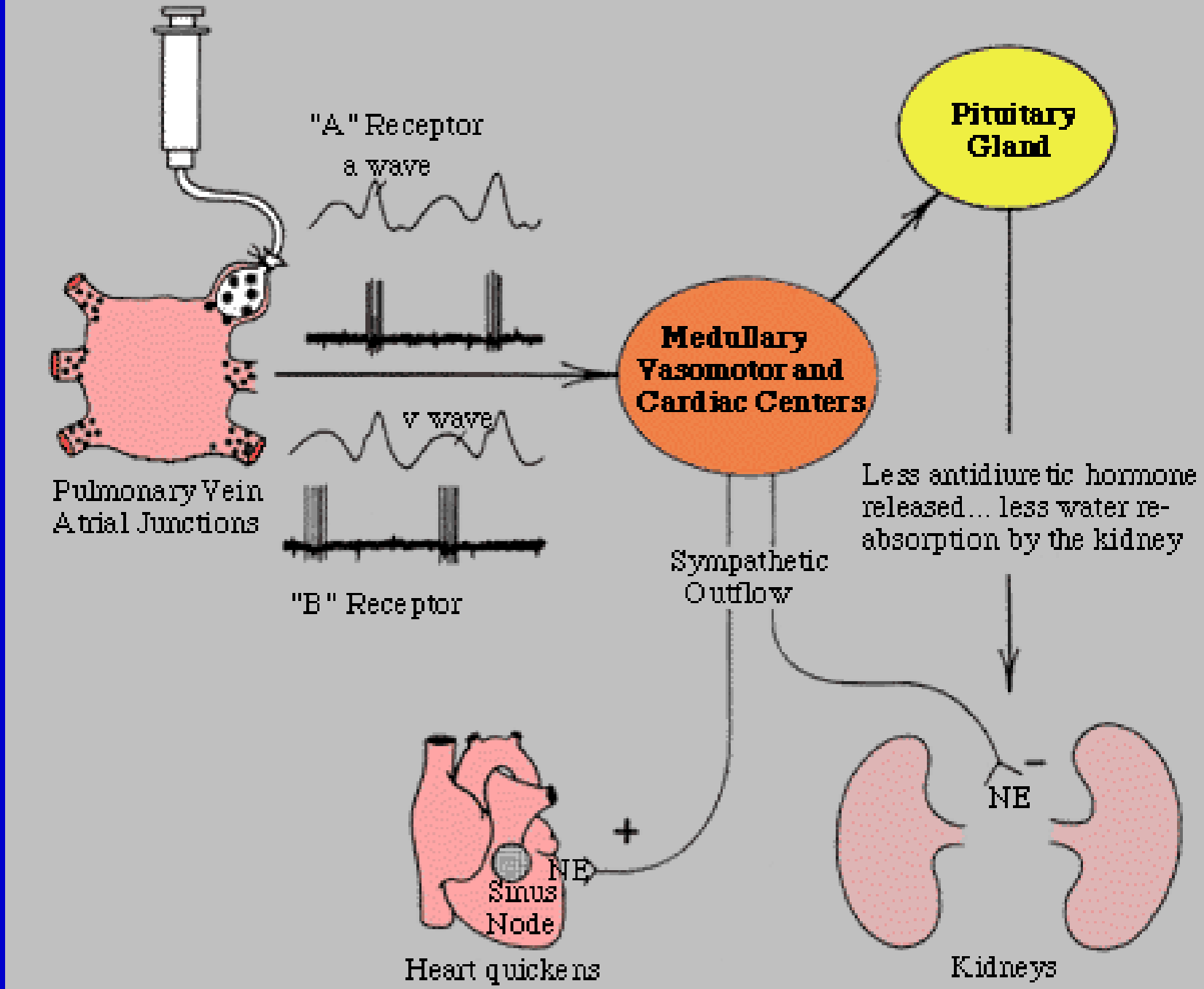
- Primarily mediated through vagal myelinated afferent fibres; activation of sympathetic afferent fibers may also occur. Increased right atrial pressure directly stretches the SA node and enhances its automaticity, increasing the heart rate

Stimulus

- Increased vagal tone and distention of the right atrium or central veins

Response

- Depends upon the preexisting heart rate
- With preexisting tachycardia, there is no effect
- Volume loading at a slow heart rate causes progressive tachycardia
- Global atrial distention in response to high pressures causes bradycardia, hypotension, and decreased systemic vascular resistance


"A" Receptor
a wave
v wave
"B" Receptor
Pulmonary Vein
Atrial Junctions
Pituitary Gland
Medullary Vasomotor and Cardiac Centers
Less antidiuretic hormone released... less water re-absorption by the kidney
Sympathetic Outflow
+
NE
Sinus Node
Heart quickens
-
NE
Kidneys

VON BEZOLD-JARISCH REFLEX

Anatomy

- Ventricular chemoreceptor and mechano-receptors with afferent pathway in unmyelinated vagal C fibres

Stimulus

- Noxius stimuli to either ventricle, associated with myocardial ischaemia, profound hypovolemia, coronary reperfusion, aortic steosis, neuroaxial anesthesia associated with sympathetic blockade or even vasovagal syncope

Response

- Hypotension, bradycardia, parasympathetically induced coronary vasodilatation and inhibition of sympathetic outflow from vasomotor centres

Clinical significance

- Reperfusion of previously ischemic tissue elicits reflex
- Natriuretic peptide receptors stimulated by endogenous ANP or BNP may modulate Bezold Jarisch Reflex
- Thus, Bezold Jarisch reflex may be less pronounced in patients with cardiac hypertrophy or atrial fibrillation

VALSALVA MANEUVER

Anatomy:

- Carotid-afferent nerve of hering (glossophyrangeal)
- Aortic – vagus
- Cardiovascular centres in medulla

Stimulus

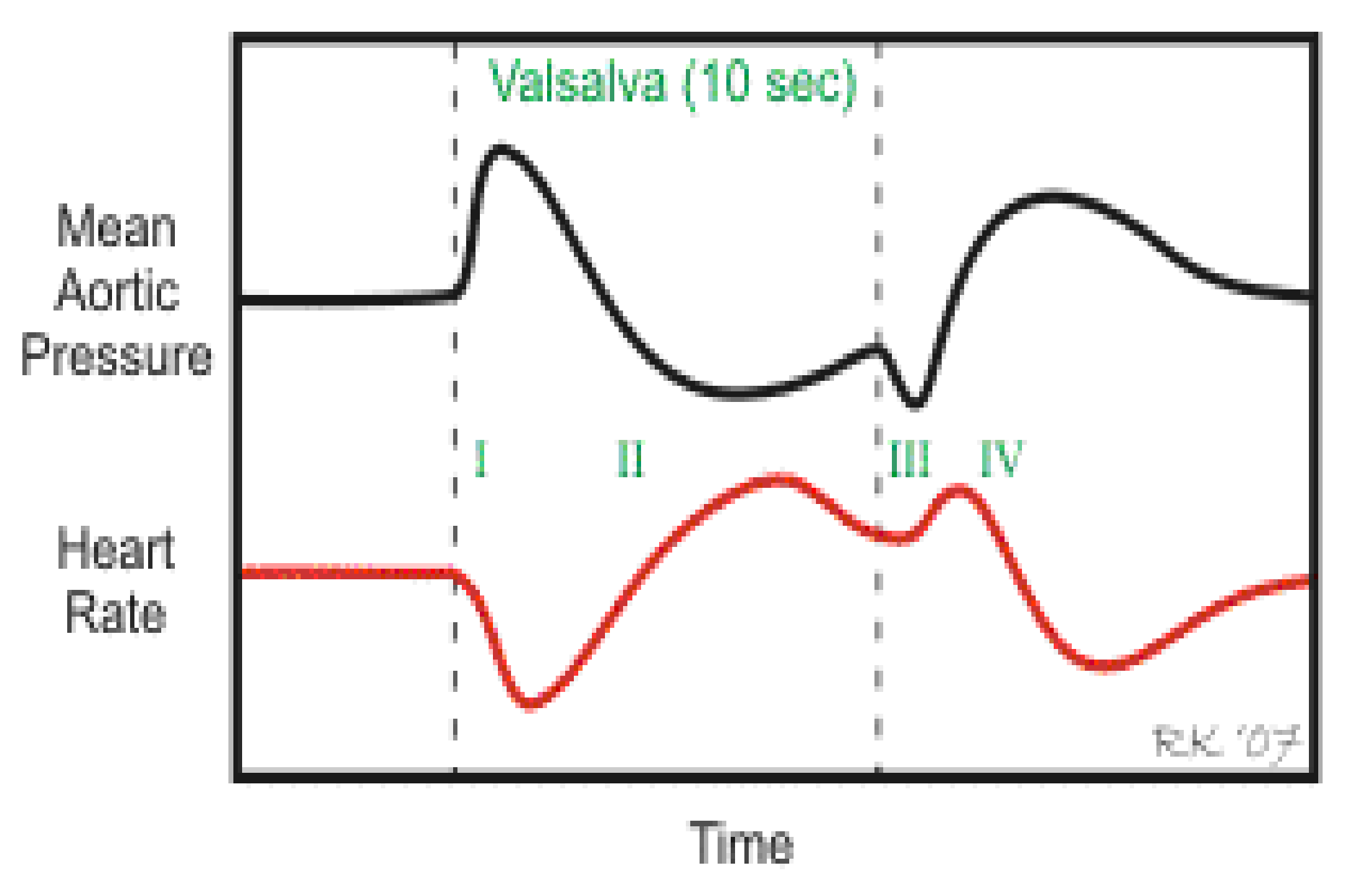
- Forced expiration against a closed glottis

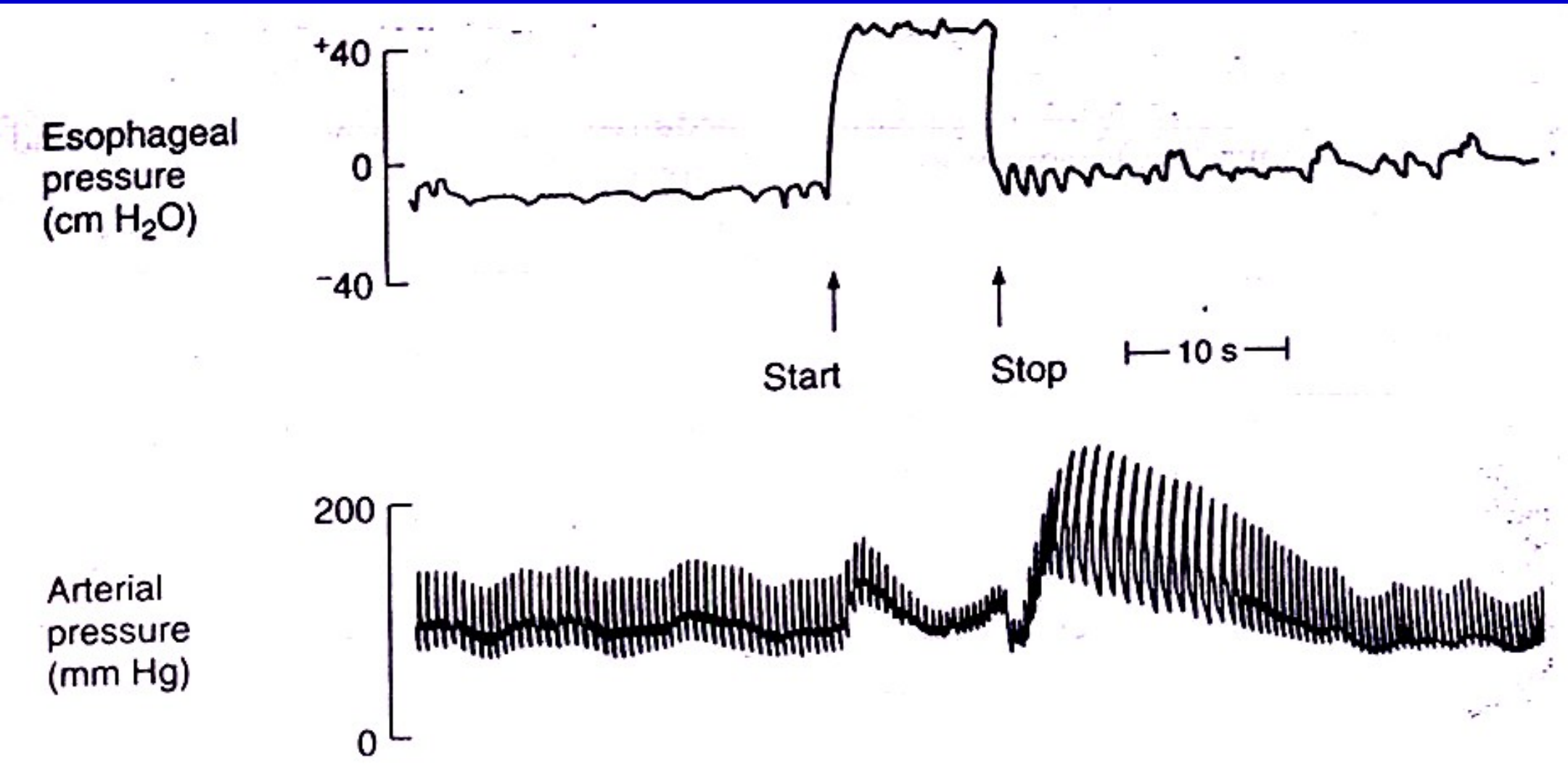
Response

- Increased venous pressure in head, upper extremitis, with decreased right heart venous return causing decreased blood pressure and cardiac output and reflex increase in heart rate

Clinical significance

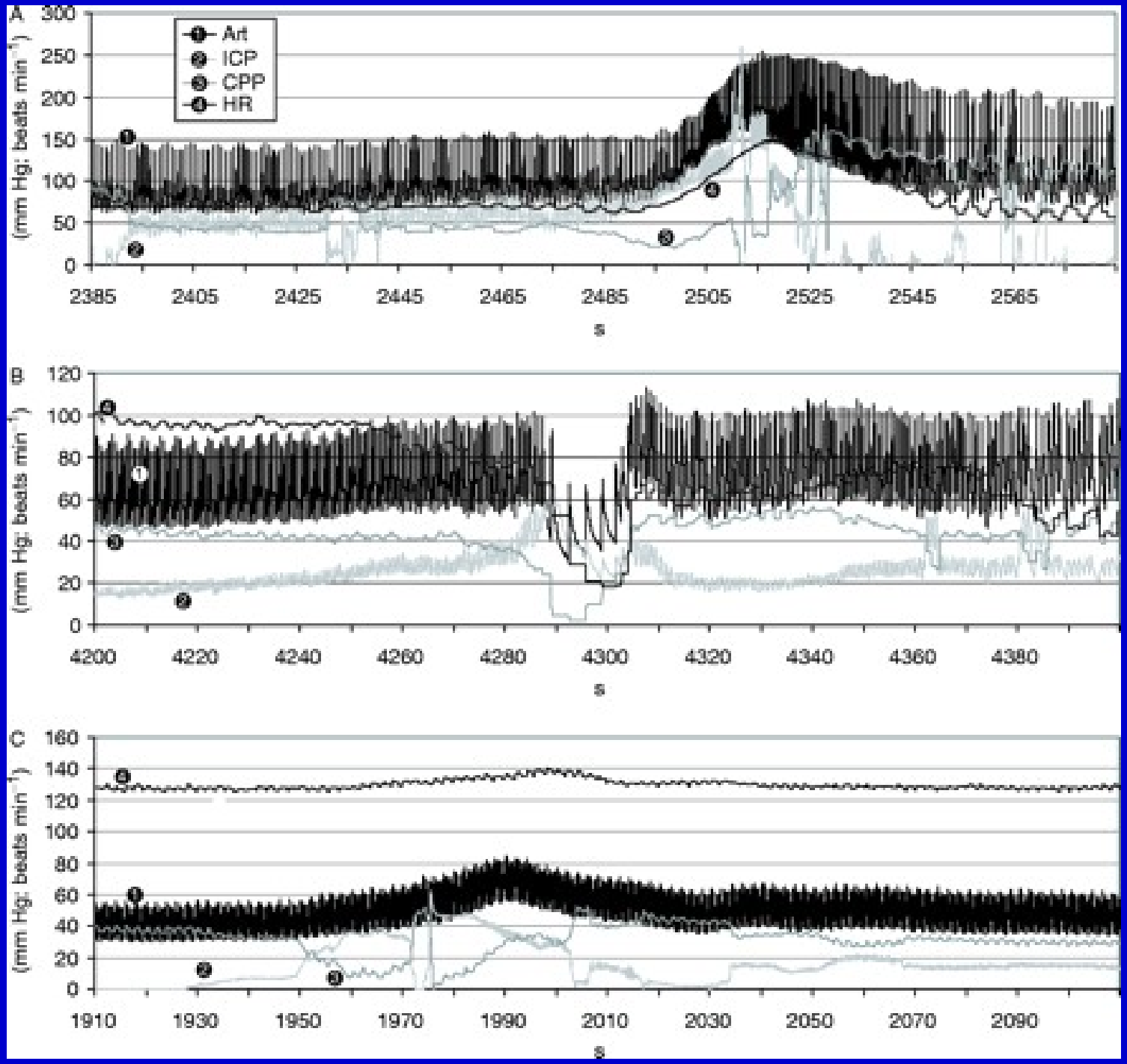
- Glottic opening increases venous return to right heart, resulting in force full right and then ventricular contraction, followed by transient bradycardia
- In patient with autonomic insufficiency heart rate changes are absent
- Patients with primary hyperaldosteronism also fail to show the heart rate changes and blood pressure rise – reasons still obscure
- Their response to valsalva maneuver returns to normal after removal of adosterone secreting tumour


Valsalva (10 sec)
Mean Aortic Pressure
Heart Rate
I
II
III
IV
RK '07
Time


Esophageal pressure (cm H_2O)
+40
0
-40
Start
Stop
10 s
Arterial pressure (mm Hg)
200
0

CUSHING'S REFLEX

- **Anatomy:** Increased cerebrospinal fluid pressure compresses cerebral arteries
- **Stimulus:** Cerebral ischemia secondary to increased CSF pressure
- **Response:** An increase in arterial pressure sufficient to reperfuse the brain
- Intense sympathetic activity causes severe peripheral vasoconstriction
- As a result of high vascular tone reflex bradycardia mediated by baroreceptor will ensure


A
Art
ICP
CPP
HR
(mm Hg; beats min-1)
300
250
200
150
100
50
0
2385
2405
2425
2445
2465
2485
2505
2525
2545
2565
s
B
(mm Hg; beats min-1)
120
100
80
60
40
20
0
4200
4220
4240
4260
4280
4300
4320
4340
4360
4380
s
C
(mm Hg; beats min-1)
160
140
120
100
80
60
40
20
0
1910
1930
1950
1970
1990
2010
2030
2050
2070
2090
s

OCCULO CARDIAC REFLEX

Anatomy

- Afferent fibres run with the short or long ciliary nerves to the ciliary ganglion and then with the opthalmic division of trigeminal nerve to the gasserian ganglion

Stimulus

- Traction on the extraoccular muscle or pressure on the globe

Response

- Bradycardia, hypotension, nausea

Clinical significance

- Demonstrated in 30-90% of patients undergoing ophthalmic surgery
- Transient cardiac arrest may occur as 1 in 2200 strabismus surgeries

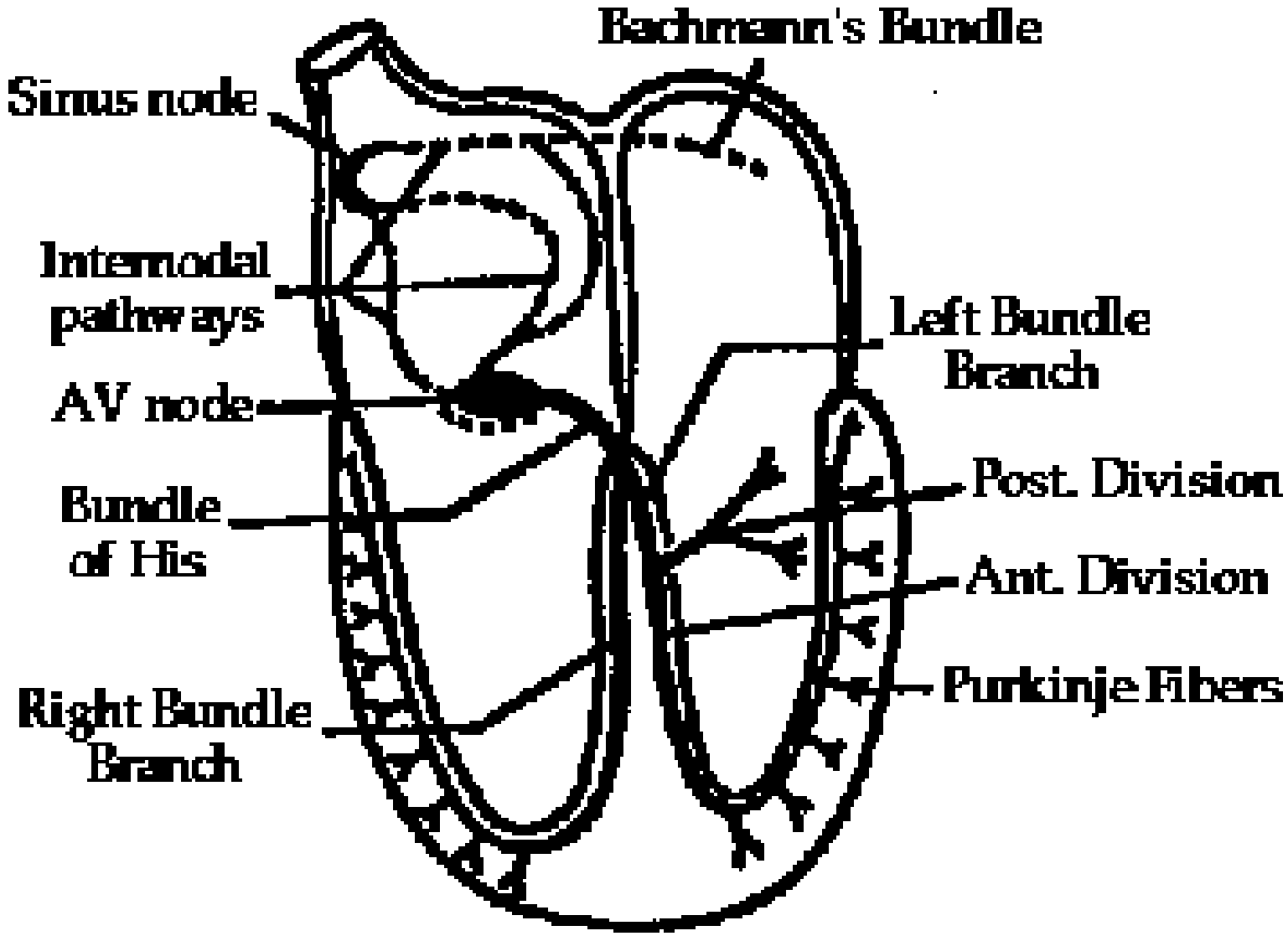
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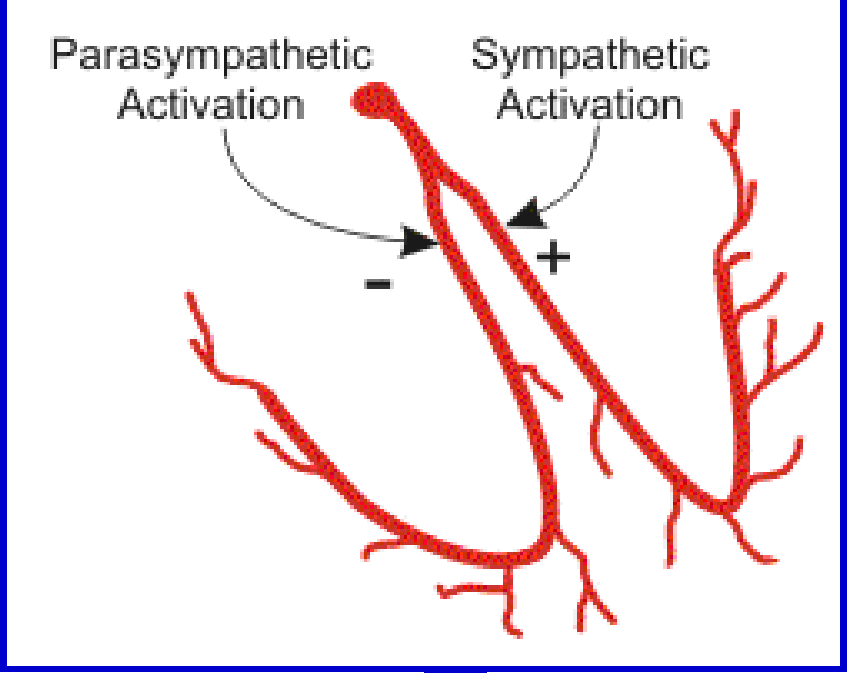
CARDIAC CONDUCTION SYSTEM

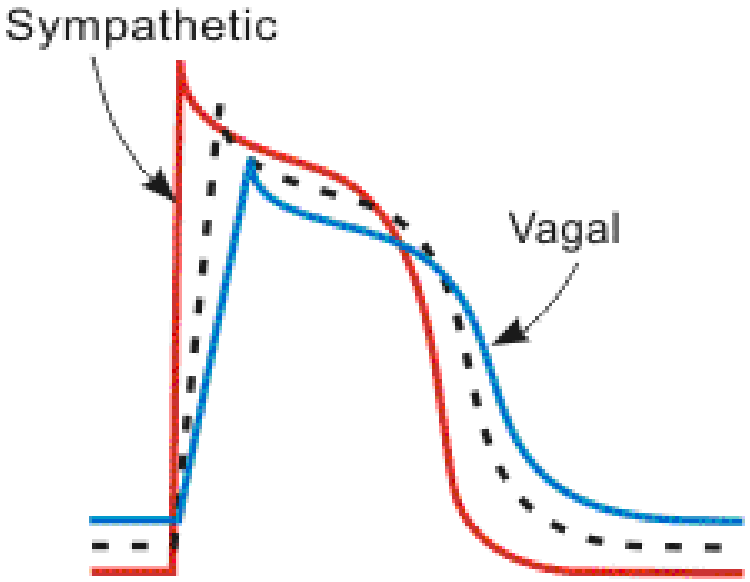
- SA node, AV node, bundle of his, right and left bundle branches and the purkinje system

- SA Node is located in right atrial wall at junction of RA with SVC
- SA Node
  - Spider and spindle pacemaker cell types are found in centre
  - Atrial cells are found in periphery
- SA node and AV node are connected via 3 tracts
  - Ant (Bachmann’s bundle)
  - Middle
  - Posterior internodal systems

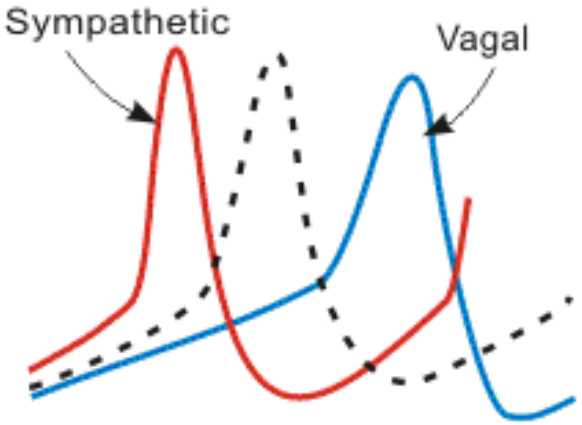
- AV node – located in floor of right atrium near ostium of coronary sinus
  - AV node consists of 4 areas
  - A-N transitional zone
  - Compact node
  - Posterior nodal extension
  - Lower nodal cell bundle


Bachmann's Bundle
Sinus node
Internodal pathways
AV node
Bundle of His
Right Bundle Branch
Left Bundle Branch
Post. Division
Ant. Division
Purkinje Fibers



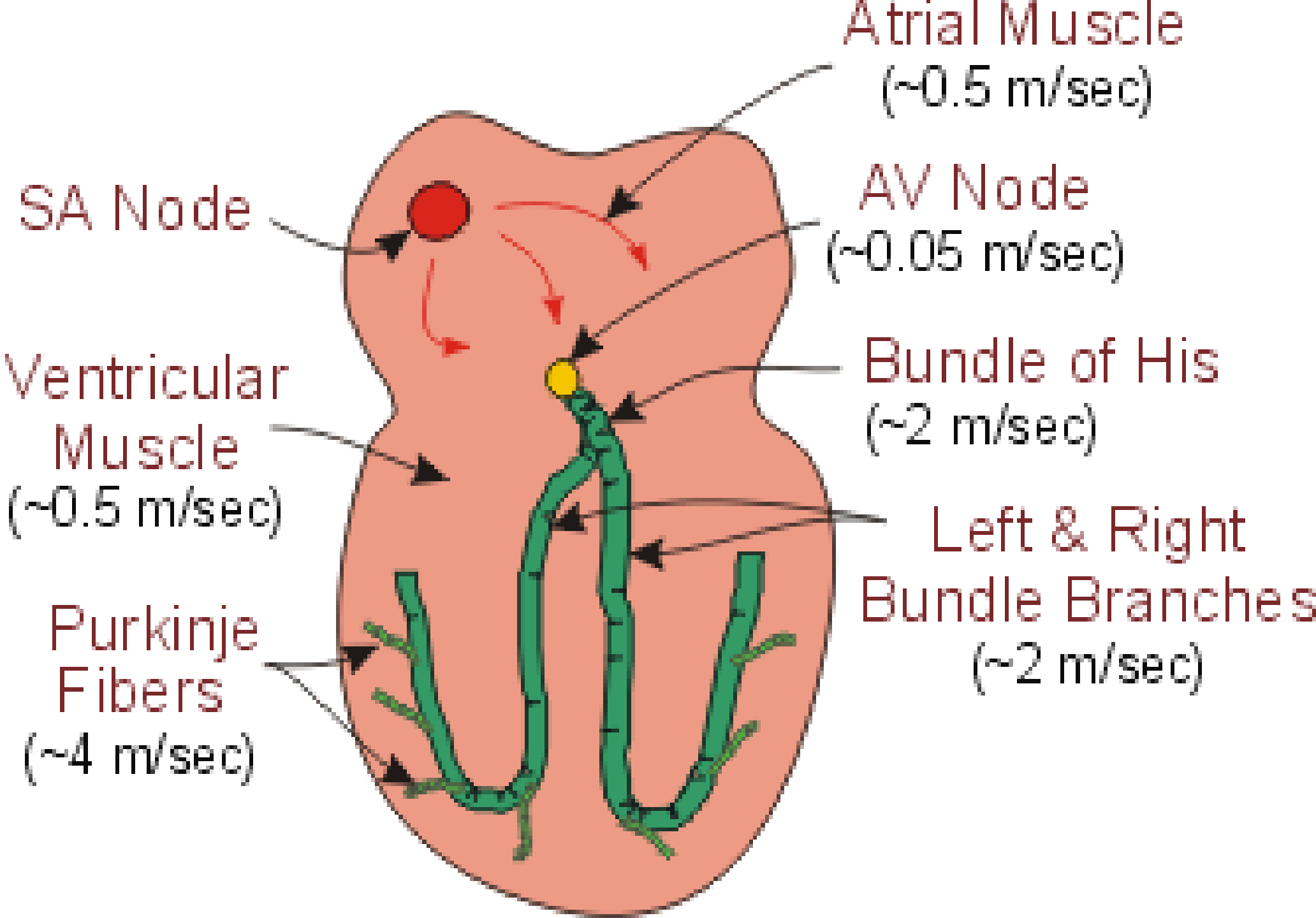


Myocyte and Purkinje Cell
Action Potentials

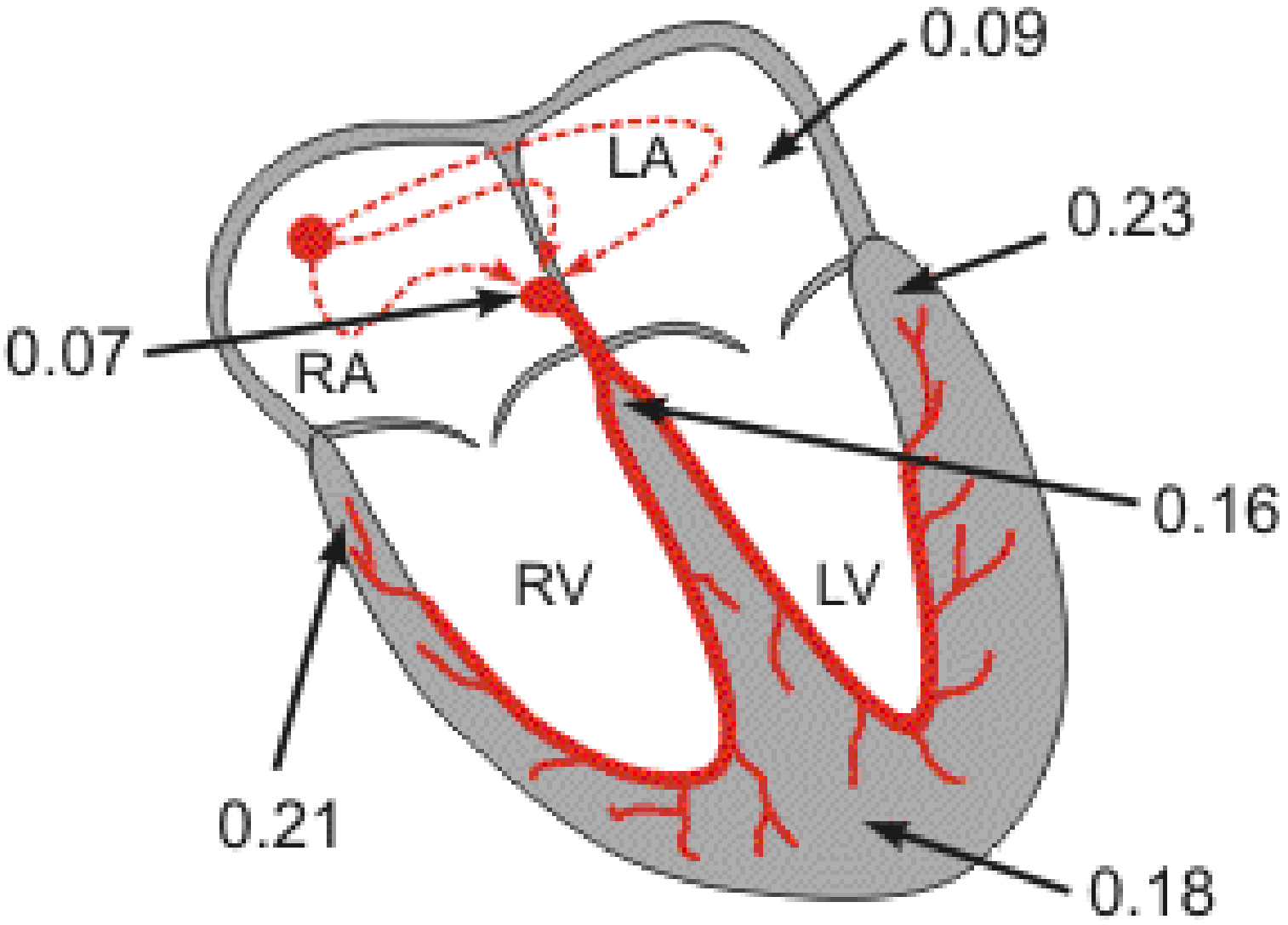


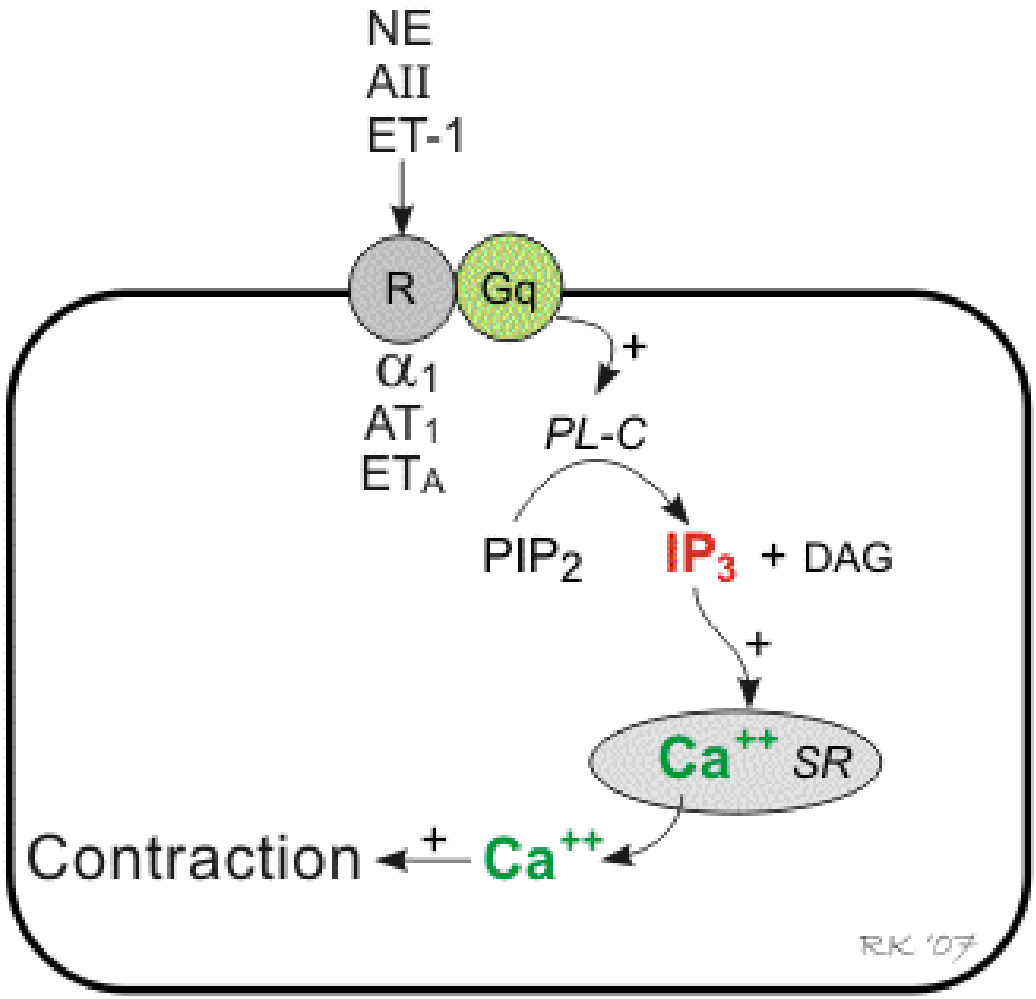
Nodal Cell Action Potentials

Effects of Parasympathetic (Vagal)
and Sympathetic Nerve Activation
on Conduction Velocity

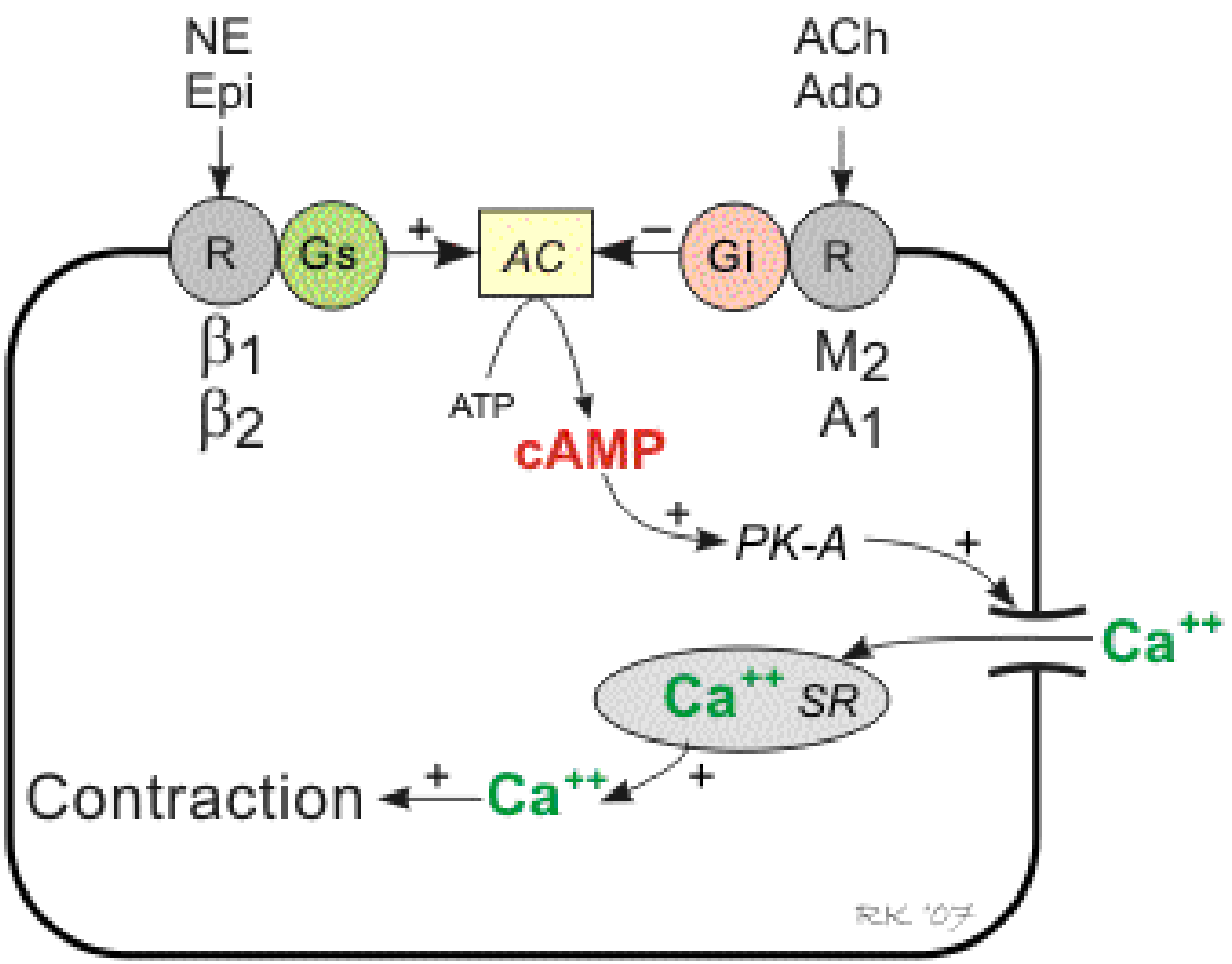

Atrial Muscle
(~0.5 m/sec)
SA Node
AV Node
(~0.05 m/sec)
Bundle of His
(~2 m/sec)
Ventricular
Muscle
(~0.5 m/sec)
Left & Right
Bundle Branches
(~2 m/sec)
Purkinje
Fibers
(~4 m/sec)

Cardiac Activation Times
(seconds)





R, receptor; Gq, phospholipase C-coupled Gq-protein; PL-C, phopholipase C; PIP_2, phosphatidylinositol biphosphate; IP_3, inositol triphosphate; DAG, diacylglycerol; SR, sarcoplasmic reticulum; NE, norepinephrine; AII, angiotensin II; ET-1, endothelin-1; α_1, alpha1-adrenoceptor; AT_1, type 1 angiotensin receptor; ET_A, type A endothelin receptor.



R, receptor; Gs and Gi, stimulatory and inhibitory G-proteins; AC, adenylyl cyclase; PK-A, protein kinase A; SR, sarcoplasmic reticulum; α and β, alpha and beta-adrenoceptors; Epi, epinephrine; NE, norepinephrine; ACh, acetylcholine; M_2, muscarinic receptor; A_1, adenosine (Ado)