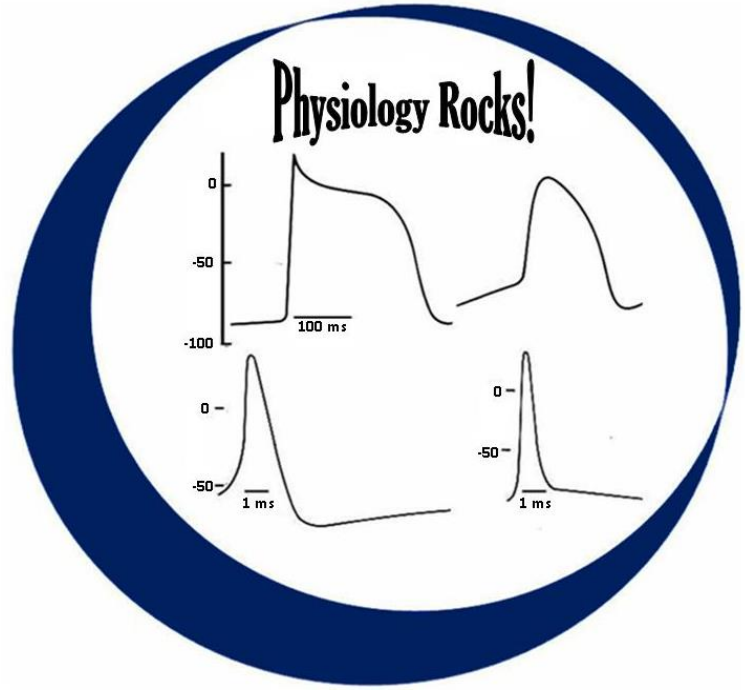
Lecture 14
Cardiovascular Controls and Reflexes



HN Mayrovitz PhD
mayrovit@nova.edu
mayrovitz.com

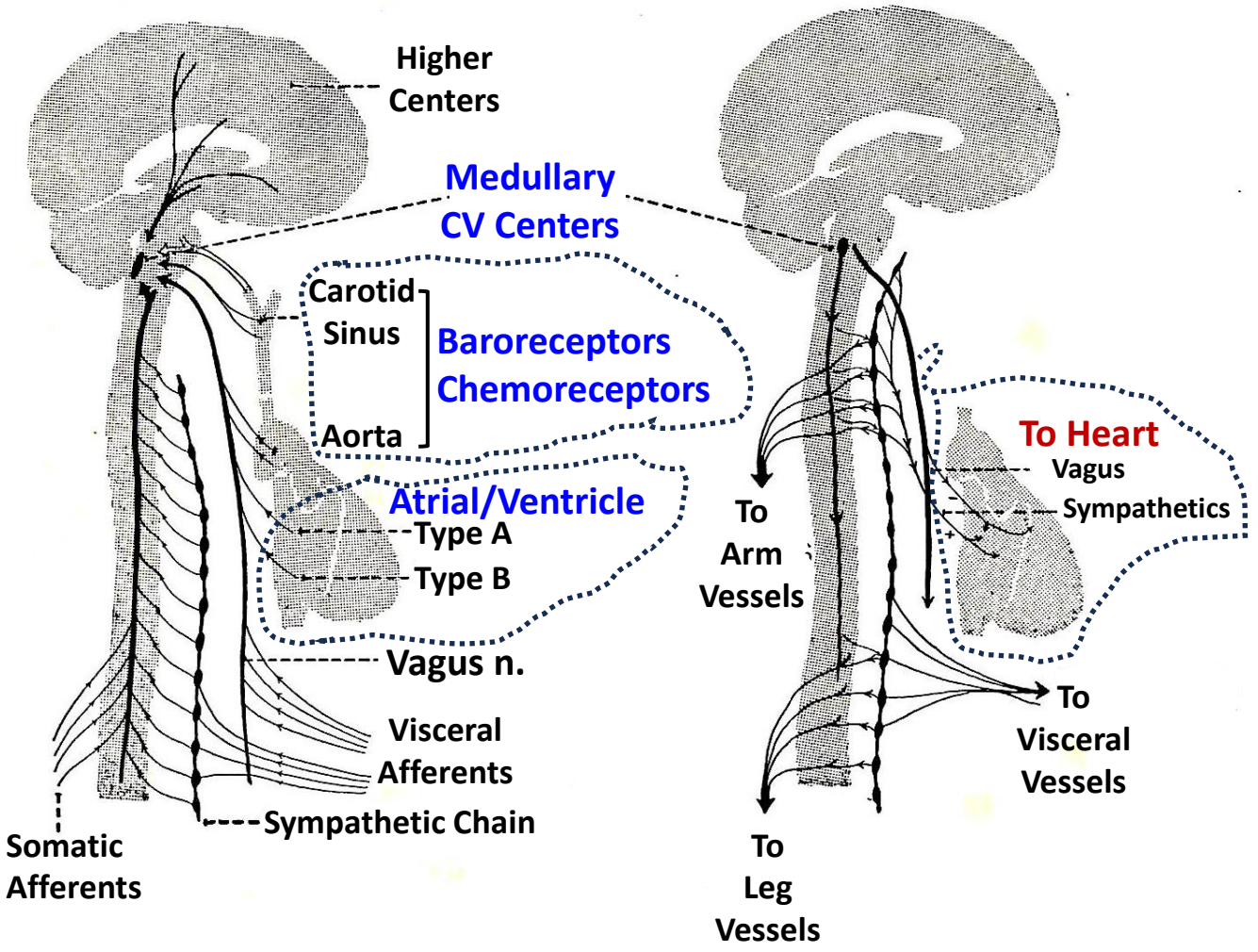
Topics

- Neural control overview
- Baroreceptors and their functions and responses
- Peripheral chemoreceptors and their functions and responses
- Central chemoreceptors and their functions and responses
- Cardiopulmonary low-pressure receptors
- Renin-Angiotensin-Aldosterone System (RAAS)
- Natriuretic Peptide System (NPS)
- Renal responses to blood pressure changes
- Hypotension pathways
- Hemorrhage responses

Cardiovascular Main Neural Control Pathways

Afferent nerves to vasomotor centers

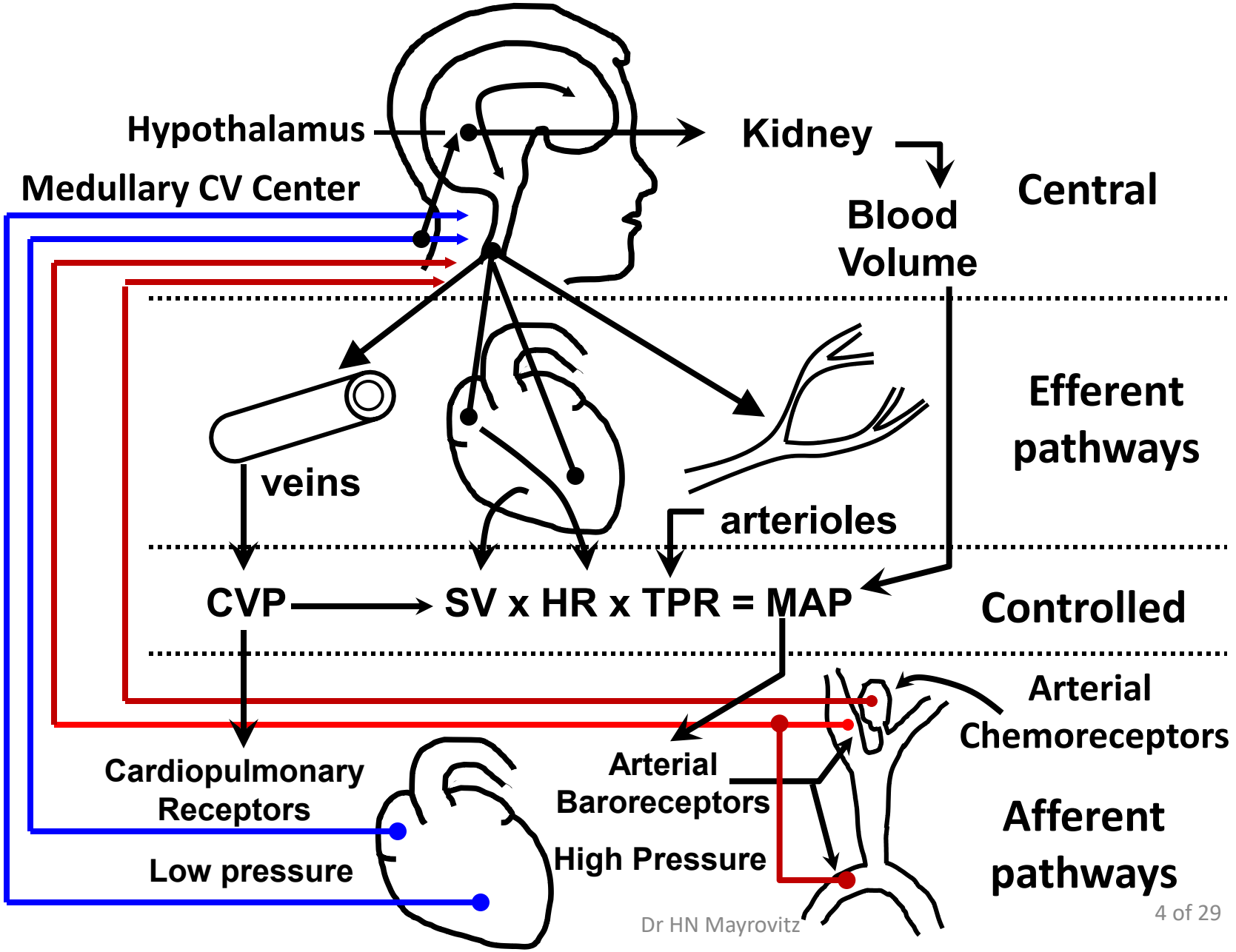
Efferent outflow from vasomotor centers



Preview of Take home

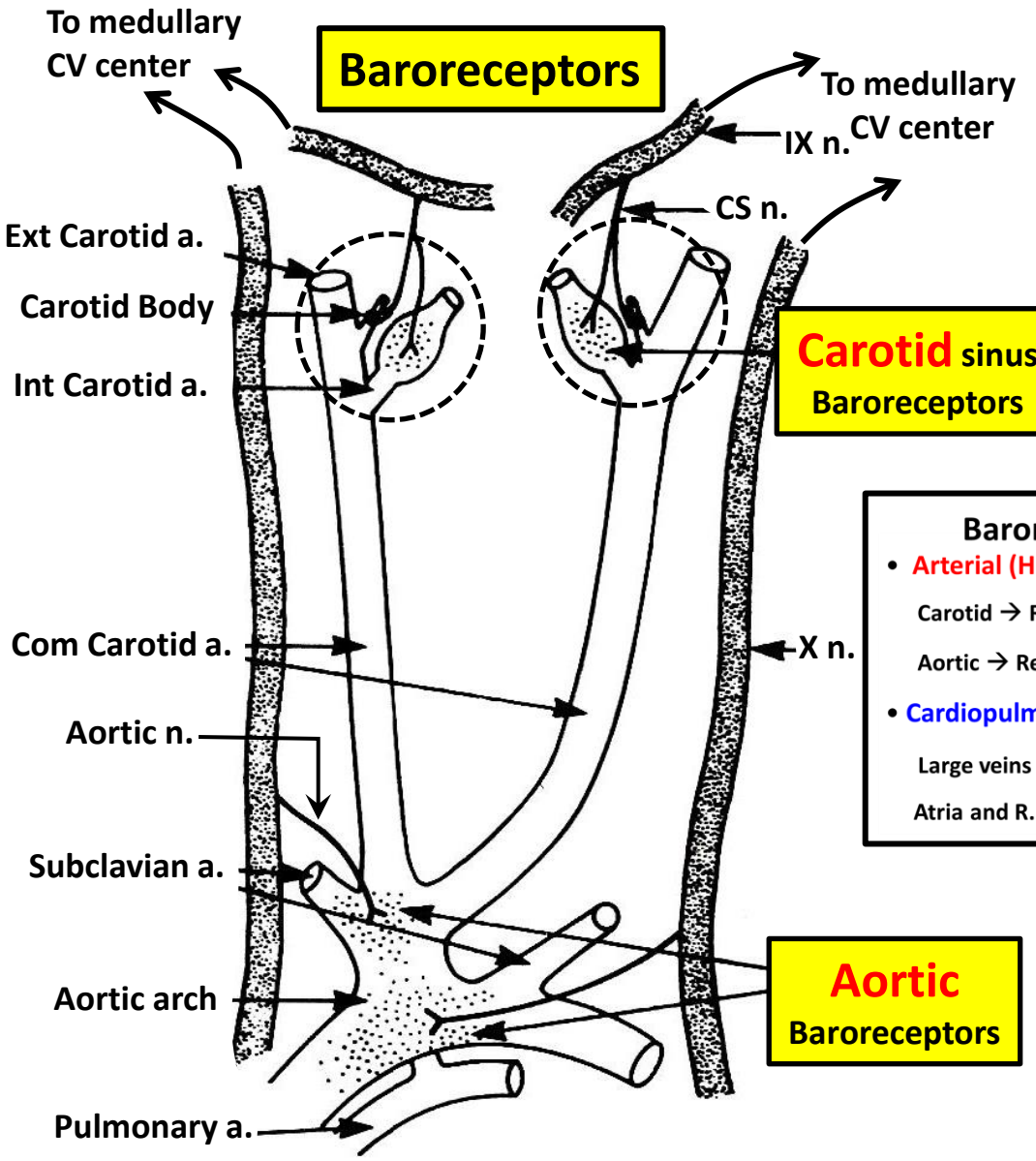
- Carotid and aortic baroreceptors are sensitive to pressure and its rate of change
- Peripheral chemoreceptors sensitive to pH, PO_2, and PCO_2
- Afferent feedback from these and other sensors monitor and respond to changes
- Efferent neural outflows to the heart affect HR (SA node), action potential properties, and myocardial contractility
- Efferent neural outflows to vessels affect constrictive state (vessel tone)
- Atrial and ventricular afferent nerve traffic depends on the volume changes within the cardiac chambers

Cardiovascular Control: With Feedback



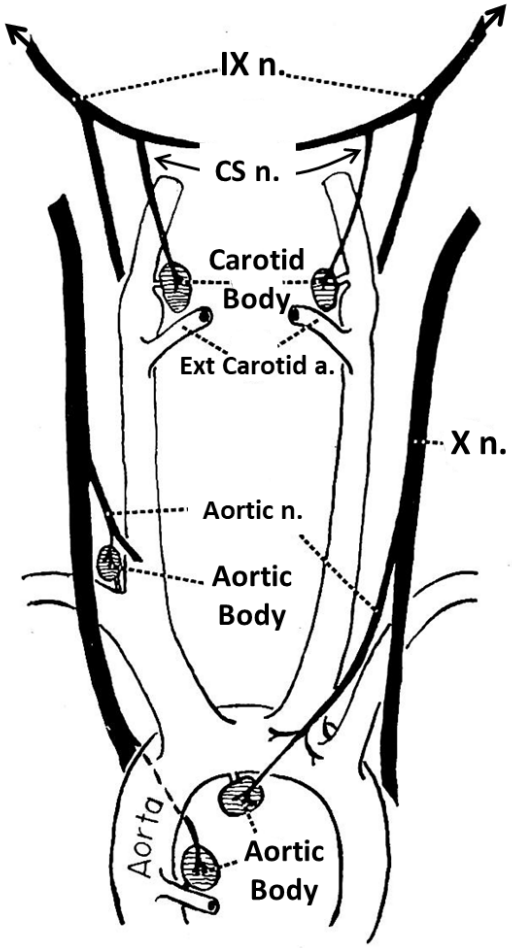
Baroreceptors and Chemoreceptors

Baroreceptors & Chemoreceptors: Closer Look



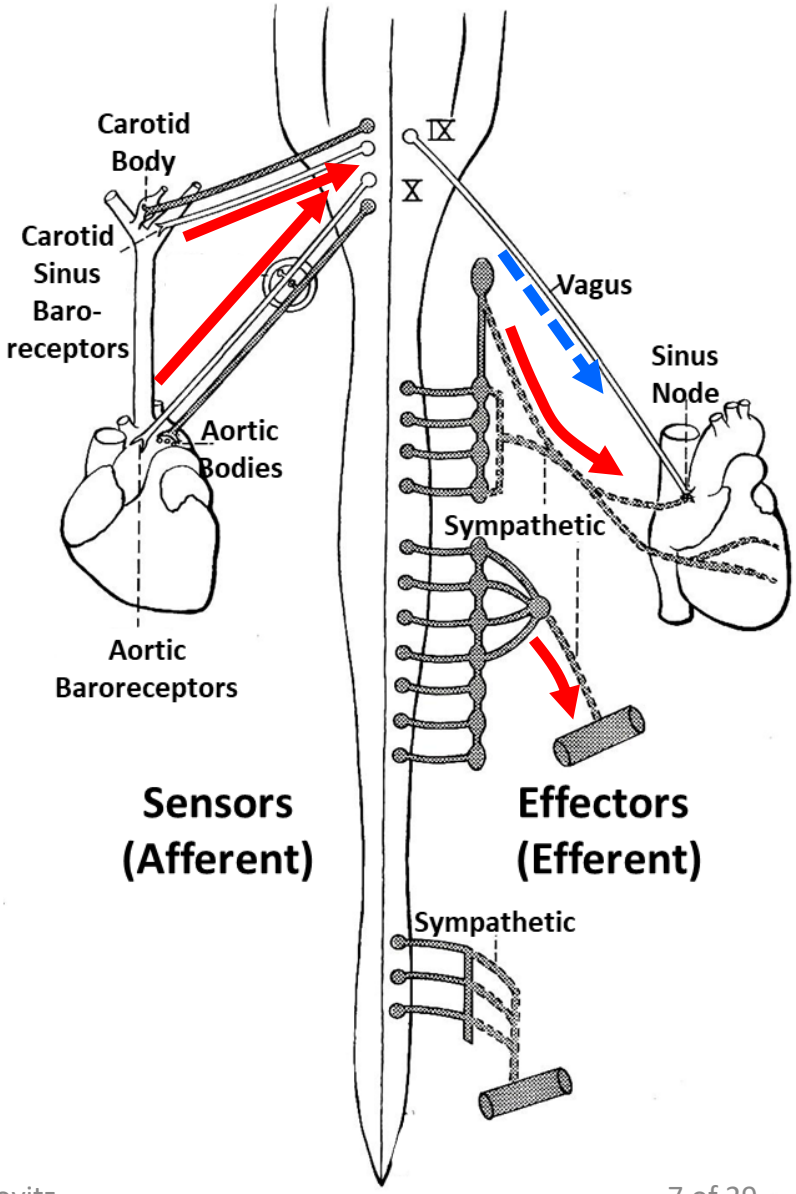
Baroreceptors

- **Arterial (High Pressure)**
  - Carotid → Respond to ± BP → ∓ BP
  - Aortic → Respond to + BP → - BP
- **Cardiopulmonary (Low Pressure)**
  - Large veins: ± Volume
  - Atria and R. Ventricle: ∓ Volume

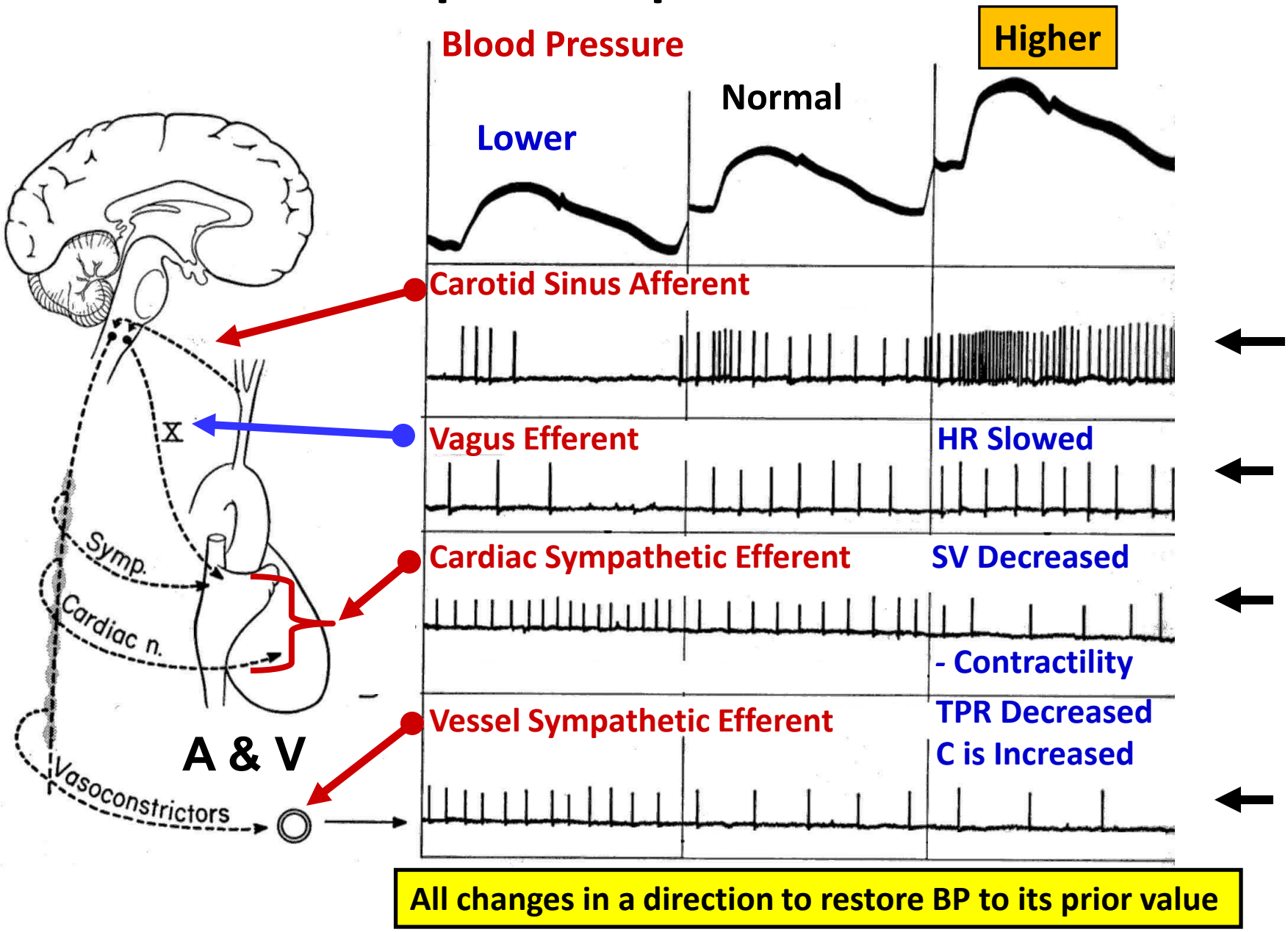


Carotid Baroreceptor Response Overview

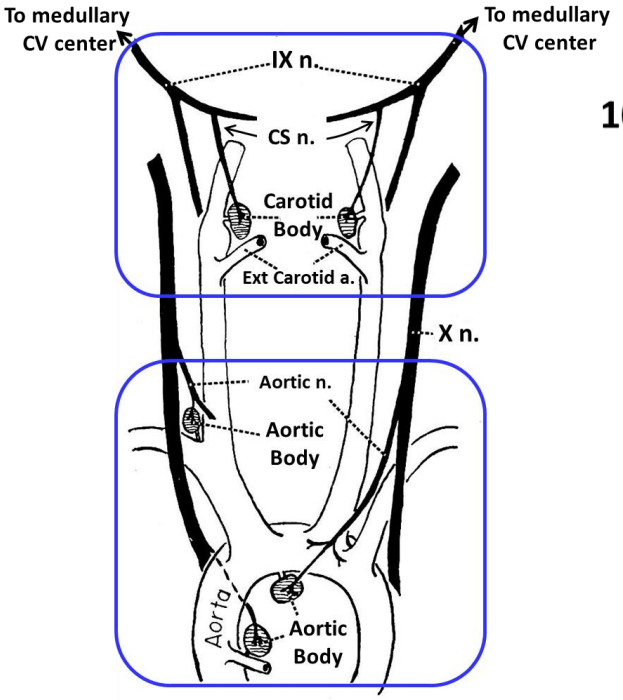
- *Increased blood pressure* causes increased afferent nerve firing to the medullary CV control center (MCVC)
- MCVC center actions alter efferent nerve traffic to heart and vessels
- HR is slowed via increased vagus impulses and contractility is reduced via reduced sympathetic impulses
- Action on blood vessels is to reduce TPR and increase venous compliance via decreased sympathetic excitation
- Opposite if blood pressure decreases
- Afferent nerve traffic depends on the amount and rate of change of BP
- Direction of change is to return BP to its prior value via negative feedback

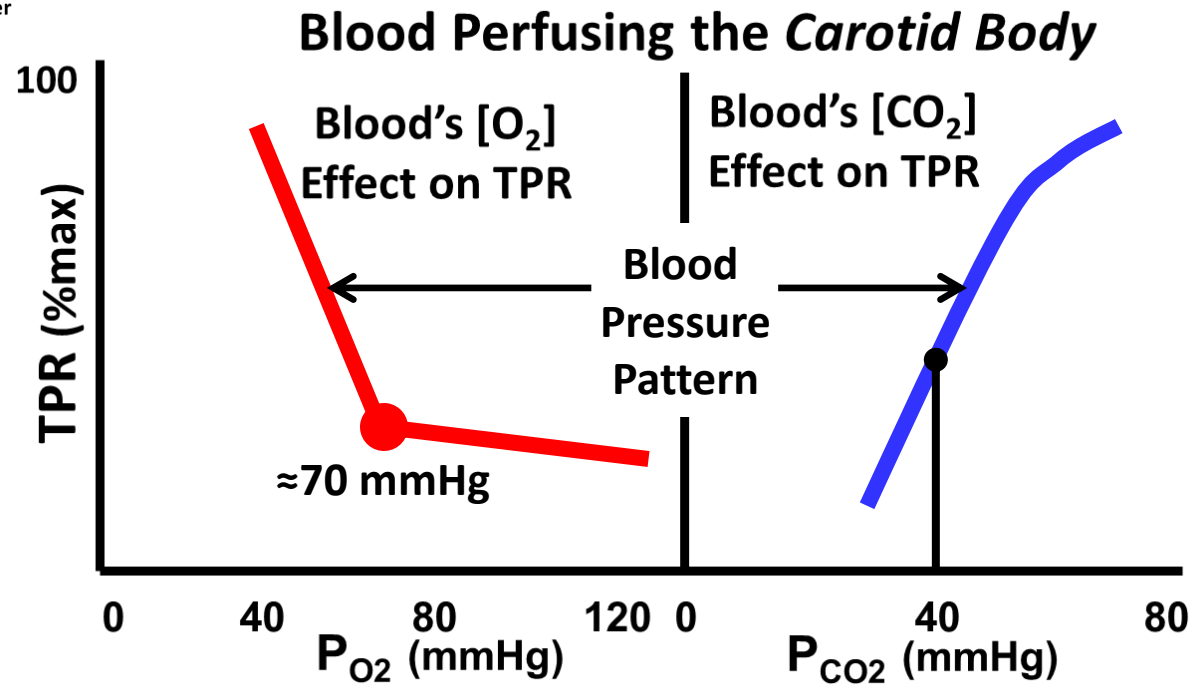


Carotid Baroreceptor Response to Pulse Pressure



Peripheral Chemoreceptor Responses

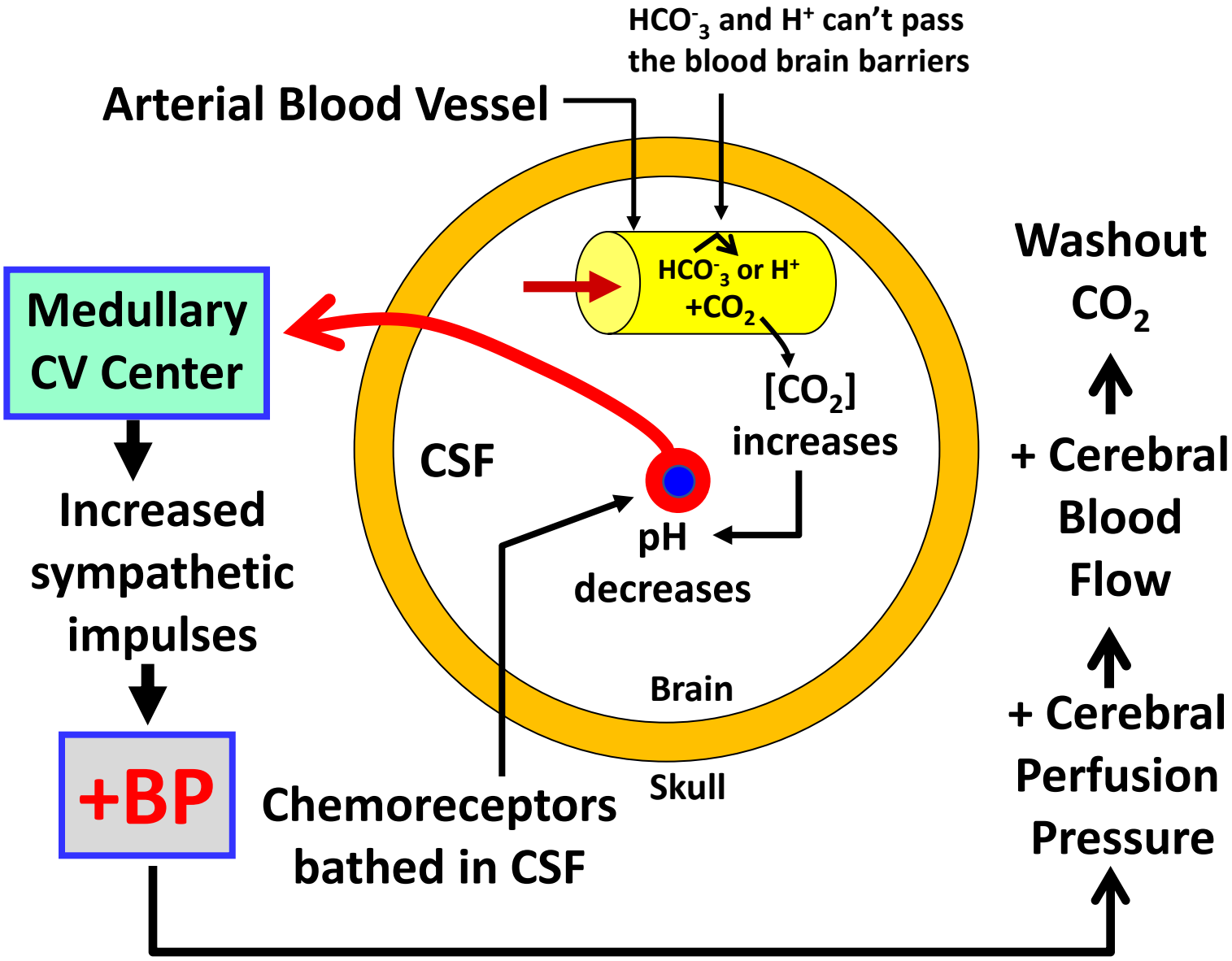




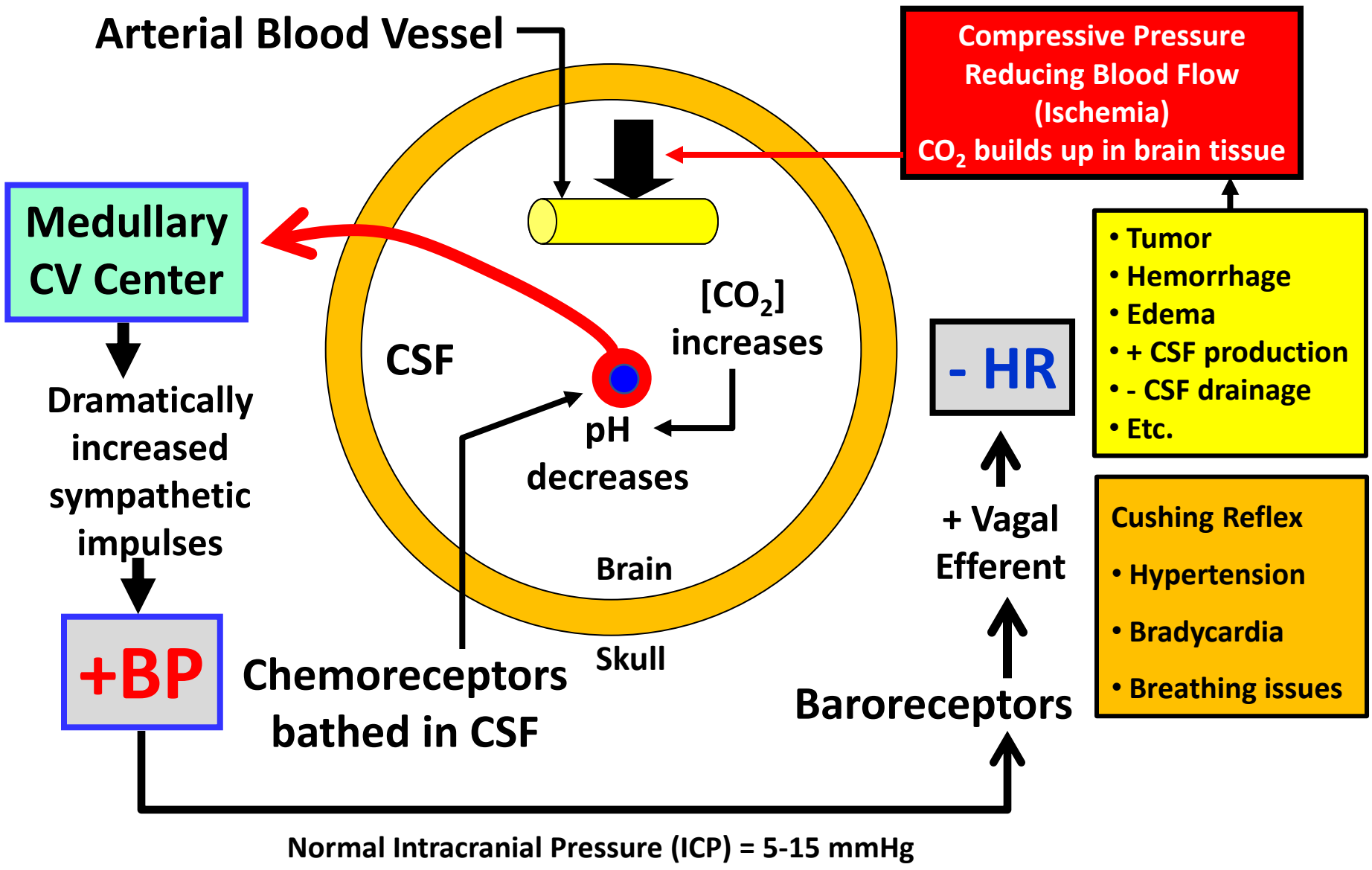
Take Home:

- **Peripheral chemoreceptors located in carotid and aortic bodies sense changes in blood oxygen pressure (PO_2), carbon dioxide pressure (PCO_2) and blood pH.**
- **A decrease in PO_2 to about 70 mmHg causes a steep increase in TPR thereby causing arterial BP to increase.**
- **Changes in TPR occur with increases in PCO_2 and decreases in pH.**

Central Chemoreceptors: Response to +Arterial CO_2



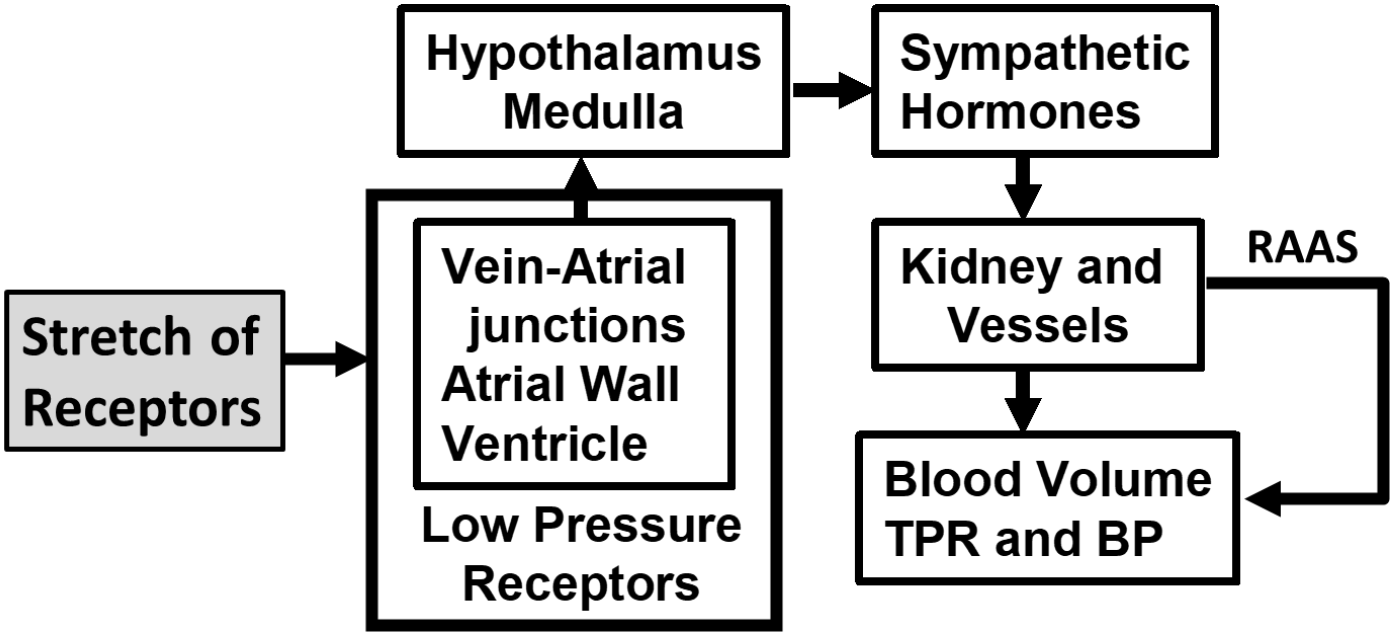
Central Chemoreceptors: Cushing Reflex



Low Pressure Baroreceptors

Cardio-Pulmonary Low-Pressure Receptors

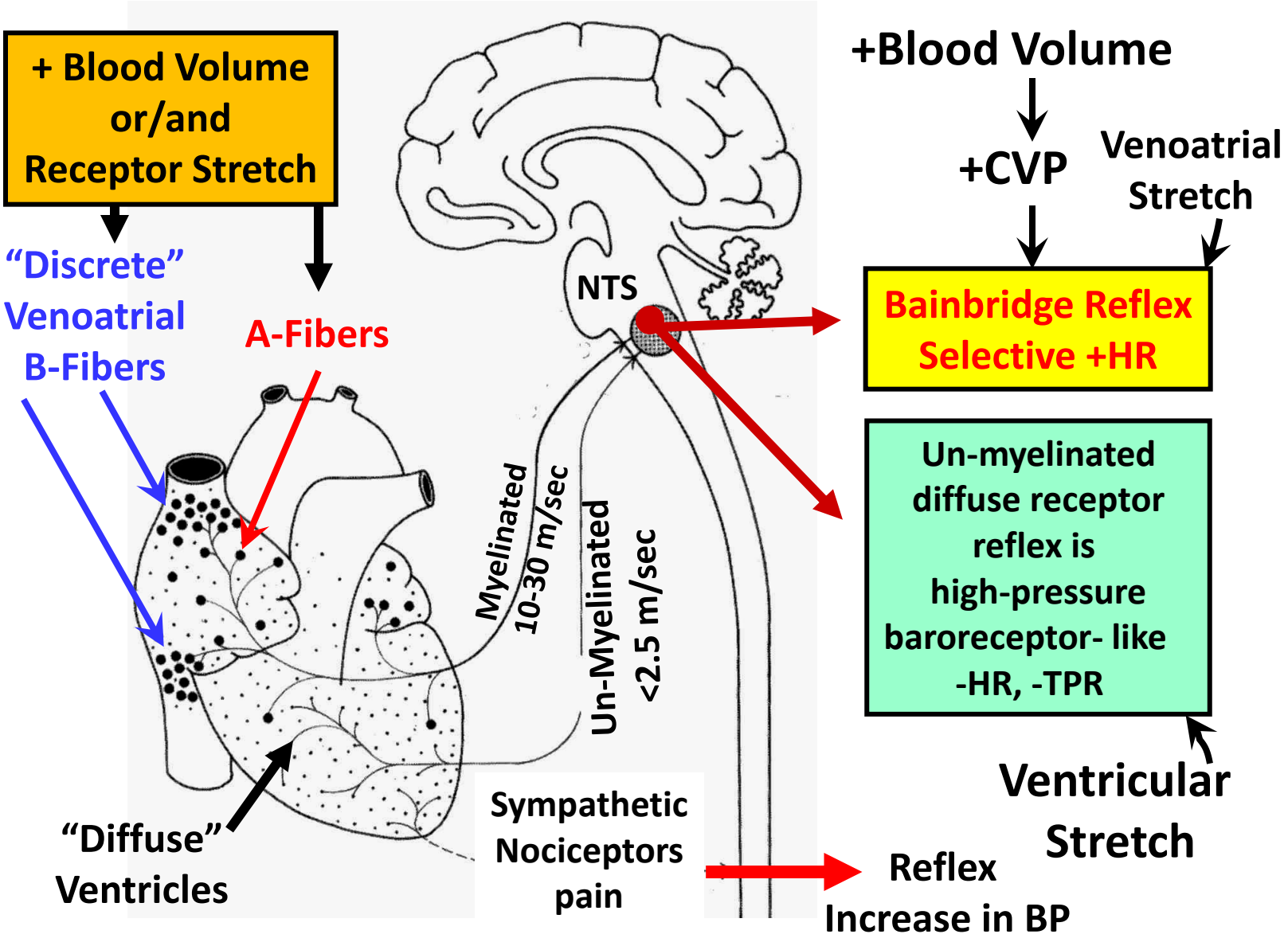
Low Pressure Receptors more Involved in "Longer-Term" BP Control in conjunction with Kidney Blood Volume Control



Cardiopulmonary (Low Pressure) Reflex

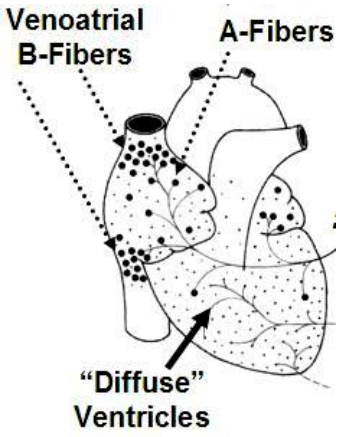
Large veins
Atria and R. Ventricle
} ± Volume → ∓ Volume

Low Pressure Receptor/Reflex Overview



NTS = Nucleus Tractus Solatarius

Low Pressure Receptors – Information Input



Atrial Systole

A-Fibers
Right Atrium

Heart Rate monitor

Afferent signals to brain "monitoring" HR

B-Fibers
SVC/IVC

Volume monitor

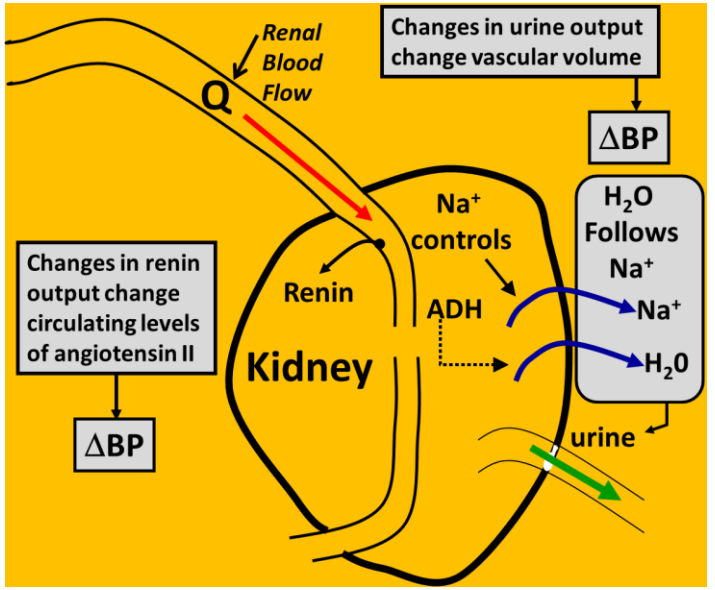
Atrial Filling - CVP Signaling
Bainbridge Reflex

Renin-Angiotensin-Aldosterone System (RAAS)

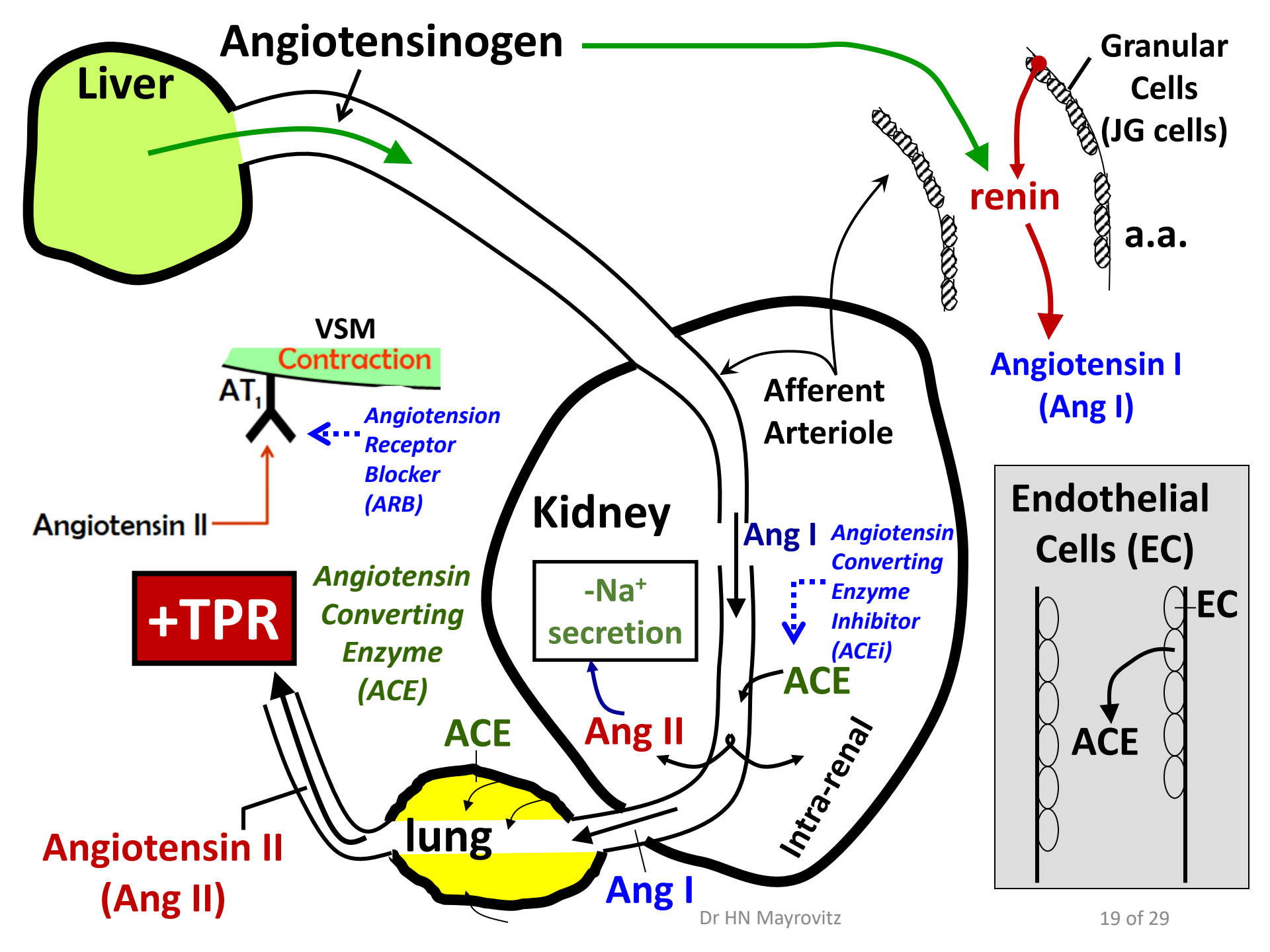
Renin-Angiotensin-Aldosterone System (RAAS)

The major "Players"

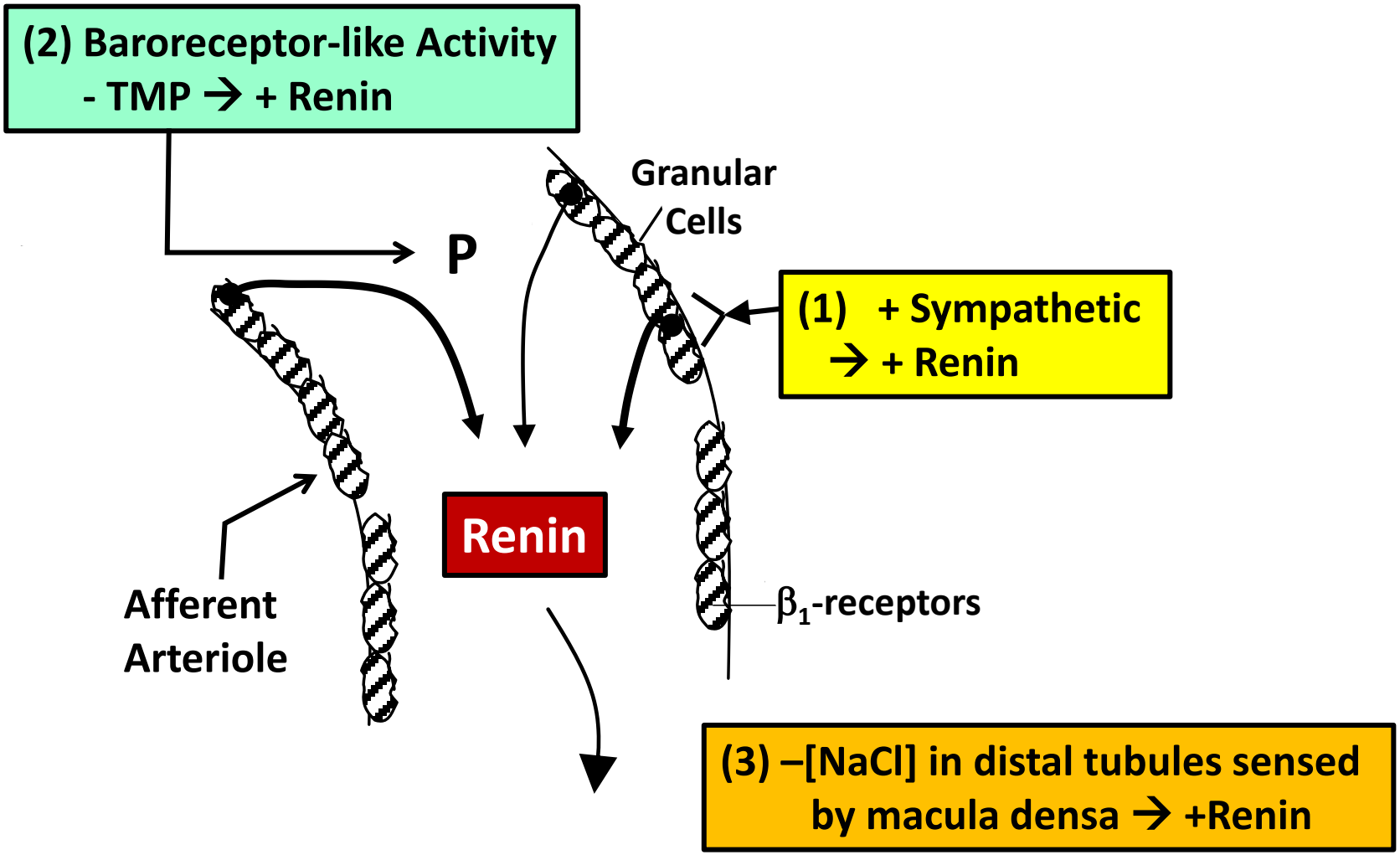
- Angiotensinogen → Protein made in and released from liver
- Renin → Proteolytic enzyme → released from kidney
- Angiotensin I → (ANG I) → Kidney → made by renin acting on angiotensinogen
- Angiotensin Converting Enzyme (ACE) → released in kidney and lung
- Angiotensin II → (ANG II) → ACE acting on ANG I → A constrictive peptide
- Antidiuretic Hormone (ADH) also called Vasopressin → from pituitary
  - → Vasoconstrictive action
  - → Promotes water reabsorption in kidney
- Aldosterone → steroid hormone → adrenal cortex
  - → Promotes Na^+ reabsorption (and H_2O) in kidney
  - → Promotes K^+ excretion in kidney
  - → Increased by ANG II

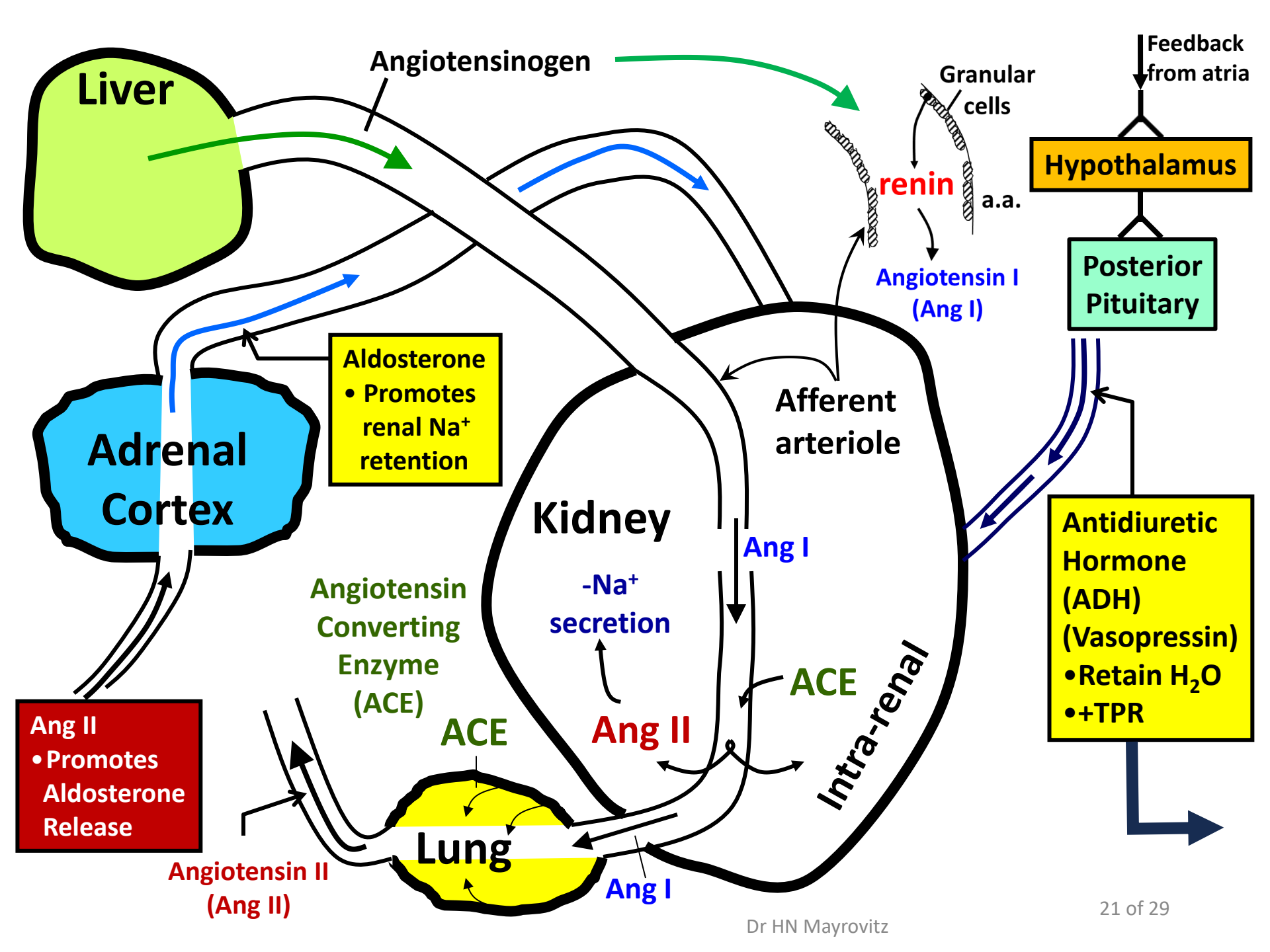


Renin-Angiotensin

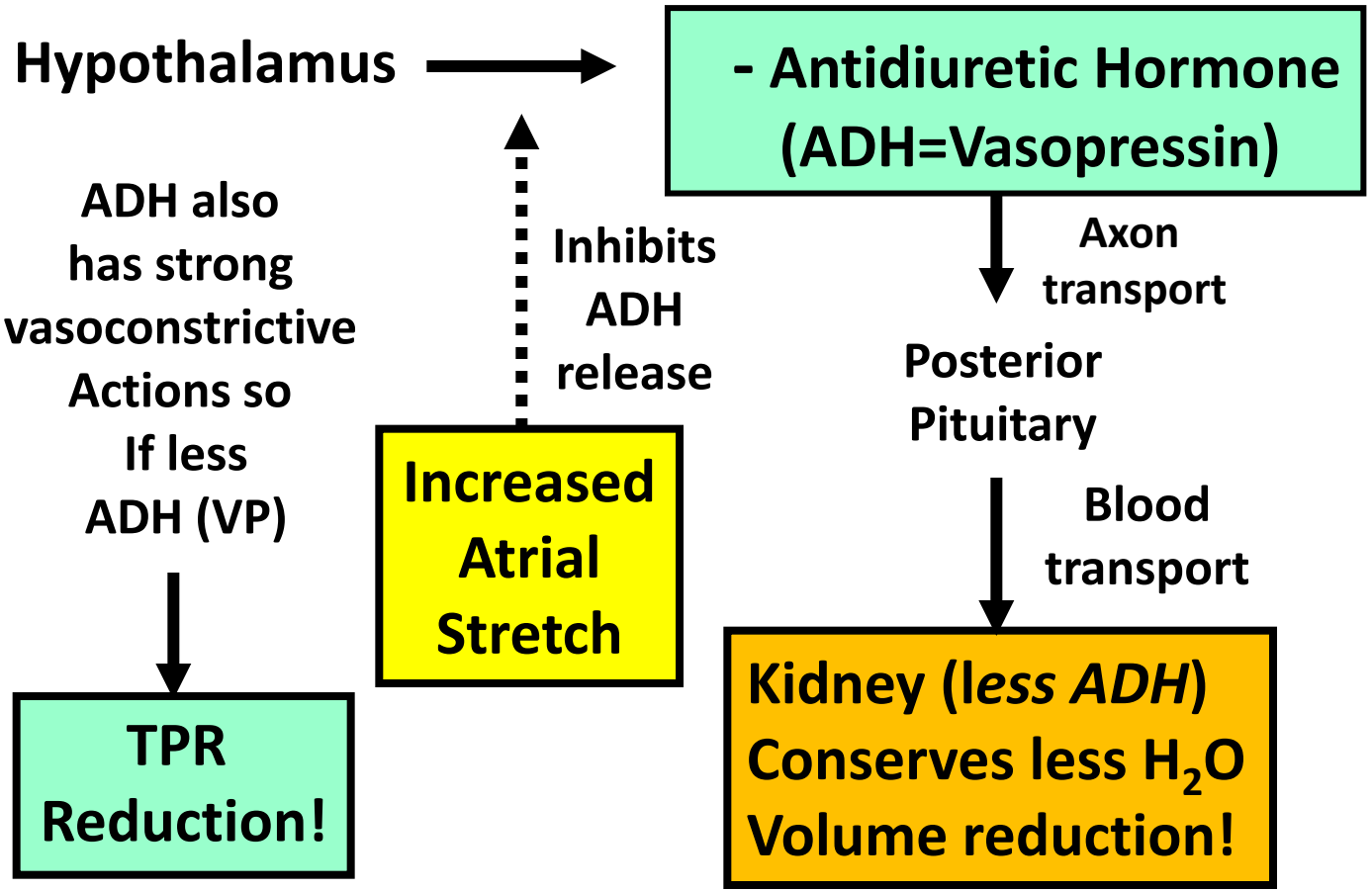

Angiotensinogen
Liver
Granular Cells (JG cells)
renin
a.a.
Angiotensin I (Ang I)
VSM
Contraction
AT1
Angiotension Receptor Blocker (ARB)
Angiotensin II
Afferent Arteriole
Kidney
Ang I
Angiotensin Converting Enzyme Inhibitor (ACEi)
-Na+ secretion
+TPR
Angiotensin Converting Enzyme (ACE)
ACE
Ang II
ACE
Intra-renal
lung
Angiotensin II (Ang II)
Ang I
Endothelial Cells (EC)
EC
ACE

Three Main Factors Affecting Renin Regulation




Liver
Angiotensinogen
Granular cells
Feedback from atria
renin
a.a.
Hypothalamus
Angiotensin I (Ang I)
Posterior Pituitary
Aldosterone
• Promotes renal Na^+ retention
Adrenal Cortex
Afferent arteriole
Kidney
Ang I
$-Na^+$ secretion
Antidiuretic Hormone (ADH) (Vasopressin)
• Retain H_2O
• +TPR
Angiotensin Converting Enzyme (ACE)
ACE
Ang II
ACE
Intra-renal
Ang II
• Promotes Aldosterone Release
Lung
Angiotensin II (Ang II)
Ang I

Antidiuretic Hormone (Vasopressin)



Reduced blood volume and TPR → Reduced BP

Natriuretic Peptide System (NPS)

Natriuretic Peptide System (NPS)

- As a general principle NPS actions tend to counterbalance RAAS actions
- BNP or the inactive NT-proBNP is used as a marker for CHF
- BNP used to track Acute Coronary Syndrome severity and progression

Three main peptides involved in the NPS

(1) Atrial Natriuretic Peptide (ANP) → ANP released from atrial myocytes

(2) B-type (or Brain) Natriuretic Peptide (BNP) → released from ventricle myocytes

(3) C-type Natriuretic Peptide (CNP) → released from EC → local vasodilation

ANP & BNP

(1) Stored as a long-chain polypeptide (ProBNP).

(2) Release stimulated by stretch, ANG II, + sympathetic nerve stimulation (SNS).

NPS effects due to actions on Natriuretic Peptide Receptors (NPR)

(1) ANP and BNP both selectively bind to receptor NPR-A; cause similar responses

(2) CNP binds to receptor NPR-B.

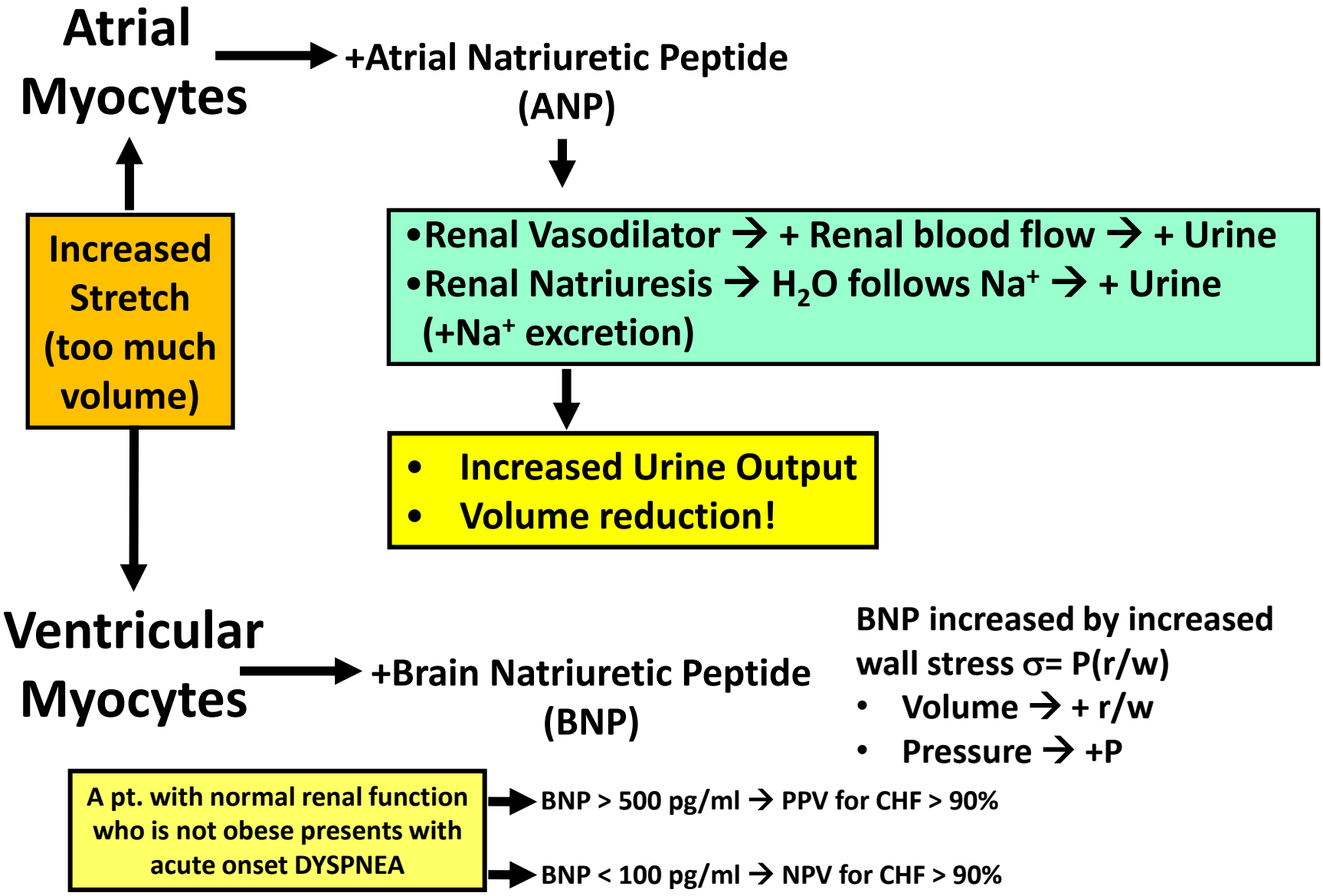
(3) Both receptor types use cGMP as a 2nd messenger.

(4) Each peptide cleared by

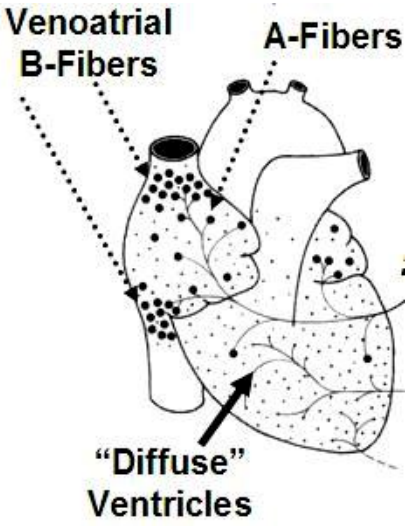
- (a) enzymatic action of neutral endopeptidase (NEP) or by
- (b) binding to a 3rd receptor (NPR-C) that internally degrades peptides.

(5) Half life of BNP is ~ 20 minutes and that of NT-proBNP is about 120 minutes.

Atrial and Brain Natriuretic Peptide (ANP/BNP)



Neural Response to +Atrial Stretch



+ Heart Rate → "Bainbridge Reflex"

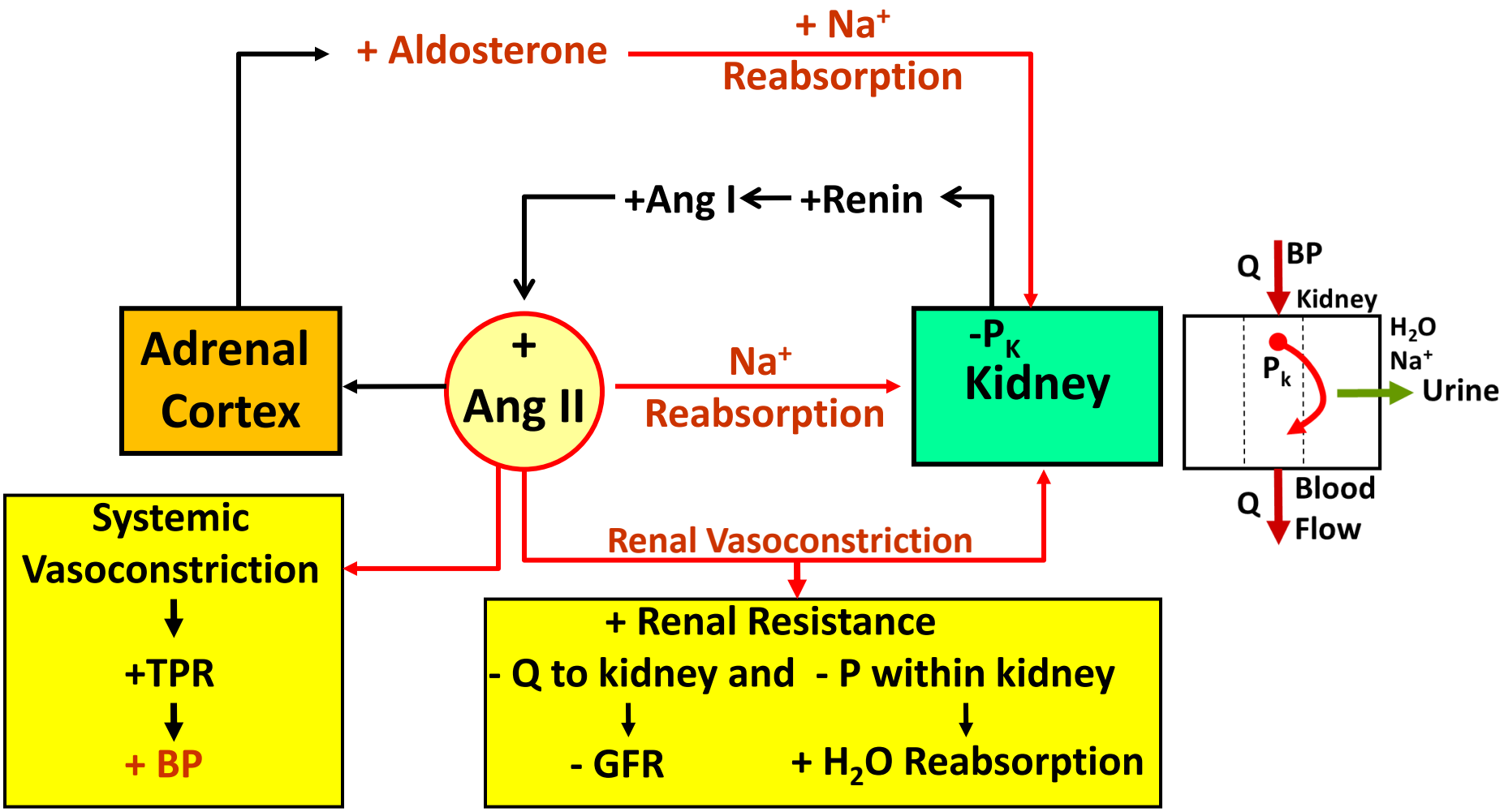
- Sympathetic Only to kidney

Renal Vasodilation and Reflex tachycardia

+ Renal Blood Flow
+ Urine Output (Diuresis)

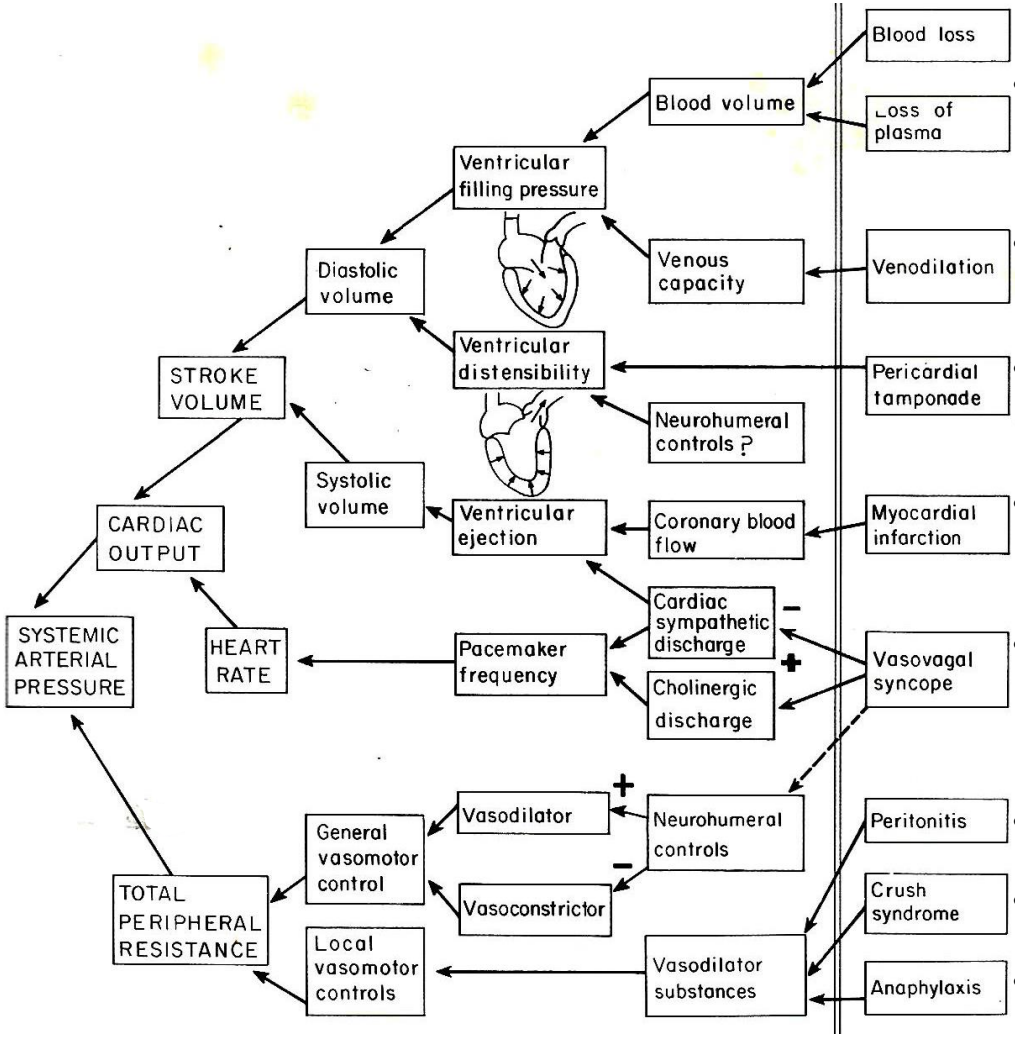
Volume Reduction!

Renal Responses to Decreased Blood Pressure



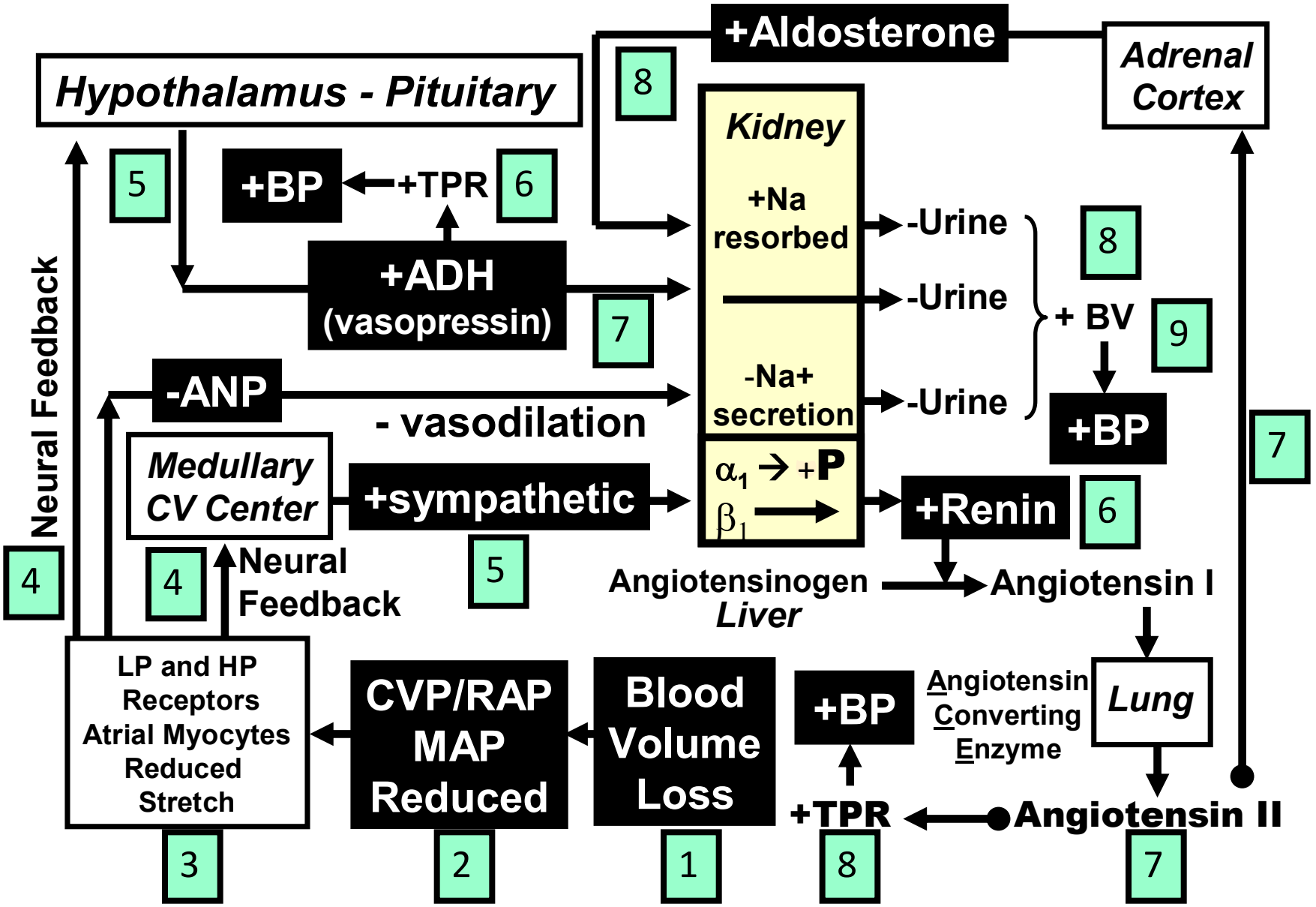
Arterial Hypotension Events and Possible Shock

Various causes and pathways to BP decrease shown, but compensatory responses not shown



- **Hemorrhage of sufficient amount either of whole blood or plasma so that volume decreased**
- **Excess blood sequestered into venous system not available for circulation**
- **Fluid build-up between pericardium and myocardium causing external compression**
- **Inadequate blood flow and/or functional myocardium to support adequate function**
- **Inappropriate intense vagal discharge causing decreased HR and slightly reduced contractility**
- **Perforation – Cirrhosis – Kidney disease**
- **Crush injury and release → Rhabdomyolysis**
- **Foods – Meds – Insects - Allergic**

Hemorrhage / Blood Loss Pathways



End CV Physiology Lecture 14