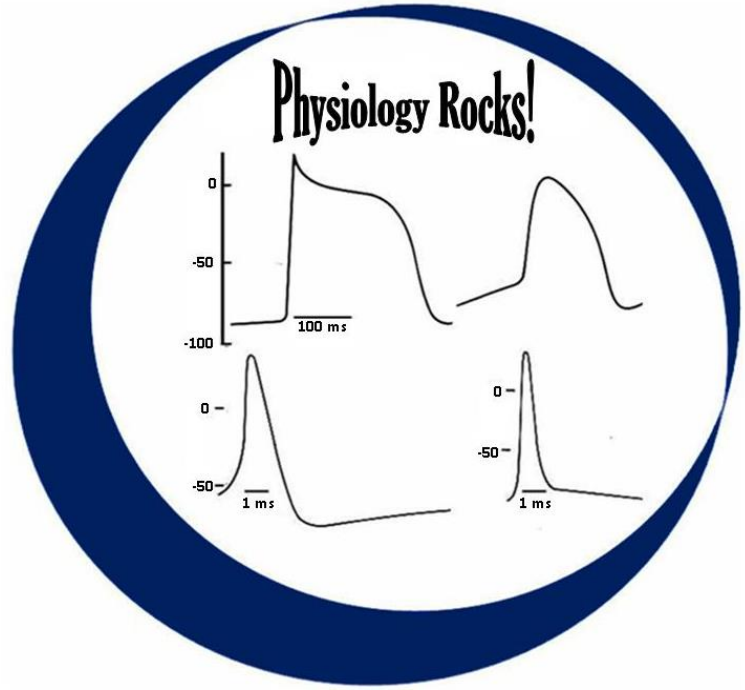
Lecture 14
Cardiovascular Controls and Reflexes



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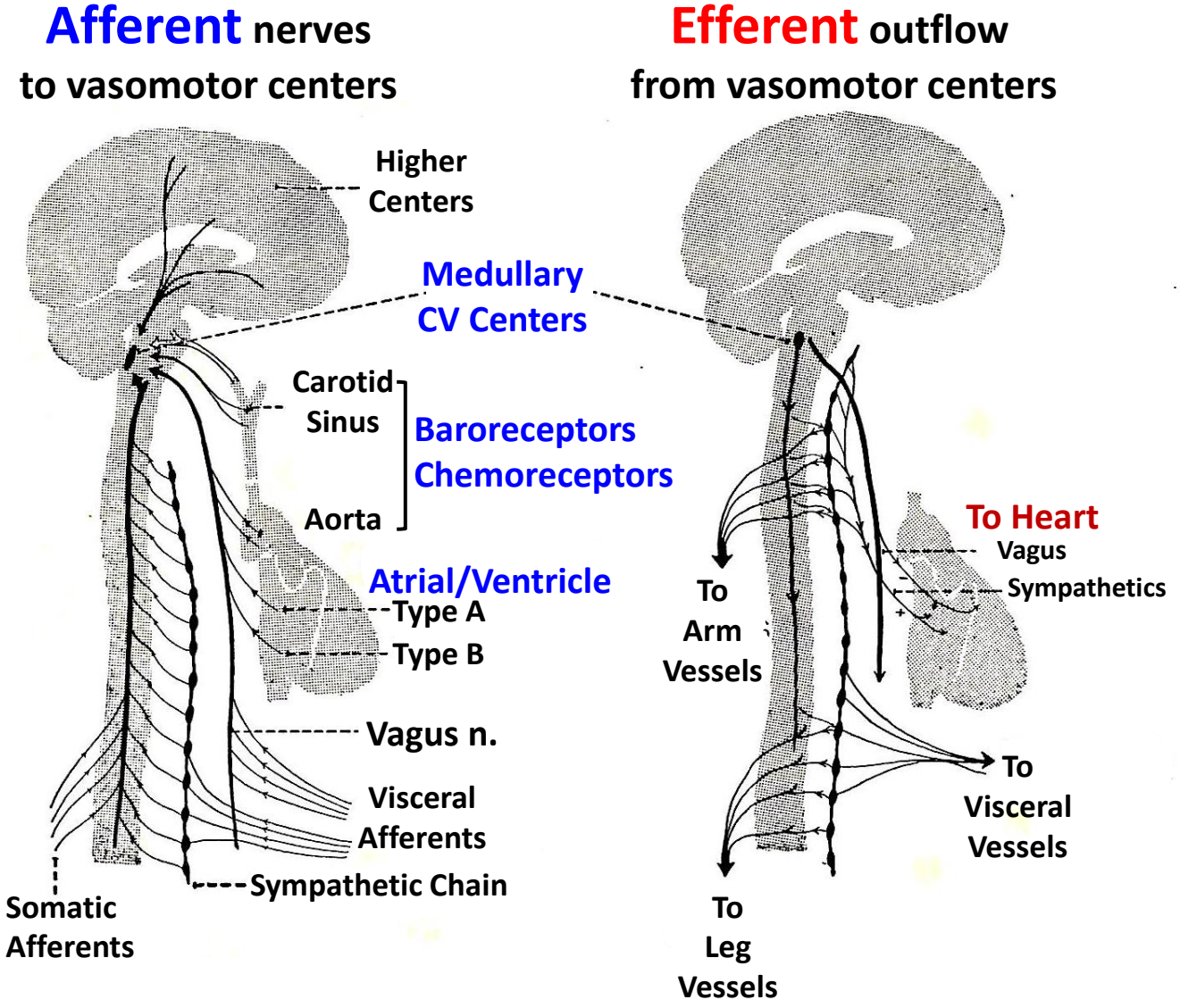
Topics

- 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

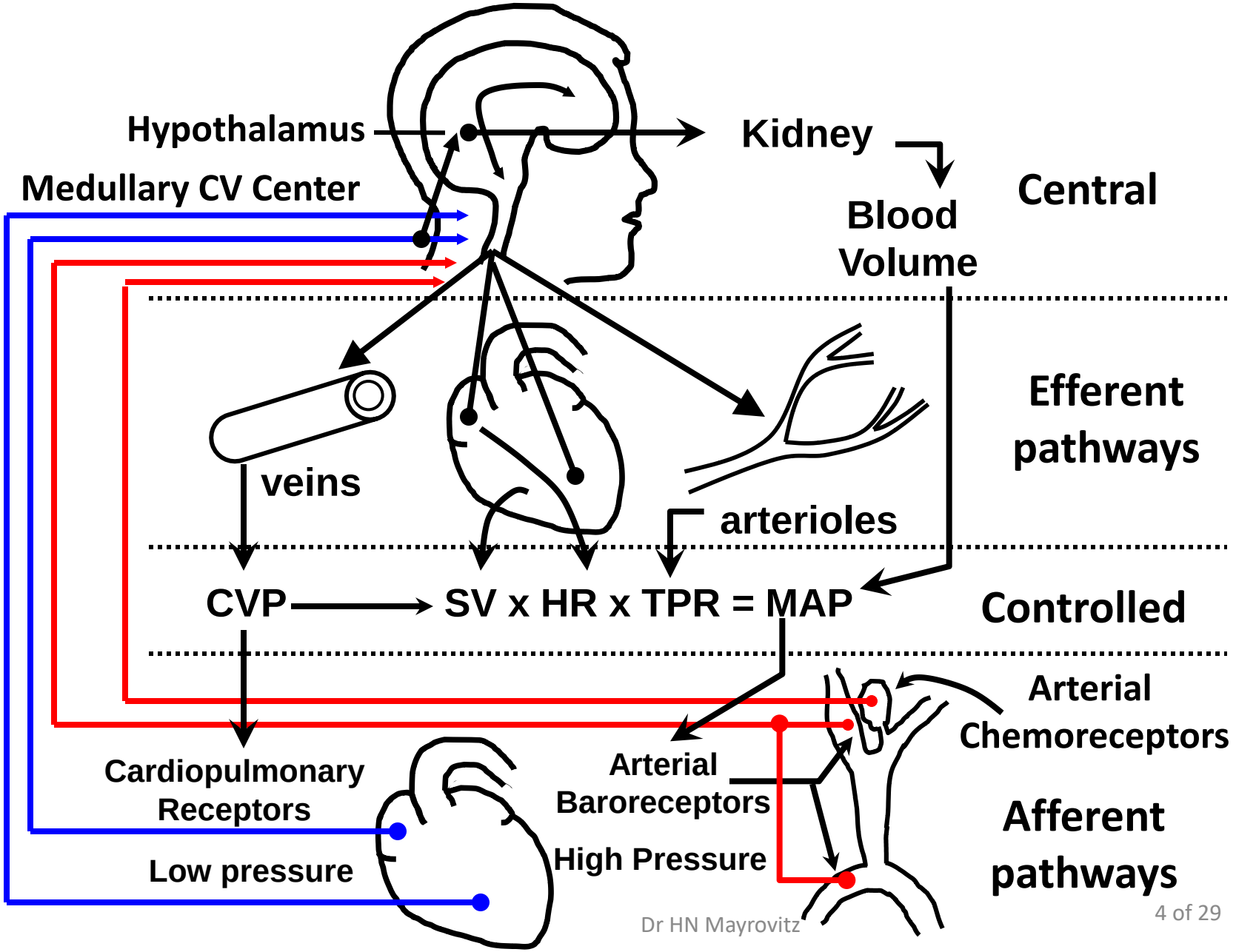
Cardiovascular Main Neural Control Pathways

Take Home



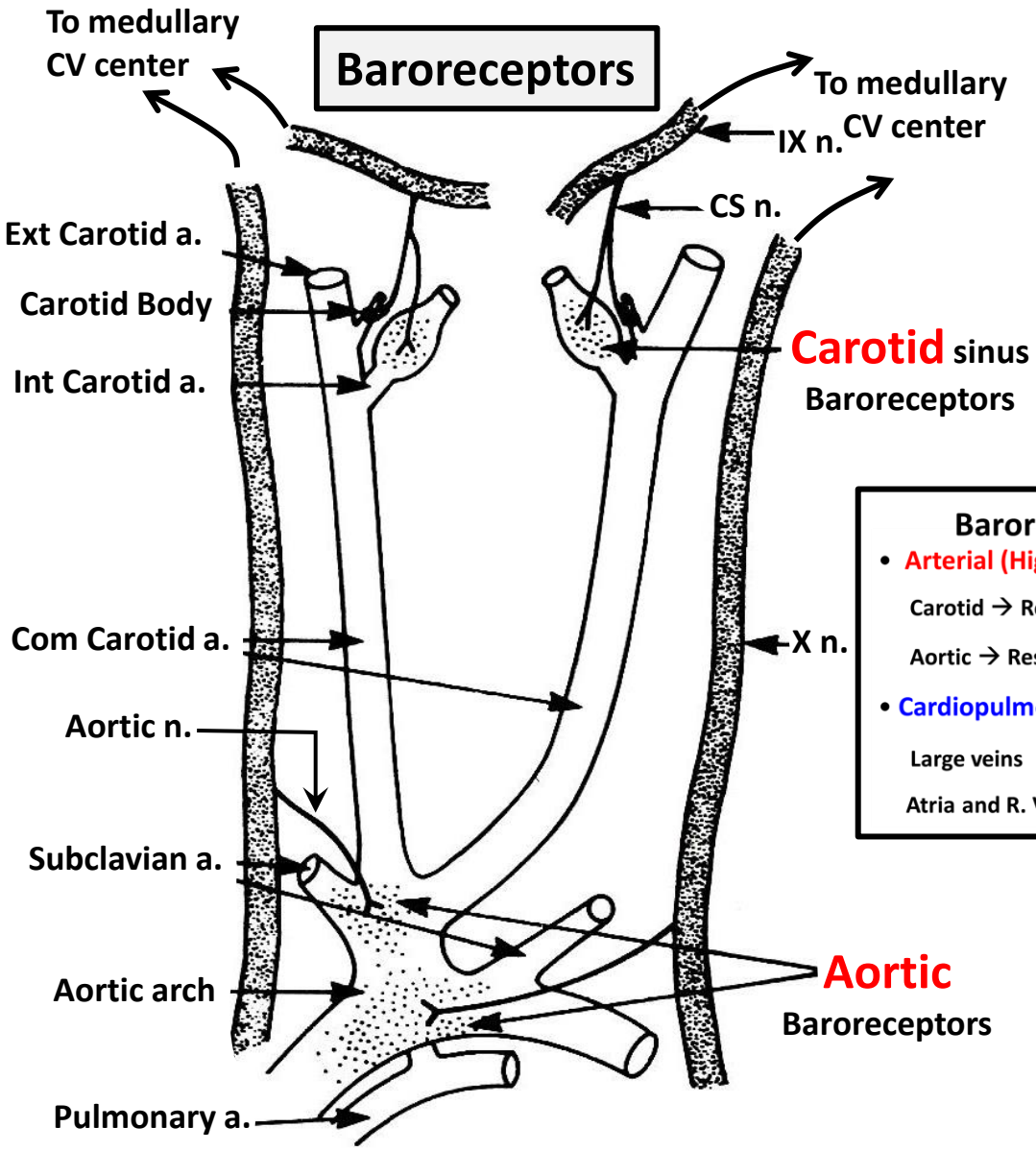
- Carotid and aortic baroreceptors are sensitive to pressure and its rate of change
- Peripheral chemoreceptors sensitive to pH, PO_2 and PCO_2
- Afferent feedback from these and other sensors monitor and respond to changes
- Efferent neural outflows to heart affect HR (SA node), action potential properties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

Cardiovascular Control: With Feedback



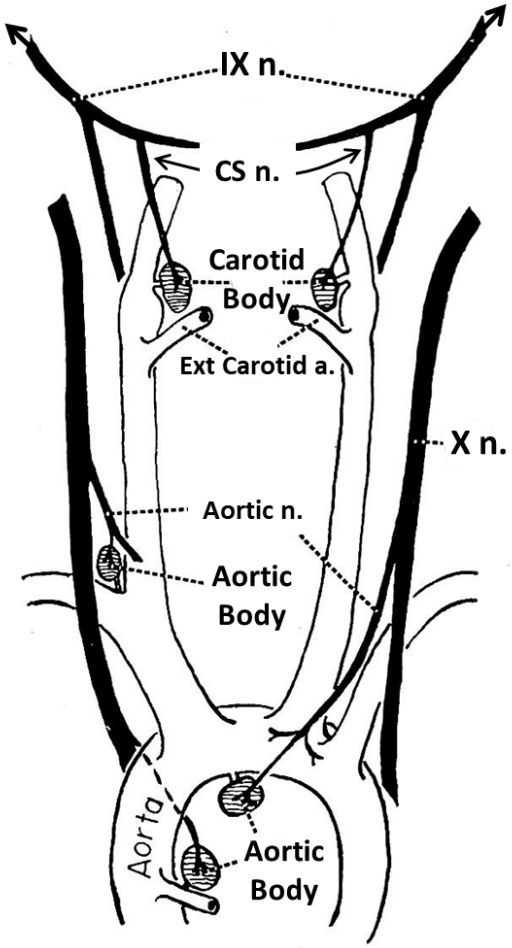
Baroreceptors and Chemoreceptors

Baroreceptors and Chemoreceptors



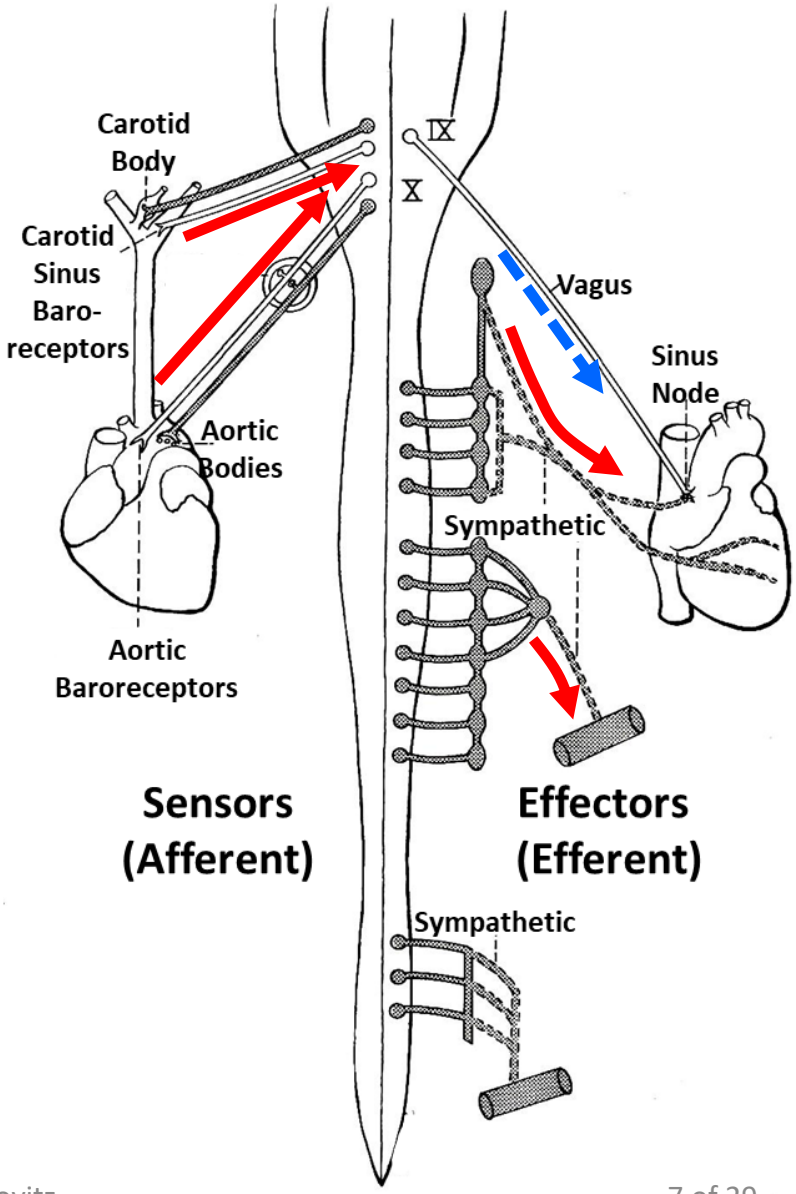
Baroreceptors

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

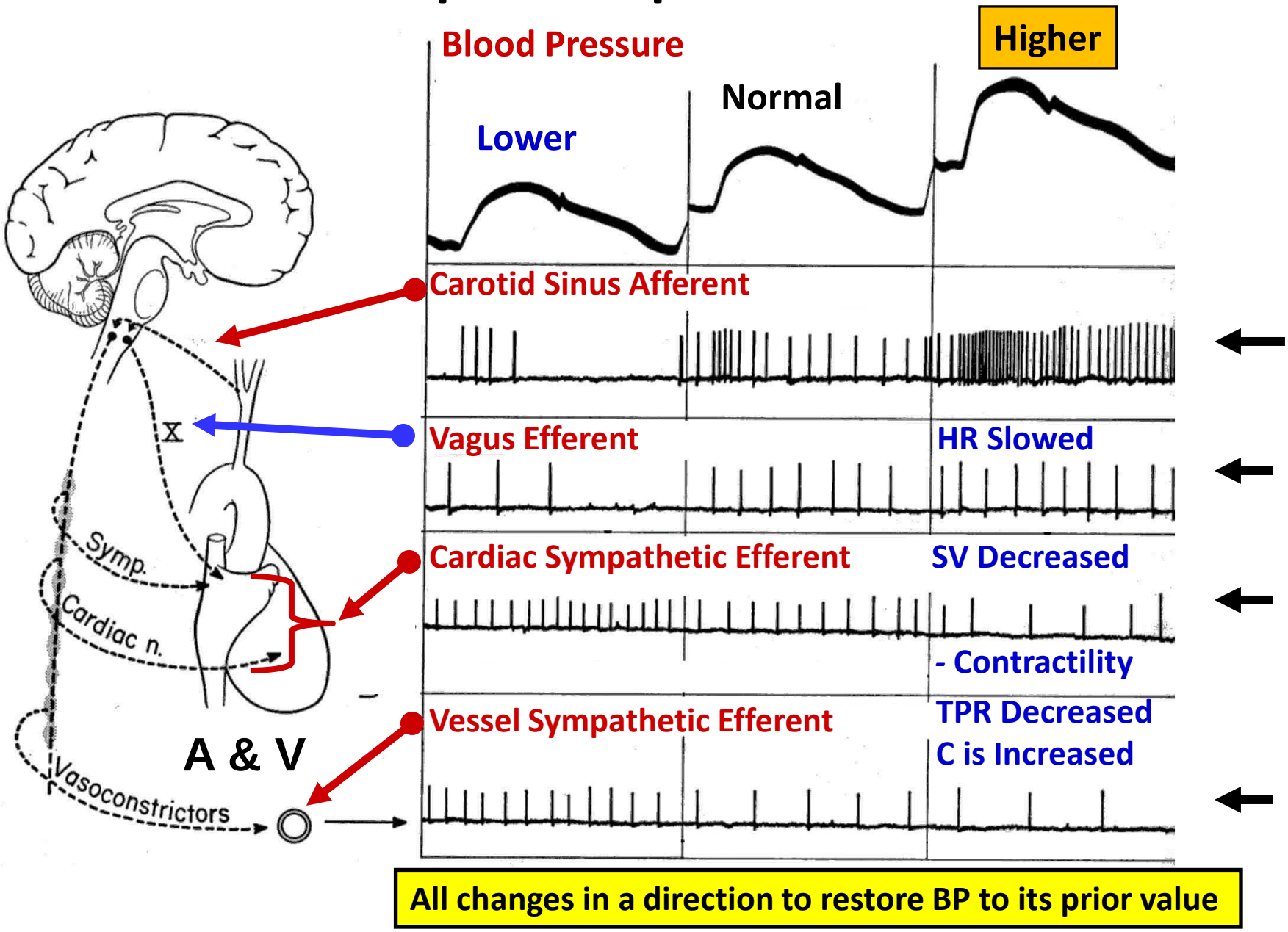


Carotid Baroreceptor Response Overview

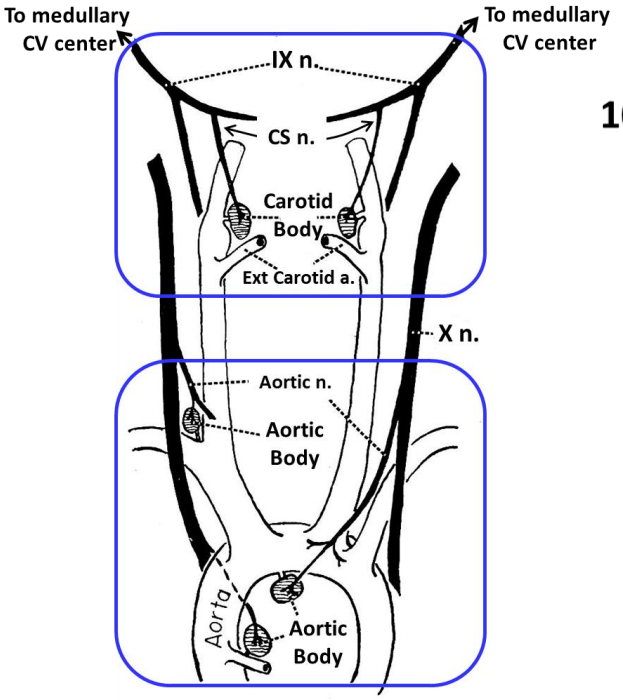
- *Increased blood pressure* causes increased afferent nerve firing to the medullary CV control center (MCVC)
- MCVC center actions alter efferent nerve traffic to heart and vessels
- HR is slowed via increased vagus impulses and contractility is reduced via reduced sympathetic impulses
- Action on blood 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

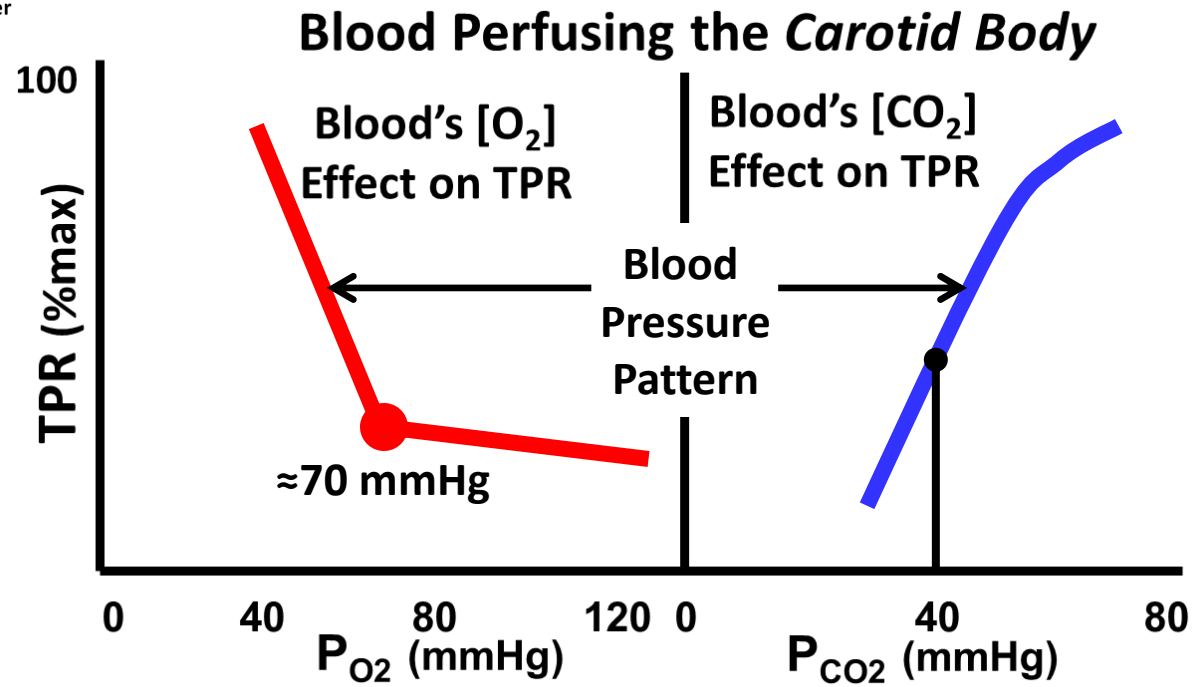


Carotid Baroreceptor Response to Pulse Pressure



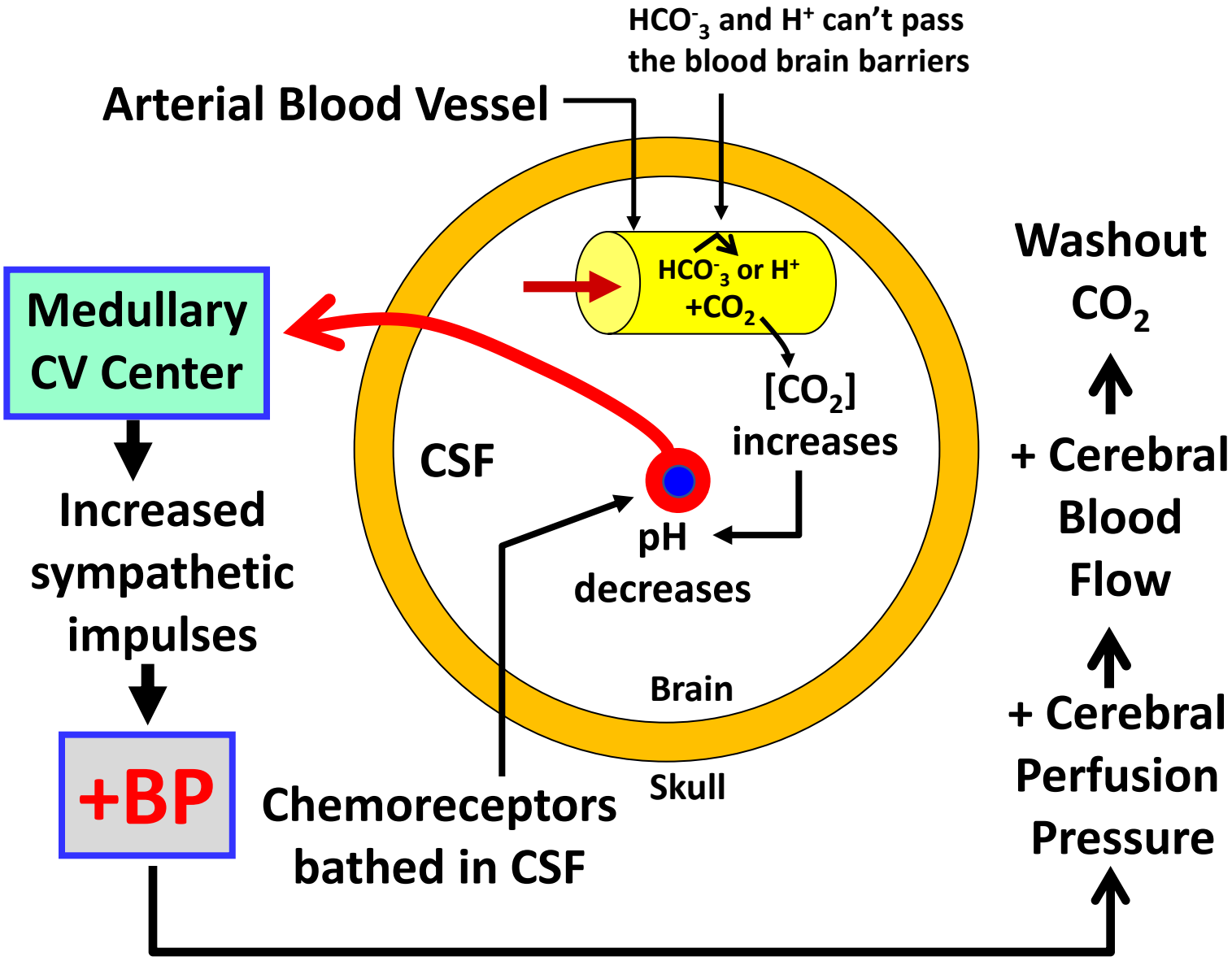
Peripheral Chemoreceptor Responses



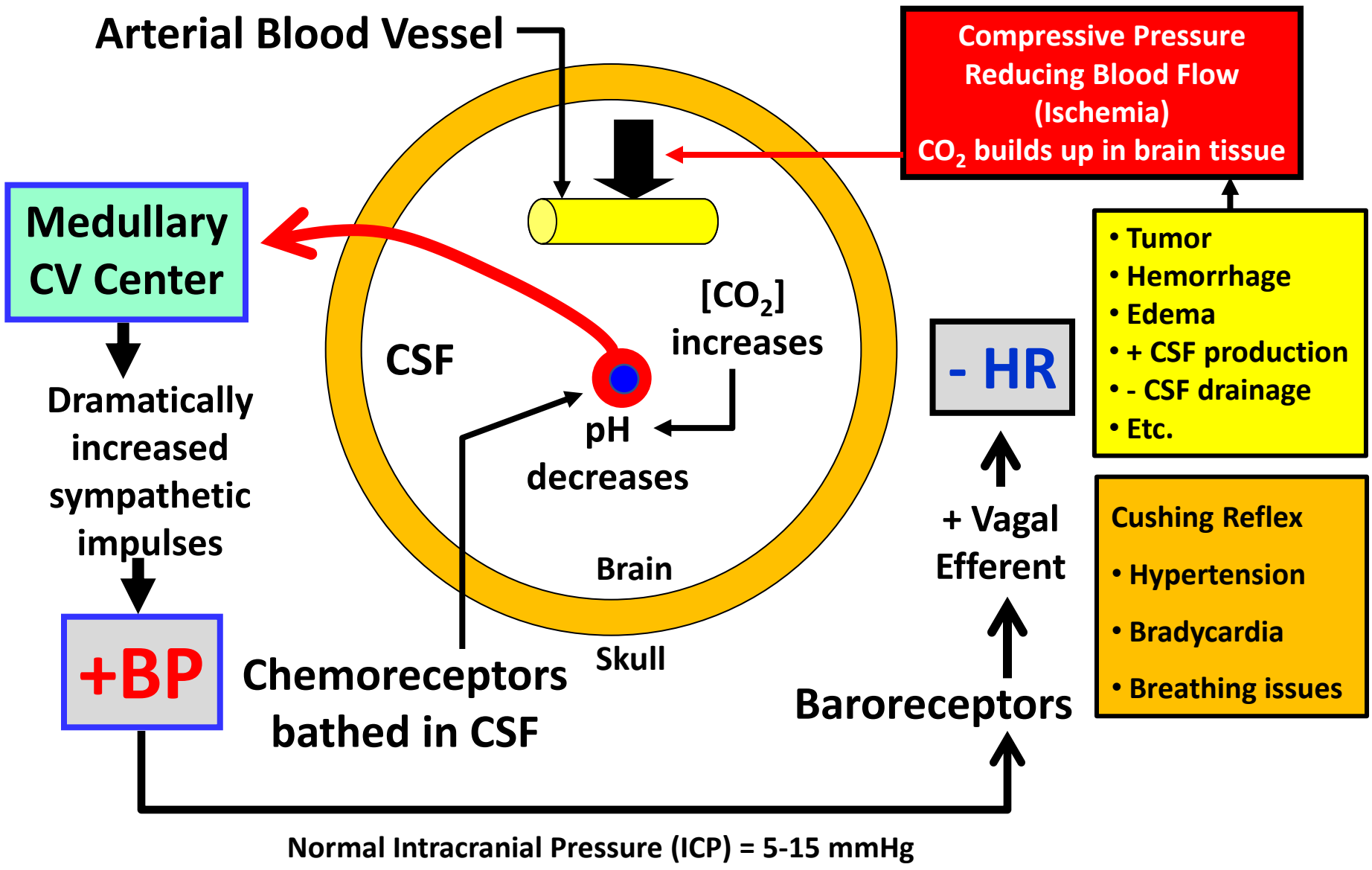


Peripheral chemoreceptors located in carotid and aortic bodies sense changes in blood oxygen pressure (Po_2), carbon dioxide pressure (Pco_2) and blood pH. A decrease in Po_2 to about 70 mmHg causes a steep increase in TPR thereby causing arterial BP to increase. Changes in TPR occur with increases in Pco_2 and decreases in pH.

Central Chemoreceptors: Response to +Arterial CO_2



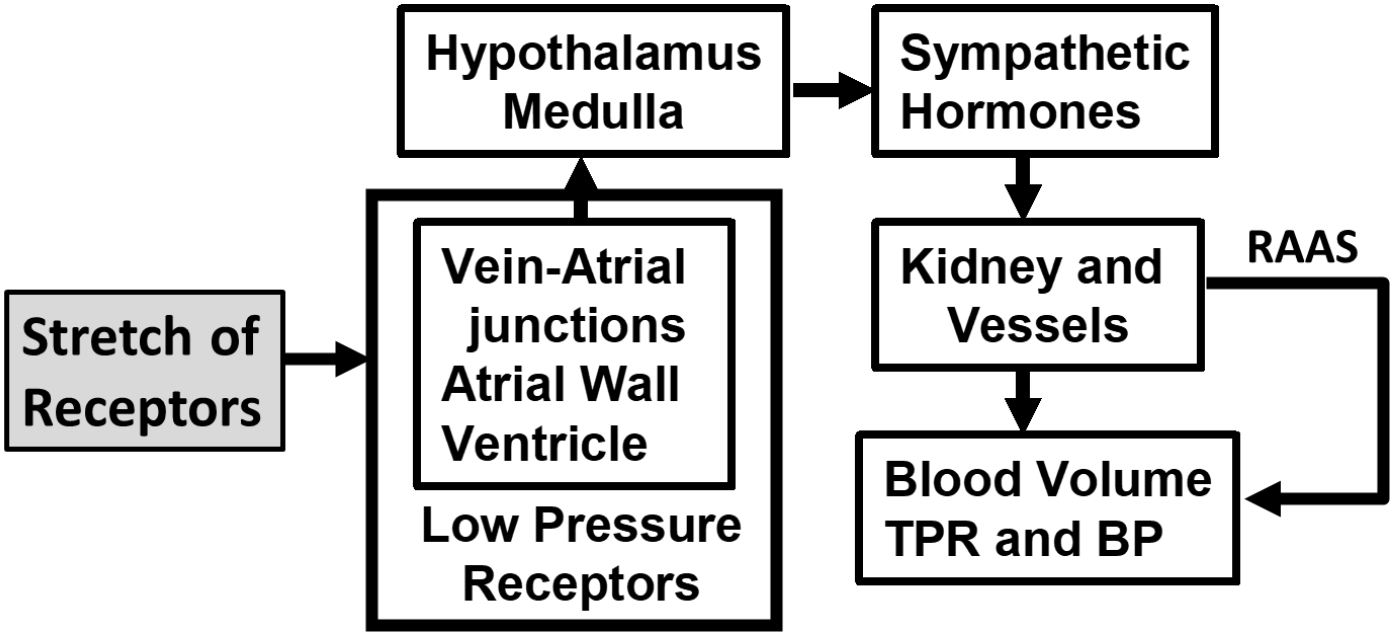
Central Chemoreceptors: Cushing Reflex



Low Pressure Baroreceptors

Cardio-Pulmonary Low-Pressure Receptors

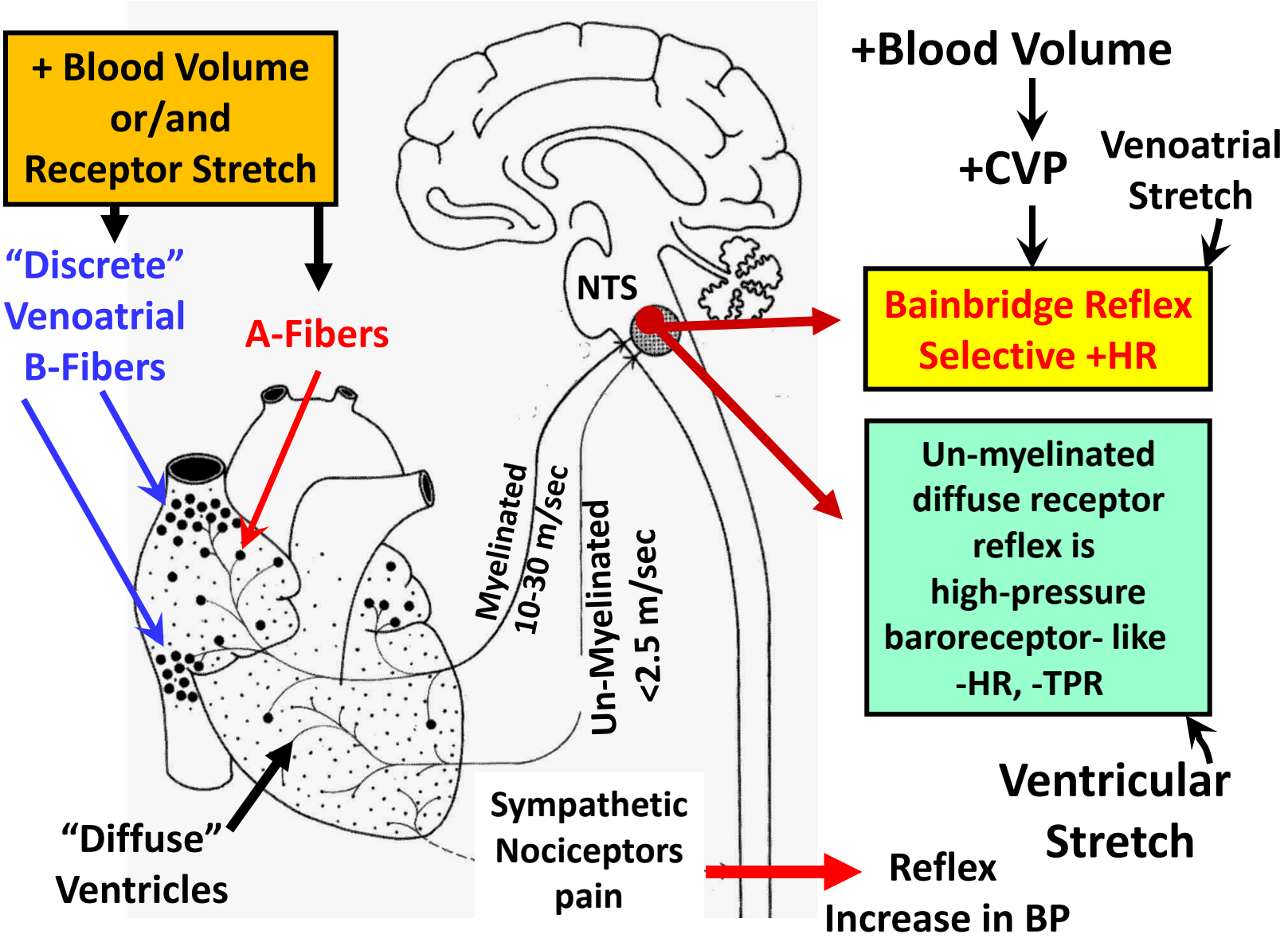
Low Pressure Receptors more Involved in "Longer-Term" BP Control in conjunction with Kidney Blood Volume Control



Cardiopulmonary (Low Pressure) Reflex

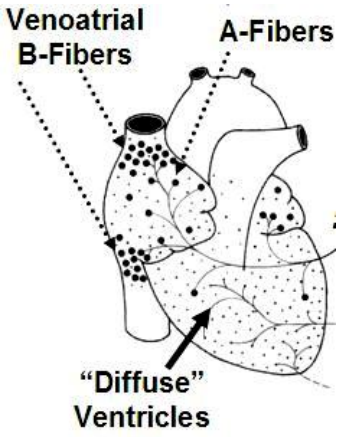
Large veins
Atria and R. Ventricle
} ± Volume → ∓ Volume

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)

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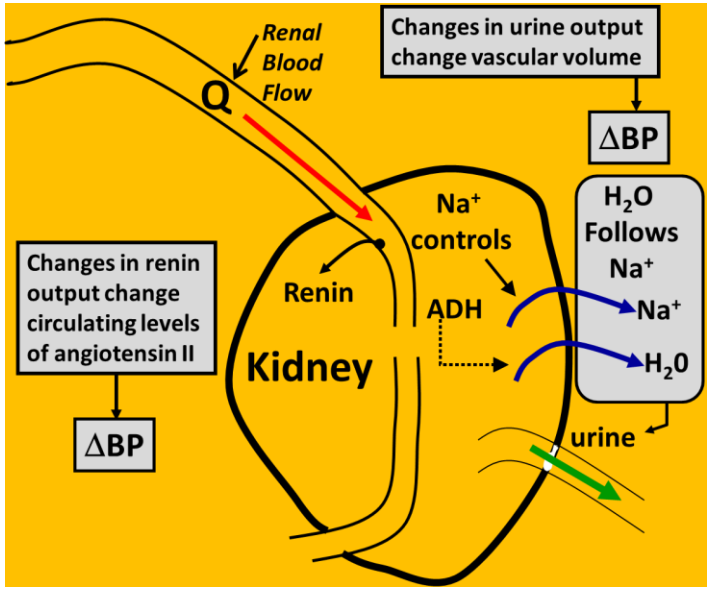
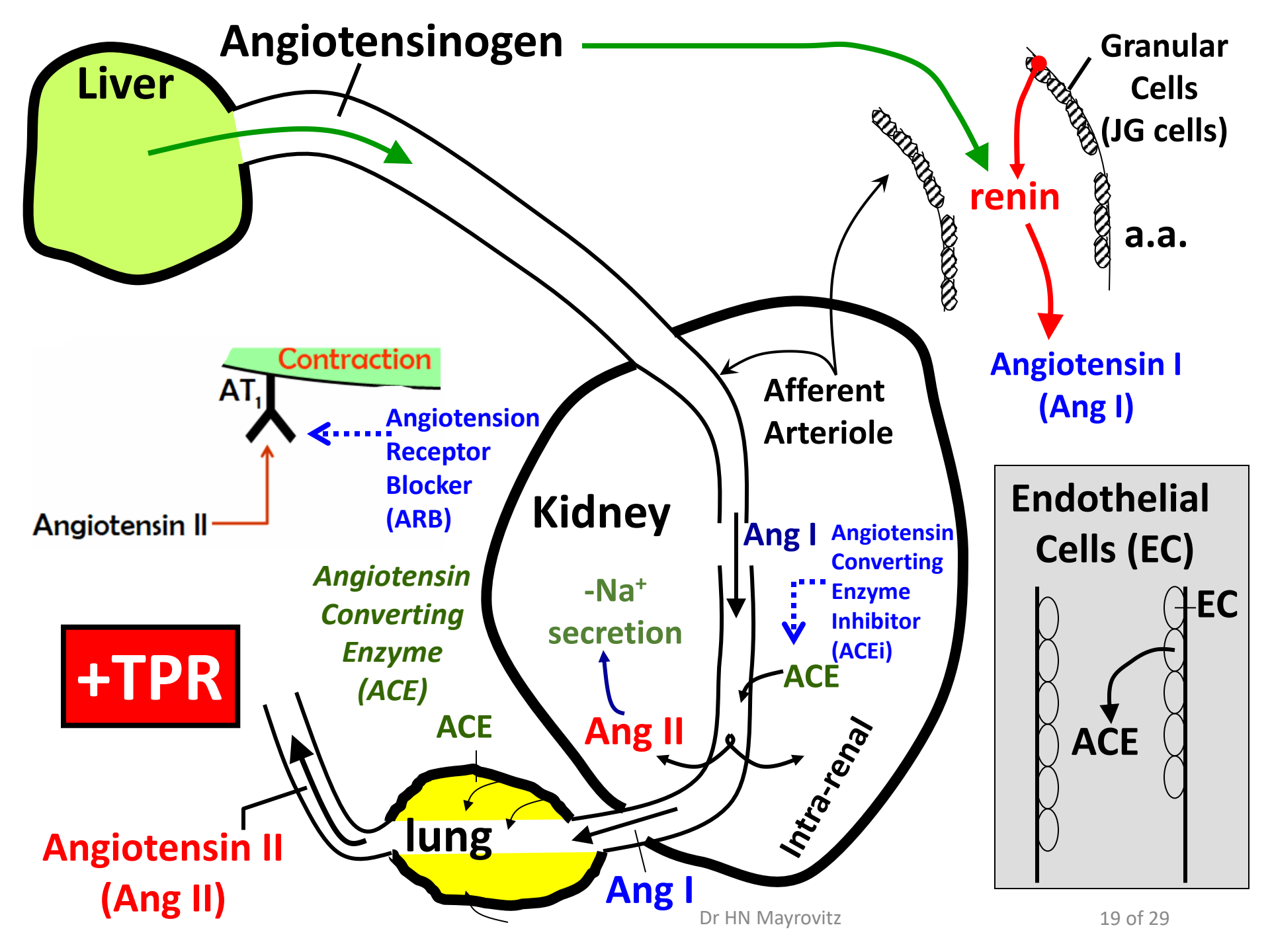
The major "Players"

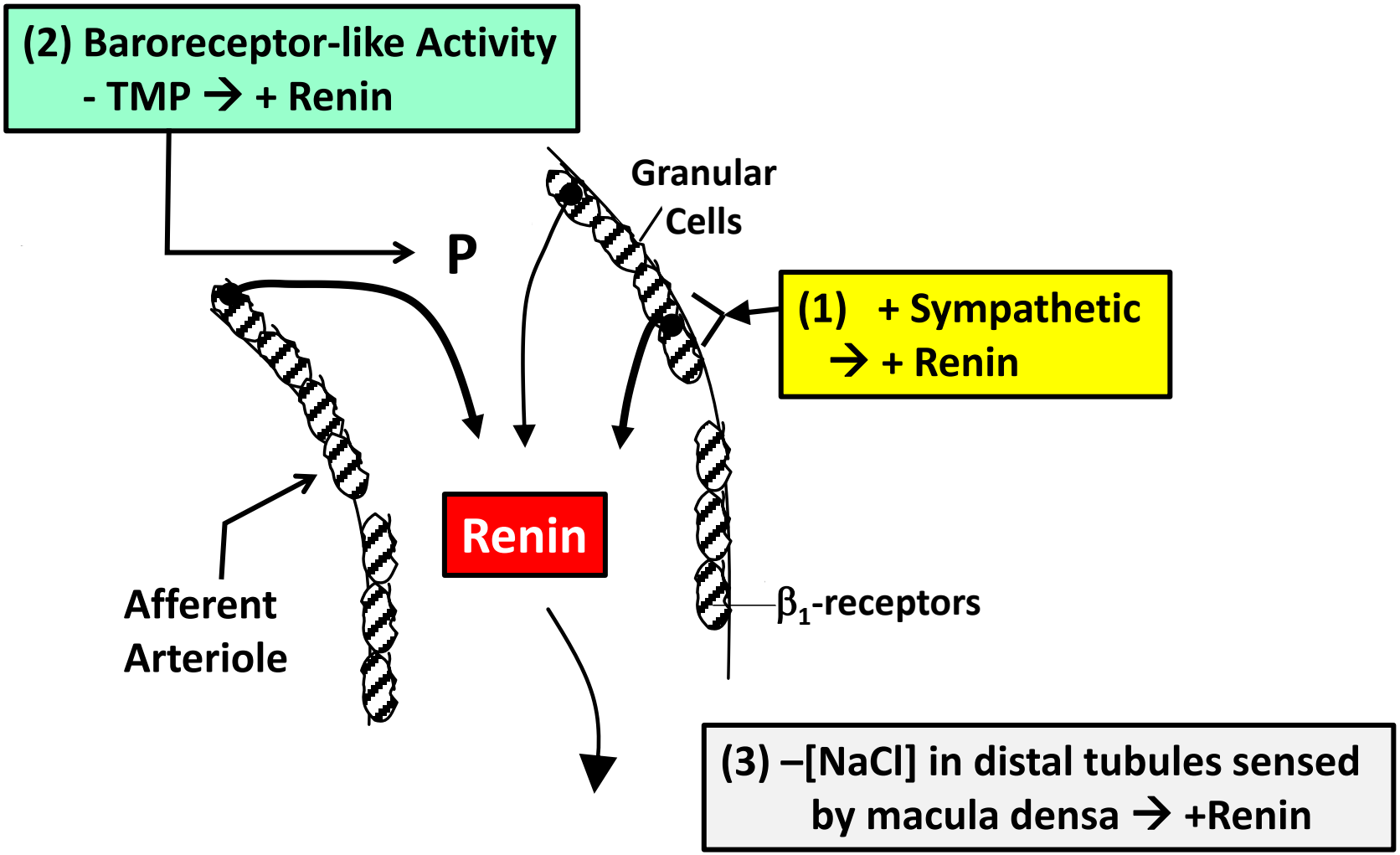
- Angiotensinogen → Protein made in and released from liver
- Renin → Proteolytic enzyme → released from kidney
- Angiotensin I → (ANG I) → Kidney → made by renin acting on angiotensinogen
- Angiotensin Converting Enzyme (ACE) → released in kidney and lung
- Angiotensin II → (ANG II) → ACE acting on 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

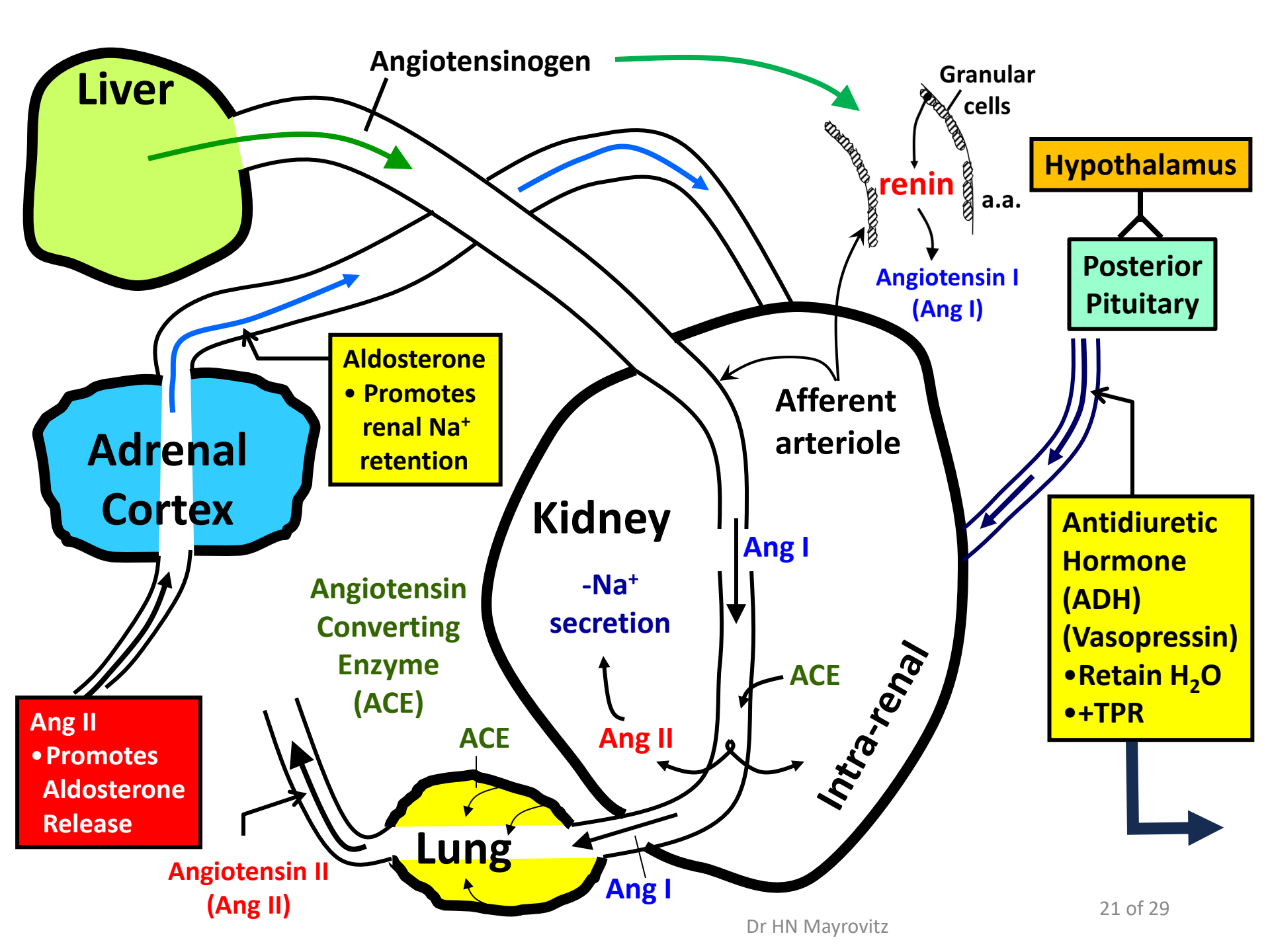


Renin-Angiotensin

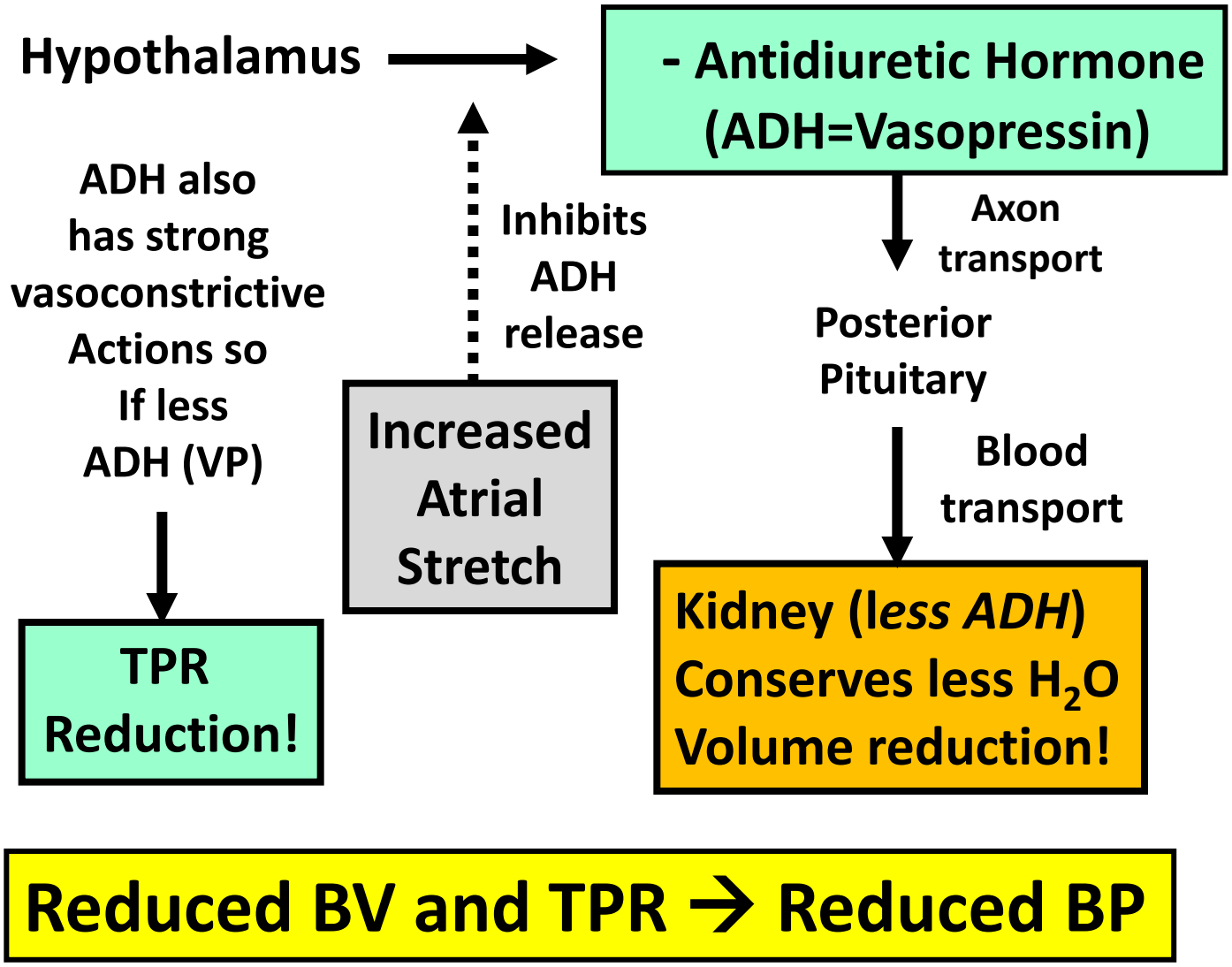

Angiotensinogen
Liver
Granular Cells (JG cells)
renin
a.a.
Angiotensin I (Ang I)
Contraction
AT1
Angiotension Receptor Blocker (ARB)
Angiotensin II
Afferent Arteriole
Kidney
Ang I
Angiotensin Converting Enzyme Inhibitor (ACEi)
ACE
-Na+ secretion
Angiotensin Converting Enzyme (ACE)
+TPR
ACE
Ang II
Intra-renal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

Three Main Factors Affecting Renin Regulation




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

Antidiuretic Hormone (Vasopressin)



Reduced BV 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 vent 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.

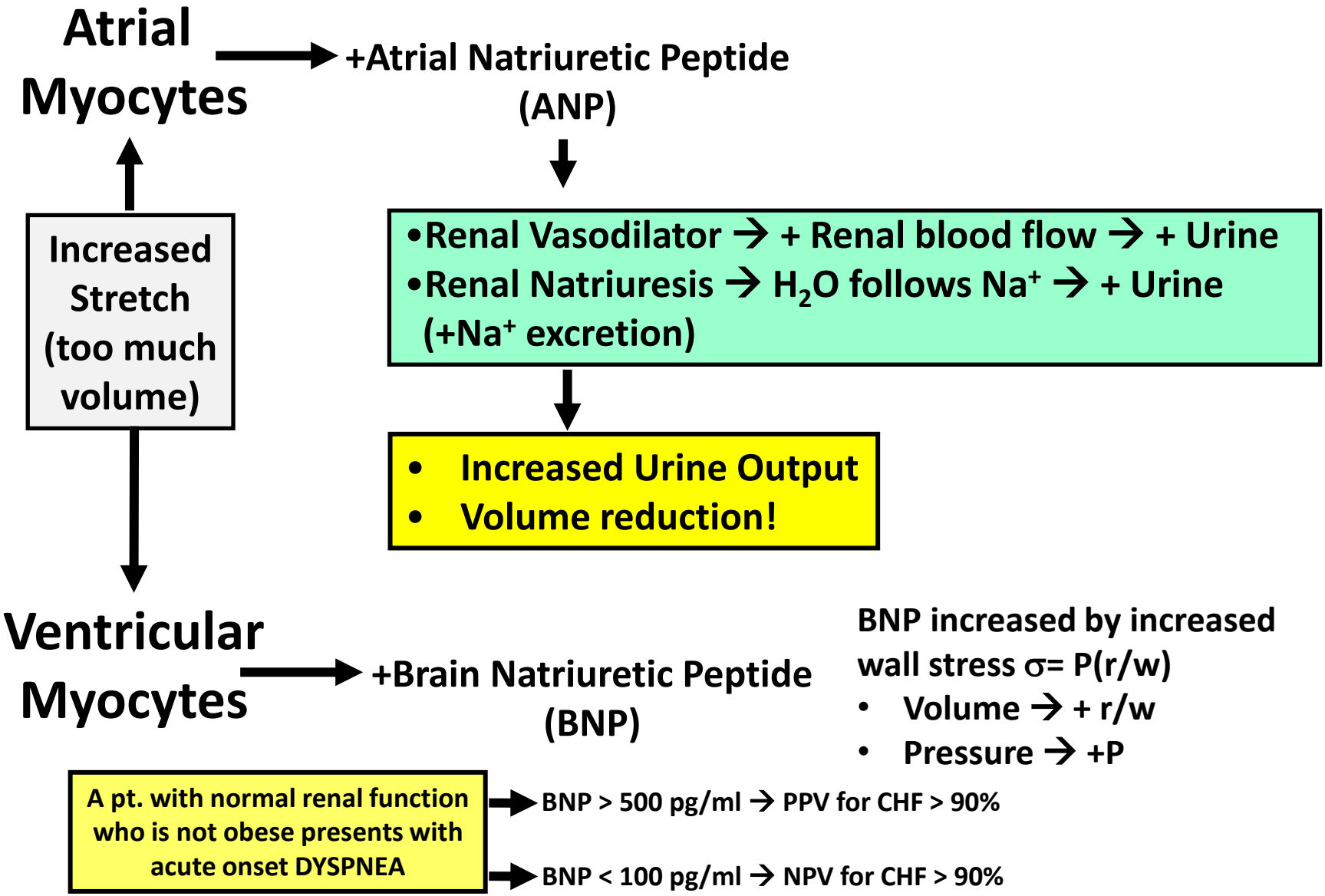
(3) Both receptor types use cGMP as a 2nd messenger.

(4) Each peptide cleared by

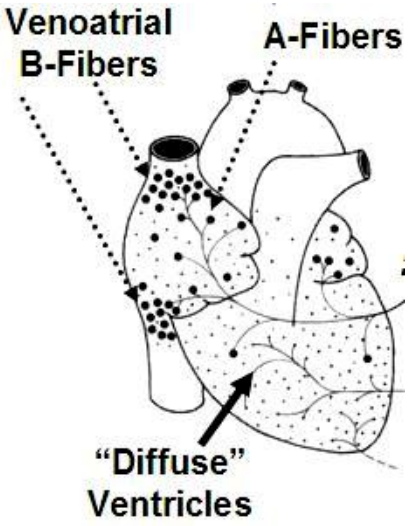
- (a) enzymatic action of neutral endopeptidase (NEP) or by
- (b) binding to a 3rd receptor (NPR-C) that internally degrades peptides.

(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"

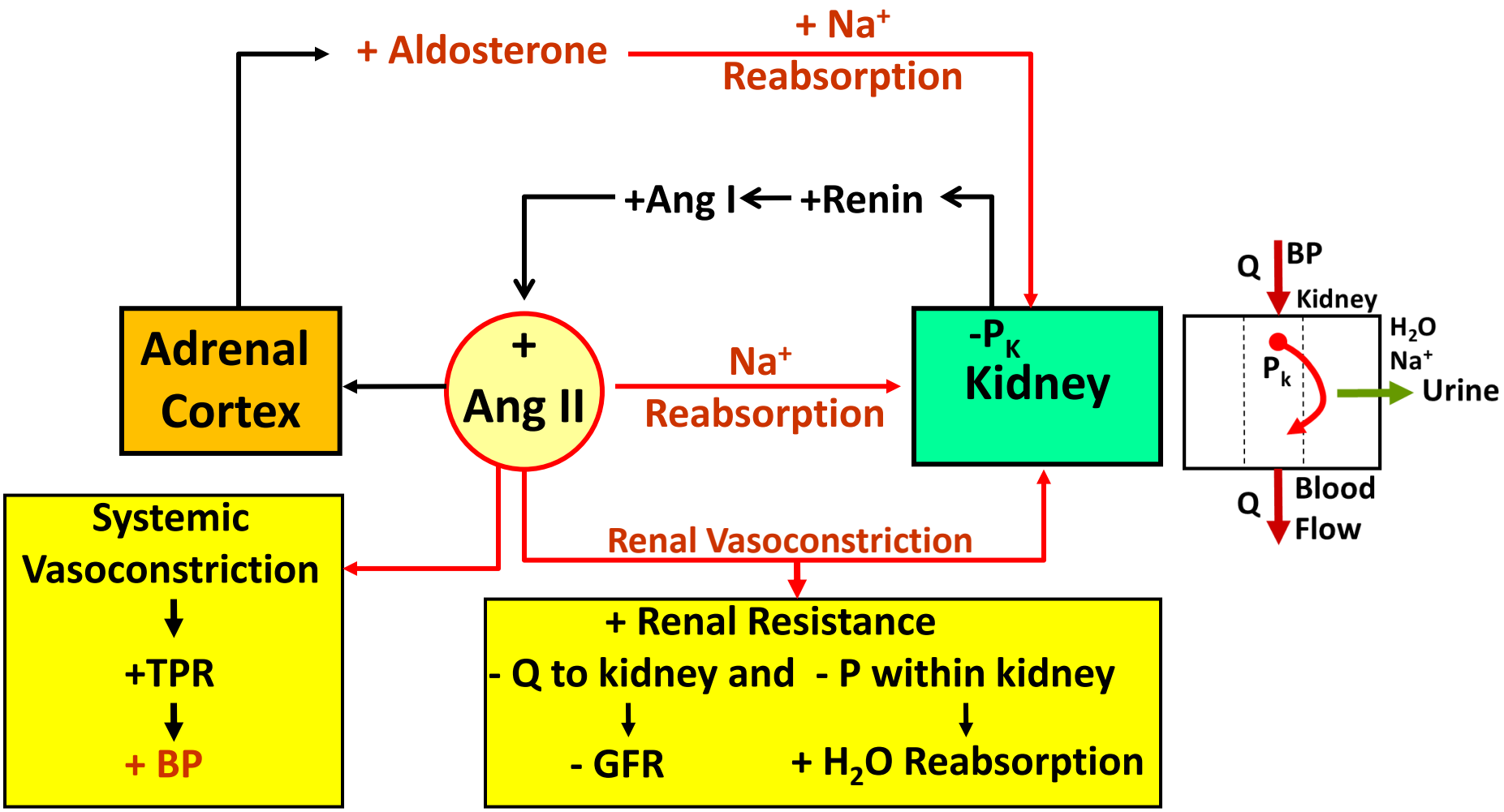
- Sympathetic Only to kidney

Renal Vasodilation and Reflex tachycardia

+ Renal Blood Flow
+ Urine Output (Diuresis)

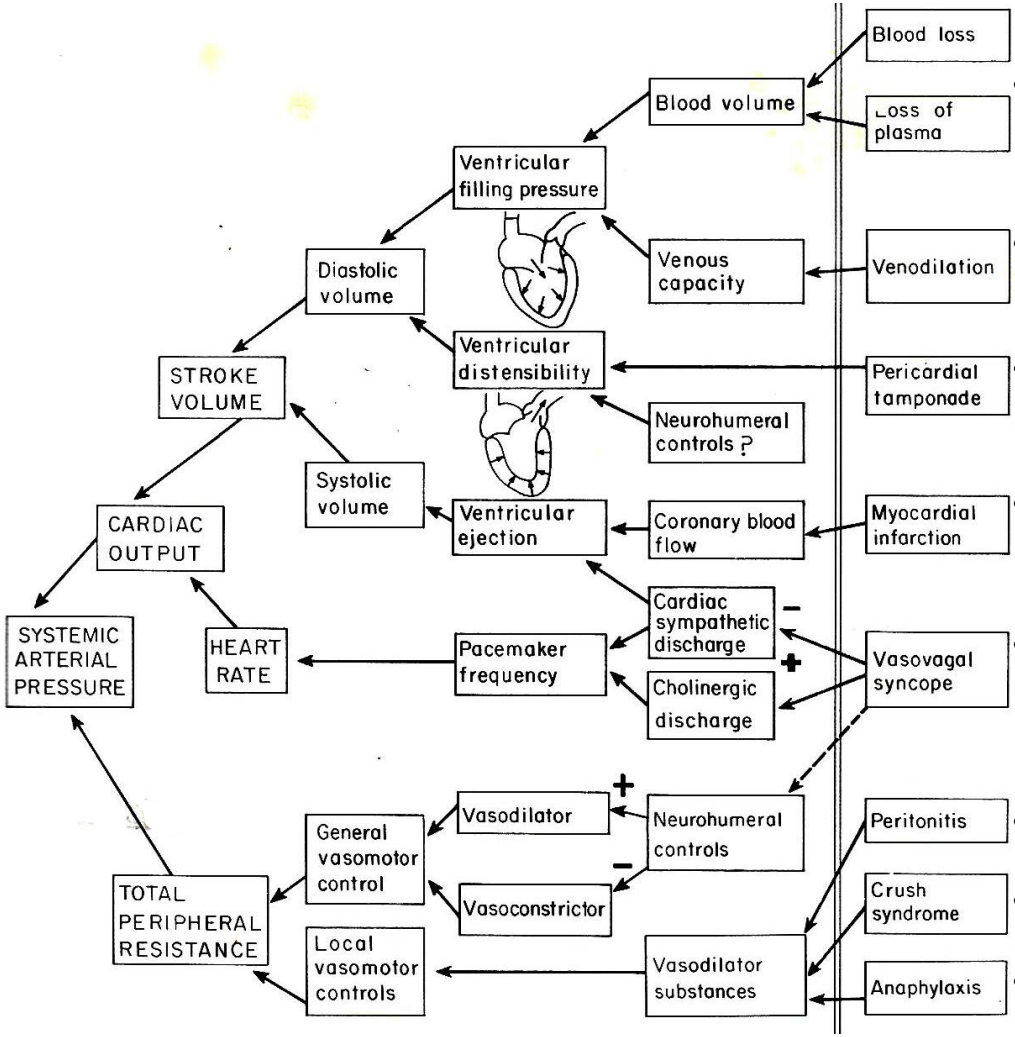
Volume Reduction!

Renal Responses to Decreased Blood Pressure



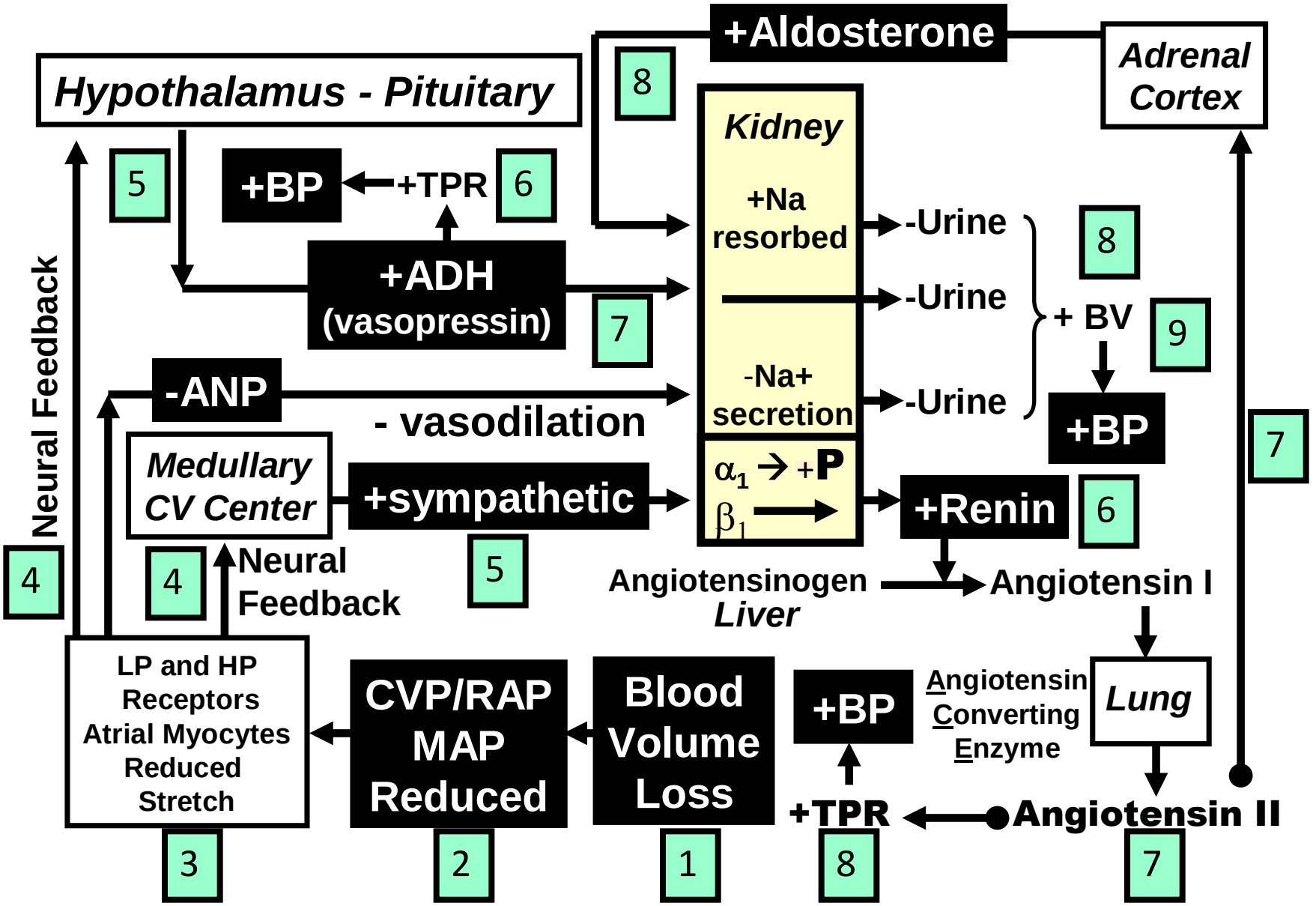
Arterial Hypotension Events and Possible Shock

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

Dr HN Mayrovitz