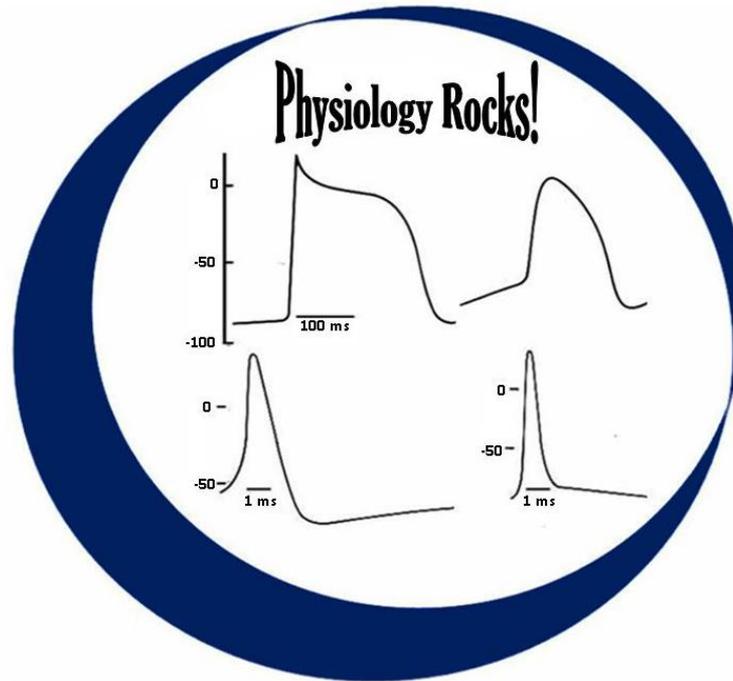


Lecture 13

Peripheral Vascular Control



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Topics

“Part 1”

- **Receptor Review for Vasodilation and Vasoconstriction**
- **Important Role of Calcium**
- **Important Role of Potassium**
- **Endothelial-Vascular Smooth Muscle Interaction**
- **Endothelial Vasoactive Substances → NO and Endothelin**

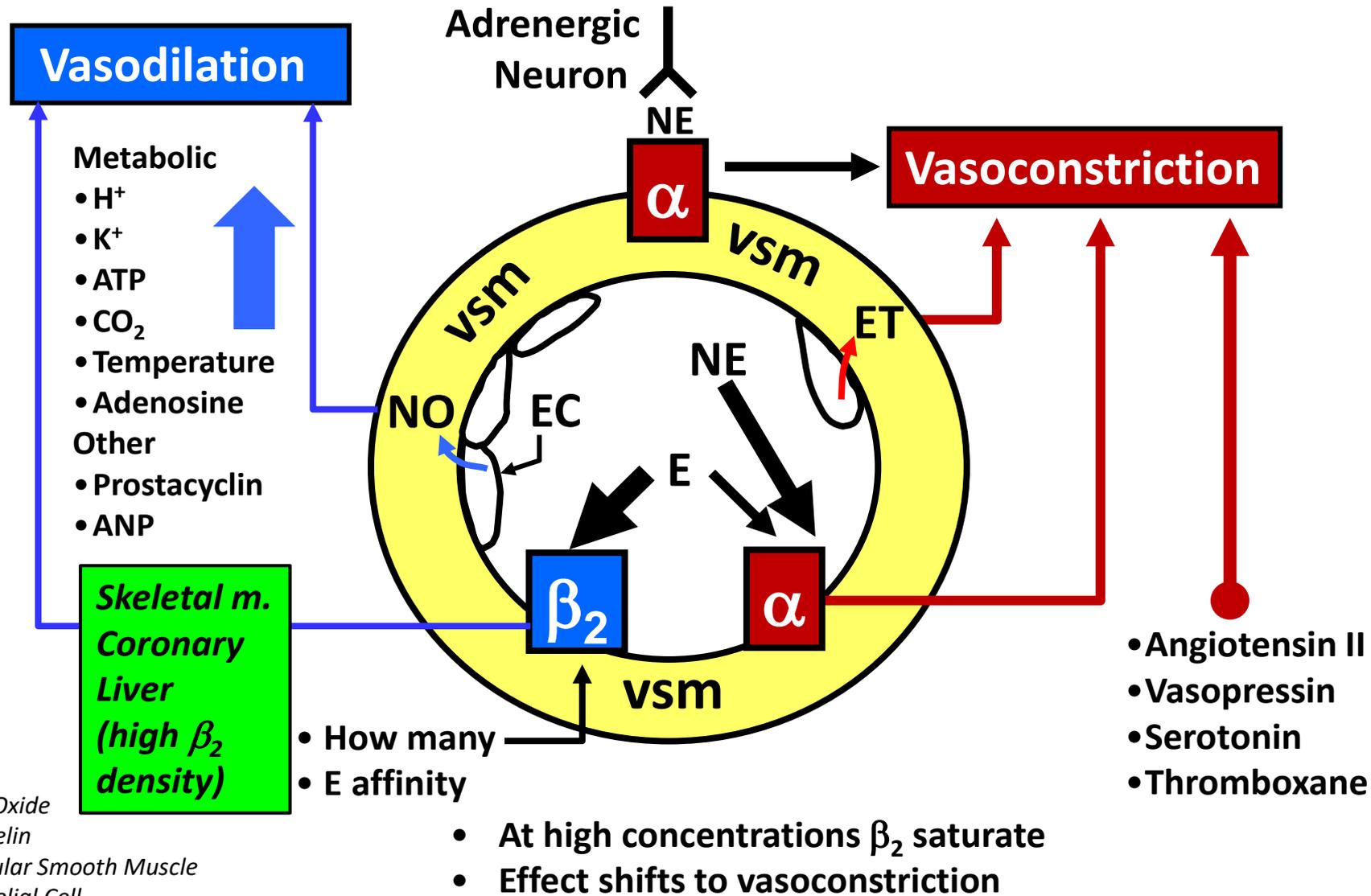
“Part 2” → Local Controls

- **Myogenic Response**
- **Autoregulation**
- **Metabolic Control**
- **Reactive Hyperemia**

“Part 3” → Selected circulations

- **Muscle**
- **Skin**
- **Cerebral**
- **Splanchnic**

Vascular Control: Neural-Humoral-Local



NO=Nitric Oxide
 ET=Endothelin
 VSM=Vascular Smooth Muscle
 EC=Endothelial Cell
 E=Epinephrine
 NE=Norepinephrine
 ANP=Atrial Natriuretic Protein

VSM Ion Mechanisms: Overview

VSM tone and contractile state depends on Ion movements through

- Voltage Operated Channels (VOC)
- Receptor Operated Channels (ROC)

VOC → Voltage- Gated (Electromechanical Coupling)

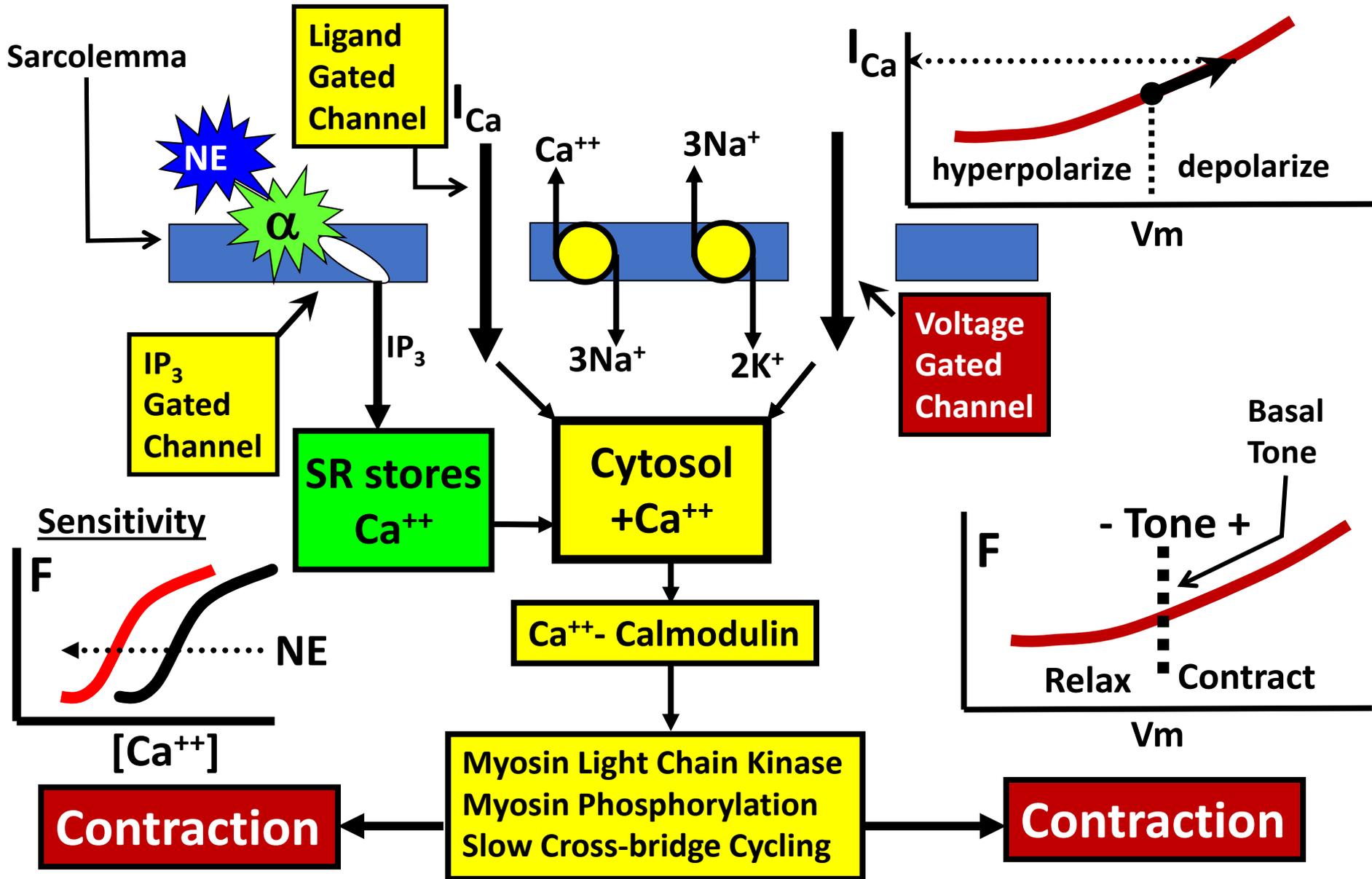
- Hyperpolarization → Vasodilation
- Depolarization → Vasoconstriction

ROC → Ligand-Gated (Pharmaco-mechanical Coupling)

Ca^{++} is a major player in VSM Contraction with increased contraction dependent on increased availability and less contraction (dilatation) the opposite.

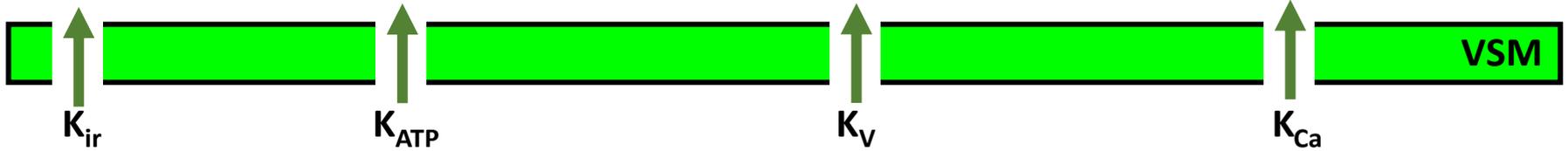
- Ca^{++} entry via ROC's and VOC's
- Ca^{++} release from SR stores
- Ca^{++} removal via SR & membrane "pumps"
- Ca^{++} sensitivity of VSM

Vascular Smooth Muscle: Dominant Role of Ca^{++}



*IP₃=inositol triphosphate
SR=sarcoplasmic reticulum*

Potassium Ion Channels in Vascular Smooth Muscle

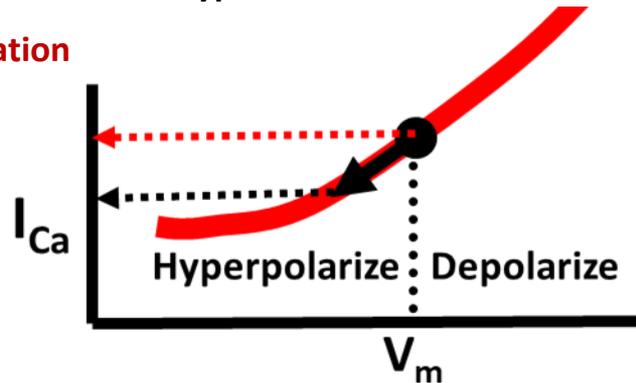


- Inward Rectifying (K_{ir})
- **Open state probability (OSP) increases if $+[K]_o$**
- Causes hyperpolarization
- Less Ca^{++} entry
- **Vasodilation**
- $[K]_o$ is increased with metabolic activity
- Basis of metabolic hyperemia

- ATP-Dependent (K_{ATP})
- OSP increases if intracellular ATP decreases (++) Ischemia
- **OSP increases if +ADP, + $[H^+]$ Adenosine or Mild Hypoxia**
- **Vasodilation**

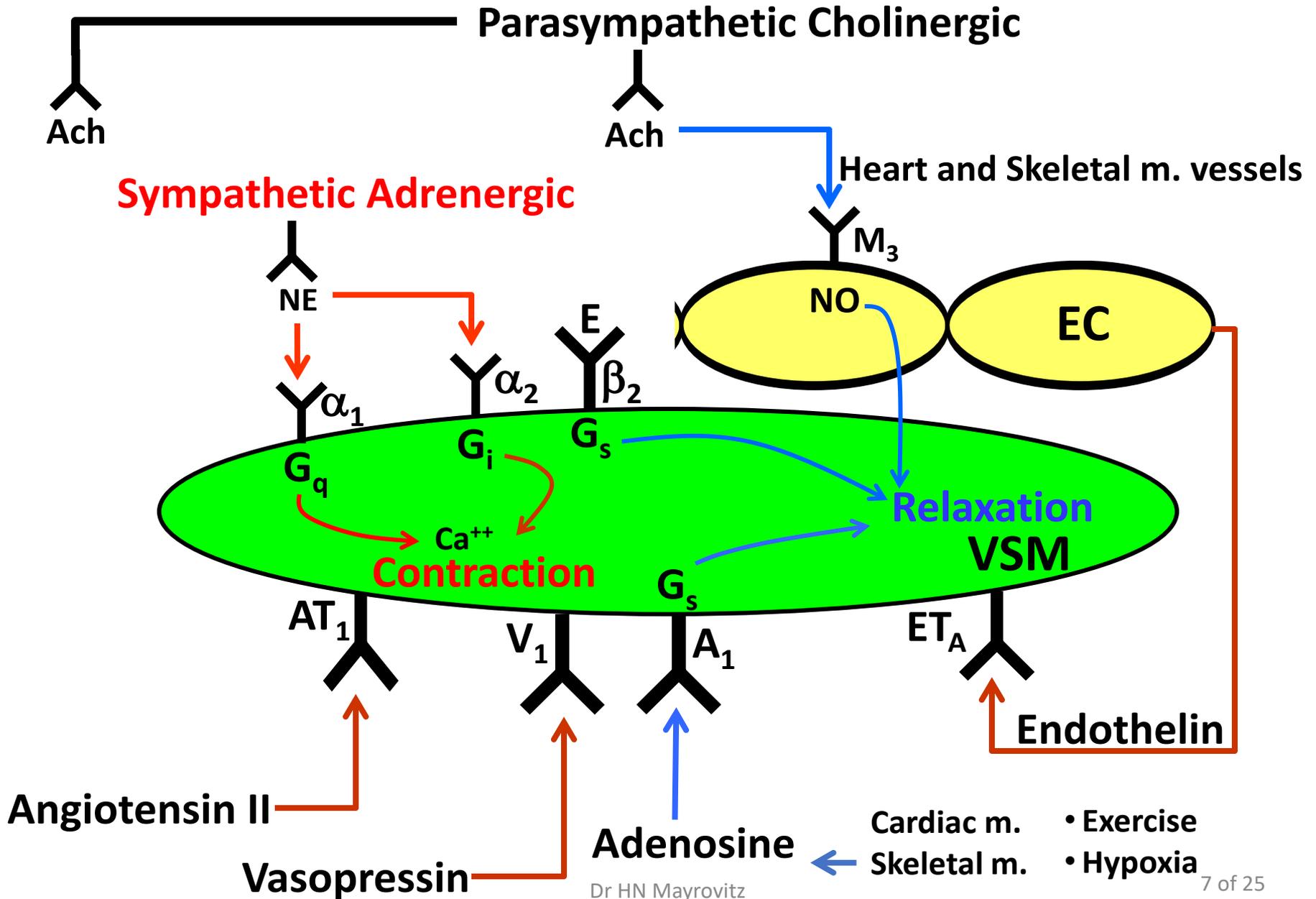
- Voltage-gated (K_V)
- Mainly in arterioles
- **+OSP with depolarization**
- **Vasodilation**

- Calcium-activated (K_{Ca})
- **Activated by increased intracellular Ca^{++} and membrane depolarization**
- **Vasodilation**



- Increased I_k (K^+ outward flux) of any of these channels causes hyperpolarization
- Hyperpolarization reduces open-state probability (OSP) of voltage-gated Ca^{++} channels
- As a result, inward I_{Ca} decreases, causing $[Ca^{++}]$ to decrease
- Result is vasodilation, directly or counteracting depolarization-induced vasoconstriction

Summary of Some Major CV-Related Receptors



Local Control Processes

- **Myogenic Response**
- **Autoregulation**
- **Metabolic → Active or Functional Hyperemia**
- **Reactive Hyperemia**

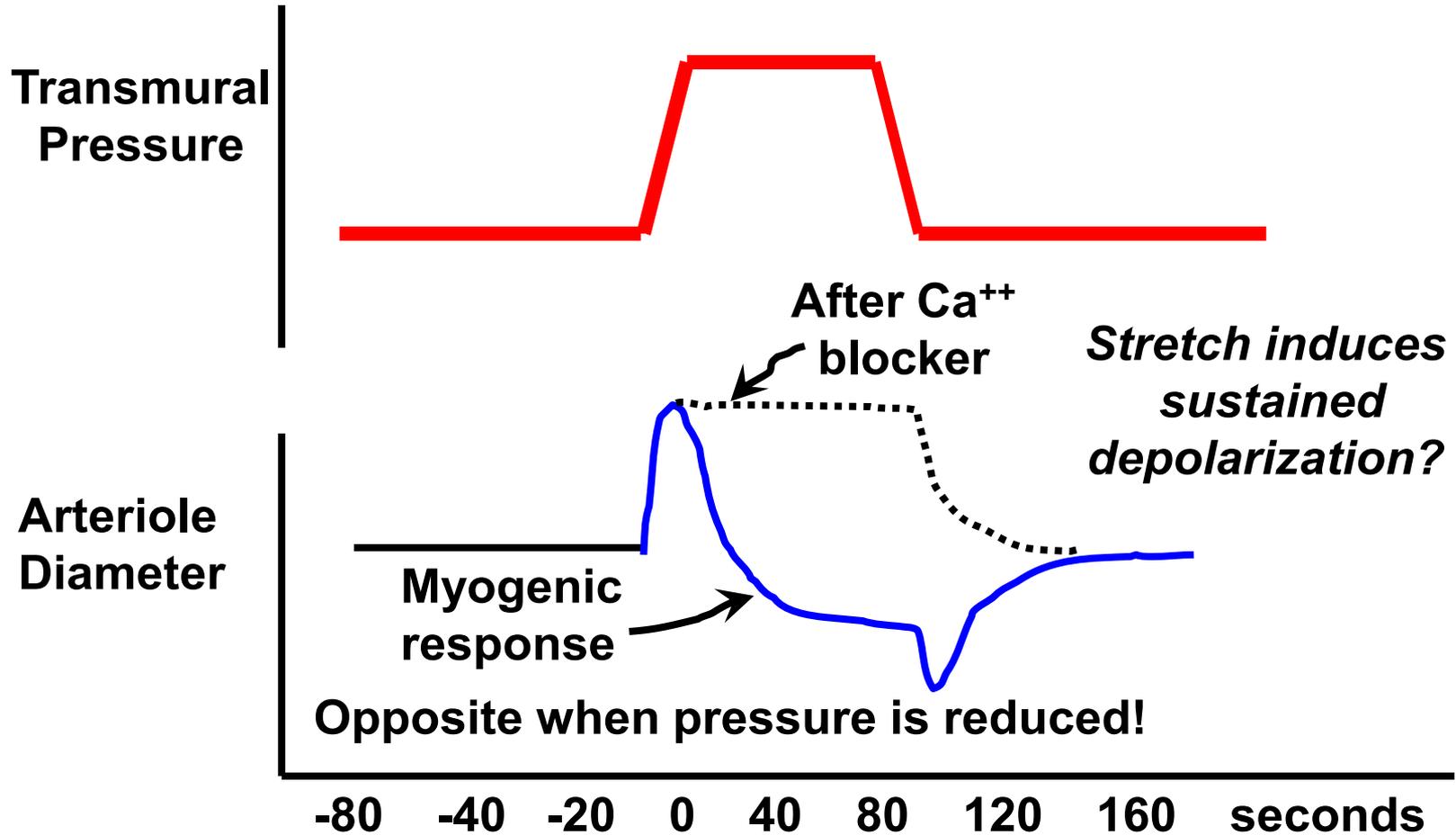
Myogenic Response: **Defined**

- **Active vascular smooth muscle change in response to a change in transmural pressure (TMP)**
- **Direction of active change opposes stretch caused by the initiating pressure change**

If TMP increases  Vasoconstriction

If TMP decreases  Vasodilation

Myogenic Response: Illustrated

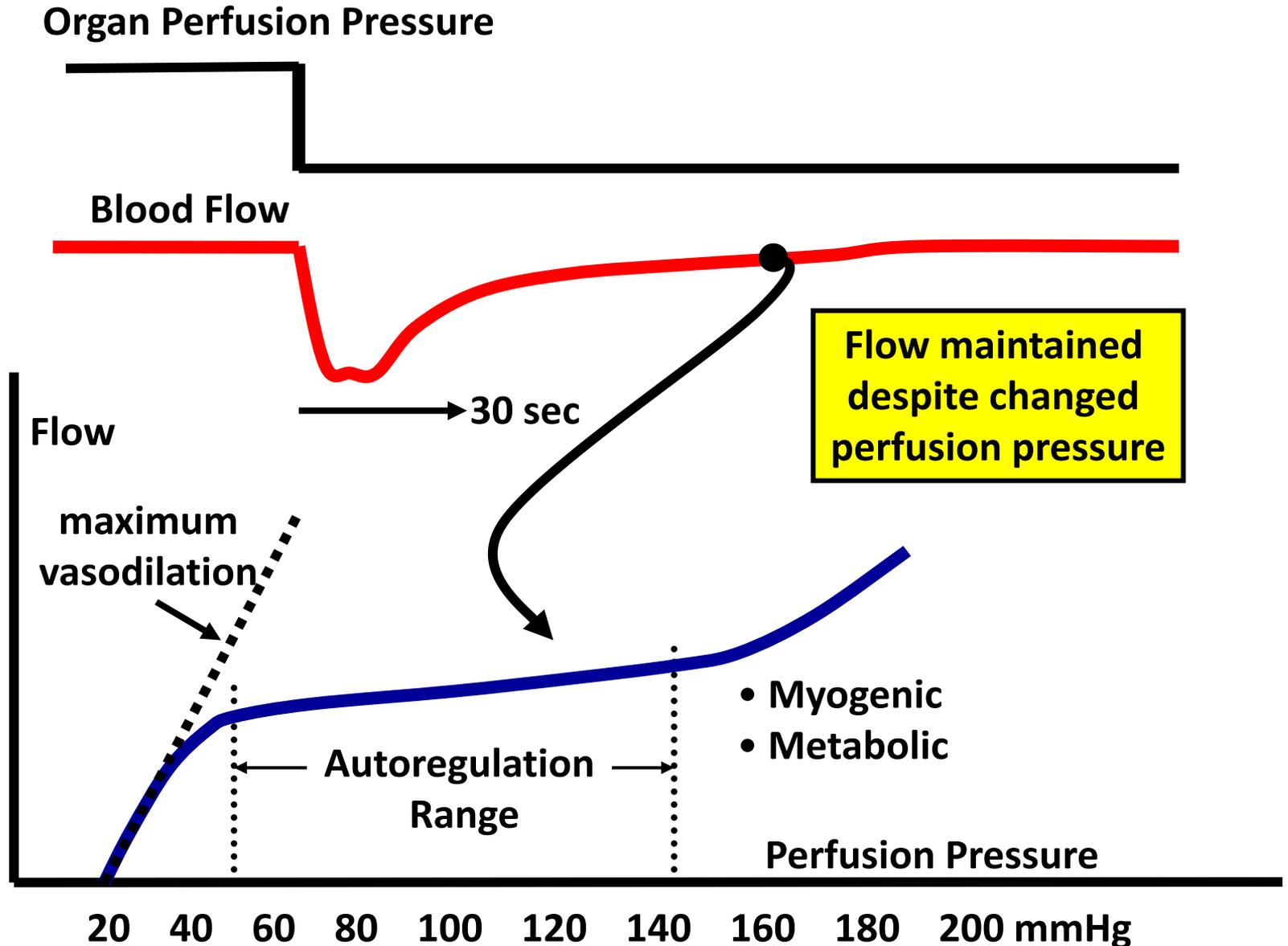


Autoregulation of Blood Flow: **Defined**

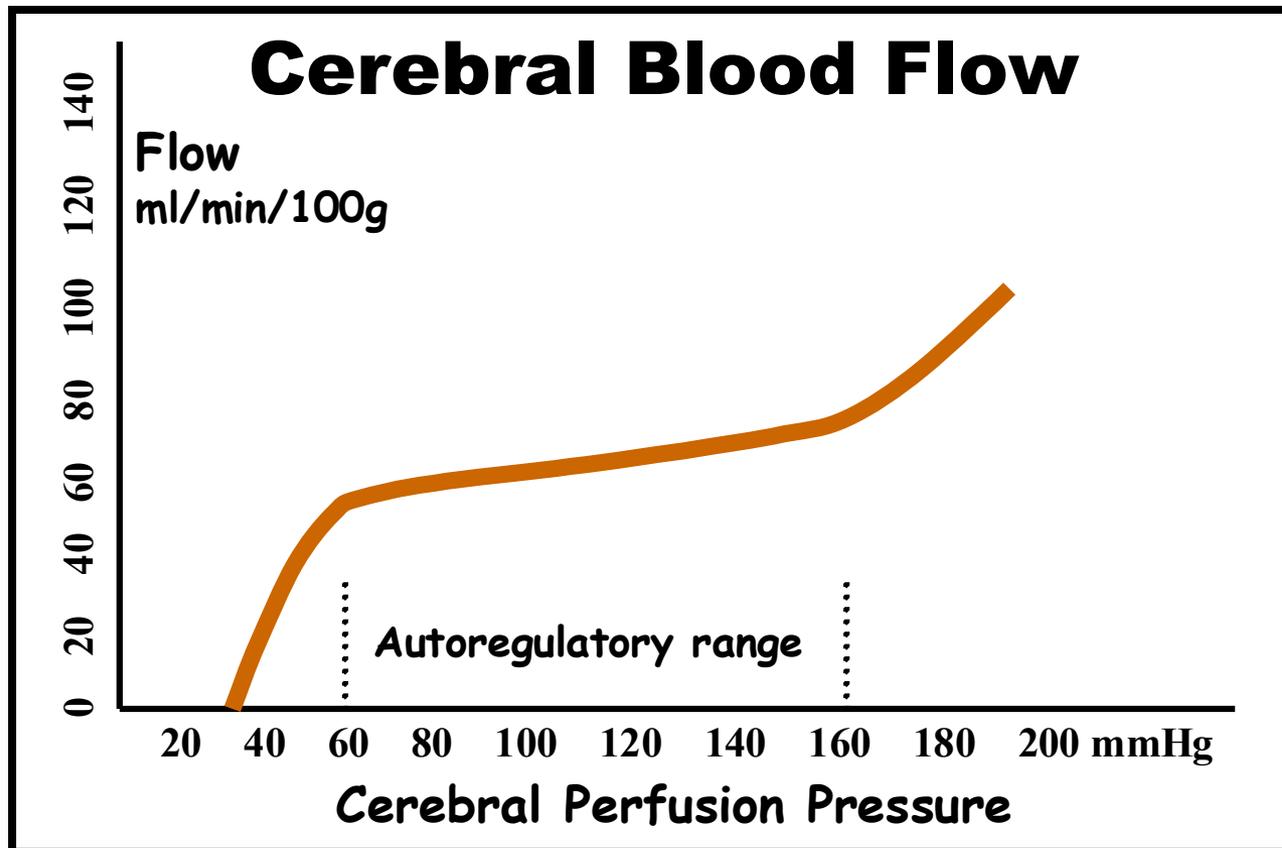
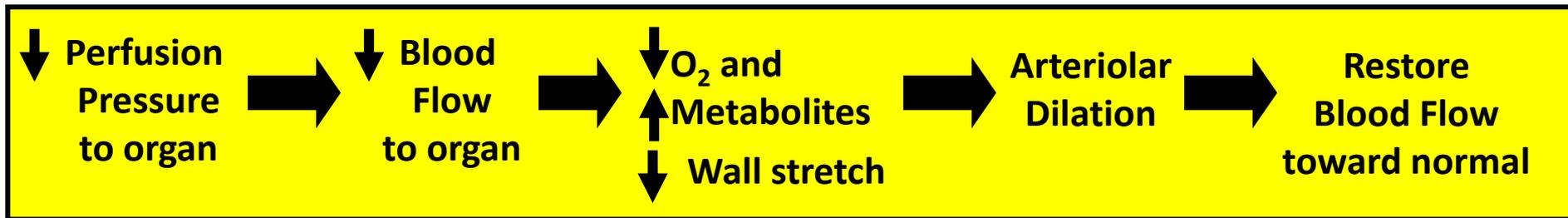
- **Tendency to maintain blood flow despite changes in perfusion pressure**
- **Change is produced by increases or decreases in vascular resistance of the organ**

- **If perfusion pressure decreases so does resistance**
- **If perfusion pressure increases so does resistance**

Autoregulation of Blood Flow: Illustrated



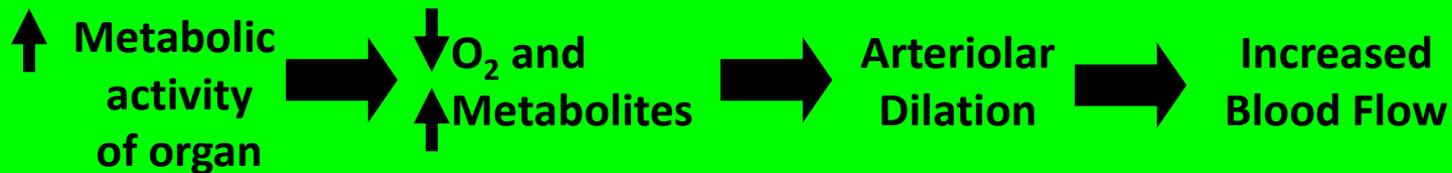
Autoregulation of Blood Flow: Reviewed



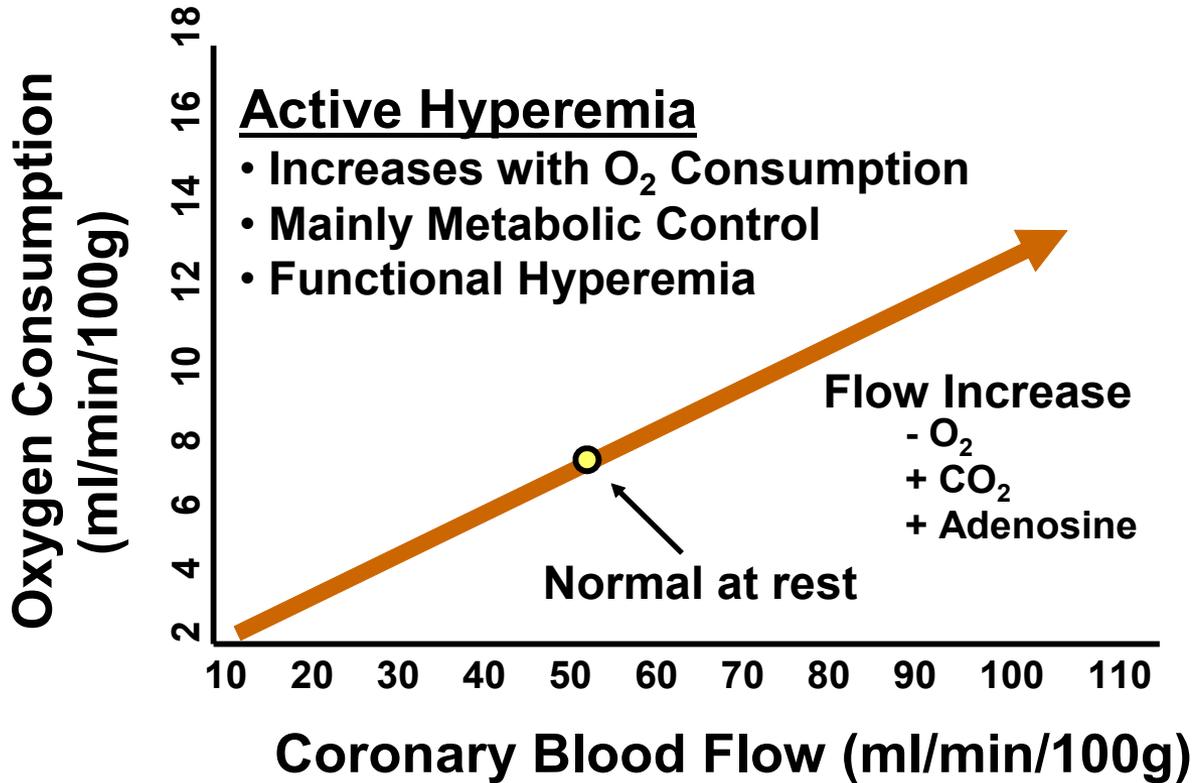
Metabolic Control: **Defined**

- Local blood flow change to match metabolic demand
- Referred to as **Active or Functional Hyperemia**

- Increased metabolic demand → **increased flow**
- Decreased metabolic demand → **decreased flow**

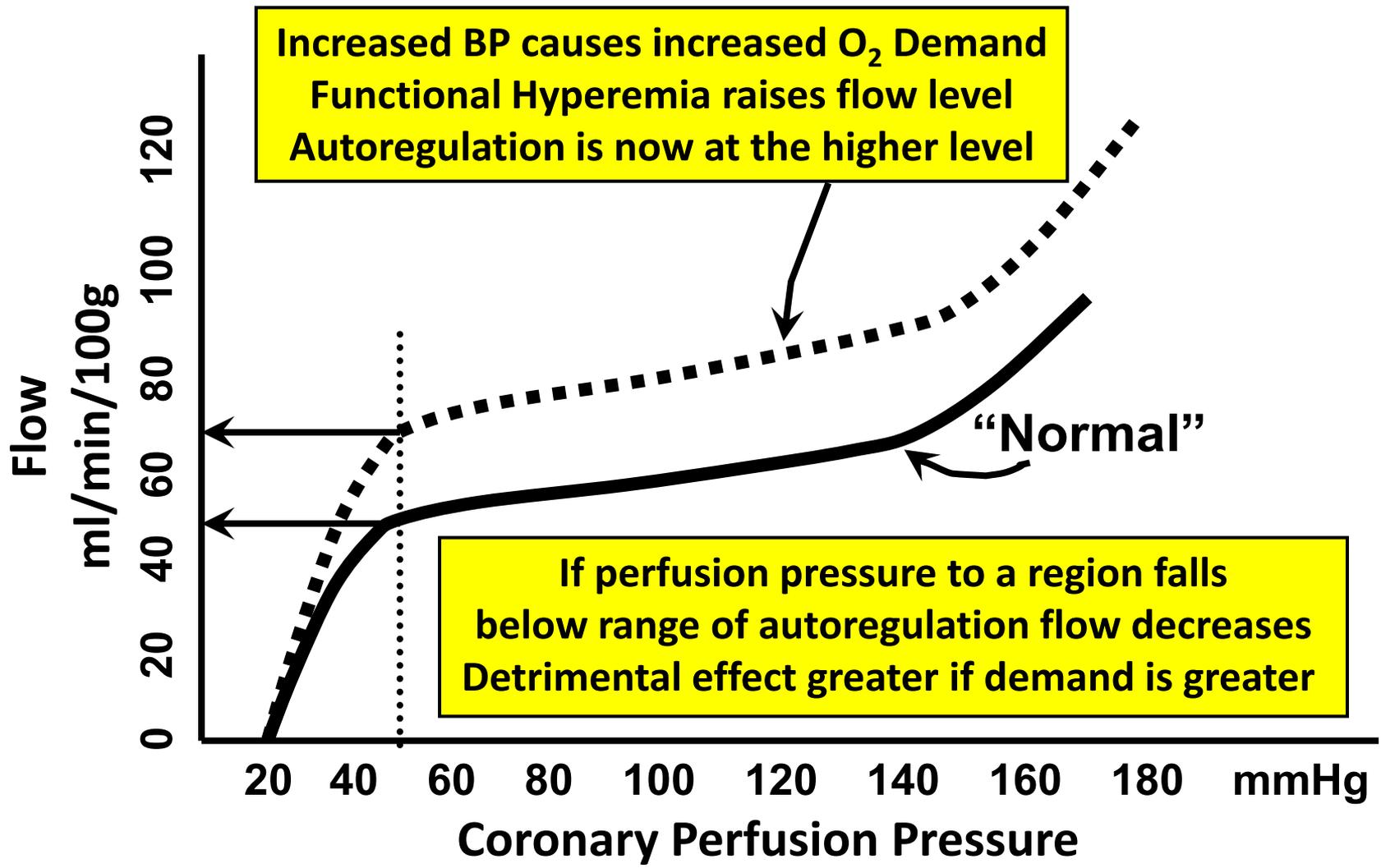


Metabolic Control: Illustrated



- Local Hypoxia
- Adenosine
- Local Acidosis
- CO₂
- Histamine
- Bradykinin
- Prostaglandins

Functional and Autoregulation Combined

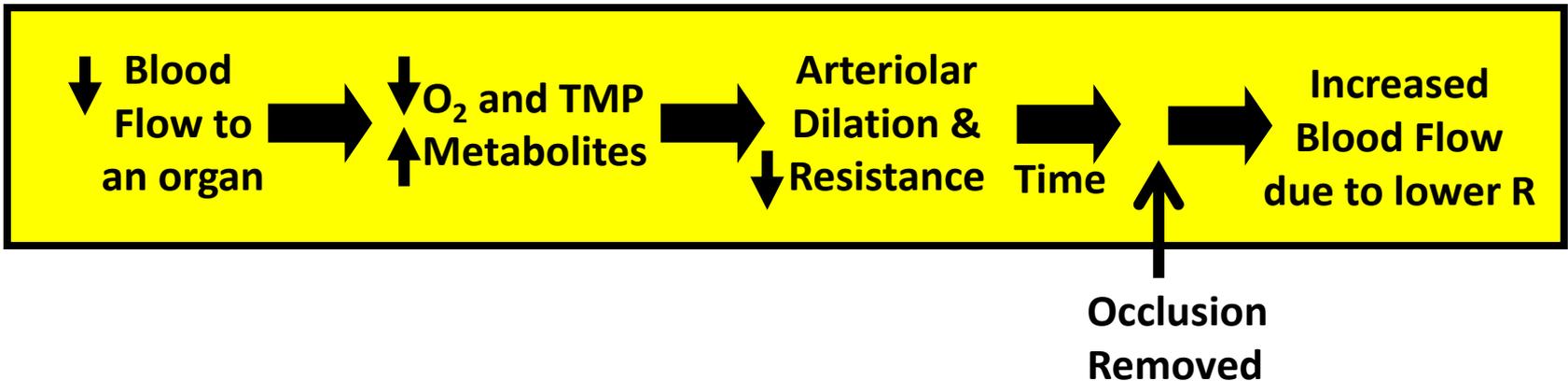


Reactive Hyperemia: Defined

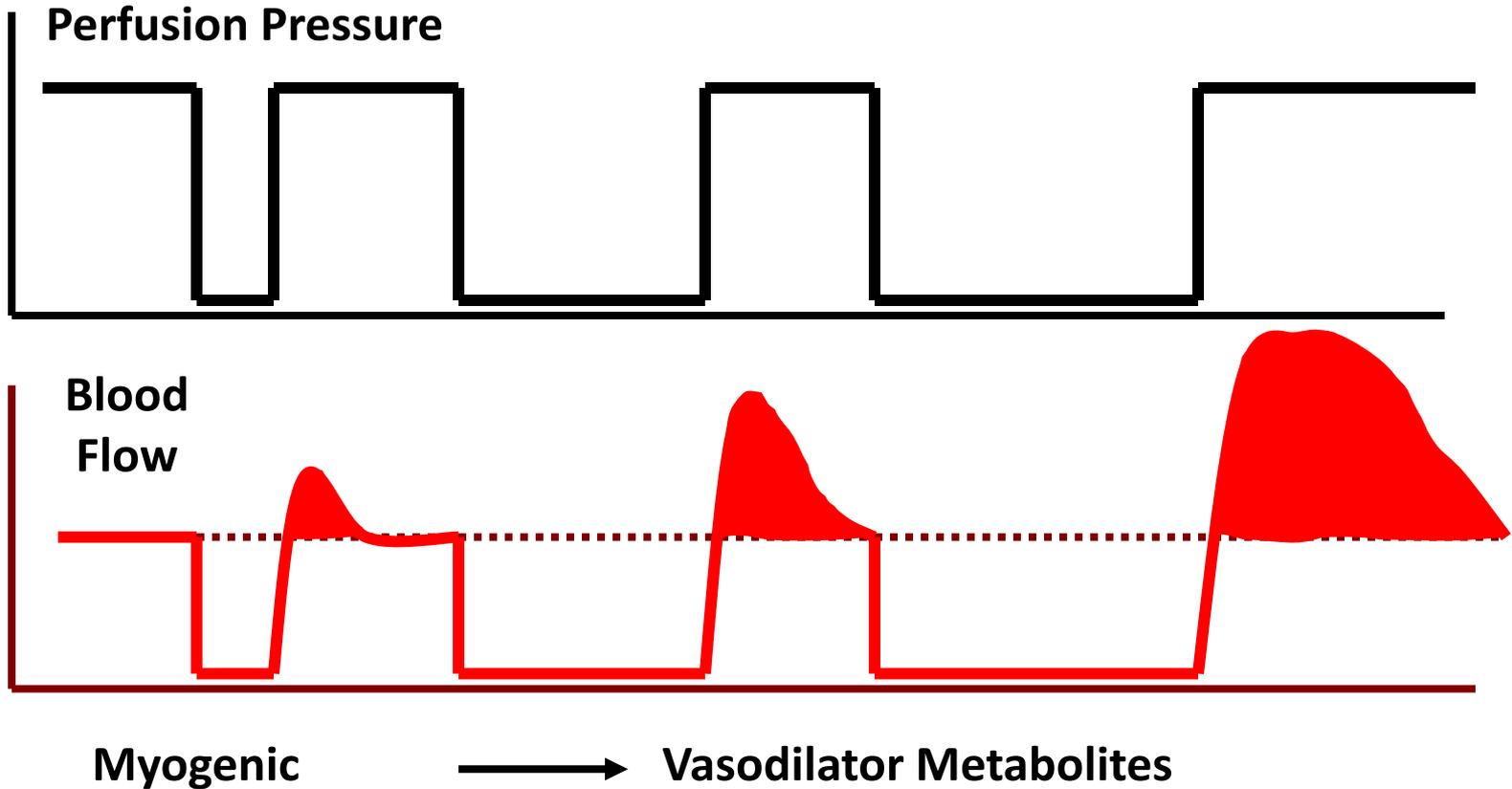
- Excess blood flow increase in response to prior deprivation
- Prior blood flow deficit usually transient then restored

Examples include

- Clearing of a clot or embolus
- Opening a blocked artery
- Release of a clamp in surgery



Reactive Hyperemia: Illustrated

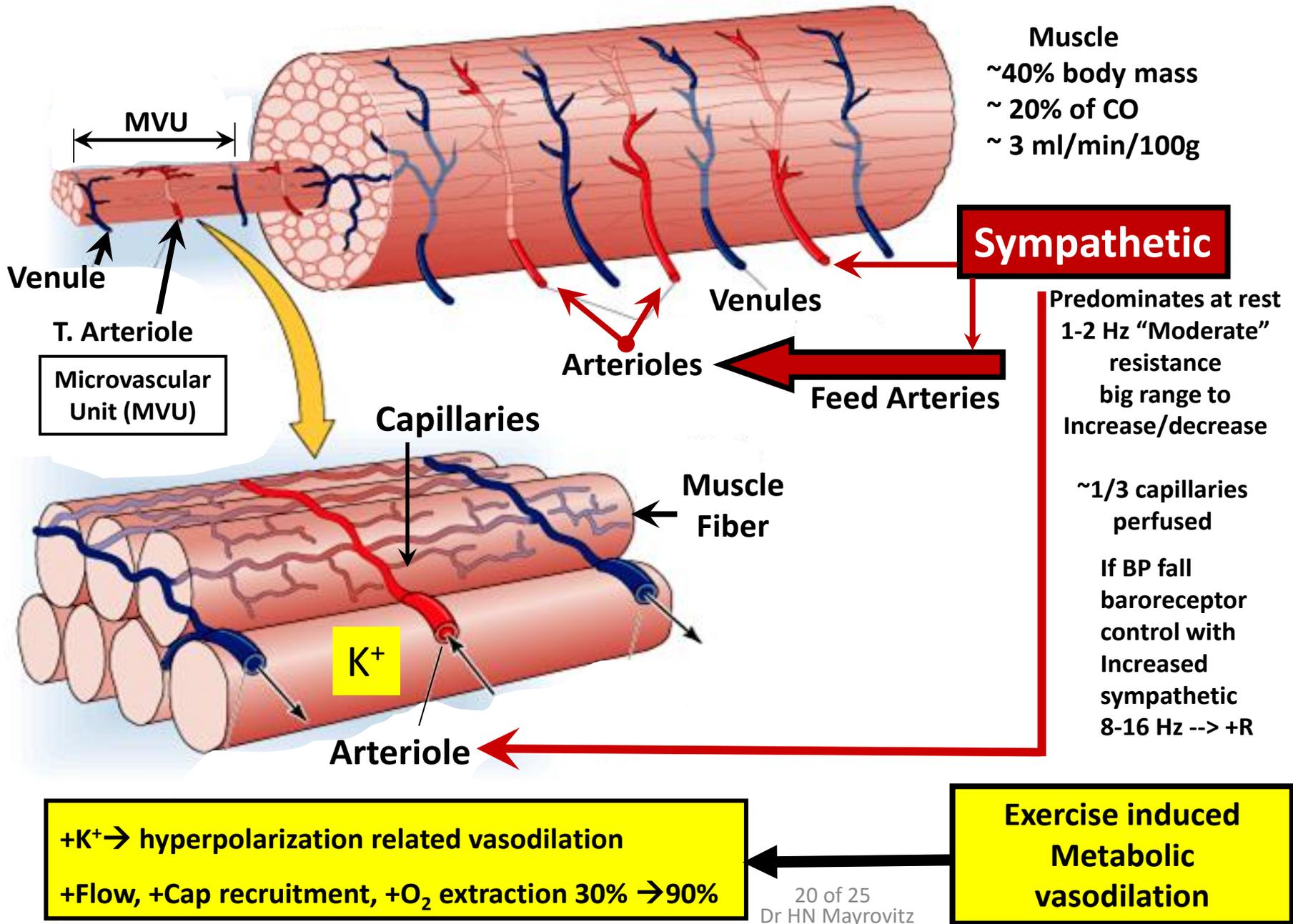


Skeletal Muscle Control

Neurogenic

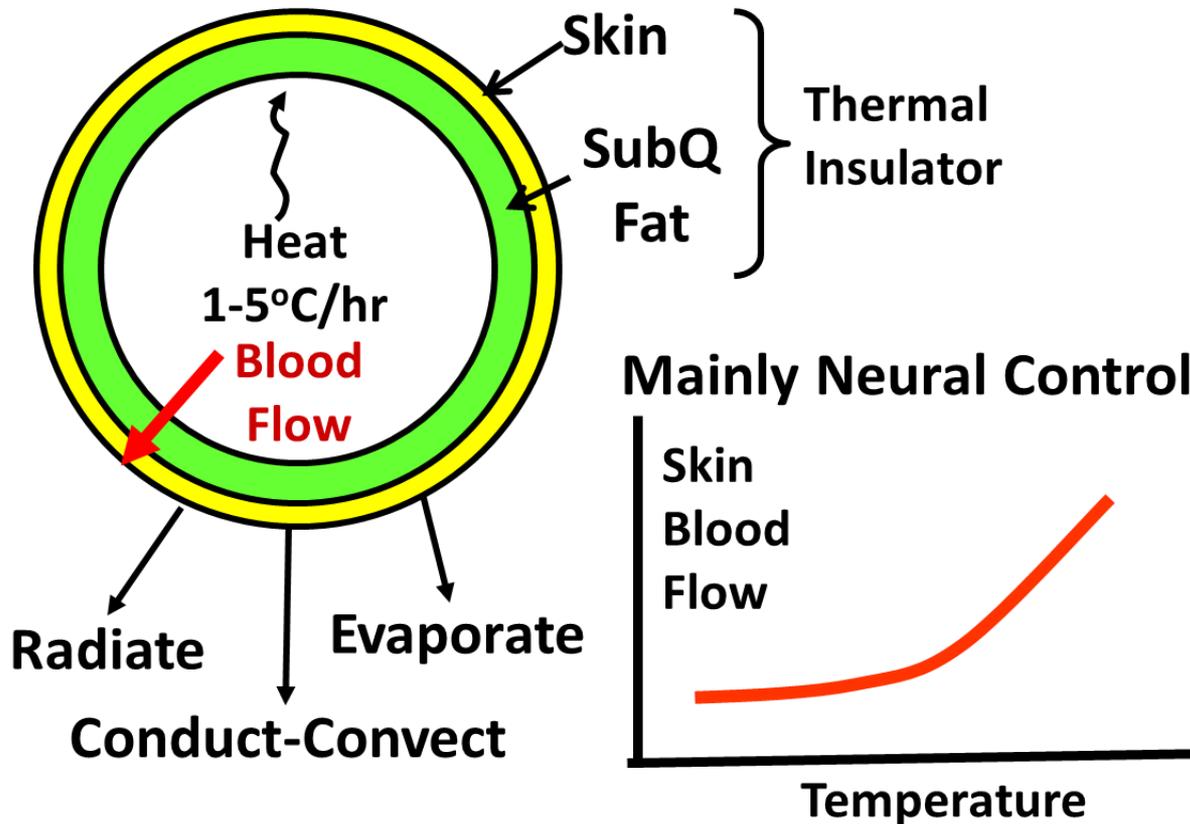
Metabolic

Skeletal Muscle

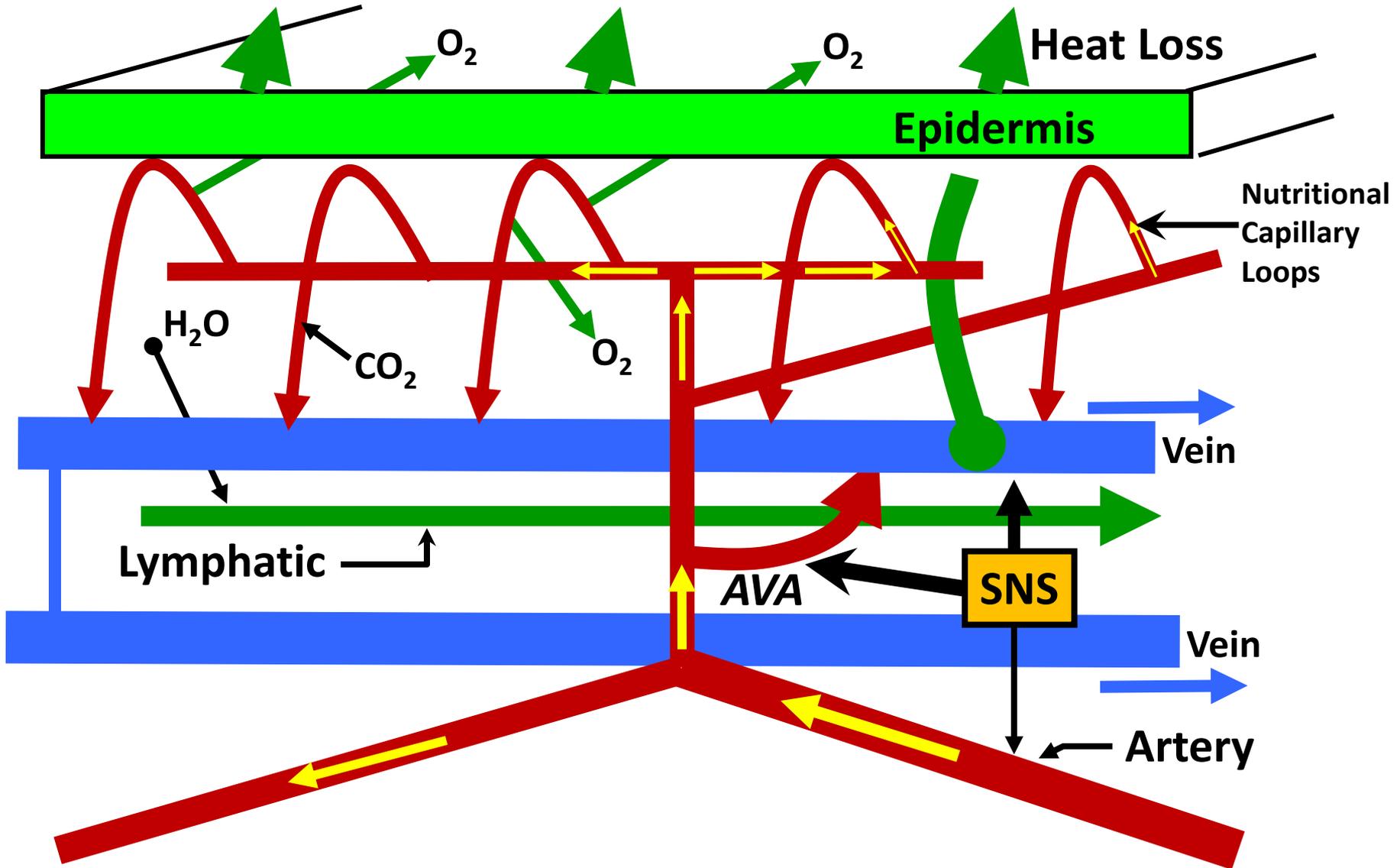


Cutaneous Control

Mainly Thermal but also Nutritional

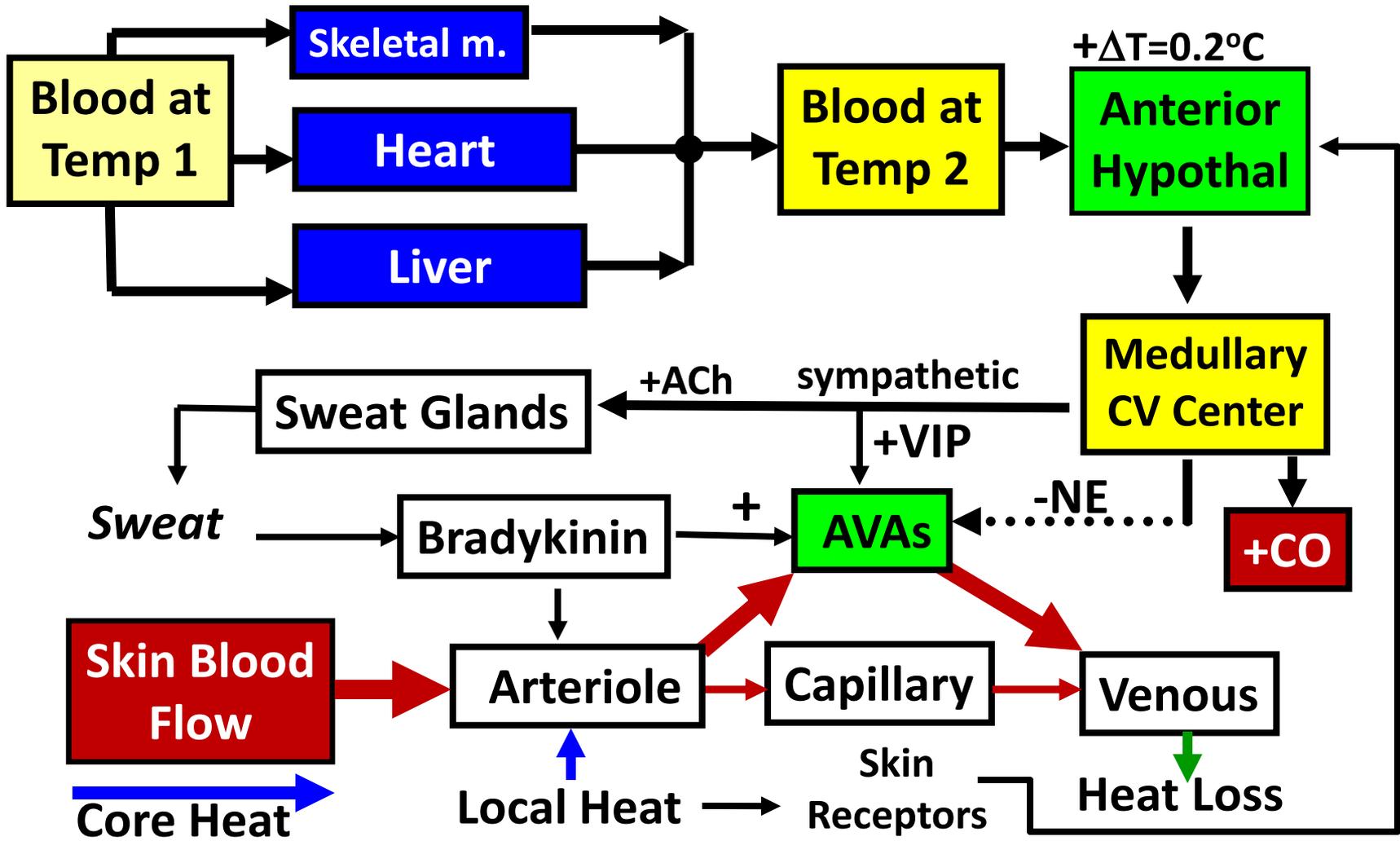


Skin Circulation Features



SNS = sympathetic nervous system
AVA = arterial venous anastomosis

Skin Overall Control



Cerebral Circulation

Main Function

Maintain O_2 and glucose) to hypoxia-intolerant grey matter

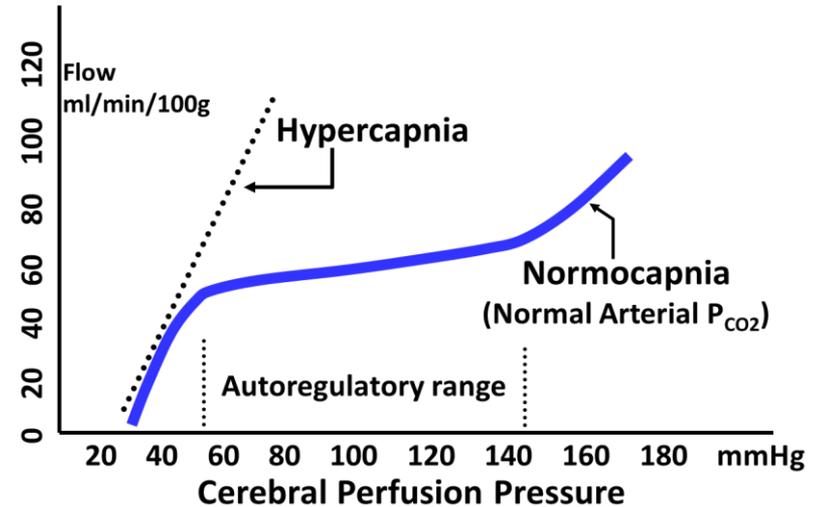
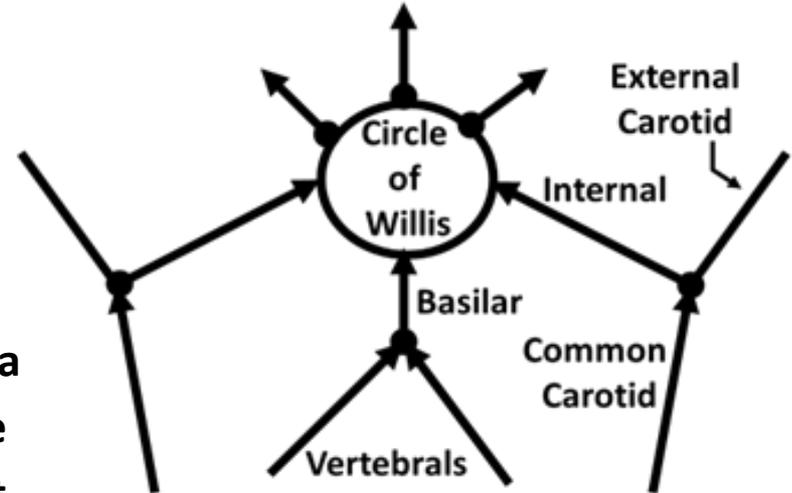
Main Features

- Circle of Willis - Anatomical guard against ischemia
- High capillary density - Small O_2 diffusion distance
- Blood brain barrier - Stable neuronal environment
- High Basal Blood Flow ~ 50 ml/min/100g
- Brain can control own flow by changing BP
- Highly developed blood flow autoregulation

Metabolic control with physiological vasodilators

- Increased CO_2
- Decreased O_2 ($P_{O_2} < 50$ mmHg)
- Increased K^+ or H^+
- Adenosine due to low O_2

(Ischemia – Hypoxemia – Hypotension)



Splanchnic Circulation

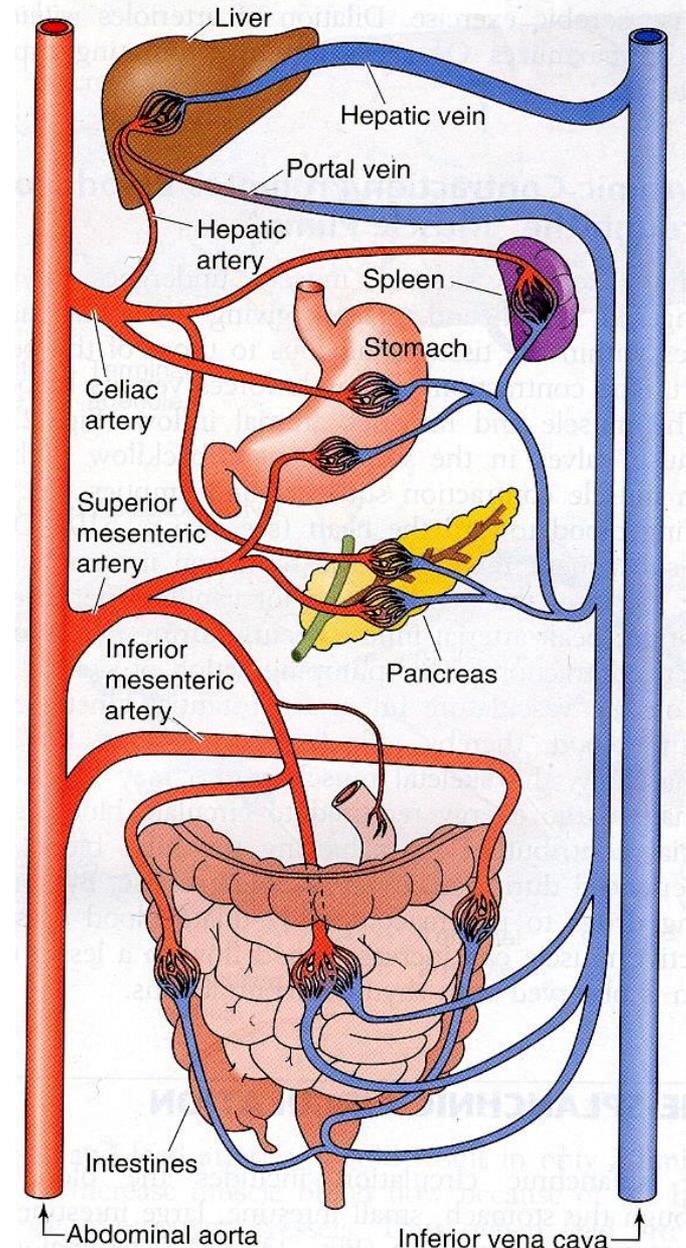
Blood flow through

- Liver
- Spleen
- Stomach
- Pancreas
- Large Intestines
- Small Intestines

Highly Interconnected
Vascular Supply

In each gut region

- Blood Flow ~ Metabolic Activity in that region ~ Digestive and Absorptive Activities in region
- Blood Flow increases sequentially from stomach to more distal segments in accordance with activity



End CV Physiology Lecture 13