Central (systemic) regulation

Neuronal regulatory mechanisms

- Vasoconstrictor fibers (VC).
  - Sympathetic (VC) → They discharge contanously leading to VC → called sympathetic VC tone.
  - parasympathetic VC fibers. no VC parasympathetic directly.
    - Coronary vasoconstriction. after bradycardia. (indirectly)
- Vasodilator fibers (VD)
  - Sympathetic (VD):- all sympathetic innervation to blood vessels is VC, except:
    1. Coronary vessels:- ↑HR → ↓ O2 tention → accumulation of metabolites → VD
    2. Skeletal muscles:- VD → cholinergic → Ach, they start operate even befor the start of the exercise.
       - cativated by Sudden strong emotions → ↓VD "markid" → ↓BP → Syncope.
    3. Splanchnic areas: Stimulat β receptor to VD
    4. Sympathetic to sweat gland: VD → it controlled by heat loss center.
  - Parasympathetic VD fibers: the only parasympathetic which are definitely VD → genital organ (sacral out-flow)
    - VD to salivary gland by facial and glossopharyngeal nerve by increasing metabolic activity.
  - Antidromic vasodilator impulges:- when pain receptors are stimulated by pain → produces dilatation of adjecent blood vessels. (reflex)
    - dont involve CNS

* impulse of pain travel toword CNS, until they reach abrach, they travel along it (antidromically), when they reach arterioles, they releas Substance P → VD → so inflammation area become red.

Hormonal regulatory mechanisms

- Circulating VC substances:-
  1. Catecholamine
     - noradrenaline → α → VC
     - Adrenaline → β_2 → VD
  2. Renin-angiotension-system "↑BP, ↑aldosterone" ← the goal
     - strong arteriolar VC so ↑ prephral resistance.
     - Aldosterone release → from suprarenal gland
     - ↑ ADH (vasopression) → from pituitary gland
     - Stimulate NA release from postganglioinc sympathatic fibers.
     - ↑ thirst sensation
     - Salt and water retention
     - Some angiotechsion II → angiotension III
  3. ADH → (vasopression / Antidiuretic hormone) → goal → ↑ blood volume, ↓ urine volume
     - act on V_1 receptor in vascular SM → ↑BP
     - act on V_2 receptor in nephron → ↑ permeability to water, urea, some solute → ↑BP

↑BP / ↑BF

- Circulation VD substances
  1. Kinins. ← Kininogen.
     - plasma Kinins (bradyKinin)
     - tissue Kinin (Kalidin
     - Action:- ① Positive chemotaxis ② ↑ permeapility ③ ↑ pain receptor ④ VD in active salivary gland. ⑤ Contraction of smooth muscle in respiratory system.
  2. Atrial Natriuretic Peptide :- from atria
     - created when (goal ↓BP) → ① NaCl intack increased ② Blood volume increased ③ Immersion in water up to neck ↑VR ④ ↑ central venous pressure ⑤ ↑ Itra ventricular pressure.
     - Action → natriuresis, VD, ↓aldosterone, ↓renin, ↓ADH

*regulation of diameter of arterioles *

local regulation mechanisms

- O2 tension
  - ↓O2 VD
  - in polmonary: ↓O2 VC
  - •O2 lack most potent VD in heart.
  - • Co2 lack most potent VD in CNS.
- Metabolites "Co2, acidosis, ↓PH, adenosine, K+ osmolarity"
  - ↳Active hyperimia
    - accumilation metabolits
    - ↳due to active tissue → ↑BF (VD)
  - ↳Reactive hyperemia (passive)
    - ↳↑BF → accumulation metabolites due to temporary occlusion (congestion blood / cyanosis)
- Intrinsic (Autoregulation)
  - we tissue needs
  - ① Myogenic → VD → ↑BF → stimulate strech receptor
    - ↳ depolarization → VC → ↓BF
    - ↳releasing endothelins.
  - ② Metabolic → ↓BF → DV due to accumulation of waste productes → ↑BF to remove their effect. "example → sympathatic VD to coronary vessels indirectly"
- local vasoconstctor substances
  - Seratonin "which released from platlets"
- Local temperature.
  - ↓ tempreture → VC
  - ↑temperature → VD
- Substances released by the endothelium
  - → Thromoboxane A2 and prostacyclin.
    - ↳ Prostacyclin → endothilum → VD + inhibit platlet aggregation.
    - ↳ Thromoboxane A2 → platlets → VC + increase platlet aggregation.
    - * Aspirin (acytyle sallcylic acid) → irreversable inhibition of cycloygenase enzyme.
      - ↳So stop forming platlet and that is lead to ↓ formation of thromoboxane A2 → VD
  - → Endothelins
    - → by streatching vesseles.
    - → potant VC
  - →Actions
    - → +ve intotropic effect on cardiac muscle (VC)
    - → coronaries (VC)
    - → Drease renal blood flow and ↑ renal vascular resistance. (VC of renal vesseles)
    - → contraction to vascular smooth muscle espeally veins (VC)
    - → Stimulate aldosterone and catecholamine release. → aldosterone (↑BF)
  - → Endothelium - derived - relaxing factor (EDRF)
    - ↳ NO "nitric oxide" → ↑ cGMP (VD)
    - ↳released by endothelium under effect of bradyKinin, substance P, VIP
    - ↳Its deficiency as in cases of endothelium injury → ↑ Thromboxane A2 (VC) → causing VC, atherosclerosis, hypertension, impotence (failure of erection by VD in male genital organs)

*Charaters of arteriole
- → ↑SM layer
- → great resistance
- → sensitive to chemicals like hormone, gases
- → has sympathatic and parasympathic fibers
- → Its endothilum synthesize chemial mediator
- → only site at which artiroles can be seen in retina.

*Function of arterioles
- → preipheral resistance
- → control Blood flow by changing their diameter.