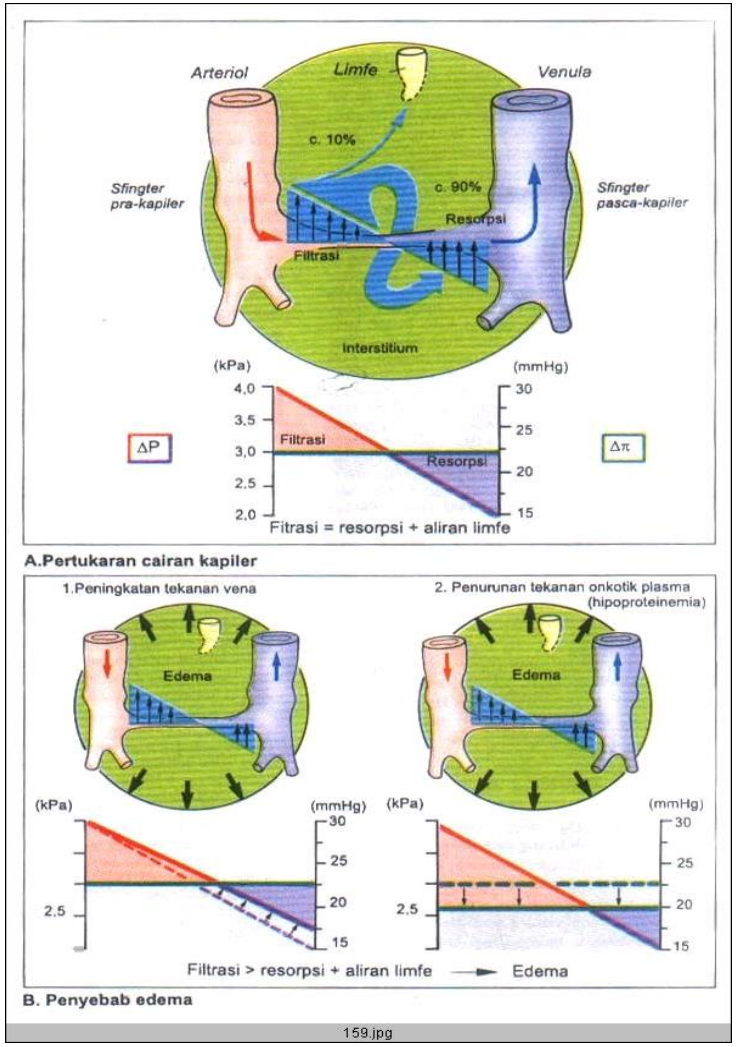
Pengaturan Tekanan Darah

dr Hadi Sarosa

Sirkulasi Sistemik

- Kapiler
  - Pertukaran zat (transport aktif & pasif)
- Venula (pembuluh kapasitan)
  - Menampung darah (compliance tinggi)
- Vena



Pengaturan Lokal

Proporsional mengatur kebutuhan jaringan

Akut

- Perubahan kebutuhan oksigen
  - VASODILATOR (CO_2, Asam laktat, adenosin, histamin, K^+, H^+, Ca^+)
  - TEORI KEBUTUHAN OKSIGEN

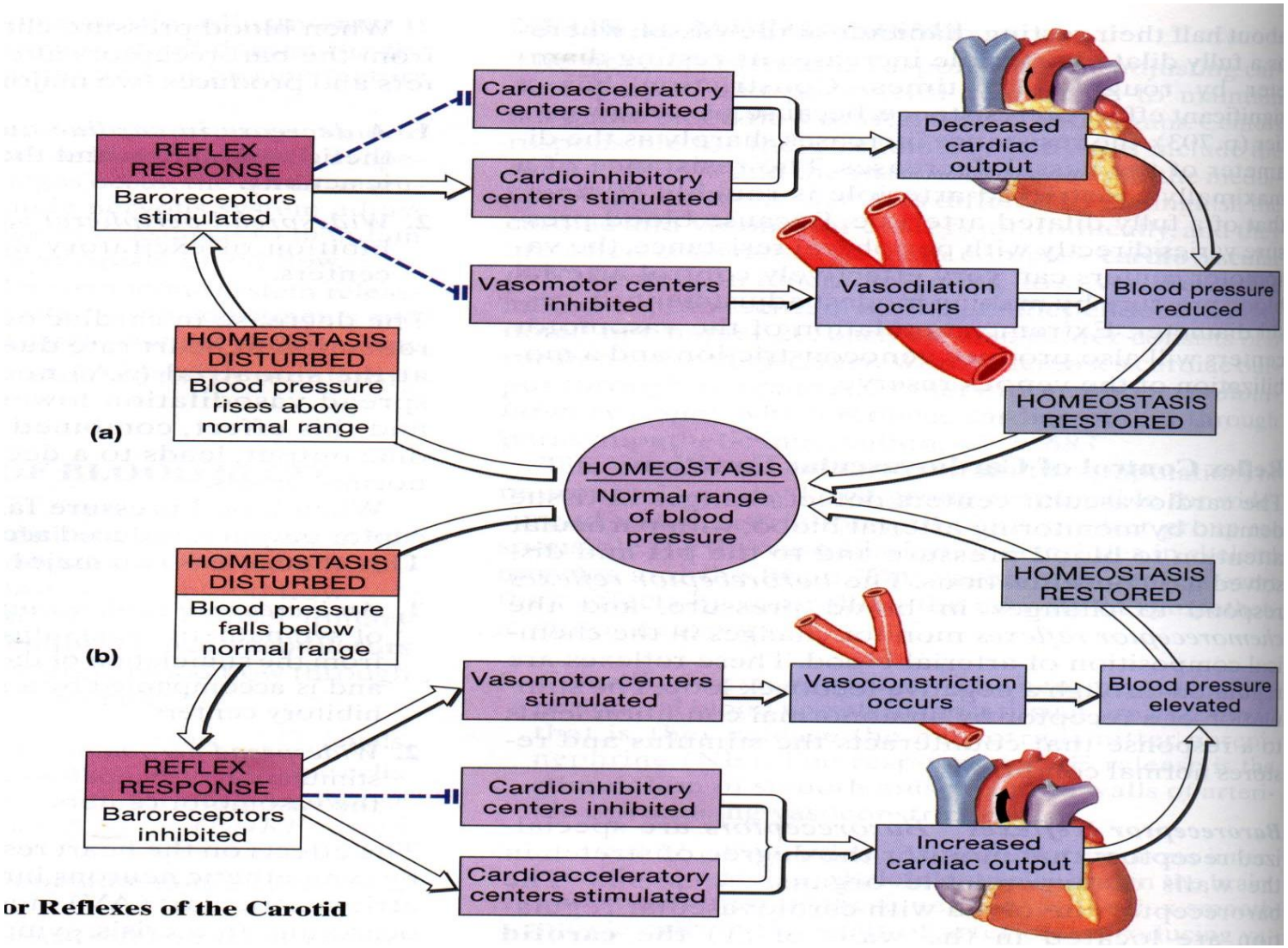
    - reactive hyperemia

    - active hiperemia

Jangka panjang

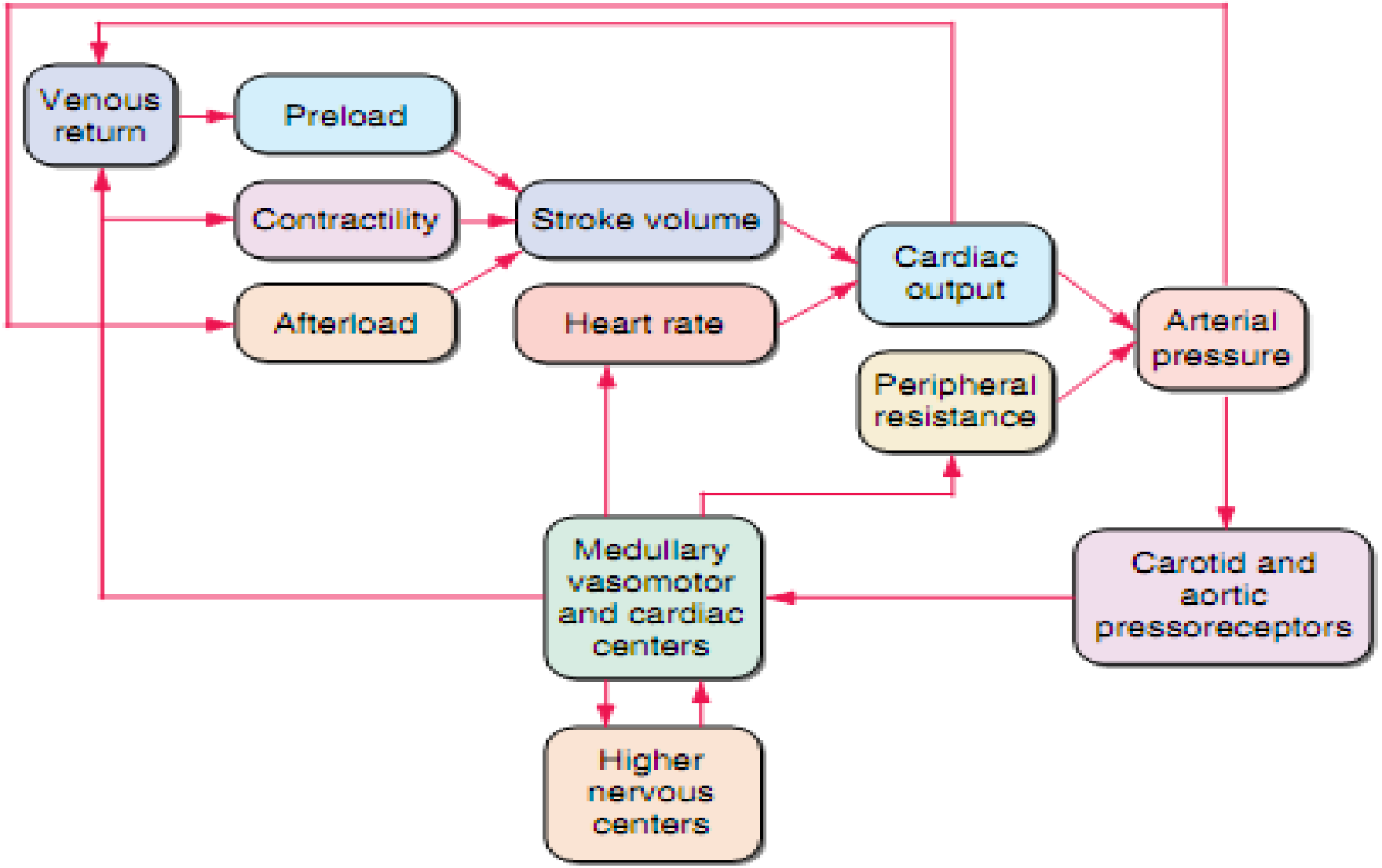
- Perubahan vaskularisasi jaringan
  - Angiogenesis
  - pertumbuhan pembuluh darah baru

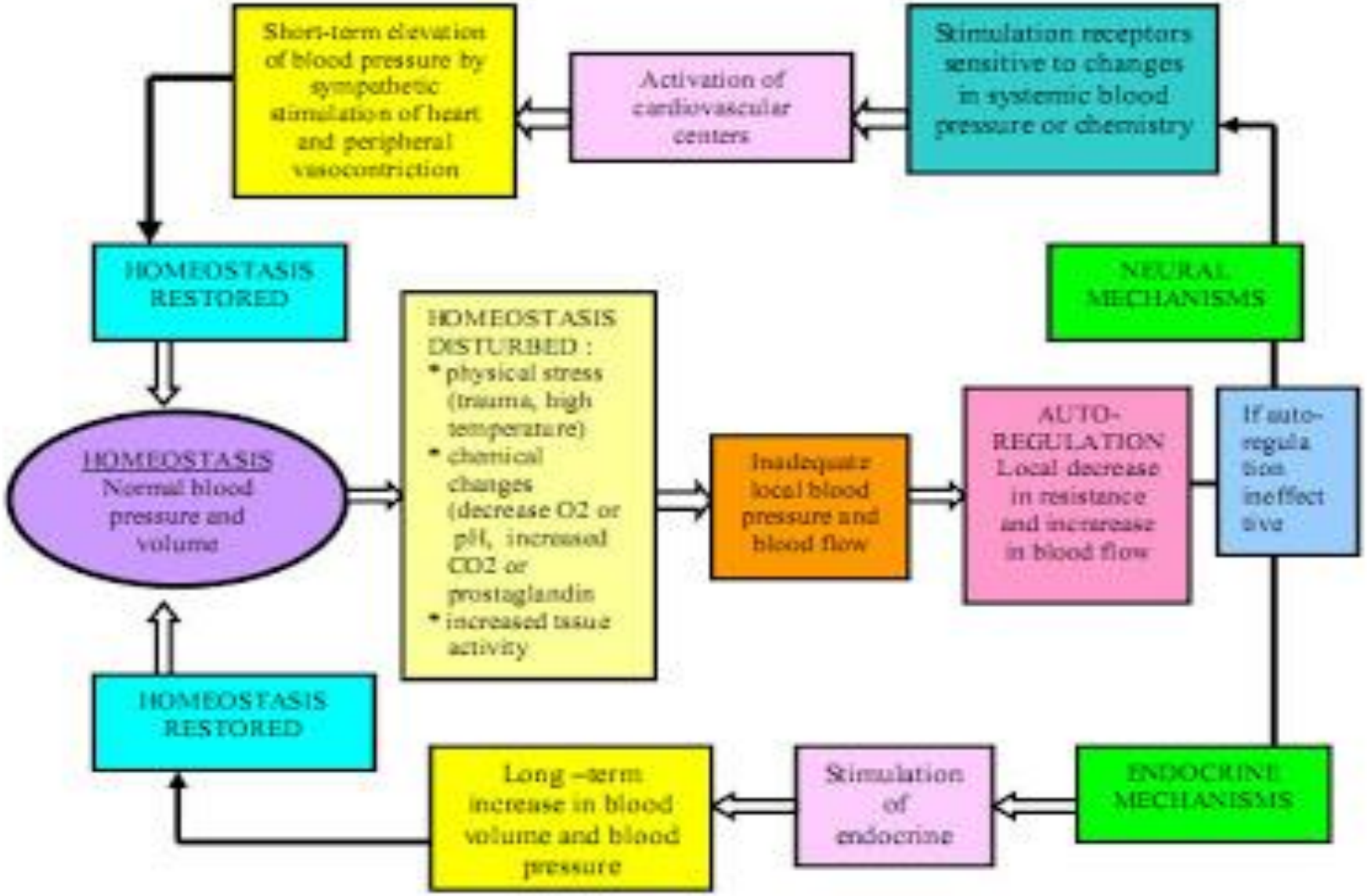
Pengaturan Tekanan Darah



or Reflexes of the Carotid

Pengaturan Tekanan Darah

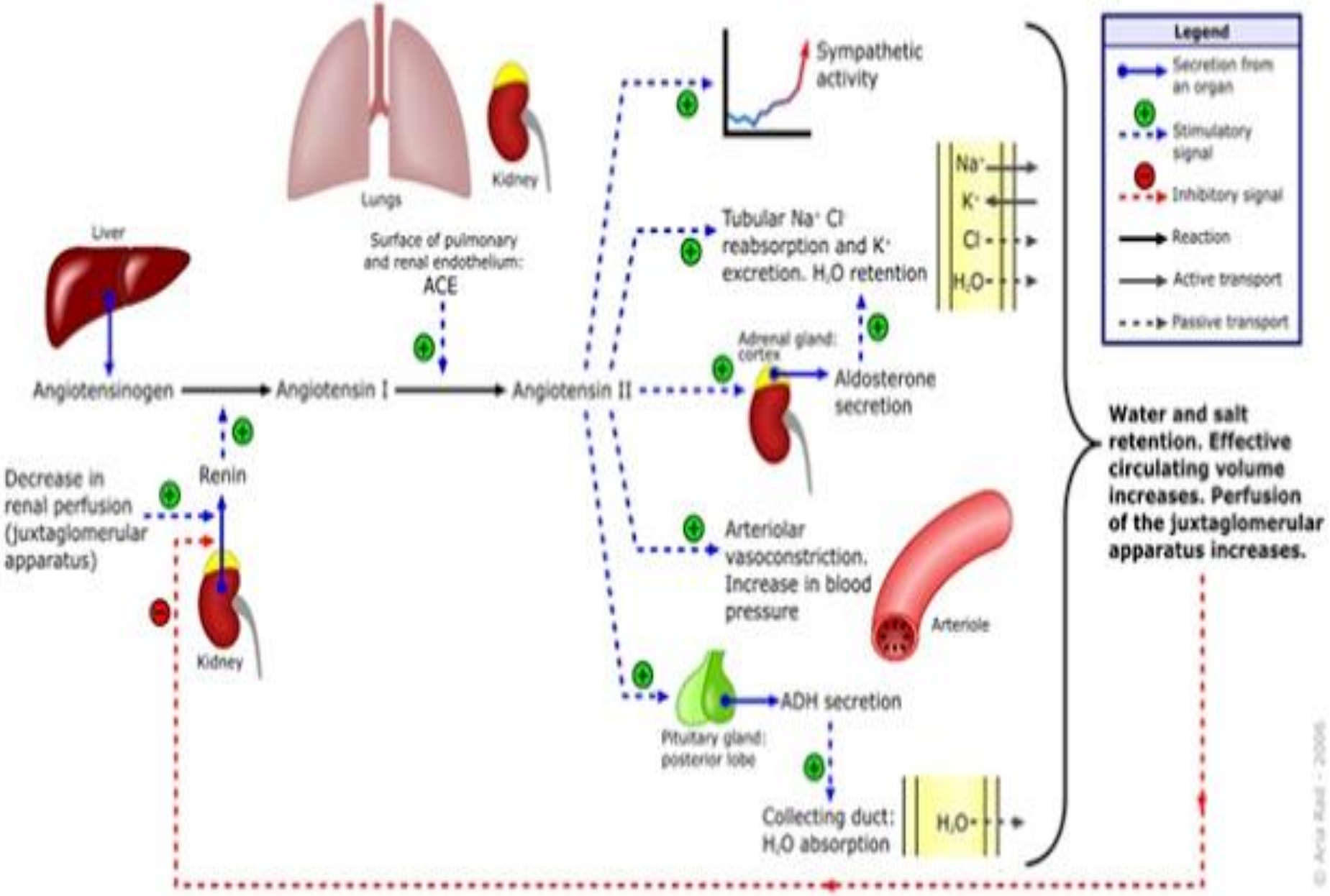




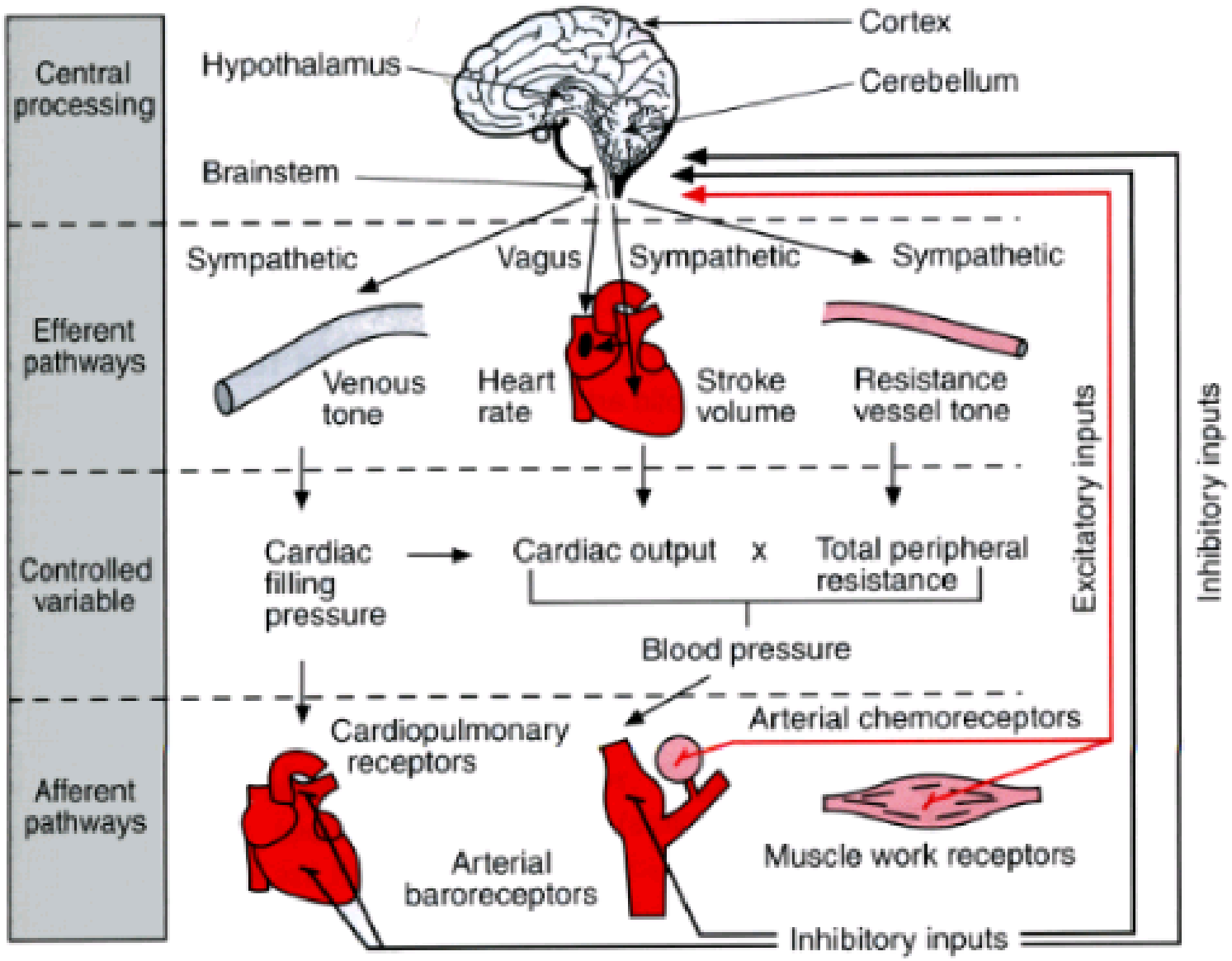
Gambar 1. Diagram regulasi homeostasis untuk mempertahankan tekanan darah dan aliran darah

(Sumber : Martini 2001, yang dimodifikasi)

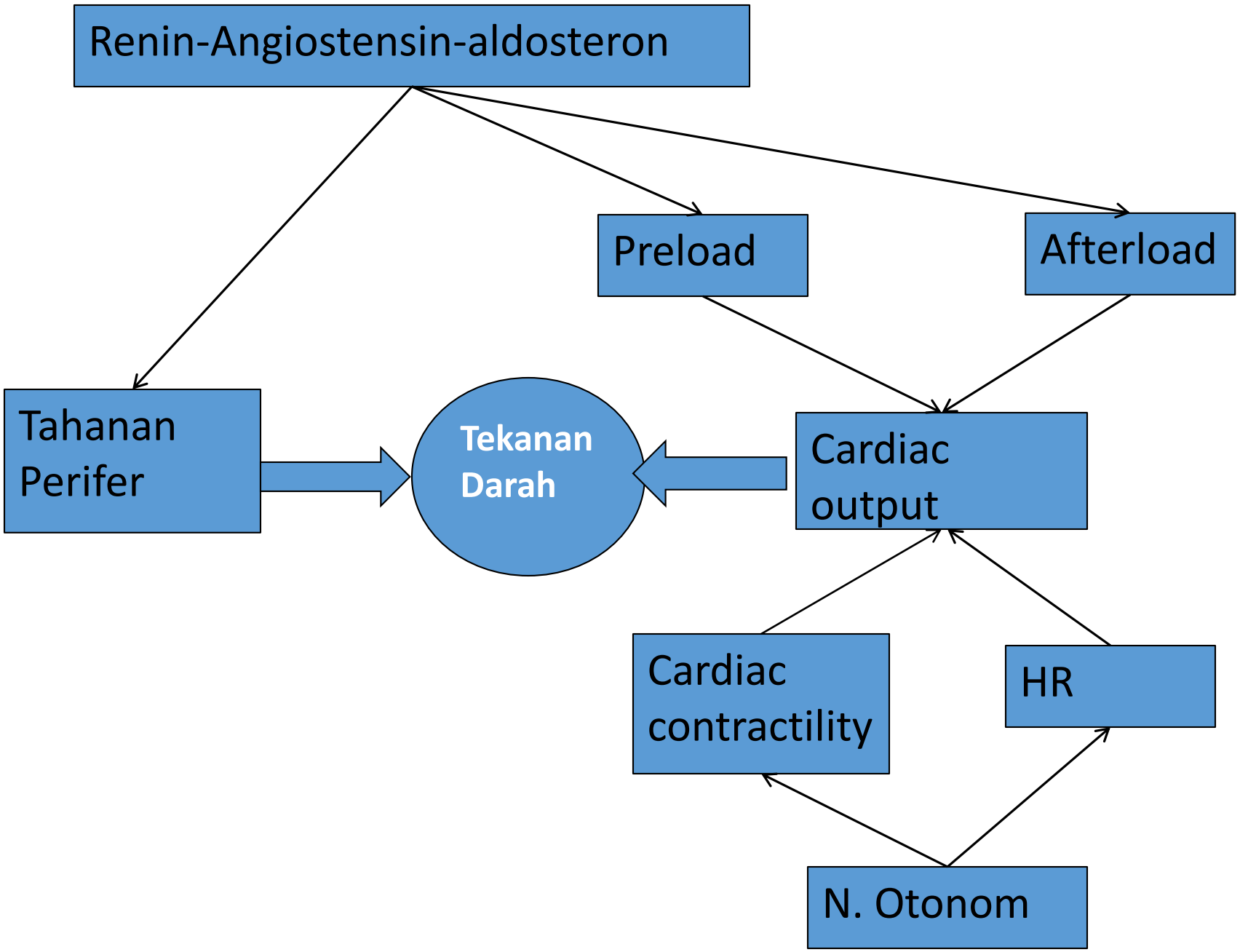
Renin-angiotensin-aldosterone system



Overview of short-term control mechanisms



From: Introduction to Cardiovascular physiology. J.R. Levick. Arnold 4th edition (2003


Renin-Angiostensin-aldosteron
Preload
Afterload
Tahanan Perifer
Tekanan Darah
Cardiac output
Cardiac contractility
HR
N. Otonom

Arteriola	Contriction	Dilatation
Local factors	Decreased local temperatur Autoregulation	Increased CO2 & decreased O2 Increased K, adenosin, lactate Decreased pH Increased temp
Endothelial Products	Endothelin-1 Locally released platelet serotonin Tromboxane A2	NO Kinin Prostacyclin
Circulating hormones	Epinephrine (except in skeletal muscle and liver) Norephinephrine Na-K ATPase inhibitor Neuropeptide Y	Ephinefrin (skeletal, liver) Histamin ANP Substance P VIP CGRP alpha
Neural factor	noredrenergic	Decreased noradrenergic Cholinergic to skeletal muscle

TEKANAN DARAH DIPENGARUHI OLEH :

- Cairan darah :
  - Cairan darah bertambah, tekanan meningkat
  - Cairan darah berkurang , tekanan turun
  - Darah lebih kental tekanan meningkat

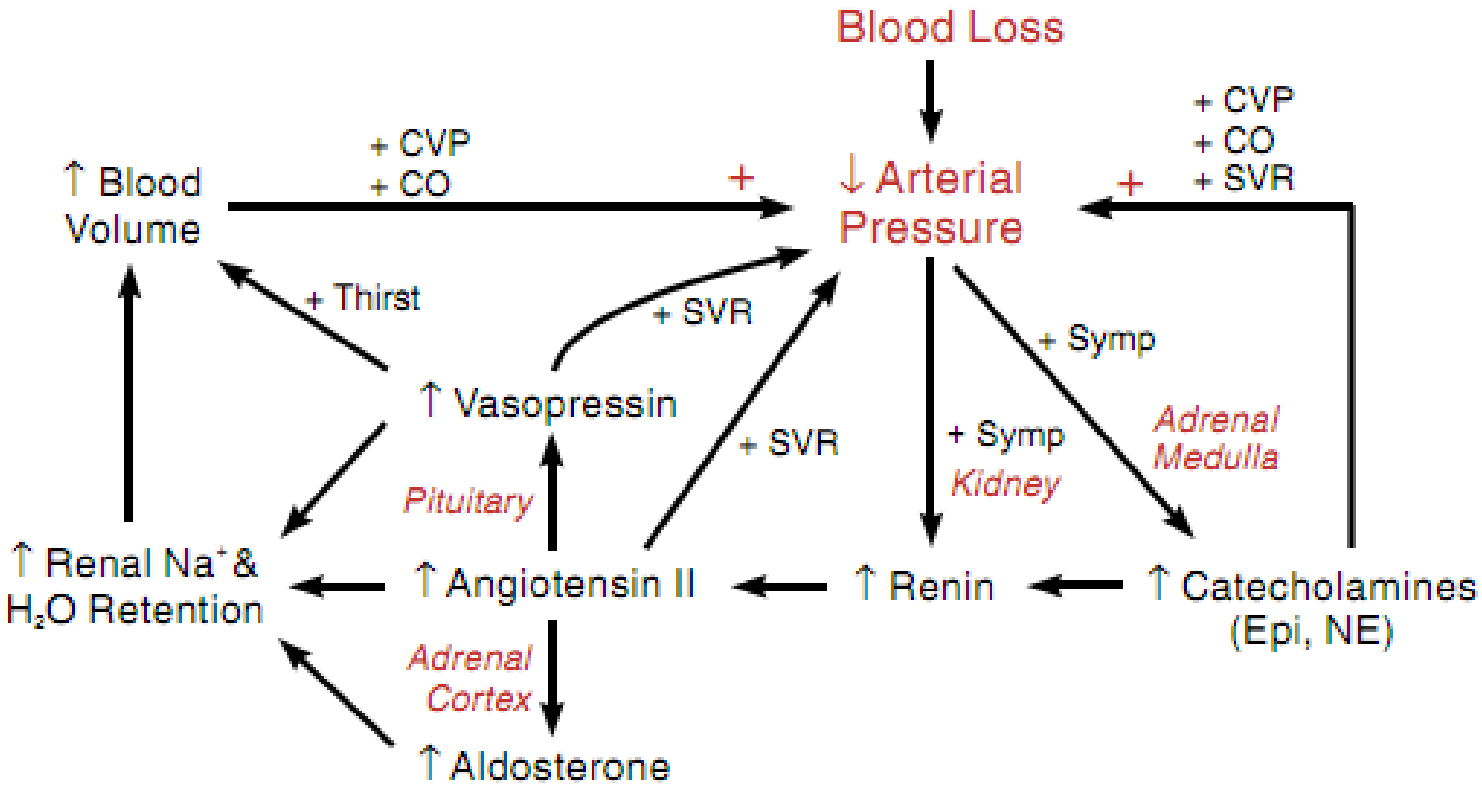


FIGURE 9-6 Activation of humoral mechanisms following acute blood loss (hemorrhage). Decreased arterial pressure activates the sympathetic nervous system (*+Symp*) (baroreceptor reflex). Renin release is stimulated by the enhanced sympathetic activity, increased circulating catecholamines, and hypotension, which leads to the formation of angiotensin II and aldosterone. Vasopressin release from the posterior pituitary is stimulated by angiotensin II, reduced atrial pressure (not shown), and increased sympathetic activity (not shown). These hormones act together to increase blood volume through their renal actions (sodium and water retention), which increases central venous pressure (*+CVP*) and cardiac output (*+CO*). Angiotensin II and vasopressin also increase systemic vascular resistance (*+SVR*). Increased circulating catecholamines (*Epi*, epinephrine; *NE*, norepinephrine) reinforce the effects of sympathetic activation on the heart and vasculature. These changes in systemic vascular resistance, central venous pressure, and cardiac output partially restore the arterial pressure.

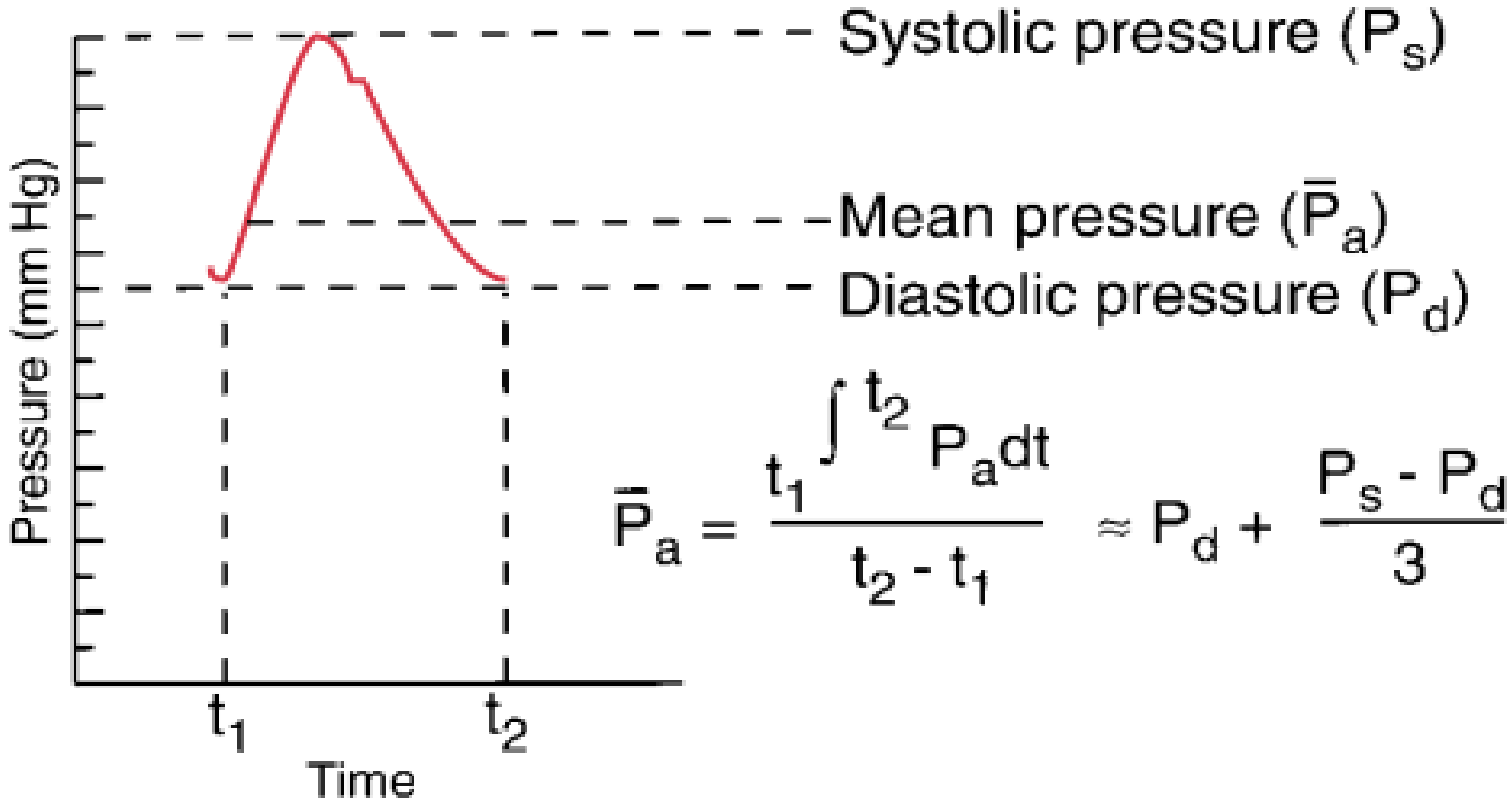


FIGURE 15.1 **Definition of mean arterial pressure.** Mean pressure is the area under the pressure curve divided by the time interval. This can be approximated as the diastolic pressure plus one-third pressure.

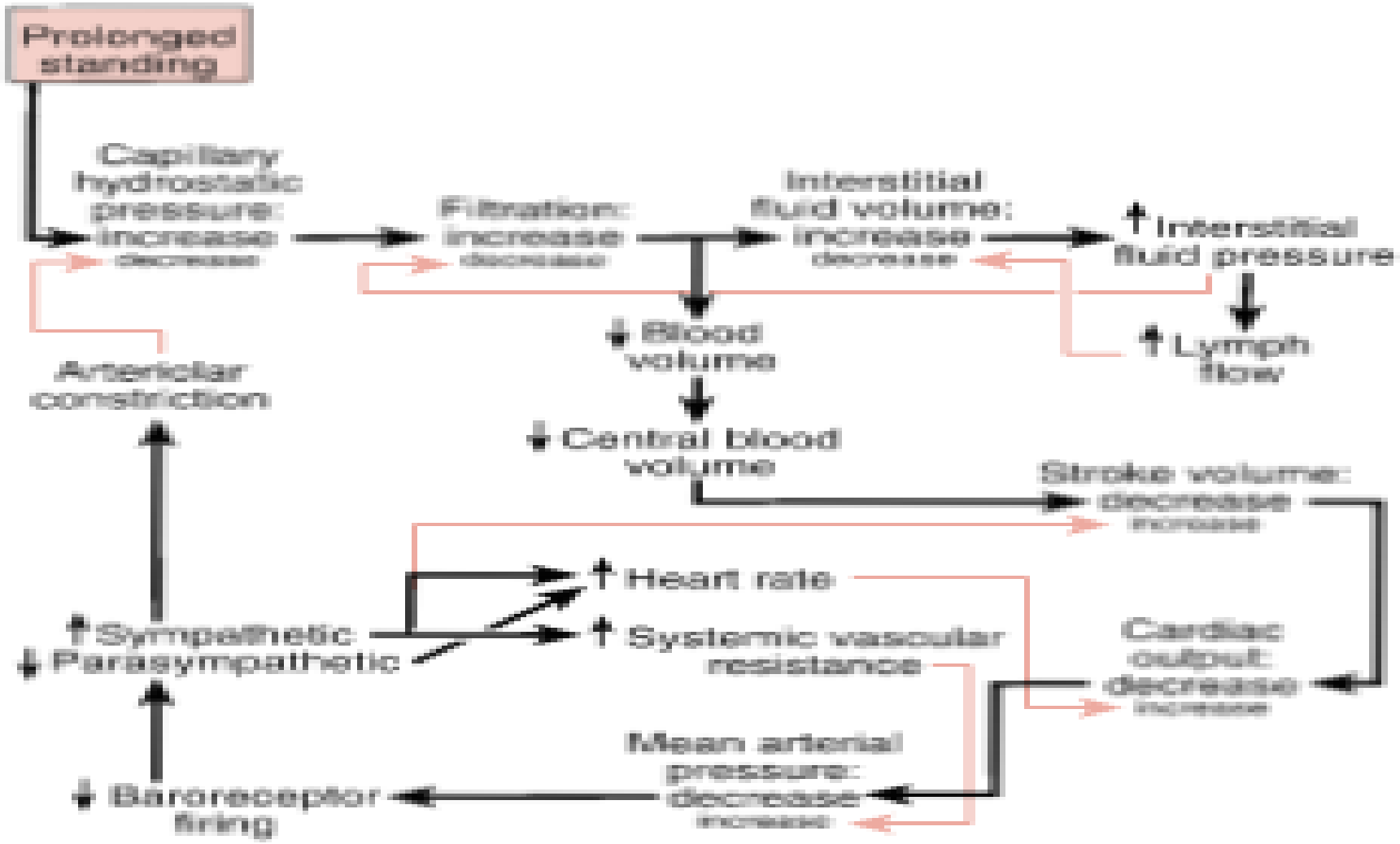


FIGURE 18.12 **Effects of prolonged standing.** With prolonged standing, capillary filtration reduces venous return. Without the compensatory events that result in the changes shown in small type, prolonged standing would inevitably lead to fainting.

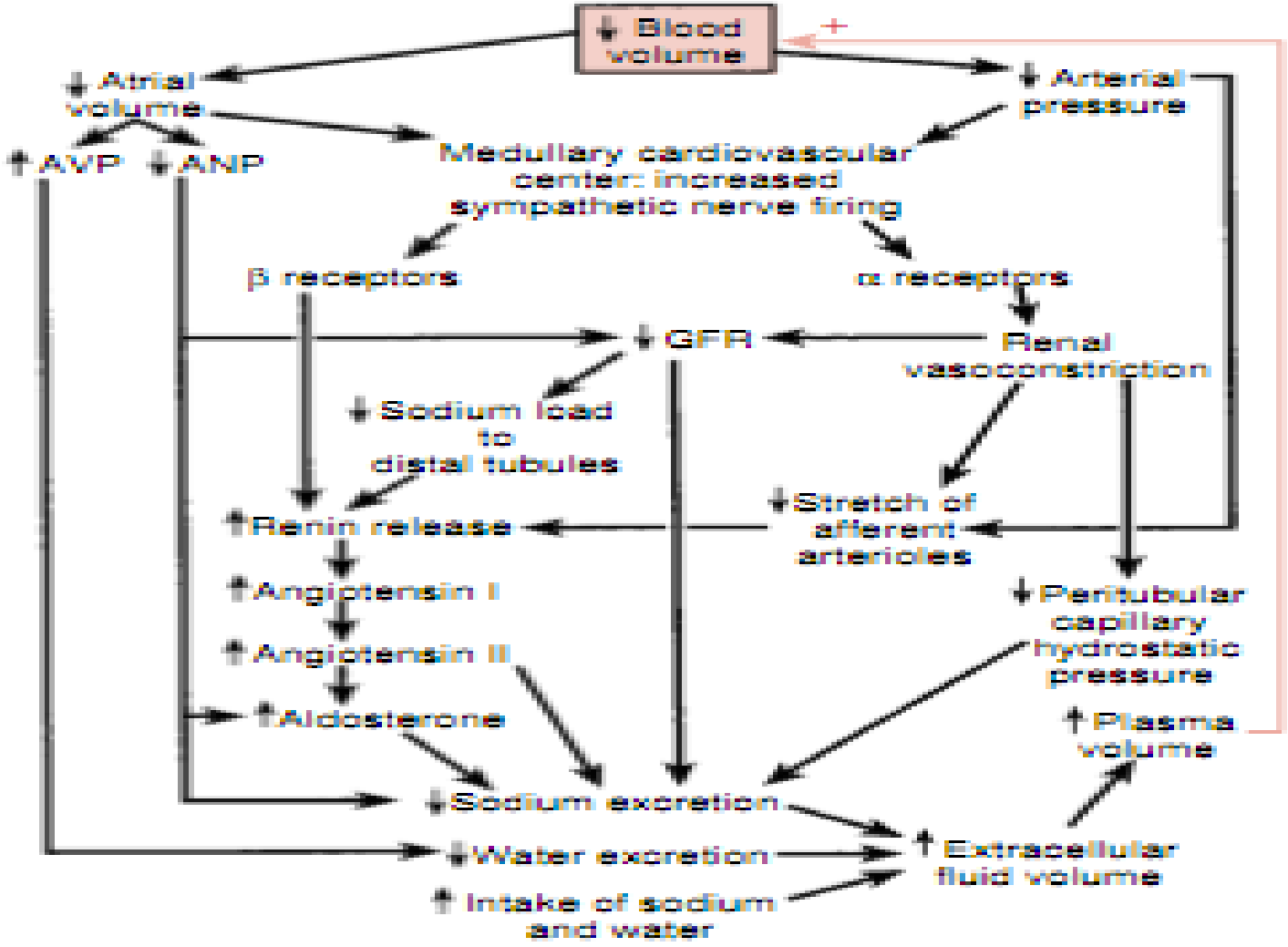


FIGURE 18.13 **Regulation of blood volume.** Blood loss influences sodium and water excretion by the kidney via several pathways. All these pathways, combined with an increased intake of salt and water, restore the extracellular fluid volume and, eventually, blood volume. These responses occur later than those shown in Figures 18.10, 18.11, and 18.12. The pathways responsible for stimulating an increased intake of salt and water are not shown. AVP, arginine vasopressin, ANP, atrial natriuretic peptide, GFR, glomerular filtration rate.

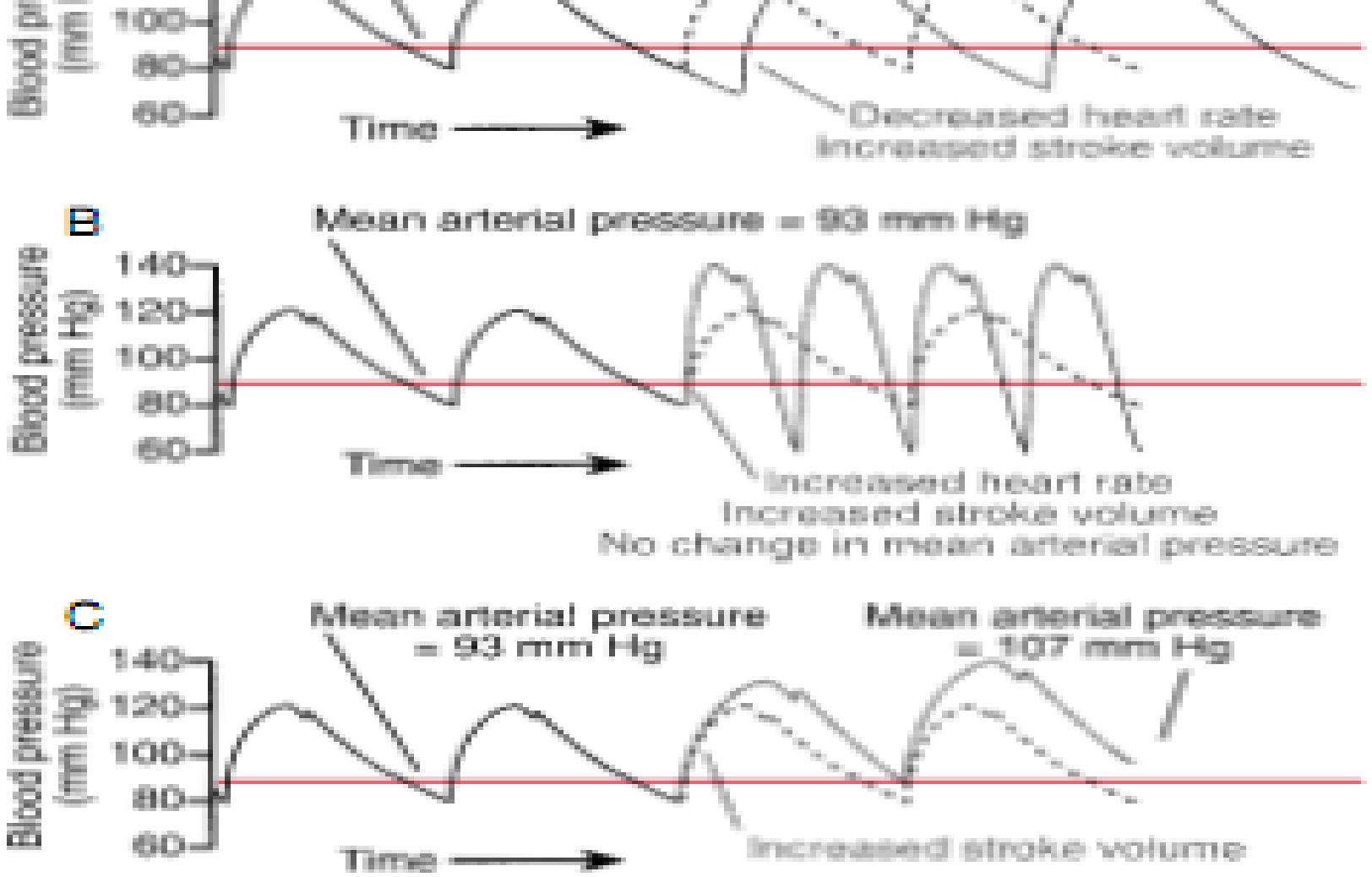


FIGURE 16.9 **Effects of** A, Effect of increased stroke volume on arterial pressure with constant cardiac output and SVR. When cardiac output is held constant by lowering heart rate, there is no change in mean arterial pressure (93 mm Hg) and systolic pressure increases while diastolic pressure decreases. B, Effect of increased heart rate *and* stroke volume with no change in mean arterial pressure because of decreased SVR. After the first two beats, stroke volume and heart rate are in-

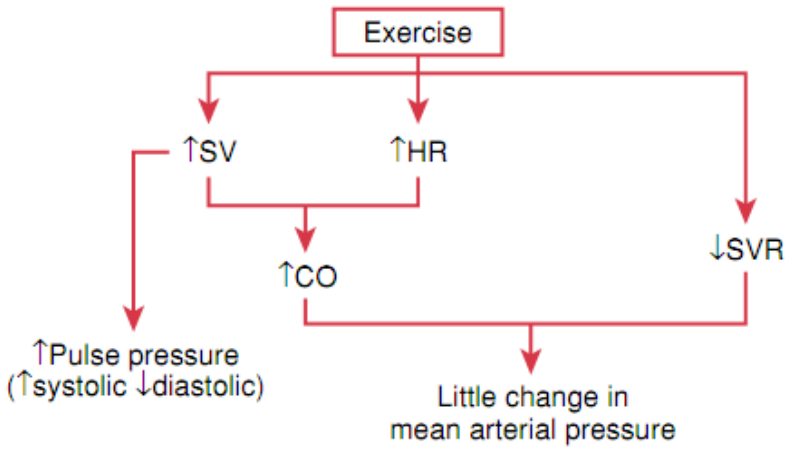
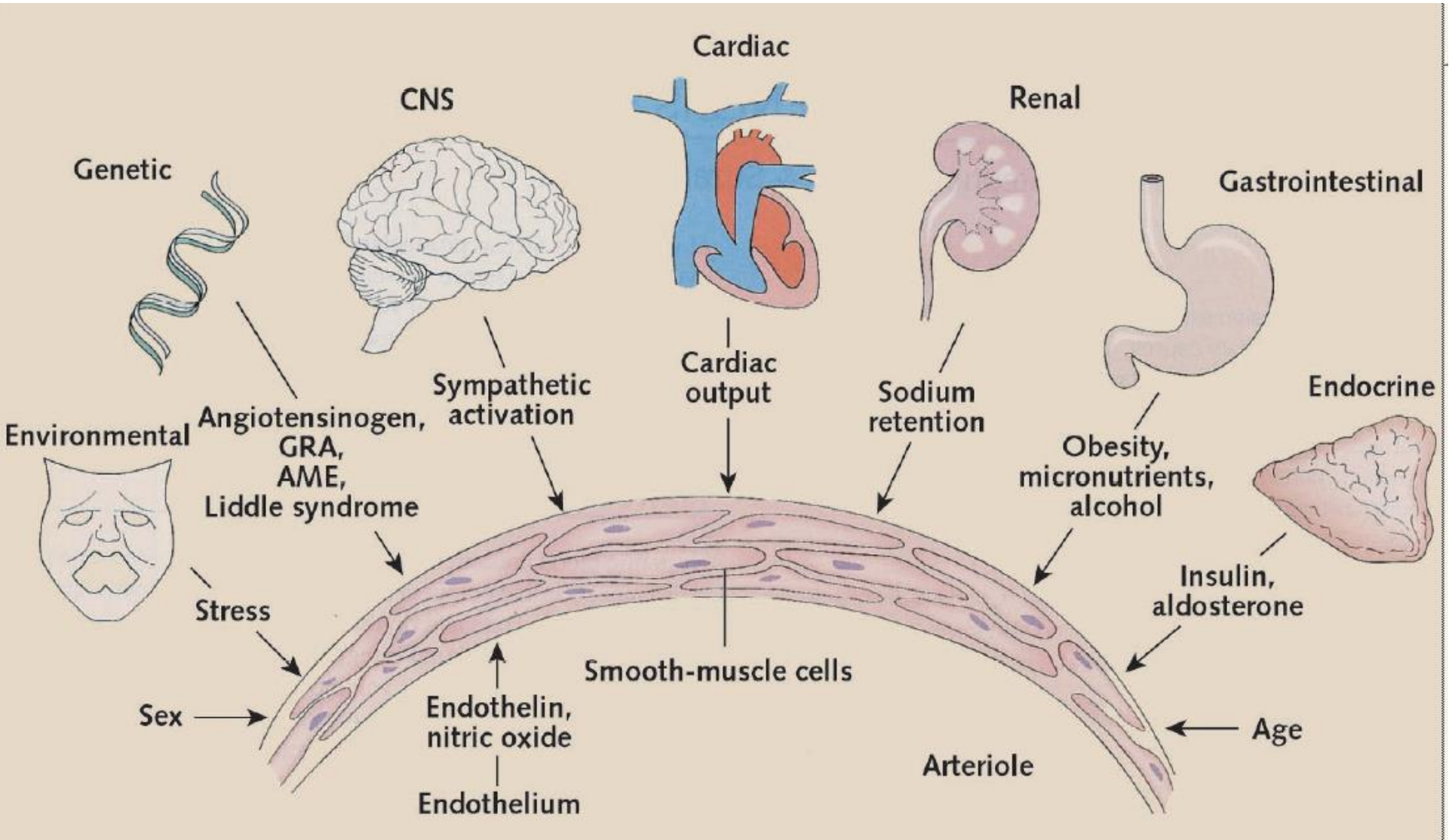
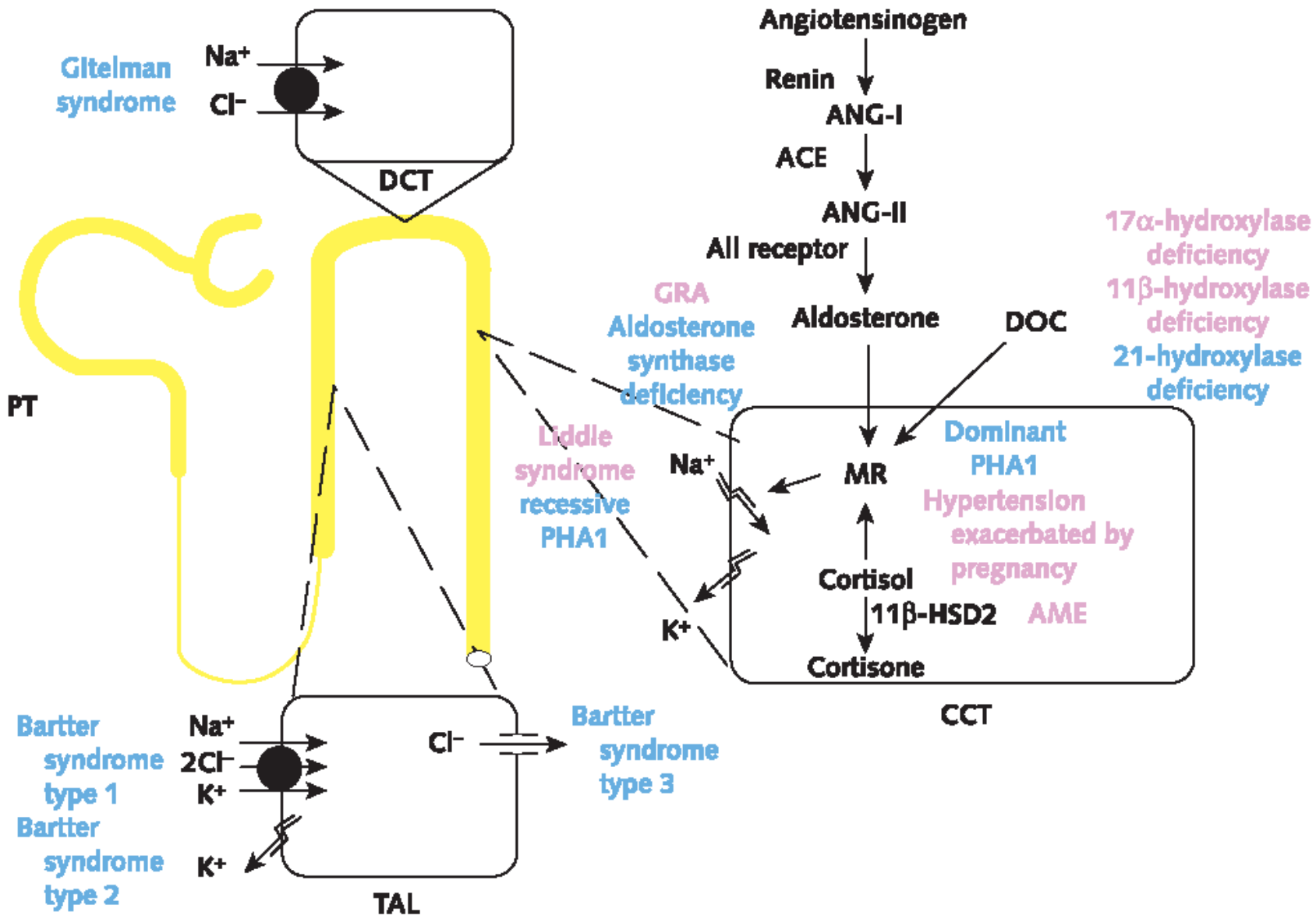


FIGURE 15.4 **Effect of dynamic exercise on mean arterial pressure and pulse pressure.** Heart rate (HR) and stroke volume (SV) increase, resulting in an increase in cardiac output (CO). However, dilation of resistance vessels in skeletal muscle lowers systemic vascular resistance (SVR), balancing the increase in cardiac output and causing little change in mean arterial pressure.

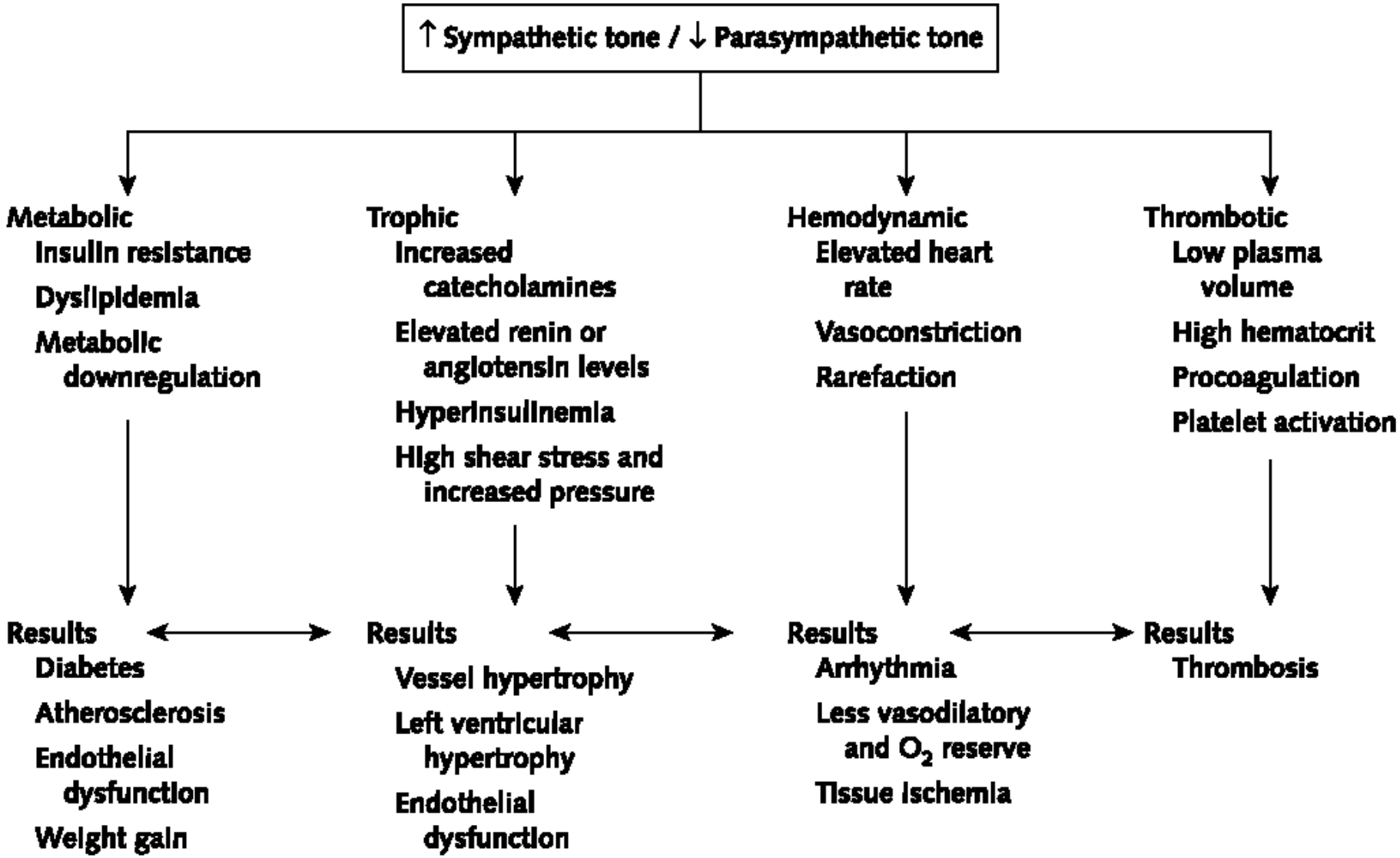
Skema penyebab hipertensi

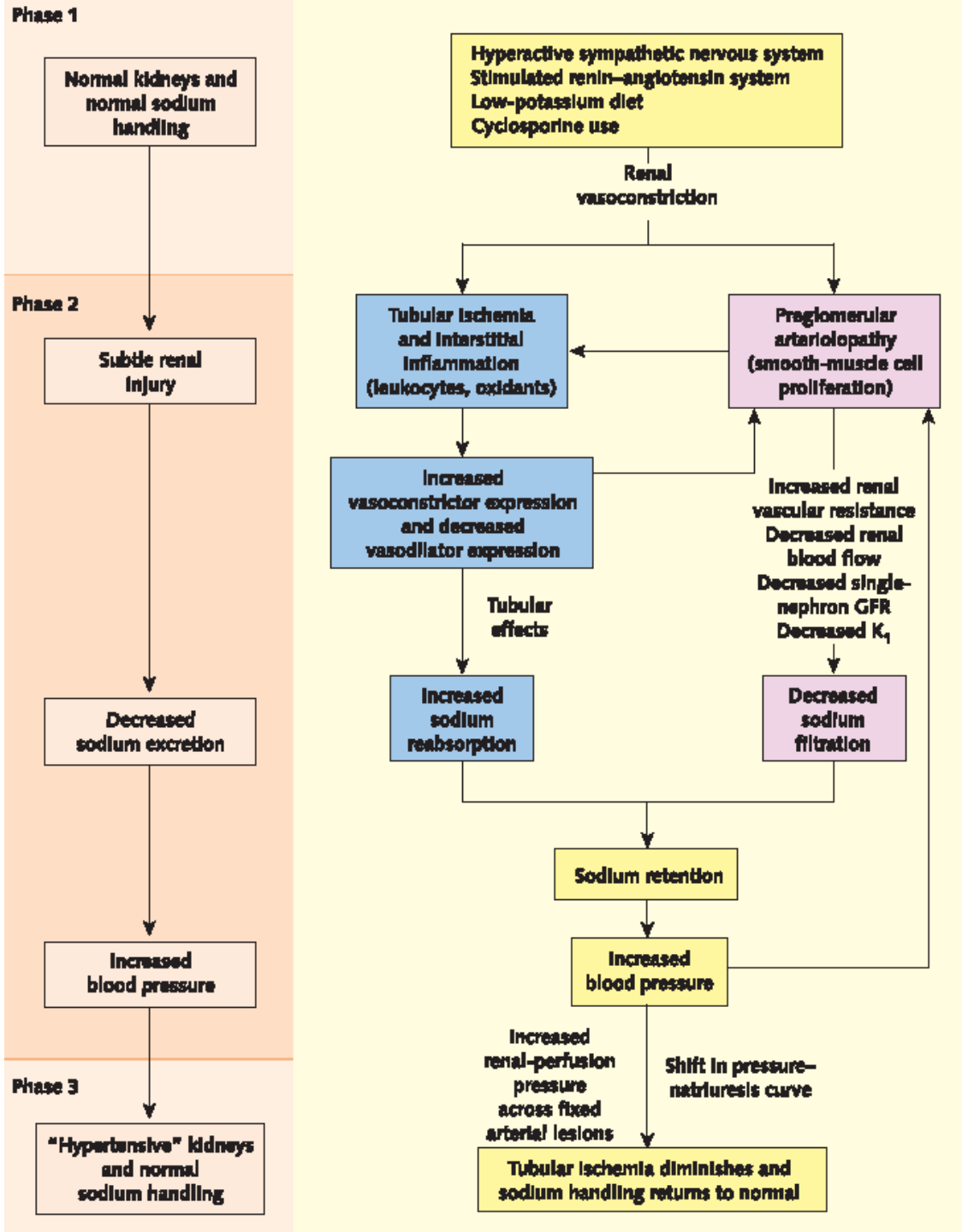


Kelainan gen yg dapat sebabkan hipertensi



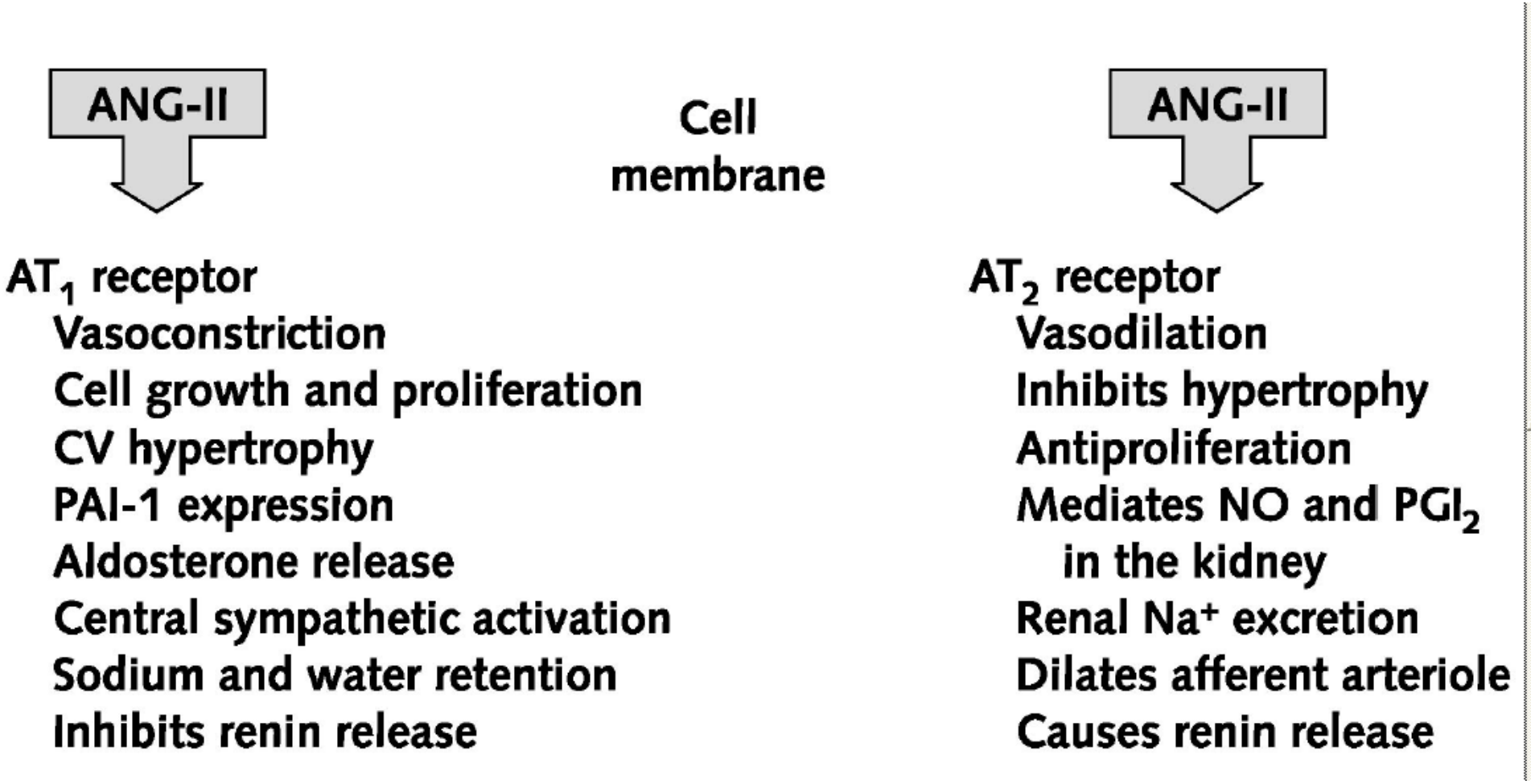
Peranan sistem simpatis





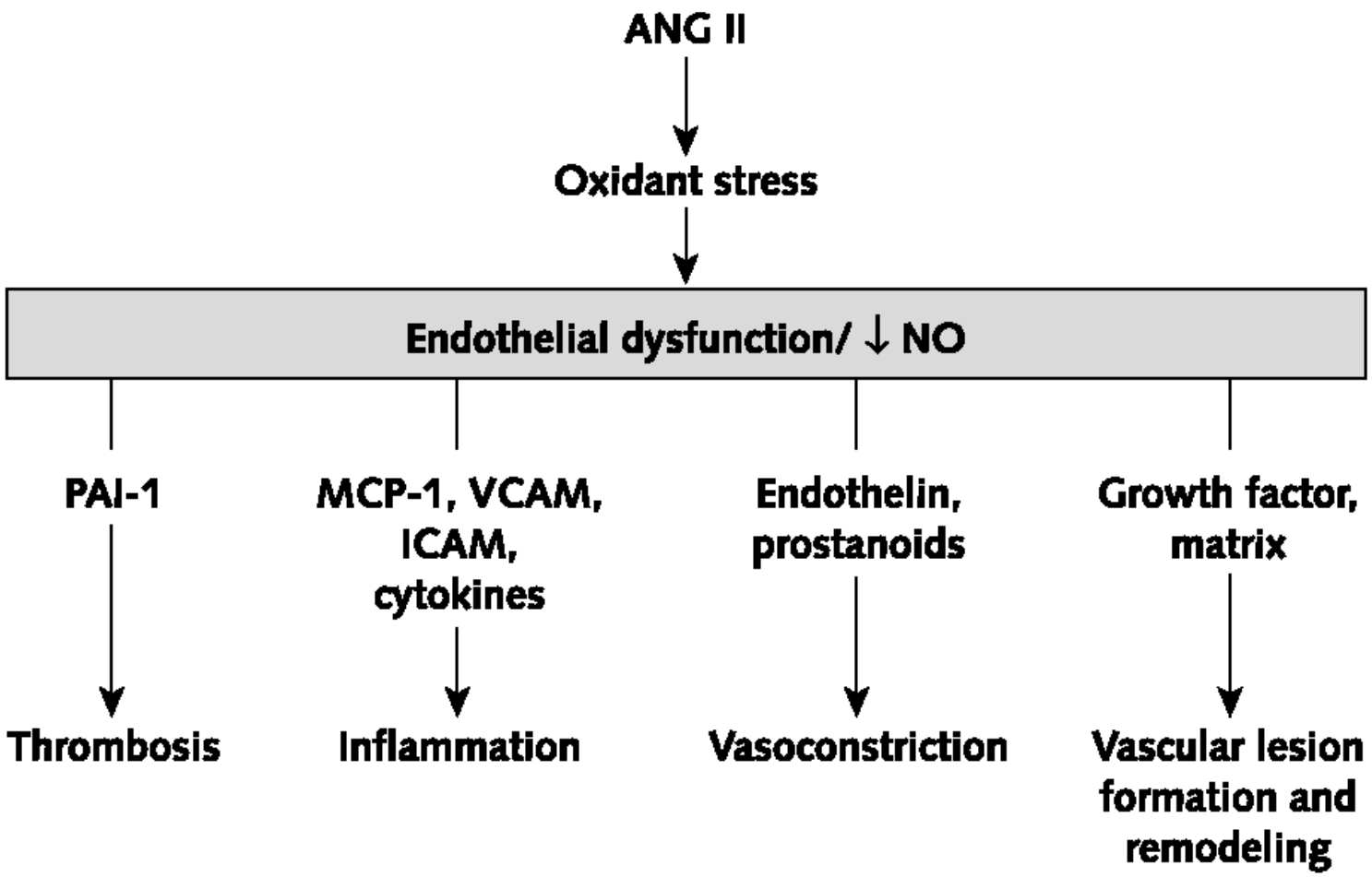
Gangguan pada ginjal

Sistem Renin-angiotensin



PAI-1 (plasminogen activator inhibitir-1)

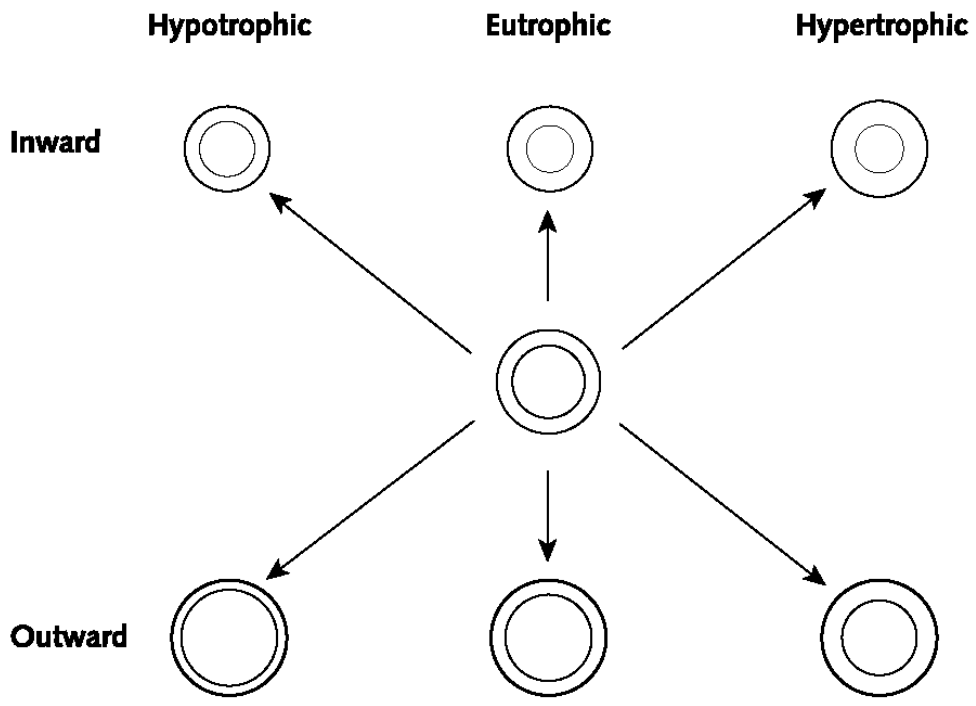
Pengaruh angiotensin II pada kerusakan vasa



- ICAM (intercellular adhesion molecul)
- MCP (monocyte chemoattractan protein)
- VCAM (vascular cell adhesion melecul)

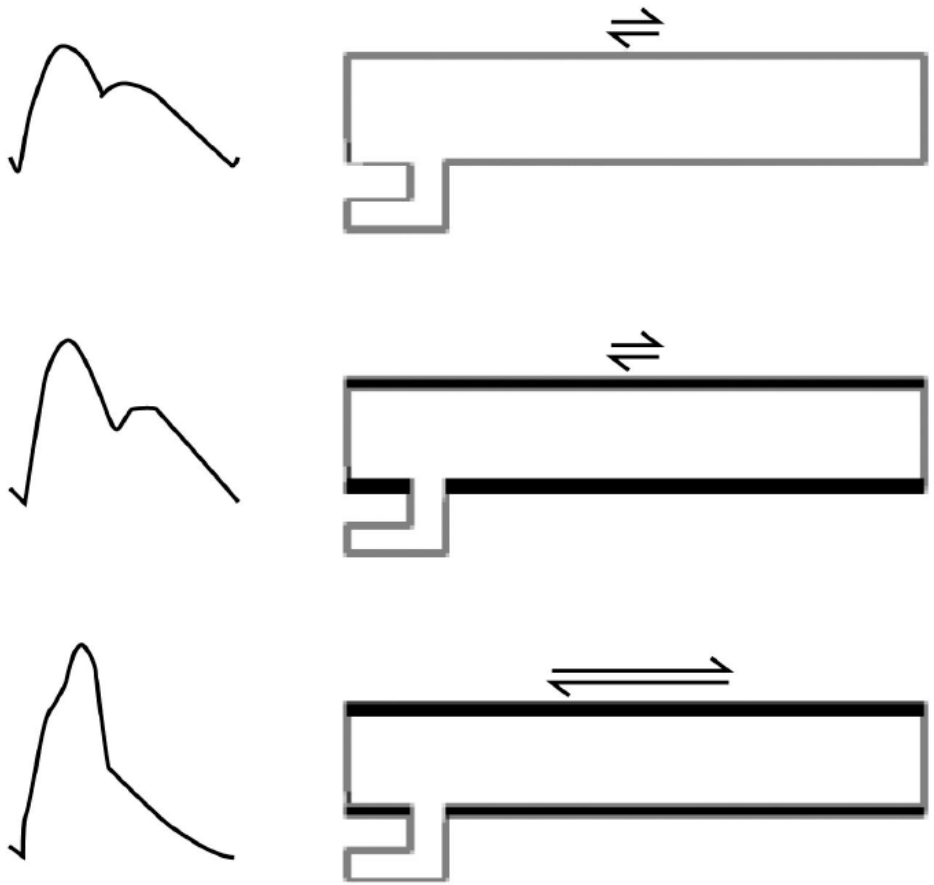
Perubahan pada vasa

- Vasa normal ditengah
  - Perubahan dapat berupa hipertropic, eutropic dan hypotropic
  - Perubahan dapat sebabkan diameter mengecil atau melebar



Perubahan pada vasa

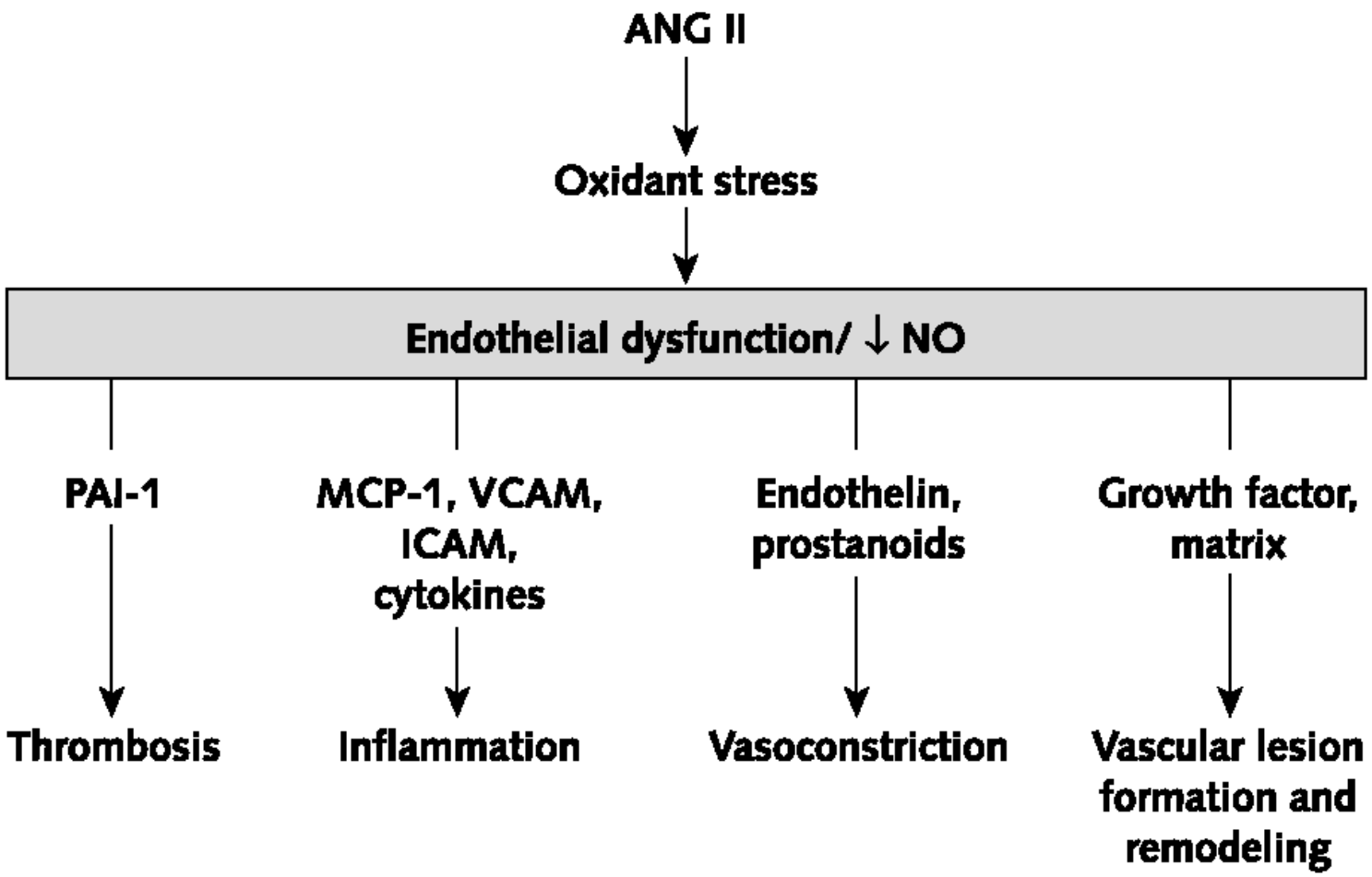
- Pembuluh darah yang kaku akan menyebebkan perubahan pada gambaran pulsus (denyut nadi)
- Pembuluh darah yang kaku akan sebabkan jantung bekerja lebih keras sehingga tekanan darah naik



Disfungsi endotel

- NO diphasilkan oleh sel endotel normal akibat dari berbagaui rangsang termasuk tekanan darah tinggi, robekan endotel, regangan denyut nadi.
- Fungsi NO
  - merupakan vasodilator yg poten
  - Sebagai inibitor agregasi dan inhibisi trombosit (platelet)
  - Supresor dalam proliferasi dan migrasi sel otot polos

Pengaruh angiotensin II pada kerusakan vasa



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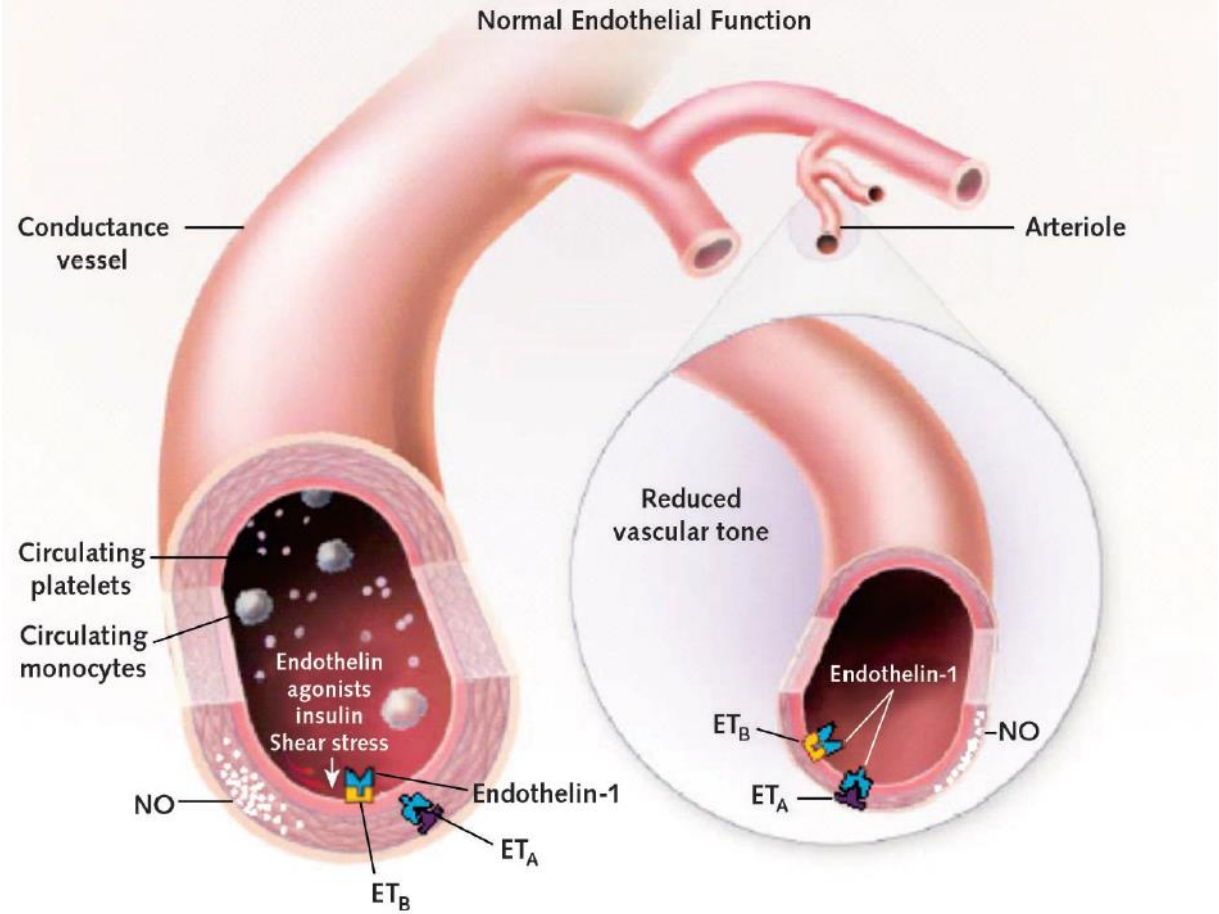
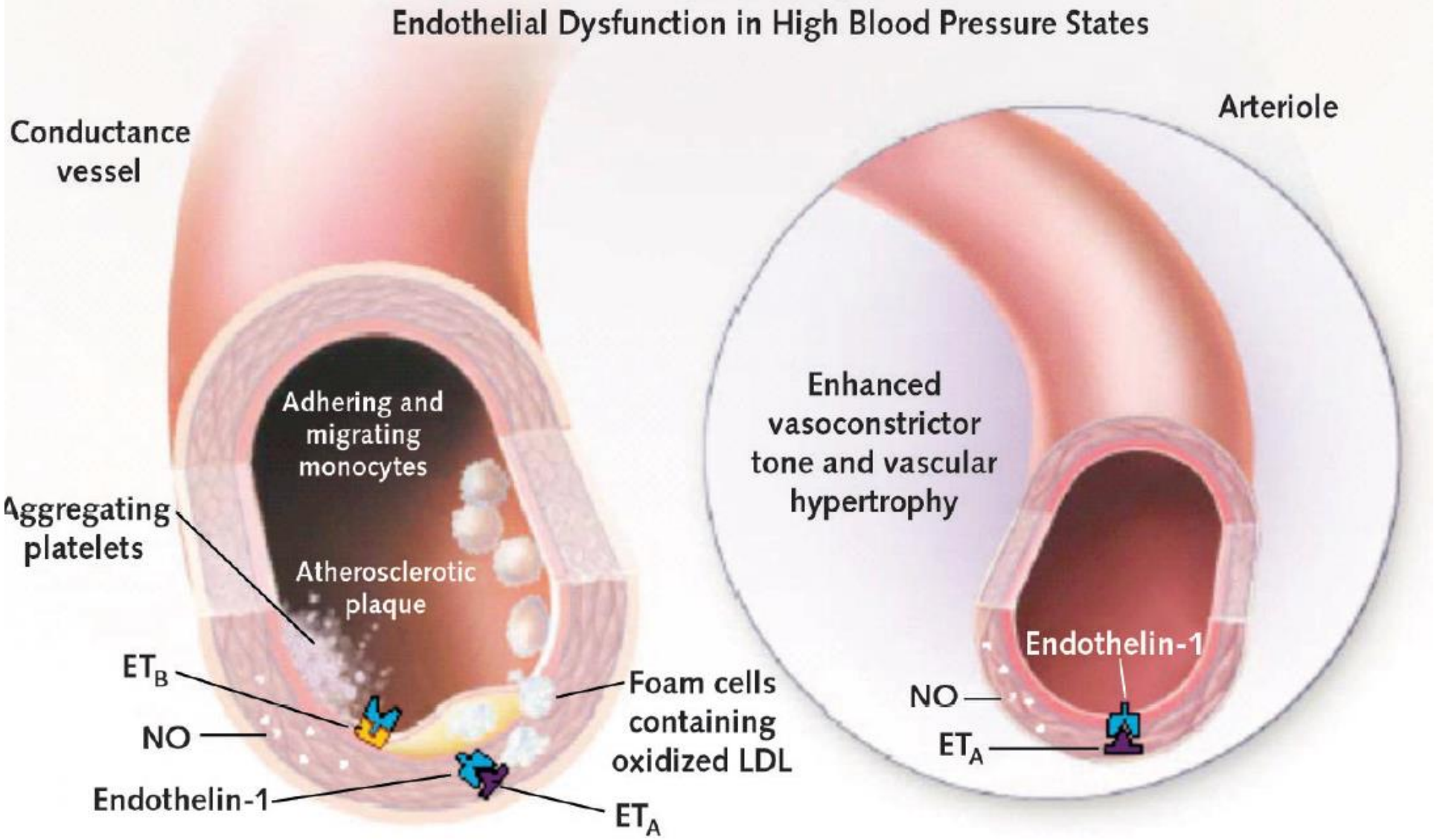
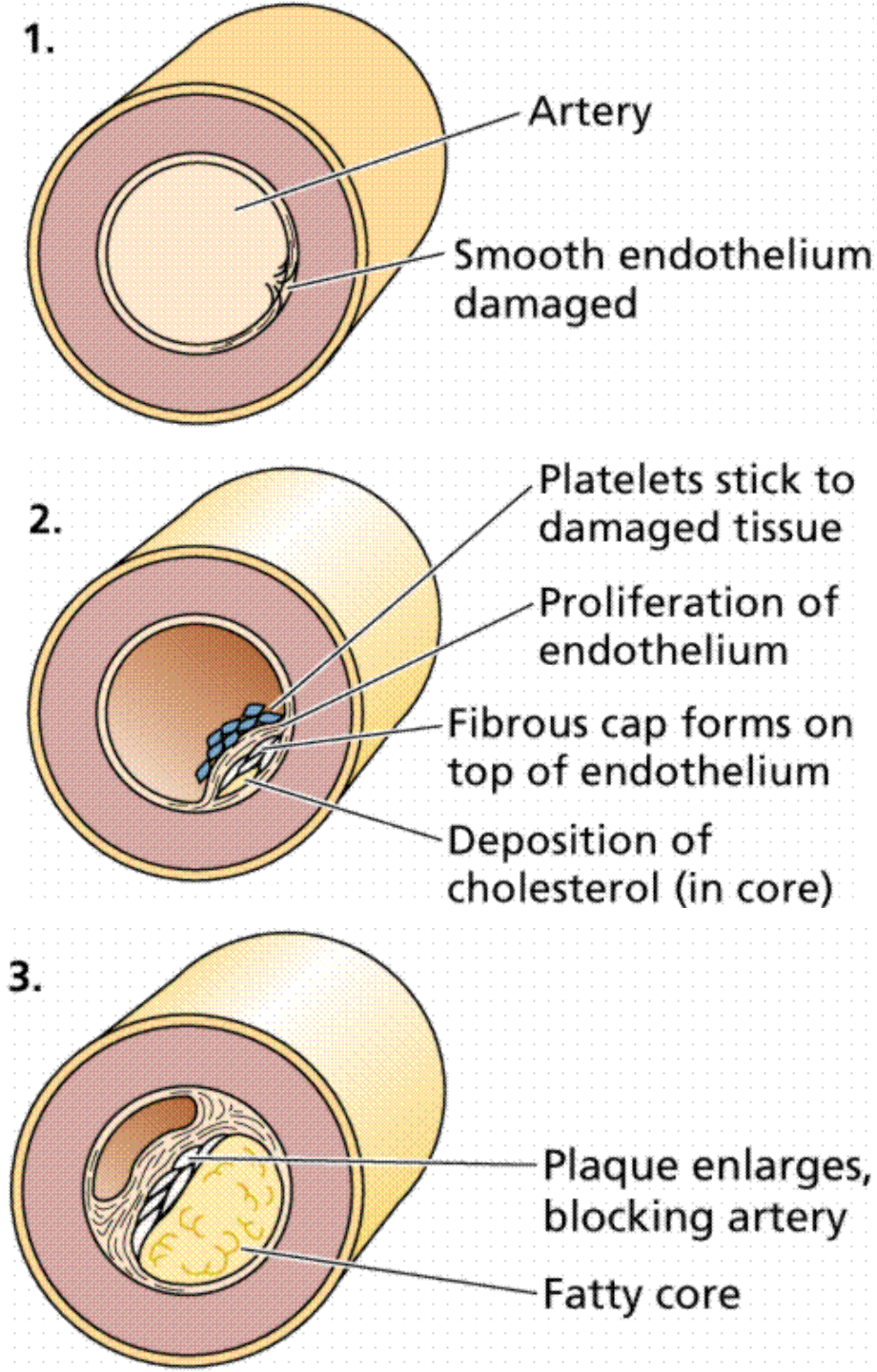


- Pada aliran darah normal trombosit & monosit beredar bebas, oksidasi dari LDL dicegah oleh NO
- Pada saat kontraksi ringan (ok pengaruh endothelin-1 pada ET_A & ET_B akan dihasilkan NO



- Pada hipertensi akan mengurangi produksi NO sehingga akan terjadi oksidasi LDL , adesi trobosit & monosit sehingga terjadi arterio sklerosis.


1.
Artery
Smooth endothelium damaged
2.
Platelets stick to damaged tissue
Proliferation of endothelium
Fibrous cap forms on top of endothelium
Deposition of cholesterol (in core)
3.
Plaque enlarges, blocking artery
Fatty core