RAPID CONTROL OF ARTERIAL PRESSURE

1. NERVOUS SYSTEM
2. BARORECEPTORS (BUFFER SYSTEM)
3. CHEMORECEPTORS
4. ATRIAL AND PULMONARY ARTERY REFLEXES (BAINBRIDGE REFLEX)
5. CNS ISCHEMIC RESPONSE
Autonomic Nervous System

- Sympathetic Nervous System
1- Vasoconstrictor area upper medulla
2- Vasodilator area lower medulla they inhibit vasoconstrictor activity
3- Sensory area posteriolateral portions of medulla and lower pons, receive signal from circulation through vagus and glossopharyngeal and it control activity of both vasoconstrictor and vasodilator areas

Of the vasomotor center
4- Regulation of heart rate through sympathetic and parasympathetic nerves. HR and strength of contraction increase when vasoconstrictor occurs.
5- Continuous partial constriction of the blood vessels called sympathetic vasoconstrictor tone
Control of vasomotor center

Many parts of the brain can excite or inhibit the vasomotor center: Pons, mesencephalon, diencephalon, hypothalamus, parts of cerebral cortex.
BARORECEPTORS
REGULATION OF BP

Baroreceptors
Afferent nerves
Medulla oblongata center
Sympathetic efferents
SA node, ventricles,
Arteriolar smooth muscle
Venous smooth muscle
REGULATION OF BP

Blood pressure

Firing of baroreceptors in carotid arteries and aorta

Sensory neurons

Cardiovascular control center in medulla oblongata

Sympathetic output

Parasympathetic output

Less NE released

α receptor

Arteriolar smooth muscle

Vasodilation

Ventricular myocardium

Force of contraction

SA node

Heart rate

Peripheral resistance

Cardiac output

Blood pressure

Negative feedback

NE: norepinephrine
ACh: acetylcholine
SA: sinoatrial
LONG–TERM REGULATION OF ARTERIAL PRESSURE

1. RENAL BODY FLUID SYSTEM
Response of baroreceptors

1- No response between 0-60 mmHg
2- Respond to rapidly changes than to stationary pressure
3- Changes during changes in body posture
4- Pressure buffer system
5- Resetting in long term regulation of BP
Chemoceptors

1- Stimulated at low pressure about 80mm Hg because decrease blood flow and so the oxygen with increase of CO2 and H

2- They excite the vasomotor center to increase the BP
Atrial reflex

1- Strech or volume reflex which decrease ADH through hypothalamus

2- Baibrige reflex: Stretch receptors transmit afferent signals through vagus nerve to the medulla and the efferent through vagal and sympathetic to increase heart rate and strength of contraction
CNS Ischemic response

1- Cerebral ischemia
2- Emergency pressure control system
3- Become significant at low pressure at 15-20mm Hg
LONG-TERM REGULATION OF ARTERIAL PRESSURE

1. RENAL BODY FLUID SYSTEM
Renal-Body Fluid System for Arterial Pressure Control

- Quantitation of Pressure Diuresis as a Basis for Arterial Pressure Control

Fig. 19.1 Typical renal urinary output curve showing pressure diuresis when the arterial pressure rises above normal
Renal-Body Fluid System for Arterial Pressure Control

- **Arterial Pressure Control---”Near Infinite Feedback Gain”**

Fig. 19.3 Analysis of arterial pressure regulation by equating the “renal output curve” with the “salt and water intake curve”
Fig. 19.4 Two ways in which the arterial pressure can be increased:

A: by shifting the renal output curve in the right hand direction toward a higher pressure level

B: by increasing the intake level of salt and water
Renal-Body Fluid System (cont.)

- Chronic Renal Output Curve is Much Steeper Than the Acute Curve
Renal-Body Fluid System (cont.)

- Failure of Increased Total Peripheral Resistance to Elevate the Long-Term Level of Arterial Pressure if Fluid Intake and Renal Function Do Not Change
Renal-Body Fluid System (cont.)

- Increased Fluid Volume Can Elevate Arterial Pressure by Increasing Cardiac Output or Total Peripheral Resistance
Figure 19.8. The average effect on arterial pressure of drinking 0.9 percent saline solution is removed. (Modified from Langston JB, Guyton AC, Douglas BH: Function in partially nephrectomized dogs. Circ Res 12:508, 1963. By permission of the American Heart Association, Inc.)

Figure 19.9. Progressive changes in important circulatory system variables during the first few weeks of volume-loading hypertension. Note especially the initial increase in cardiac output as the basic cause of the hypertension. Subsequently, the autoregulation mechanism returns the cardiac output almost to normal while simultaneously causing a secondary increase in total peripheral resistance. (Modified from Guyton AC: Arterial Pressure and Hypertension. Philadelphia: WB Saunders, 1980.)
THE RENIN- ANGIOTENSIN SYSTEM
Components of the Renin-Angiotensin System

Fig. 19.10 Renin-angiotensin vasoconstrictor mechanism for arterial pressure control
under the influence of angiotensin II. This shift is caused by both the direct effects of angiotensin II on the kidney and the indirect effect acting through aldosterone secretion, as explained earlier.

Finally, note the two equilibrium points, one for zero angiotensin showing an arterial pressure level of 75 mm Hg, and one for elevated angiotensin showing a pressure level of 115 mm Hg. Therefore, the effect of angiotensin to cause renal retention of salt and water can have a powerful effect in promoting chronic elevation of the arterial pressure.

Role of the Renin-Angiotensin System in Maintaining a Normal Arterial Pressure Despite Large Variations in Salt Intake

One of the most important features of the renin-angiotensin system is its ability to maintain a normal arterial pressure in the face of wide variations in salt intake.
Renin-Angiotensin System

- **Large Variation in Salt Intake**

  - Increased salt intake
  - Increased extracellular volume
  - Increased arterial pressure
  - Decreased renin and angiotensin
  - Decreased renal retention of salt and water
  - Return of extracellular volume almost to normal
  - Return of arterial pressure almost to normal
Figure 19-17. Approximate potency of various arterial pressure control mechanisms at different time intervals after the onset of a disturbance to the arterial pressure. Note especially the infinite gain (\(\infty\)) of the renal body fluid pressure control mechanism that occurs after a few weeks' time. CNS, central nervous system. (Modified from Guyton AC: Arterial Pressure and Hypertension. Philadelphia: WB Saunders, 1980.)

Pressure control mechanisms are almost entirely acute nervous reflexes or other nervous responses. Note in Figure 19-17 the three mechanisms that show responses within seconds. They are (1) the baroreceptor feedback mechanism, (2) the central nervous system ischemic mechanism, and (3) the chemoreceptor mechanism. Not only do these mechanisms begin to react within seconds, but they are also powerful. After any acute fall in pressure, as might be caused by severe hemorrhage, the nervous mechanisms combine to cause (1) constriction of the veins and transfer of blood into the heart, (2) increased heart rate and contractility of the heart to provide greater pumping capacity by the heart, and (3) constriction of most peripheral arterioles to impede flow of blood out of the arteries. All these effects occur almost instantly to help bring blood pressure back into a survival range.