

Physiology

Lec.(4)

Arterial blood pressure

The blood pressure provides the driving force for circulation of blood, it has been defined as the lateral pressure exerted by the blood on the wall of blood vessels

Systolic pressure:-

It is the maximum pressure during systolic phase of the cardiac cycle and in healthy adults it ranges from 110-140mmHg. the average being 120mmHg

Diastolic pressure:-

It is the maximum pressure during ventricular diastole and its normal range is 60-90mmHg. with an average value of 80mmHg in adults

The difference between systolic and diastolic pressure is called pulse pressure and it is about 40mmHg

Mean blood pressure = Diastolic pressure + $\frac{1}{3}$ pulse pressure

Physiological variations

Blood pressure shows certain physiological variations

1-Age-blood pressure is relatively low in children and it increases with age. in old age blood pressure rises, the systolic rise being greater than diastolic and this is due to arteriosclerosis

2-sex-in female blood pressure is lower than males but after menopause this difference is abolished

3-Diurnal variation-during sleep and rest blood pressure is reduced

4-Digestion there is a rise of blood pressure during digestion

5-Posture –change of posture from recumbent to erect position causes a rise of diastolic pressure due to baroreceptor mechanism

6-Exercise –muscular exercise results in a rise of systolic blood pressure due to increased venous return but there is no alteration in the diastolic blood pressure

7-Emotion –emotional excitement activates the sympathetic nervous system and consequently produces a rise of systolic blood pressure

Factors influencing blood pressure

Blood pressure is the resultant of cardiac output and peripheral resistance

$$BP = CO \times PR$$

Nervous control of circulation is exerted almost through autonomic nervous system, sympathetic nerves innervate blood vessels and heart, sympathetic stimulation of small arteries and arterioles increases vascular resistance and decreases the rate of blood flow through the tissues, innervation of the large vessels especially the veins makes it possible for sympathetic stimulation to decrease the volume of the vessels.

Sympathetic fibers also go to the heart and stimulate the activity of the heart, increasing both the rate and strength of pumping, Parasympathetic stimulation decreases heart rate and heart muscle contractility, its main role in controlling the circulation

Is to markedly decrease heart rate and slightly decrease heart muscle contractility

One of the most important functions of sympathetic nervous system is to provide rapid control of arterial pressure by causing vasoconstriction and stimulation of the heart. at the same time that sympathetic activity is increased ,there is a reciprocal Inhibition of parasympathetic vagal signals to the heart that also contribute to a greater heart rate

There are three major changes that take place to increase arterial pressure through stimulation of autonomic nervous system

1-arterioles are constricted, causing increased total peripheral resistance and raising blood pressure

2-the veins and large vessels of the circulation are constricted, displacing blood from peripheral vessels toward the heart and causing the heart to pump with greater force, which also help to raise arterial pressure

3-enhancing heart pumping an important characteristic of nervous control is that it is very rapid, beginning within seconds ,conversely ,sudden inhibition of nervous stimulation can decrease the arterial pressure within seconds

Reflex Mechanisms for Maintaining Normal Arterial Pressure

Aside from the exercise and stress functions of the autonomic nervous system to increase arterial pressure there are other control mechanisms such as.

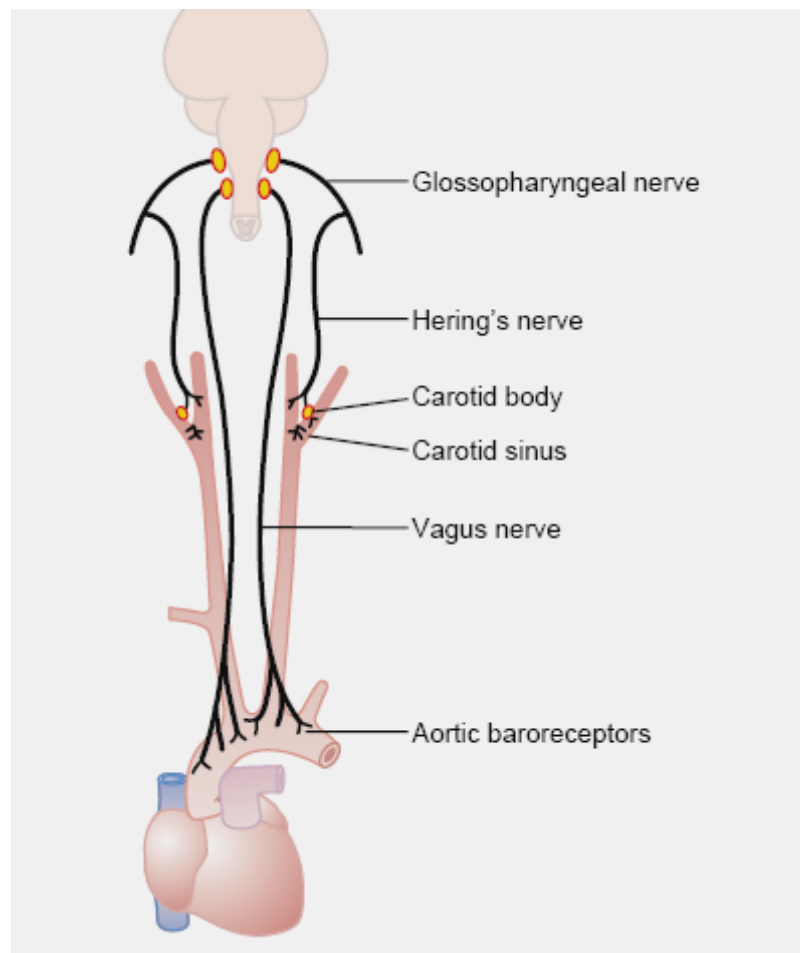
The Baroreceptor Arterial Pressure Control System—Baroreceptor Reflexes

, this reflex is initiated by stretch receptors, called either *baroreceptors* or *press receptors*, located at specific points in the walls of several large systemic arteries. A rise in arterial pressure stretches the baroreceptors and causes them to transmit signals into the central nervous system. "Feedback" signals are then sent back through the autonomic nervous system to the circulation to reduce arterial pressure downward toward the normal level.

Physiologic Anatomy of the Baroreceptors and Their Innervation.

Baroreceptors are spray-type nerve endings that lie in the walls of the arteries; they are stimulated when stretched. A few baroreceptors are located in the wall of almost every large artery of the thoracic and neck regions; but, as shown in Figure baroreceptors are extremely abundant in (1) the wall of each internal carotid artery slightly above the carotid bifurcation, an area known as the *carotid sinus*, and (2) the wall of the aortic arch.

Figure shows that signals from the "carotid baroreceptors" are transmitted through very small *Hering's nerves* to the



glossopharyngeal nerves in the high neck, and then to the *tractus solitarius* in the medullary area of the brain stem. Signals from the aortic baroreceptors" in the arch of the aorta are transmitted through the *vagus nerves* also to the same tractus solitarius

Circulatory Reflex Initiated by the Baroreceptors. After the baroreceptor signals have entered the tractus solitarius of the medulla, secondary signals *inhibit the vasoconstrictor center* of the medulla and *excite the vagal parasympathetic center*. The net effects are (1) *vasodilation* of the veins and arterioles throughout the peripheral circulatory system and (2) *decreased heart rate* and *strength of heart contraction*. Therefore, excitation of the baroreceptors by high pressure in the arteries reflexly *causes the arterial pressure to decrease* because of both a decrease in peripheral resistance and a decrease in cardiac output. Conversely, low pressure has opposite effects, reflexly causing the pressure to rise back toward normal.

Function of the Baroreceptors During Changes in Body

Posture. The ability of the baroreceptors to maintain relatively constant arterial pressure in the upper body is important when a person stands up after having been lying down. Immediately on standing, the arterial pressure in the head and upper part of the body tends to fall, and marked reduction of this pressure could cause loss of consciousness. However, the falling pressure at the baroreceptors elicits an immediate reflex, resulting in strong sympathetic discharge throughout the body. This minimizes the decrease in pressure in the head and upper body.

. Because the baroreceptor system opposes either increases or decreases in arterial pressure, it is called a *pressure buffer system*, and primary purpose of the arterial baroreceptor system is to reduce the minute by minute variation in arterial pressure to about one third that which would occur were the baroreceptor system not present..

Control of Arterial Pressure by the Carotid and Aortic chemoreceptors

The *chemoreceptors* are chemosensitive cells sensitive to oxygen lack, carbon dioxide excess, and hydrogen ion excess. They are (two *carotid bodies*, one of which lies in the bifurcation of each common carotid artery, and usually one to three *aortic bodies* adjacent to the aorta).when blood pressure falls below acritical level,the chemoreceptors become stimulated because diminished blood flow causes decreased oxygen as well as excess buildup of carbon dioxide and hydrogen ions the signals transmitted from chemoreceptors excite the vasomotor center and this elevates the arterial pressure back toward normal

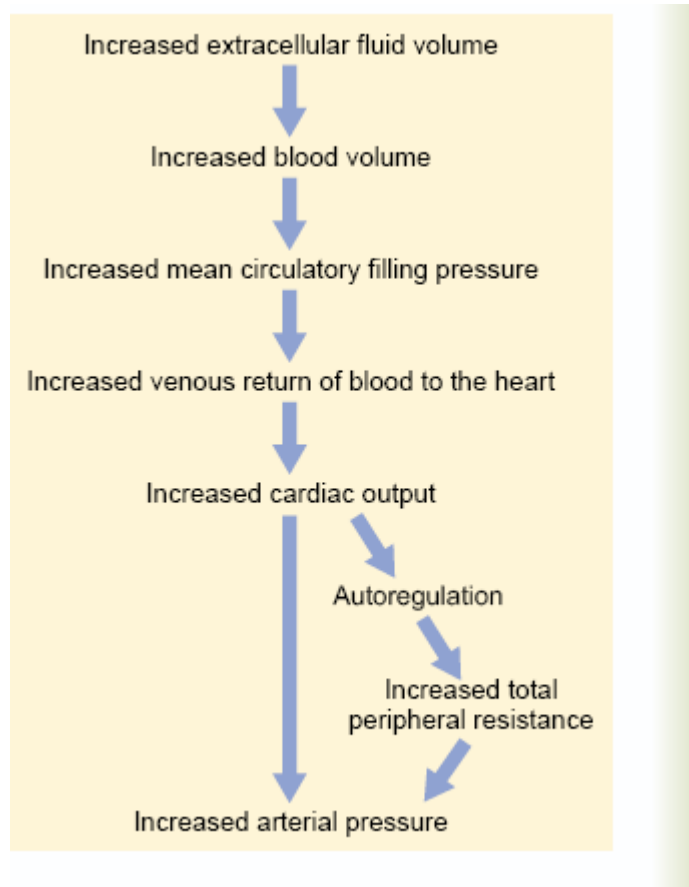
The central nervous system ischemic response

When blood flow to the vasomotor center in the lower brain stem becomes sufficiently decreased to cause cerebral ischemia(I.e.,nutritional deficiency) the neurons of the vasomotor center become strongly excited.when this occurs,systemic arterial pressure often rises as high as the heart can pump.this response is an emergency arterial pressure control system that acts rapidly and powerfully to prevent further decline

Renal–Body Fluid (long term control of arterial pressure)

the Figure . The sequential events are (1) increased extra cellular fluid volume (2) increases the blood volume, which (3) increases the mean circulatory filling pressure, which (4) increases venous return of blood to the heart, which (5) increases cardiac output, which (6) increases arterial pressure.

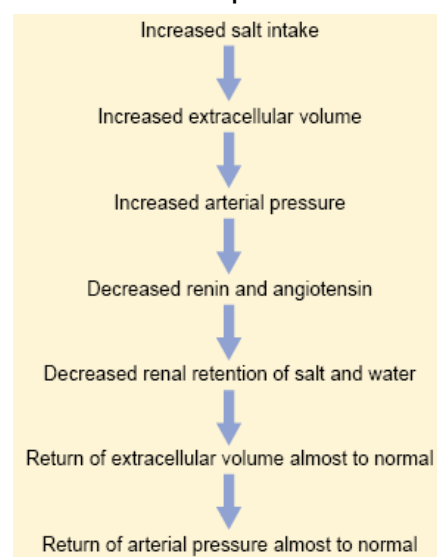
Note especially in this schema the two ways in which an increase in cardiac output can increase the arterial pressure. One of these is the direct effect of increased cardiac output to increase the pressure, and the other is an indirect effect to raise total peripheral vascular resistance through *autoregulation* of blood flow.



Importance of Salt (NaCl) in the Renal–Body Fluid Schema for Arterial Pressure Regulation

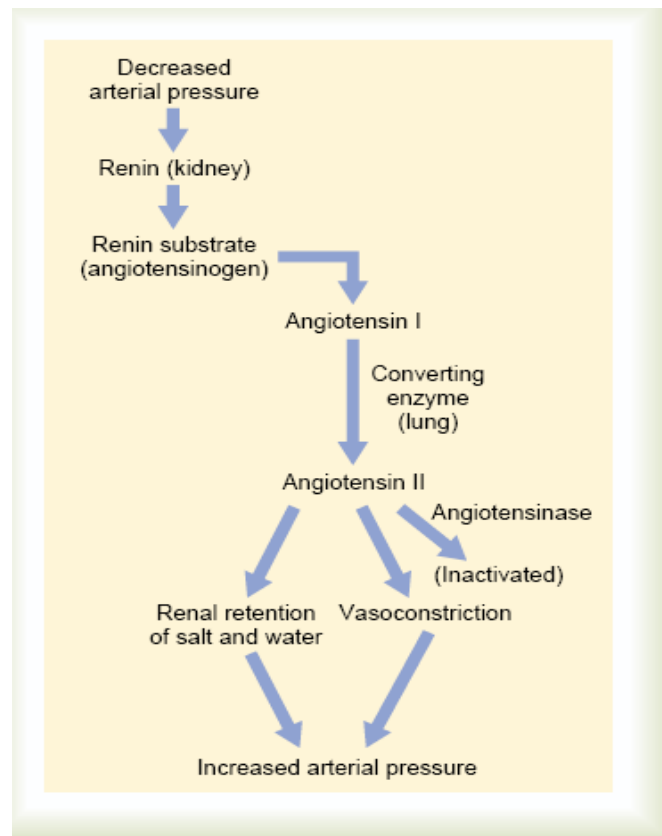
1. When there is excess salt in the extra cellular fluid, the osmolality of the fluid increases, and this in turn stimulates the thirst center in the brain, making the person drink extra amounts of water to return the extra cellular salt concentration to normal. This increases the extra cellular fluid volume.

2. The increase in osmolality caused by the excess salt in the extra cellular fluid also stimulates the –posterior pituitary gland secretory mechanism to secrete increased quantities of *antidiuretic hormone*. The antidiuretic hormone then causes the kidneys to reabsorb greatly increased quantities of water from the renal tubular fluid, thereby diminishing the excreted volume of urine but increasing the extracellular fluid volume. Thus, for these important reasons, the amount of salt that accumulates in the body is the main determinant of the extracellular fluid volume. Because only small increases in extracellular fluid and blood volume can often increase the arterial pressure greatly, accumulation of even a small amount of extra salt in the body can lead to considerable elevation of arterial pressure.



The Renin-Angiotensin System: Its Role in Pressure Control and in Hypertension Aside from the capability of the kidneys to control arterial pressure through changes in extra cellular fluid volume, the kidneys also have another powerful mechanism for controlling pressure. It is the renin angiotensin system.

Renin is a protein enzyme released by the kidneys when the arterial pressure falls too low. In turn, it raises the arterial pressure in several ways, thus helping to correct the initial fall in pressure



Hypertension in Preeclampsia (Toxemia of Pregnancy).

Approximately 5 to 10 per cent of expectant mothers develop a syndrome called *preeclampsia* (also called *toxemia of pregnancy*). One of the manifestations of preeclampsia is hypertension that usually subsides after delivery of the baby. Although the precise causes of preeclampsia are not completely understood, ischemia of the placenta and subsequent release by the placenta of toxic factors are believed to play a role in causing many of the manifestations of this disorder, including hypertension in the mother. Substances released by the ischemic placenta, in turn, cause dysfunction of vascular endothelial cells throughout the body, including the blood vessels of the kidneys. This *endothelial dysfunction decreases release of nitric oxide* and other vasodilator substances, causing vasoconstriction, decreased rate of fluid filtration from the glomeruli into the renal tubules, impaired renal pressure natriuresis, and development of hypertension. Another pathological abnormality that may contribute to hypertension in preeclampsia is thickening of the kidney glomerular membranes (perhaps caused by an autoimmune process), which also reduces the rate of glomerular fluid filtration. For obvious

reasons, the arterial pressure level required to cause normal formation of urine becomes elevated, and the long-term level of arterial pressure becomes correspondingly elevated. These patients are especially prone to extra degrees of hypertension when they have excess salt intake.

Neurogenic Hypertension. *Acute neurogenic hypertension*

can be caused by strong *stimulation of the sympathetic nervous system*. For instance, when a person becomes excited for any reason or at times during states of anxiety, the sympathetic system becomes excessively stimulated, peripheral vasoconstriction occurs everywhere in the body, and *acute* hypertension ensues.

"Primary (Essential) Hypertension"

About 90 to 95 per cent of all people who have hypertension are said to have "primary hypertension," also widely known as "essential hypertension" by many clinicians. These terms mean simply that *the hypertension is of unknown origin*, in contrast to those forms of hypertension that are *secondary* to known causes, such as renal artery stenosis. In some patients with primary hypertension, there is a strong hereditary tendency

In most patients, *excess weight gain* and *sedentary lifestyle* appear to play a major role in causing hypertension. The majority of patients with hypertension are overweight, and studies of different populations suggest that excess weight gain and obesity may account for as much as 65 to 70 percent of the risk for developing primary hypertension. Clinical studies have clearly shown the value of weight loss for reducing blood pressure in most patients with hypertension. In fact, new clinical guidelines for treating hypertension recommend increased physical activity and weight loss as a first step in treating most patients with hypertension. Some of the characteristics of primary hypertension caused by excess weight gain and obesity include:

1. Cardiac output is increased due, in part, to the additional blood flow required for the extra adipose tissue. However, blood flow in the heart, kidneys, gastrointestinal tract, and skeletal muscle also increases with weight gain due to increased metabolic rate and growth of the organs and tissues in response to their increased metabolic demand

2. Sympathetic nerve activity, especially in the kidneys, is increased in overweight patients. The causes of increased sympathetic activity in obesity are not fully understood, but recent studies suggest that hormones, such as *leptin*, released from fat cells may directly stimulate multiple regions of the hypothalamus, which, in turn, have an excitatory influence on the vasomotor centers of the brain medulla.

3. Angiotensin II and aldosterone levels are increased two- to threefold in many obese patients. This may be caused partly by increased sympathetic nerve stimulation, which increases renin release by the **kidneys** and therefore formation of angiotensin II, which, in turn, stimulates the adrenal gland to secrete aldosterone.

4. The renal-pressure natriuresis mechanism is impaired, and the kidneys will not excrete adequate amounts of salt and water unless

the arterial pressure is high or unless kidney function is somehow improved.