# Introduction to THE REGULATION OF BLOOD PRESSURE AND HEART RATE

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By

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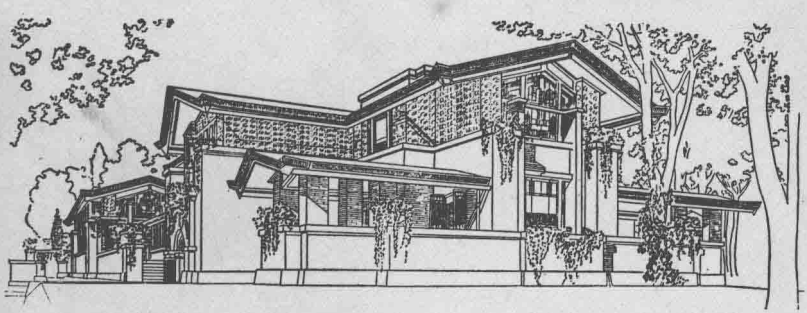

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*Introduction to*

# THE REGULATION OF BLOOD PRESSURE AND HEART RATE

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# FOREWORD

FOR many years physiologists have been occupied with the study of the mechanisms which regulate the arterial blood pressure and the heart rate. A precise and detailed knowledge of the physiological regulation of arterial pressure is indeed the key, not only to the physiology of normal circulation, but perhaps also to the pathogenesis of chronic arterial hypertension, which is undoubtedly one of the most frequent and dangerous clinical abnormalities.

*Introduction to*

# THE REGULATION OF BLOOD PRESSURE AND HEART RATE

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*Chapter One*

# PHYSIOLOGICAL CONTROL OF ARTERIAL BLOOD PRESSURE

THE arterial pressure is the lateral pressure exerted by the blood on the walls of the large arteries.

Normal Blood Pressure: The arterial pressure in the large arteries is characterized by a systolic pressure, an increase of pressure induced by the systolic contraction of the left heart ventricle, and by a diastolic pressure, a drop of arterial pressure occurring during the diastolic arrest of the heart between two contractions. The mean arterial pressure represents the average between the systolic rise and the diastolic fall of pressure (see Fig. 1).

These arterial pressures are not the same in different age groups. The systolic pressure ranges from 75 to 90 mm. mercury during infancy, from 90 to 110 mm. in childhood, and 100 to 120 mm. about puberty. In adults, the average normal systolic pressure is usually stated to be 125 to 130 mm. In old people, it is between 140 and 150 mm.

The diastolic pressure ranges around 50 mm. mercury during the first five years of life, and after that remains fairly constant at 60 mm. to 80 mm.

The systolic pressure is mainly determined by the cardiac output, the circulating blood volume, the blood viscosity, the elasticity of the large arteries, and the peripheral resistance, i.e. the tone or degree of contraction of the small blood vessels: the arterioles and to a lesser extent the capillaries.

The diastolic blood pressure is chiefly determined by the peripheral vascular resistance and the elastic contraction of the large arteries during the heart diastole.

The systolic and diastolic pressures as well as the

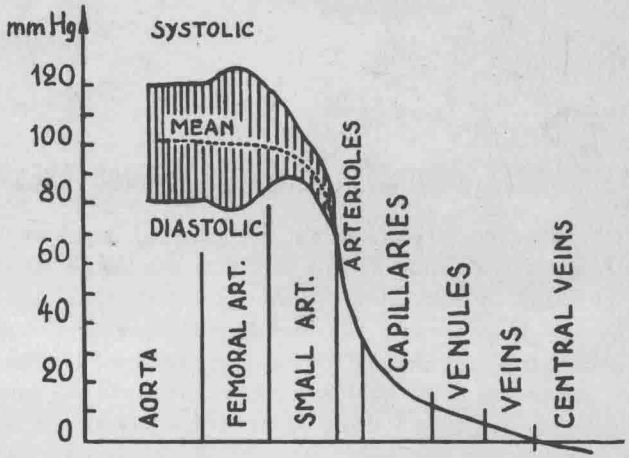


**Fig. 1. Diagram of Pressure Areas of the Circulation. Systolic, diastolic and mean blood pressures in millimeters of mercury.**

mean pressure, vary in the different parts of the vascular bed (Fig. 1). These pressures are highest in the aorta, drop somewhat in the more peripheral branches of the large arteries, are lower in the arterioles, and still lower in the capillaries. In these small vessels, fluctuations of systolic and diastolic pressure are nearly absent and the blood pressure and flow are fairly constant. In the veins the pressure is still lower and may even be negative in the larger channels near the heart, especially in diastole and inspiration. The following table gives the average height of the mean (not systolic) pressure at different parts of the vascular system in a young adult man, in the horizontal position:

| | |
|---|---|
| Large arteries | 65 - 110 mm. Mercury |
| Medium arteries | 85 mm. |
| Arterioles | 30 - 50 mm. |
| Capillaries | 10 - 30 mm. |
| Small veins (arm) | 9 mm. |
| Portal vein | 10 mm. |
| Large veins near to the heart. | 0 - 8 mm. |

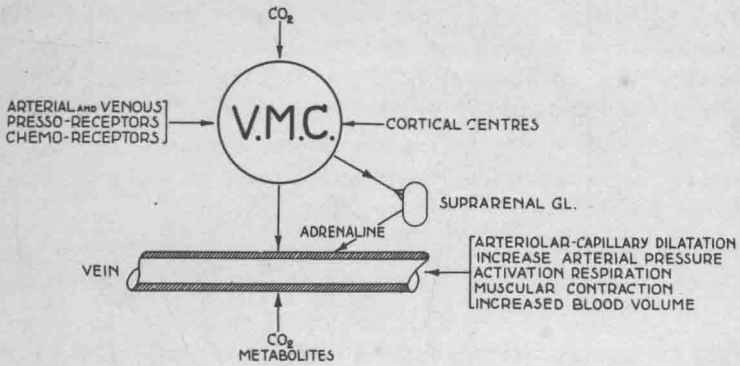


**Fig. 2. Regulation of Venous Tone and Pressure. V.M.C.: Venous vasomotor centers.**

To a great extent the blood pressure in the large arteries is the product of the cardiac output and the peripheral resistance. The circulating blood volume, the viscosity of the blood, and the elasticity of the large arteries are fairly constant factors, and thus, under normal conditions, influence the arterial pressure only to a minor degree.

The term cardiac output means the output of blood per ventricle of the heart per minute. The outputs of the two ventricles under physiological conditions are exactly the same. The cardiac output is determined chiefly by the following factors:

A. The venous return.
B. The force of the heart beat.
C. The rate of the heart.
D. The arterial pressure.

A. The venous return, i.e., the amount of blood streaming to the right side of the heart, depends mainly on the following factors:

1. The tone of the wall of the veins, which is controlled

by nerve impulses (venomotor nerves connected with the vasomotor centers) and by local chemical factors such as adrenaline, the $CO_2$ - and $O_2$ - content of the blood, and the metabolites (Fig. 2).

2. The tone and contraction of the striated muscles, which squeeze upon their contained veins and thus drive the blood onward toward the heart.

3. The negative pressure in the pleural cavity induced by each inspiration, which aspirates the blood from the veins toward the heart.

4. The contractions of the diaphragm, which at each inspiration raise the intra-abdominal pressure and thus squeeze the venous blood out of the splanchnic area toward the heart.

5. The tone of the arterioles, capillaries and veins, which if contracted, retain a smaller amount of blood, or if widely dilated, retain a larger proportion of the blood volume.

6. The gravity assists the return of the venous blood from organs above the heart level, but hinders the venous return from dependent regions.

B. The force of the heart beat depends chiefly on:

1. The initial length of the heart muscle-fibers at the beginning of each systole. It has been found by Starling (1) that distension of the heart during diastole by increased venous return enhances the force of the systolic contraction and leads to the ejection of a larger volume of blood.

2. The duration of the diastolic pause of the heart. A long diastolic pause, by favoring recovery from the effects of the previous systolic contraction and by increasing the diastolic venous filling of the heart, increases the force and output of the subsequent systolic beat.

3. The blood flow in the coronary vessels of the heart.

The nutrition and oxygen supply of the heart depend mainly on the pressure under which the blood is forced into the coronary arteries, on the tone of the coronary arterioles and capillaries, and on the oxygen supply. The pressure of the arterial blood forced into the coronary vessels depends on the aortic blood pressure. The tone of the coronary vessels is influenced and regulated by the nerve supply and by various chemical factors. The vagus nerve conveys mainly vasoconstrictor, and the sympathetic, vasodilator fibers to the coronary arteries. The $O_2$ - and $CO_2$ - tension of the arterial blood, the hydrogen ion concentration, adrenaline, and certain metabolites also adjust the coronary vascular tone and blood supply to the needs of the heart muscle.

C. The rate of the heart is influenced and controlled by several factors:

1. The nerve supply of the heart: the vagus and sympathetic nerves.

a) The vagus nerves arise in the dorsal nucleus of the vagus, the vagal center, and run in the cervical vagus trunks to the vagal synapses in the heart, and from there to the sino-auricular and auriculo-ventricular nodes in the heart. Stimulation of the vagus induces an inhibition of the several basic functions of the heart; thus it provokes a slowing of the heart beat by decreasing the rate of impulse formation in the sino-auricular node, a marked diminution of the force of contraction of the auricles and ventricles, a reduction of the refractory period and thus of the length of the systole, and a decrease of the conductivity of the impulses through the different parts of the heart which in some conditions may progress to complete auriculo-ventricular block.

b) The sympathetic nerves to the heart arise in the intermediolateral cell column of the upper thoracic

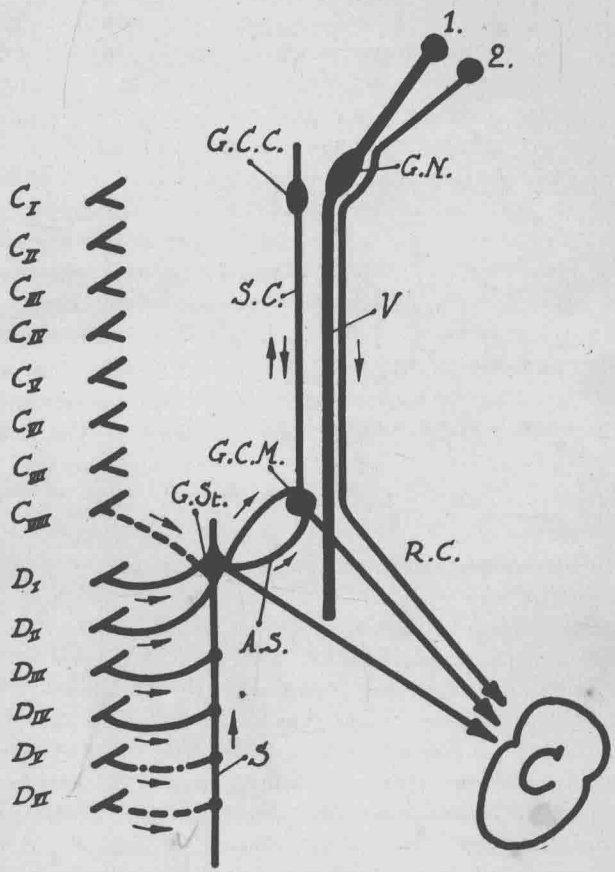


Fig. 3. Cardio-Accelerator Innervation in Dog.
1: vagus center (cardio-inhibitory innervation).
2: center of cardio-accelerator fibres running in the vagus trunk to the heart.
G.C.C.: superior cervical sympathetic ganglion.
G.N.: ganglion nodosum of vagus nerve.
S.C.; cervical sympathetic nerve.
G.C.M.: middle cervical sympathetic ganglion.
G.ST.: ganglion stellatum.
A.S.: ansa of Vieussens.
S.: sympathetic ganglionnic chain (thorax).
R.C.: rami cardiaci.
C.: heart.

segments and enter the sympathetic chain by way of the ventral spinal roots and white rami. Their sites of synapse and subsequent course vary considerably in different animal species and in man. In the dog the preganglionic fibers ascend through the chain to the stellate and middle cervical ganglia where the majority of the sympathetic synapses are located. Postganglionic fibers pass to the heart over the inferior and middle cardiac nerves as shown in Fig. 3 (2).

In man the distribution is more complex. Some fibers run in the cervical sympathetic trunk as far rostrally as the superior ganglion to give rise to a superior cardiac nerve in addition to the middle and inferior nerves described above. Other preganglionic fibers synapse in the upper thoracic chain ganglia and pass directly to the heart as a series of fine mediastinal filaments. At least in the dog and monkey, fibers functionally allied to those of the sympathetic system take origin in the bulb and descend in the vagus nerves. Stimulation of these cardiac sympathetic nerves induces an increase in the rate, in the conductivity and excitability, and in the force of contraction of the heart.

Under ordinary circumstances the vagus nerves exert a more powerful influence on the heart than do the sympathetic nerves. Thus it has been shown experimentally by Samaan (3) that moderate vagal stimulation provokes a bradycardia which masks strong cardio-accelerator stimulation. Futhermore stimulation of the cardio-accelerator nerves as well as small doses of adrenaline make the heart even more sensitive to vagal impulses. However, strong and prolonged stimulation suppresses the cardio-inhibitory action of the vagus (vagus escape). These factors may have an influence on the effects of direct and reflex stimulation of the antagonistic cardiac nerves.

The normal heart rate in adult men is about 70 beats per min. It varies considerably with age. The

following table presents the average resting heart rate in man at different ages.

| Age in years | Heart rate per minute |
|---|---|
| Foetus | 150 |
| 0 | 135 |
| 5 | 105 |
| 10 | 90 |
| 15 | 80 |
| 20 | 75 |
| 25-40 | 70 |
| 80 | 75 |

2. The chemical and physical factors chiefly affecting the heart rate are adrenaline, nor-adrenaline, thyroxine, the $O_2$ - and $CO_2$ - content, and temperature of the blood (Fig. 25).

D. The arterial pressure influences the output of the heart indirectly through its relationship to venous pressure and venous return. Thus lowered arterial pressure decreases venous pressure, in consequence of which venous return and cardiac output decrease.

The peripheral resistance is induced and controlled to a large extent by the variably contractile arterioles, especially those of the abdominal splanchnic area, and to a lesser extent by the capillaries. Since the flow of blood is ordinarily much faster in the arterioles than in the capillaries, the major resistance to flow resides in the former. However if the arterioles dilate while the capillaries contract, the peripheral resistance may then reside largely in the latter. Other factors such as viscosity also induce a resistance to the peripheral flow of blood. The degree of contraction or tone of the arterioles and probably also of the capillaries and small veins is maintained and controlled by several factors. Of prime importance are the nervous impulses which arise in the vasomotor