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Appendix
Physiological Background: Cardiac Control

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Abstract. This section provides an overview of the mechanisms existing in cardiac control in order to facilitate the understanding of the computer model described in this paper.

Autonomic Nervous System

Sympathetic system

Sympathetic nerve fibres innervating the heart are almost evenly distributed over all parts of this organ. Furthermore, the sympathetic nervous system can exert its influence on the heart by way of the catecholamines, adrenaline and noradrenaline, circulating in the blood. Sympathetic stimulation markedly increases the activity of the heart. Three effects can be distinguished:
- an increase in heart rate (positive chronotropic effect),
- an enhancement of the contractile force of the heart muscle (positive inotropic effect), increasing the capability of the heart to pump larger volumes of blood, and
- an acceleration of atrioventricular conduction (positive dromotropic effect).

Parasympathetic system

Although the parasympathetic nervous system is exceedingly important for many other autonomic functions of the body, it plays only a minor role in regulation of the circulation. Its only really important circulatory effect is the control of heart rate by way of parasympathetic fibres carried to the heart in the vagus nerves. Principally, parasympathetic stimulation causes a marked decrease in heart rate (negative chronotropic effect) and a slight decrease in heart muscle contractility (negative inotropic effect). Furthermore, the velocity of conduction in the atrioventricular node is decreased by parasympathetic stimulation (negative dromotropic effect).

One of the most important functions of nervous control of the circulation is its capability to cause rapid increases in arterial pressure and heart rate, beginning and accomplishing the circulatory adaptation within seconds. For this purpose, the entire vasoconstrictor and cardioaccelerator functions of the sympathetic nervous system are stimulated as a unit. At the same time, there is reciprocal inhibition of the parasympathetic vagal inhibitory signals to the heart.

Muscle Exercise

During heavy exercise, the muscles require greatly increased blood flow. Part of this increase results from local vasodilatation of the muscle vasculature caused by increased metabolism of the muscle cells. Additional increase results from a simultaneous elevation of arterial pressure and heart rate (leading to increased cardiac output) during the exercise. The latter mechanism is believed to result mainly from the following effect: at the same time that the motor areas of the nervous system become activated to cause exercise, most of the reticular activation system of the brain stem is also activated, which includes greatly increased stimulation of the vasoconstrictor and cardioacceleratory areas of the vasomotor centre. These raise the arterial pressure and the heart rate instantaneously to keep pace with the increase in muscle activity.

Reflex Mechanisms in Circulatory Control

Aside from the exercise and stress functions of the autonomic nervous system in circulation control, there are multiple subconscious special nervous control mechanisms that operate all the time to maintain the circulation parameters at or near their normal operating level. Almost all of them are negative feedback reflex mechanisms.

Arterial Baroreceptor Control System

Basically, this reflex is initiated by stretch receptors, called either baroreceptors or pressoreceptors, which are located in the wall of each internal carotid artery at the carotid sinus, and in the wall of the aortic arch. A rise in pressure stretches the baroreceptors and causes them to transmit signals into the central nervous system. After the baroreceptor signals have entered the medulla, secondary signals eventually inhibit the vasoconstrictor centre of the medulla and excite the vagal centre. The net effects are vasodilatation of the veins and arterioles throughout the peripheral circulatory system and decreased heart rate and strength of heart contraction. Therefore, excitation of the baroreceptors by 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. The carotid sinus baroreceptors are not stimulated by pressures between 0 and 60 mmHg, but above 60 mmHg they respond progressively more rapidly and reach a maximum at about 180 mmHg. The aortic baroreceptors operate, in general, at pressure levels about 30 mmHg higher. In the normal operating range of arterial pressure, around 100 mmHg, even a slight change in pressure causes a strong change in autonomic reflexes to readjust the arterial pressure back toward normal. Thus, the baroreceptor feedback mechanism works most effectively in the pressure range where it is most needed. The baroreceptors respond extremely rapidly to changes in arterial pressure; in fact, the rate of impulse fining increases during systole and decreases again during diastole. Furthermore, the baroreceptors respond much more to a rapidly changing pressure then to a stationary pressure.

"Resetting" of the baroreceptors

The baroreceptor control system is probably of little or no importance in long-term regulation of arterial pressure for a simple reason: The baroreceptors themselves reset in 1 to 2 days to whatever pressure level they are exposed. Prolonged regulation of arterial pressure requires other control systems, principally the renal-body fluid-pressure control system along with its associated hormonal mechanisms.

Control of arterial pressure by the carotid and aortic chemoreceptor

Closely associated with the baroreceptor system is a chemoreceptor reflex that operates in much the same way as the baroreceptor reflex except that chemoreceptors, instead of stretch receptors initiate the response. These receptors are sensitive to O2 lack, CO2 excess, or hydrogen ion excess. They are located in several small organs 1 to 2 millimetres in size: two carotid bodies, and several aortic bodies adjacent to the aorta. Each of these bodies is supplied with an abundant blood flow through a small nutrient artery, so that the chemoreceptors are always in close contact with the arterial blood. Whenever the arterial pressure falls below a critical level, the chemoreceptors become stimulated because of diminished blood flow to the bodies and therefore diminished availability of O2 as well as excess build-up CO2 and hydrogen ions that are not removed by the slow flow of blood. The signals transmitted from the chemoreceptors to the vasomotor centre excite the vasomotor centre, and this elevates the arterial pressure. The chemoreceptors are not stimulated strongly by pressure changes until the arterial pressure falls below 80 mmHg. Therefore, it is at the lower pressures that this reflex becomes especially important to help prevent still further fall in pressure.

Central nervous system (CNS) ischemic response

When blood flow to the vasomotor centre in the lower brain stem becomes decreased enough to cause nutritional deficiency, that is, to cause cerebral ischemia, the neurones in the vasomotor centre itself respond directly to the ischemia and become strongly excited. When this occurs, the systemic arterial pressure often rises to a level as high as the heart can possibly pump. This effect is believed to be caused by failure of the slowly flowing blood to carry CO2 away from the vasomotor centre; the local concentration of CO2 increases greatly and has an extremely potent effect in stimulating the sympathetic nervous control areas in the medulla: It is possible that other factors, such as the build-up of lactic acid and other acidic substances, also contribute to he marked stimulation of the vasomotor centre. The degree of sympathetic vasoconstriction caused by intense cerebral ischemia is often so great that some of the peripheral vessels become totally or almost totally occluded. The kidneys, for instance, often entirely cease their production of urine because of arteriolar constriction in response to the sympathetic discharge. Therefore, the CNS ischemic response is one of the most powerful of the sympathetic vasoconstrictor system. Despite the powerful nature of the CNS ischemic response, it does not become significant until the arterial pressure falls far below normal, down to 60 mmHg and below, reaching its greatest degree of stimulation at a pressure of 15 to 20 mmHg. Therefore, it operates principally as an emergency arterial pressure control system that acts rapidly and powerfully to prevent further decrease in arterial pressure whenever blood flow to the brain decreases dangerously close to the lethal level.

Atrial reflex control of heart rate (Bainbridge reflex)

An increase in atrial pressure also causes an increase in heart rate, sometimes increasing the heart rate as much as 75%. A small part of this increase is caused by a direct effect of the increased atrial volume to stretch the sinus node; such direct stretch can increase the heart rate as much as 15%. An additional 40 to 60% increase in heart rate is caused by a reflex called the Bainbridge reflex. The stretch receptors of the atria that elicit the Bainbridge reflex transmit their afferent signals through the vagus nerves to the medulla of the brain. Then,

efferent signals are transmitted back through both the vagal and the sympathetic nerves to increase the heart's rate and, presumably , strength of contraction. Thus, this reflex helps prevent damming of blood in the veins, atria and pulmonary circulation.