Regulation of Blood Vessels

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The cardiovascular system serves the biologic organism as a transportation and mixing system. The demand by the organism for this service varies in different tissues and at different times while the volume of blood in the organism tends to remain constant. The cardiovascular system fulfills its function, therefore, by varying the rate and the distribution of blood flow. Blood vessels play a major role in controlling the rate of flow of blood by affecting the resistance to flow, and they possess the entire responsibility for the distribution of the blood and its flow pathways. The primary aspect of blood vessels that affects the flow and distribution of blood is their caliber, i.e., their internal radius. This discussion will deal primarily with the factors that control the caliber of blood vessels and with the role of these factors in the regulation of the circulation of blood.

The concept of control of the caliber of blood vessels by the nervous system is relatively new. Moreover, the mechanisms by which the caliber of blood vessels vary as a result of nervous influences are only beginning to be understood and evaluated. Indeed, the youthfulness of many of our concepts of physiology and medicine is so striking that I cannot avoid mentioning it.

As late as 1831, Dr. E. Weber, a well-known German physician, said, "I believe that flushing and pallor could be explained if it could be taken for granted that the caliber of arteries becomes larger and smaller under nervous influence." Only slowly has evidence begun to accrue during the past century that supports the idea of nervous control of the heart and blood vessels. It was not until after the Civil War that Ludwig and Cyon noted that stimulation of the central end of the aortic nerve causes bradycardia and hypotension, and it was 65 years later, in 1927, that Hering described similar effects following stimulation of the carotid sinus nerve. Subsequently, interest in the reflex regulation of the cardiovascular system expanded rapidly.

In the 1930's Heymans and his co-workers extended Hering's observations with a series of experiments that established that receptor areas lying outside of the central nervous system are sensitive to the behavior of the cardiovascular system and reflexly, through the central nervous system, control the behavior of the cardiovascular system in such a way that the behavior seemed to be regulated.

Also in the 1930's, Bronk became interested in these receptors and, with his colleagues, obtained records of the electrical activity of the nerve fibers arising from the carotid sinus, aortic receptors, and mesenteric Pacinian corpuscles. He stated, "It is their function to signal changes in blood pressure, and it is the function of their afferent fibers to provide a means of communication between these receptors and the central nervous system. Thus the centers are kept informed regarding the pressure within the blood vessels." Heymans began a lecture with, "For many years physiologists have been and are still occupied with the study of the mechanisms which regulate blood pressure. A precise and detailed knowledge of the regulation of pressure is the key, not only to the physiology of the normal circulation, but perhaps also to the pathogenesis of chronic arterial hypertension."

The concept developed quite naturally and has subsequently been widely held that arterial blood pressure is the function that is regulated through these receptors. This regulation of blood pressure is generally considered as an established principle of cardiovascular

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Aided by grants from the National Heart Institute, U. S. Public Health Service, and the Office of Naval Research.
Electrical impulses from a single nerve fiber of a carotid sinus receptor together with a tracing of intraarterial blood pressure. (Reproduced by permission of the publisher and the authors from Bronk and Stella (1932).)

physiology. Figure 1 illustrates the well-known relationship between arterial blood pressure and receptor activity that Bronk and his co-workers found and that did much to establish the concept of blood pressure regulation.

Figure 2 is a schematic diagram, using the definitions stated in the introduction, which illustrates the concept of arterial blood pressure regulation. On the right of the diagram are the mechanisms that control blood pressure. Within each black box lies a mechanism that is considered to be a prime variable. The impulse traffic of the efferent nerves (Ie) arises from the central nervous system and commands, by mechanisms implicit in the black boxes, changes in peripheral resistance, stroke volume, and pulse rate. Thus, the product of stroke volume and pulse rate gives cardiac output, and the product of cardiac output and resistance gives blood pressure. These are among the elementary relationships that have been taught to every medical student for many years. In the top center of the diagram is the black box indicating that intrarterial pressure causes a response in the afferent sensory mechanism ("pressure receptors") such that impulses of the afferent nerves travel to the central nervous system, thus transmitting information about pressure (Ip). On the left, the integrating mechanism, located within the central nervous system, is supplied with information regarding the level of blood pressure and in turn sends out efferent information in order to maintain the blood pressure at some ideal value—the "normal" blood pressure. Experimental work has established the apparent existence of each of these black boxes with the exception of that related to the establishment of an "ideal value" for pressure. Also, the organization of the black boxes into a closed loop that is linked together as shown seems reasonable. Thus, the concept of arterial blood pressure regulation seems to be established.

Many of the workers who have developed and perpetuated the concept of arterial blood pressure regulation realized that the so-called pressure receptors were not really stimulated by pressure directly, but rather by the stretching of the vessel wall as a consequence of intravascular pressure. They apparently thought, however, that vessel-wall stretch and intravascular pressure were related in such a manner that the 2 variables are equivalent. Furthermore, the concept of arterial blood pressure regulation may be considered to be a more general or important function, and a more easily measured function than vessel-wall stretch.

In the 1940's, some experiments directed attention specifically to the relationship of the vessel-wall stretch and the nervous output of receptors lying within the vessel wall. Hanus, Kreuziger, and Asteroth, in Germany, encased the carotid sinus with a rigid cement, thus preventing stretch of the carotid sinus when the blood pressure was increased, and thereby eliminated the reflex response to the increased pressure. Also, Palme, working in Germany in the 1930's, applied epinephrine directly to the wall of the sinus and noted a marked fall in systemic blood pressure. He also stimulated efferent fibers innervating the carotid sinus and obtained similar results. Palme concluded that the reflex effects obtained by him were due to direct stimulation of the receptors by the efferent nerves, although it might have been concluded that the efferent nerve activity altered the properties of the vessel wall and that this alteration, in turn, affected the stretch of the receptors for a given pressure.

Heymans and Landgren and their respec-
tive colleagues, and many others, have since applied a great variety of substances to the wall of the carotid sinus and the general conclusion has developed that the reflex changes that occur, but that are not due to blood pressure variation, are due to changes in the properties of the vessel wall, i.e., that the wall becomes more or less distensible. Heymans published a paper in 1951 entitled, "New Aspects of Blood Pressure Regulation" and in the summary he stated, "These experiments show that the state of contraction and thus the resistance to stretch of the arterial wall where the receptors are located are the primary factors effecting the receptors which regulate systemic pressure."

During this period also many other characteristics of the receptors were discovered. For example, it has been found that the traffic of nerve impulses arising in these receptors is a function of the rate of change of pressure as well as of the pressure itself. This implies that the pulsatile nature of arterial pressure is an essential aspect of normal cardiovascular regulation. This may be of importance in the use of extracorporeal bypass-pumps to maintain the circulation during heart surgery, since the normal pulsatile nature of the arterial pressure is reduced or absent.

Our definition of regulation states that there must be some mechanism for determining the magnitude of the function that is being regulated. The concept of blood pressure regulation can only be valid if there are receptors that measure pressure; yet it has been shown that the activity of these receptors is related to vessel-wall stretch and to the rate of change of stretch rather than to pressure and its rate of change. This distinction between stretch receptors and pressure receptors...
Netnogams from multifiber preparation of carotid sinus nerve of (A) normotensive and (B) hypertensive dog during standard endosinus pressure stimulus. It can be noted that the activity of the receptors of the normal dog appears to be significantly less than that of the hypertensive dog although the pressure within the 2 sinuses is being made to vary in the same way. Breaks in top reference line at 1-second intervals. (Reproduced by permission of the publisher and the authors from McCubbin, Green, and Page (1956).?)

Figure 3

is of importance if the factors that relate vessel stretch and blood pressure are also variables under physiologic and pathologic circumstances.

The relationship between intravascular pressure and the circumference of the vessel wall is determined by the mechanical properties of the blood vessel wall. These properties are commonly referred to as vessel-wall tone or stiffness. It has been shown, as mentioned above, that these properties can be altered by placing vasoactive substances, such as epinephrine, acetylcholine, and norepinephrine on the carotid sinus wall and by stimulating the efferent nerves supplying the carotid sinus. The obvious question is, however, do these mechanical properties vary under natural circumstances? Certainly there is evidence that arteries become stiffer with age and in certain diseases. It is also known that most blood vessels of the body constrict and dilate under a variety of physiologic conditions, and it is not unreasonable to suppose that the receptor-containing vessels share in these changes. Moreover, it is known that in hypertension the properties of arteries (e.g., water and electrolyte content) change, there is vascular constriction, and the constituents of connective tissue may also change. Although there is every reason to suspect that many physiologic and pathologic conditions may affect the properties of the receptor-containing blood vessels, little research has been done in this regard.

One interesting series of experiments has, however, shown that the relationships between arterial blood pressure and traffic of the receptor impulses of the carotid sinus change during the development of renal hypertension. In 1955, McCubbin, Green, and Page produced hypertensive dogs by wrapping their kidneys. Figure 3 shows representative data taken from one of their publications. Note that the receptor nerve-impulse output with respect to pressure is significantly reduced in the hypertensive animal as compared to the normal dog. Since this receptor activity acts, via the central nervous system, to tend to reduce blood pressure, it can be concluded that there is less depressor activity of the arterial blood pressure in the hypertensive animal than in the normal animal. Thus, the pressure could be expected to be increased in the hypertensive animal as a result of a change of carotid sinus-receptor behavior. It can also be concluded that this change in behavior is associated with changes in the mechanical properties of the sinus wall.
that in such hypertension states the integrating mechanisms of the central nervous system may be altered or the properties of the circulation control mechanisms may change. Indeed, the present medical director of the American Heart Association and his colleagues produced sustained hypertension in dogs by encasing the carotid sinuses in plastic jackets that restricted pulsation of the sinus wall. Possible changes in cerebral blood flow associated with these studies, however, complicate the interpretation of the findings. In any case, the cardiovascular regulatory mechanism probably plays a vital role in the behavior of the cardiovascular system in health and disease. Clearly, however, the subject merits further investigation.

It is obvious that before this entire problem can be properly investigated and evaluated, it is necessary to understand and to be able to measure, quantitatively, the mechanical properties of blood vessel walls that relate the caliber or stretch of the wall and the intravascular pressure.

Recently methods have been devised for measuring these mechanical properties of blood vessels. In brief, the principle can be illustrated as in figure 4. The uppermost figure represents a solid strip of material, e.g., a strip of blood vessel wall. T represents a tension stress (force per unit area) tending to stretch the material. \( l_0 \) represents the initial length of the material before it is stretched and the shaded areas represent the increase in length that occurs when the tension is applied. Here the elongation of the material is a function of the amount of tension applied. But since the amount of elongation is also a function of the initial length of the material, the ratio of the increase in length to the initial length is measured instead of simply the total elongation. This ratio is called strain.

\[
\text{Strain} = \frac{\text{Change in length}}{\text{Initial length}} = \frac{\text{total } \Delta l}{l_0}
\]

The mechanical properties of this material that are of interest to us are those that determine the relationship between strain and stress, stress being the amount of tension applied to the material which in this case is related to the intraarterial blood pressure. If the material is purely elastic, i.e., has only the property of elasticity, then the relationship of stress to strain is given by

\[
\text{Stress} = (E) \cdot (\text{Strain})
\]
in which \( E \) is called the modulus of elasticity,
the elastic stiffness or elastic resistance to stretch. Steel, for example, has a very high $E$ whereas rubber has a much lower $E$. The $E$ of arteries is similar to that of rubber.

Probably no real material is purely elastic. Certainly blood vessel walls are not purely elastic, rather they are viscoelastic. Viscosity is a quality often associated with molasses and is similar to friction; therefore, the blood vessel walls have, in addition to the elastic resistance to stretch, a resistance to stretch that depends upon how rapidly the stress is applied. Therefore, in the case of a viscoelastic material

$$\text{Stress} = E \times (\text{Strain}) + R \times (\text{Rate of strain})$$

in which $R$ is the modulus of viscosity. Materials also have the property of mass or inertia. It has been found, however, that, under living conditions, the effective inertia of blood vessels is negligible and does not appreciably contribute to their stiffness or resistance to stretch.9

The cylinder shown in the middle of figure 4 represents a blood vessel in order to illustrate the fact that the wall of a blood vessel is circular rather than linear as shown above. Furthermore, it may be seen that the pressure acts radially, i.e., from the direction of the center of the cylinder, whereas the tension that stretches the wall is circular, i.e., in a different direction. The lower figure represents a piece cut from the wall of the cylinder to show the difference in the direction of the pressure stress and the tension stress. It can be shown that the 2 stresses are related by the radius of the cylinder and the wall thickness.9

$$\text{Tension} = \text{Pressure} \times \frac{\text{Radius}}{\text{Wall thickness}}$$

Thus, for the same internal pressure the stretching tension would be relatively small for a vessel of small radius and large wall thickness, and, conversely, for a vessel of large diameter and small wall thickness the tension would be relatively large for the same pressure. These relationships are important to an understanding of the properties of blood vessels, e.g., it is necessary to know the radius and wall thickness in addition to the pressure and strain if one wishes to compute the elasticity and viscosity of a blood vessel wall.

The often-used nouns, tone or stiffness of arteries can, therefore, be rigorously defined in terms of 2 mechanical properties, elasticity and viscosity. There are important distinctions between these 2 properties as they affect the stiffness of blood vessels. The elastic property is somewhat simpler in concept. It is analogous to the property of an ideal spring, in that its stiffness is independent of how rapidly it is stretched. The viscous property is analogous to viscosity of liquids; therefore, its contribution to stiffness does depend upon how rapidly the material is stretched. A viscoelastic blood vessel is therefore stiffer if pressure changed rapidly than if it changed slowly. Thus, the stiffness or tone of such a vessel would depend upon the rate of change of pressure during the cardiac cycle, i.e., upon the shape or contour of the pressure pulse.

The magnitudes and behavior of these properties of arteries in living, intact animals have been measured by simultaneously measuring the instantaneous intra-arterial pressure and vessel dimensions. The data were then analyzed by means of automatic computers. Thus, it has recently become possible to measure quantitatively and to define the "tone" or stiffness of arteries, which are their most important properties.9

Figure 5 illustrates, for example, the relationship between common carotid arterial diameter and pressure during each cardiac cycle before and after the application of norepinephrine to the vessel wall. Although the local application of norepinephrine did not change the systemic intravascular pressure, the average diameter and the pulsatile change in diameter which accompanies each pressure pulse cycle was reduced. This indicates a stiffening of the arterial wall caused by the application of norepinephrine. Analysis of such records has shown that norepinephrine can cause an increase in both the elastic and viscous stiffness of arteries by as much as 10 times.
Figure 6 illustrates the opposite effect that follows the application of acetylcholine to the wall of an artery. Here the magnitude of the average diameter and of the diameter pulsations are increased with respect to pressure, indicating a reduction in stiffness. Analysis has shown that, in arterial dilatation, although the elastic stiffness is reduced, the viscous stiffness is increased.

Such studies have emphasized several interesting characteristics of arteries under living conditions. It is sometimes thought that the diameter of arteries changes visibly and palpably during the cardiac cycle. For example, that palpation of the pulse is palpation of a changing vessel diameter. We have found, however, that the change in diameter, associated with a cardiac cycle, of even the largest and most distensible arteries of the body is very small. In most cases it is only 1 or 2 per cent of the average diameter. Thus, when one palpates an arterial pulsation, it is really the pressure changes that one feels rather than the dimensional changes of the vessel. Even though these latter changes are small, relative to the size of the vessel, their effect on the properties and behavior of the circulatory system is great. A slight change in diameter represents a larger change in the cross-sectional area of the vessel (area of a circle is proportional to the square of the diameter) and a change in the area produces a relatively larger effect on the resistance to blood flow. These small changes in diameter are also great enough to cause the receptors lying within the walls of blood vessels to have their characteristic discharge. It has been found that the elastic and viscous properties of arteries can undergo 10-fold variations and also, therefore, that the caliber and stretch of vessels undergo equivalent changes with respect to blood pressure.

Figure 7 is an illustration of the simultaneous measurements of intracarotid-sinus pressure, carotid sinus diameter, and carotid sinus receptor electrical activity that are being studied in our laboratory. Although these investigations are in a preliminary stage, they are being presented because they are pertinent to this discussion. The picture shows, from above downward, a continuous record of sinus diameter, intrasinus pressure, and electrical spikes from approximately 5 single receptor units, each having a different amplitude. On the left are the relationships before the application of norepinephrine to the wall. The strain is 0.038, i.e., the sinus is stretched 3.8 per cent during the cardiac cycle by the pulse pressure. Previously the stress-strain relationships that characterize the entire aorta and its major branches to vessels of 1 mm. in diameter have been studied. The strain or percentage stretch that the carotid sinus undergoes for a given increase in pressure, i.e., its distensibility, is considerably greater than that of any other arterial segment studied so far. As indicated in figure 7 the, pressure-elastic modulus (E_p) is approximately 1,500 Gm./cm.,^2 which is only about 50 per cent of the average values of other arteries. It should be recalled that the distensibility of a vessel is a function of its radius and wall thickness as well as of its mechanical prop-

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properties. For example, it is known that the decreased distensibility that tends to accompany the increased stiffness associated with aging is often compensated for by an increase in its radius, i.e., dilatation. The low pressure-elastic modulus (high distensibility) of the carotid sinus is due primarily to the relatively high radius-wall thickness ratio. Indeed, the mechanical properties of the wall of the carotid sinus are quite similar to those of the aorta.

The records assembled at the right side of figure 7 were obtained following the application of a solution of norepinephrine to the wall of the carotid sinus. Although the intraarterial pressure pulses were not changed, the average and pulsatile diameter of the carotid sinus decreased. This indicates that the wall of the carotid sinus became markedly stiffer. The pressure-elastic modulus increased by 40 per cent, and, since the pressure pattern did not change, the strain decreased by 40 per cent.

These records show that marked changes can occur in the physical properties of the walls of the carotid sinus and that these changes can profoundly affect the activity of receptors lying in the vessel walls, which are independent of blood pressure changes but which, in turn, cause changes in the general cardiovascular system, including blood pressure changes.

The viscoelastic properties of the vessel wall are determined by a variety of tissue constituents, viz., smooth muscle, collagenous and elastic tissue, water, electrolytes, etc. It is most likely that physiologic control of the mechanical properties of the vessel walls generally is achieved by variations in the tension exerted by the smooth muscle in the vessel walls. Thus, the 10-fold variations in the elastic and viscous moduli found in the experiments recounted above is no doubt due to variations in the contractile force exerted by the smooth muscle. Moreover, it is also likely that chronic alterations in the properties of the vessel walls occur as a result of changes in the electrolyte and the water content of the vessel walls. Such variations have been reported to be associated with a variety of conditions including hypertension. It is possible, for example, that the renal hypertensive dogs studied by the Cleveland group suffered from alterations in vessel-wall electrolyte and water content. Also it is known that alterations in the relative amounts of elastic and collagenous tissues accompany aging and certain diseases. It is also possible that the mechanical characteristics of the connective tissue itself may undergo changes.

_Circulation, Volume XXI, May 1959_
Comparison of carotid sinus wall motion (top tracing), of intracarotid sinus pressure (middle tracing), and of the electrical activity of a few fibers of the carotid sinus nerve (bottom) before (left) and after (right) application of norepinephrine to carotid sinus, without change in intrasinus pressure.

Few, if any, of these factors that affect the mechanical properties of vessels have been adequately studied, and much remains to be learned before we can assess the role of vessel-wall properties in the regulation of the circulation. It is likely that these properties will prove to be internally related to the regulation of the circulation.

We may summarize the factors affecting receptor activity as follows: 1. Receptor stimulus is probably the strain of the vessel wall in which the receptors are located. 2. The strain is a function of at least 4 biologic variables, namely, intraarterial pressure, rate of change of intraarterial pressure, elastic modulus of the vessel wall, viscous modulus of the vessel wall. 3. Any given receptor area, e.g., the carotid sinus, contains a population of receptors of which each receptor may have different thresholds or sensitivities, and perhaps some may respond differently with respect to the direction of the strain. The information that is significant to the central nervous system may be contained in the net traffic from entire populations of receptors from a variety of areas.

It is obvious that much remains to be learned about these factors and their interrelationships. It is also obvious that the concept that pressure per se is regulated is too simple and may have been misleading.

So far I have mainly discussed the receptor mechanism of the carotid sinus as an example of the manner in which regulation of the circulation may be considered. Many other "mechano-receptor" areas have been described, but the carotid sinus mechanism has been most widely studied and therefore more information about its function has accumulated. It is impossible to say, however, what the relative role of this and the numerous other receptor areas may be in the over-all regulation of the cardiovascular system. Aviado and Schmidt have reviewed studies of a variety of receptor areas that are known to affect the cardiovascular and pulmonary systems. In addition, there are probably many receptor areas yet to be discovered.

A number of receptor areas within the vascular system have been called volume-receptor areas in contrast to these other areas referred to as pressure-receptor areas. These so-called volume receptors have been found particularly in the great veins and pulmonary vessels and, when stimulated, may cause reflex variations in cardiovascular, pulmonary, and renal functions. Dr. Elkinton will comment upon the fact that such receptors play an important role in the control of the body's water and electrolyte content. In reality, the so-called pressure and volume receptors and their activity is a function of the intravascular pressure and the mechanical properties of the vessel walls that
contain the receptors. The difference in the names probably arose because in the systemic arteries pressure changes are large compared to volume changes, whereas the volume changes of the systemic veins and pulmonary vessels are large compared to pressure changes. Thus, the major factors responsible for the difference are the mechanical properties of the blood vessels.

It is instructive to consider how many functions of the cardiovascular system are affected by the mechanical properties of the blood vessels. As stated above, they affect (1) the blood pressure and blood volume, and (2) the activity of receptors located within blood vessel walls, as well as (3) the vascular resistance to flow by producing vasoconstriction and vasodilatation. They affect (4) the performance of the heart by altering the venous return and the arterial load. By their effect on blood pressure they affect (5) the exchange of fluid between the intravascular and extravascular fluid spaces. They (6) direct the flow of blood through various vascular pathways. They affect (7) renal function both through neuroreceptor mechanisms and by their controlling effect on renal blood flow. The mechanical properties of the blood vessels, therefore, must play a predominant role in the regulation, not only of functions of the cardiovascular system, but also of the entire, integrated regulation of the biologic functions of the body.

An interesting interrelationship exists between the activity of strain receptors located within the vessel walls and the mechanical properties of the vessel walls. The activity of the receptor is a function of the mechanical properties of the wall, since those properties determine the amount of strain that results from a certain amount of stress (e.g., pressure). In turn, the reflex response associated with this receptor activity results in a change in the mechanical properties of the vessel walls, i.e., this is the mechanism by which the caliber of blood vessels is controlled. This situation is similar to what would occur in the temperature regulation of the house, talked about in the introduction of this symposium, if the thermostat were so made that whenever the temperature within the house changed, an alteration of the "setting" of the thermostat would occur. The extent of this effect as well as its significance is unknown but again is an indication of our naivete regarding regulation of the cardiovascular system.

I have said little regarding the important aspect of cardiovascular regulation that is associated with the central nervous system. This has been lumped into the integrating and error-generating black box in figure 2. Dr. Rushmer has shown us that voluntary or conscious central nervous system activity can duplicate the cardiovascular responses to exercise. This, it seems to me, is an excellent example of the influence of these higher centers upon the regulatory mechanisms that may be analogous to the resetting of the thermostat by the occupant of the house mentioned during the introduction of this symposium. There is little doubt that the cardiovascular system is controlled during exercise, but at a higher level of activity. It is likely, however, that there are other factors in addition to the drive of the higher central nervous levels in establishing the "ideal" or "set" values about which regulation takes place. Indeed, the role of the central nervous system in the regulation of the functions of the cardiovascular system is still largely unknown. Some of these questions will be discussed by Dr. Folkow.

I have tried to show, in this brief period, that in considering the regulation of blood vessels there are vast areas that are poorly understood and, indeed, that fundamental concepts of what is regulated and how regulation occurs are not understood. It is evident that the diagram shown in figure 2 is woefully inadequate and that even the functions of the black boxes shown therein are not well understood.

It is not surprising, therefore, that the etiology of hypertension has escaped detection or that we cannot explain the physiologic responses to stresses such as exercise. The surface of cardiovascular physiology has hardly been scratched, and there are many fundamental, challenging problems facing investigators.

While the advances of the past 300 years
may seem disappointingly few, there is reason for optimism. The next decade may bring greater advances than those of the entire past 3 centuries. It has been said that more than 80 per cent of all the physiologists who have ever lived are now alive. It is evident in most phases of human endeavor that the rate of achievement of knowledge is, like population growth, a rapidly accelerating phenomenon.

Addendum
Two recently published books, to which the reader may wish to refer, have come to the author's attention since this discussion was prepared.34, 35

Summario in Interlingua
Le sistema cardiovasculari servi le organismi biologic com le systema de transporto e de mixtion. Le requirimentos del organismi pro iste servizio varia in differente tissus e a differente tempore durante que le volumine de sanguine de le organismi tende a remaner constante. Ergo le systema cardiovasculari executa su function per variar le intensitate e le distribuzione del fluxo de sanguine. Le vasos de sanguine ha un rolo major in determinar le intensitate del fluxo de sanguine in tanto que illos exercet un influencia super le resistentia contra le fluxo. Le vasos de sanguine ha le complete responsabilitate pro le distribution del sanguine e le circuitus de su fluxo. Le aspecto principal del vasos de sanguine que affice le fluxo e le distribution del sanguine es lor calibe.

Le presente articulo summariza le recercaes effettuate relative al factori que determina le calibe del vasos de sanguine e al rolo de iste factori in le regulation del circulation del sanguine. Le discussion concerne primarimente le mechanismi receptori del sino carotidien que es presentate como exemplo del maniera in que le regulation del circulation pot esser considerate.

Le autor formula le conclusion general que in le campo del regulation del vasos sanguinear i existe vaste areas que es mal comprendite a iste tempore e de facto que conceptos fundamental con respecto a qual processos es regulate e a como le regulation occurre es non ancora sufficientemente clar.

References